CORRIGENDA

"The Choleretic Action of Genebile* in a Dog" Vol. 46 No. 2, p.149 — A. Immelman, C.J. Roos and N.C. Owen: Fig. 1 should depict a line drawing of a catheter secured in a bile duct. The histogram printed as Fig. 1 should, in fact, be read as part of Fig. 2.

1. "Arsenic resistance in species of multi-host ticks in the Republic of South Africa and Swaziland" Vol. 46 No. 4. Contents/Inhoud Senior Author should read **"M.D. MATTHEWSON".**
2. "The resistance of a field strain of *Haemonchus contortus* to five Benzimidazole anthelmintics in current use" Vol. 46, No. 4: p369 line 19 should read: *"Trichostrongylus colubriformis"*; p371, line 10 of "Discussion" should read "Theodorides *et al.*"

JOURNAL OF THE SOUTH AFRICAN VETERINARY ASSOCIATION

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TYDSKRIF VAN DIE SUID-AFRIKAANSE VETERINÊRE VERENIGING

Die TYDSKRIF is die offisiële mondstuk en eiendom van 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. Kopeireg word op al die letterkundige inhoud van die Tydskrif voorbehou.

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Geldelike steun deur die Departement Nasionale Onderwys word met dank erken.

EDITORIAL

YOUR JOURNAL

Readers who are familiar with this Journal will undoubtedly notice the obviously different cover and general appearance of this number. This is partly due to the fact that the journal is now being set by Messrs Engravaplate (Pty) Ltd and printed by Instacopy Services (Pty) Ltd, both of Durban.

There are also a few other changes. The back stapling will eliminate some of the problems, such as loss of pages, with which readers previously had to contend when the journal was bound and glued to a square back. The print has been changed a little, and some print sizes are also a little different to the previous volumes. We think the change is an improvement, with increased legibility.

Regarding the style and presentation of articles, you will notice that an Abstract now replaces the summary to which you have become accustomed. The Abstract is in the exact form used by the *Veterinary Bulletin* and will provide a little more detailed information than the summaries. Readers will also note that the full names of the journals are to be quoted under "References" in the future. Prospective and future authors should particularly take note of these two changes.

With the assistance of the S A V A office, our advertisers and their agents, and of course the printers, the Editorial Committee sincerely hopes that this Journal will again be appearing regularly in March, June, September and December of each year.

If this Journal does not contain all that members of the S A V A and subscribers wish it to, please bear in mind that the Committee can only publish what it receives for publication. We welcome *all* contributions and hope to provide in the needs of all sectors of the veterinary profession. Members of the S A V A should remember that it is *their* journal. They have a moral obligation to first submit their articles to *this* journal. This is the one and only journal and official organ of the S A V A, inclusive of all its Branches and Groups. It is and can only be what members make of it.

Finally, bear in mind that making mistakes is the inevitable lot of anyone who tries to do something. The Editorial Committee welcomes ideas and constructive criticism concerning the Journal and how to improve its value to you as reader.

VAN DIE REDAKSIE

U TYDSKRIF

Lesers wat met hierdie Tydskrif bekend is sal ongetwyfeld die verandering in die algemene voorkoms en buiteblad daarvan opgemerk het. Ten dele volg dit op die feit dat die tydskrif nou deur mnre Engravaplate (Pty) Ltd geset en deur Instacopy Services (Pty) Ltd, albei van Durban, gedruk word.

Maar daar is ook ander veranderinge. Deur die blad op die rugkant met kramme te laat bind sal die probleem van losrakende blaaie, soos wat met die platrugbindvorm van vorige jaargange ondervind is, iets van die verlede wees. Die letterstyl is ook ietwat verander, en so ook die lettergroottes. Ons meen dis 'n verbetering wat ook groter leesbaarheid in die hand sal werk.

Betreffende die styl en aanbiedingsvorm van referate sal u merk dat 'n uittreksel (Abstract) die gebruiklike opsomming vervang. Dit sal ietwat meer inligting verskaf, en is geskryf in die formaat wat deur die *Veterinary Bulletin* gebruik word. Lesers sal ook oplet dat die volle name van die tydskrifte wat onder "Verwysings" aangehaal word, voortaan in plaas van die verkorte vorm gebruik sal word. Voornemende outeurs moet gerus op hierdie twee veranderings ag slaan.

Met behulp van die kantoor van die S A V V en die samewerking van ons adverteerders en hulle agente asook natuurlik die drukkers vertrou die Redaksiekomitee dat die Joernaal weereens gereeld aan die begin van elke Maart, Junie, September en Desember van elke jaar sal verskyn.

Indien hierdie joernaal nie aan die vereistes van inskrywers en lede van die S A V V voldoen nie moet daar tog onthou word dat die Redaksie slegs dit kan publiseer wat vir publikasie aangebied word. Ons verwelkom *alle* bydraes en sou graag aan die benodigdhede van alle sektore van die professie wil voldoen. Lede van die S A V V moet steeds in gedagte hou dat dit *hulle* tydskrif is. Lede het 'n morele verpligting om hulle artikels eers vir publikasie in *hierdie* tydskrif aan te bied. Die Tydskrif is die enigste joernaal en amptelike mondstuk van die S A V V, sy Groepe en Takke. Die Tydskrif is en kan ook niks meer wees as wat die lede daarvan maak nie.

Ten slotte, onthou maar dat enige persoon wat iets probeer doen, noodwendig ook foute maak. Die Redaksie verwelkom gedagtes en opbouende kritiek aangaande die Tydskrif en wyses waarop dit vir u as leser meer waardevol gemaak kan word.

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OPENINGSREDE DEUR SY EDELE J.P. VAN DER SPUY, MINISTER VAN NASIONALE OPVOEDING, TYDENS DIE KONGRES VAN DIE SUID-AFRIKAANSE VETERINÊRE VERENIGING, DURBAN, 8 SEPTEMBER 1975

OPENING ADDRESS BY HIS HONOUR J.P. VAN DER SPUY, MINISTER OF NATIONAL EDUCATION, OF THE CONGRESS OF THE SOUTH AFRICAN VETERINARY ASSOCIATION, DURBAN, 8 SEPTEMBER 1975

Die veeartsenykundige professie in die Republiek van Suid-Afrika het groot roem binne en buite ons grense verwerf hoewel dit maar op 'n betreklike kort geskiedenis kan terugkyk. Dis grotendeels aan Sir Arnold Theiler te danke dat die Fakulteit Veeartsenykunde in 1920 te Onderstepoort gestig is, akademies in verbintenis met die destydse Transvaalse Universiteitskollege. Die Fakulteit was 'n integrerende deel van die Laboratorium te Onderstepoort (of Navorsingsinstituut vir Veeartsenykunde soos dit jare later herdoop is). Die dosente was almal navorsers en die doel van die vyfjarige studie was baie duidelik: die opleiding van navorsers en staatsveeartse. Dit was logies omdat dit feitlik *al* werk kringe was wat in Suid-Afrika vir veeartse beskikbaar was, en die paar graduandi jaarliks was genoeg om in dié behoeftes te voorsien.

As gevolg van die aanbevelings van die Brinkkommissie is die Fakulteit van Veeartsenykunde egter op 1 April 1973 uitgelyf uit die Departement van Landbou-tegniese Dienste en het dit 'n volwaardige onderdeel van die Universiteit van Pretoria geword. Op daardie dag het ook die verbintenis tussen die Fakulteit en die Departement van Nasionale Opvoeding ontstaan wat my in meer en meer intieme aanraking gebring het met probleme waarvoor u professie te staan gekom het. Ek het my dit onder andere ten doel gestel om op hoogte te kom van die opleiding van lede van 'n professie wat 'n baie belangrike rol moet speel in die voedselproduksie van die land met sy snel toenemende bevolking.

Dit is daarom vir my 'n genoeë dat ek vanaand saam met u Vereniging kan vergader wat so nou gemoeid is met die opleiding van veeartse, nagraadse studie en die voortsetting van vakgerigte opleiding nadat basiese kwalifikasies verwerf is. Ek dank u opreg vir die gewaardeerde uitnodiging om die openingsrede te lewer en u vriendelike verwelkoming.

Die veearts moet in sy opleiding eerstens bekwaam word om onmiddellik nadat hy gekwalifiseer het, 'n standaard van professionele bekwaamheid te handhaaf wat die Veeartsraad sal bevredig. Verder mag daar met die universitêre opleiding van die veearts nooit uit die oog verloor word dat die bevolking van die Republiek van Suid-Afrika 'n groeiende behoefte aan diereprodukte sal hê nie. Van die veeartsenykundige professie word verwag dat hy sal bedra tot die voeding van die mens deur die bestryding van dieresiektes en die bevordering van diereproduksie. Die naam Malthus is alombekend en reeds in 1798 het hy gewaarsku teen die rampspoedige gevolge wat wag omdat die mensdom vinniger, vermenigvuldig as wat landbouproduksie vermeerder. Dit is waar dat hy die tegnologiese ontwikkeling van die wêreld onderskat het en dat dit in 'n groot mate 'n katastrofe kan afwend. Veeproduksie is 'n onderafdeling van landbouproduksie, maar alles wat moontlik is, moet gedoen word om dit tegnologies op 'n hoër peil te bring.

Die Republiek van Suid-Afrika is in 'n besondere situasie. Eerstens word sy veebedryf deur feitlik al die siektes geteister wat eie is aan tropiese Afrika sowel as dié van ander lande. Tweedens is sy voedseldiere meer afhanklik van weiding in die veld as van graanvoer en aangeplante weidings omdat die beperkte bewerkbare grond nodig is vir menslike voedselproduksie. Hierdie tendens kom trouens alhoemeer voor ook in die res van die wêreld. Die afhanklikheid van die natuurlike weiding in ons land bring egter mee dat die diere vir langdurige tye moet leef van droë, veselagtige voer wat kan lei tot tekortsiektes en metaboliese steurnisse. Derdens het die Republiek wat veeartsenykundige opleiding en navorsing betref, nog altyd 'n leiersposisie beklee ten opsigte van sy buurstate en is hy geroepe om dit te handhaaf.

Gelukkig kon die graduandi wat tot dusver aan die Fakulteit van Veeartsenykunde opgelei is, hulself goed laat geld op die terreine waar hulle beweeg het, en alles wat moontlik is moet gedoen word om dié toedrag van sake te laat voortduur.

Die veeartsprofessie het ten doel om die veebedryf en daardeur die gemeenskap te dien. As sulks is daar verskillende faktore wat 'n invloed uitoefen op sekere afdelings van die professie. So byvoorbeeld sal die ekonomiese klimaat in die land met besondere verwysing na die landbou-ekonomie die private praktyk beïnvloed. Hierdie en ook nog ander faktore val buite die beheer van die veearts, maar die belangrikste element wat die voortbestaan van 'n professionele groep verseker, is sy wetenskaplike status en prestasies. Dit word beïnvloed deur voorgraadse en nagraadse opleiding, voortgesette studie en spesialisasie, en hieraan kan beslis baie gedoen word.

The shortage of veterinarians in South Africa has been convincingly brought to the notice of the relevant authorities. You are aware of the fact that the arrangements have been finalised for creating facilities to admit 90 students per year to the existing Faculty of Veterinary Science. This represents a doubling of the present number of admissions. I must thank the South African Veterinary Association for promising its support to ensure the success of this venture. No doubt the South African Agricultural Union

which has been pressing for the training of more veterinarians will be pleased with the developments.

You are also aware of the fact that the Minister of Bantu Education has announced his intention of establishing a faculty of veterinary science for the training of Bantu veterinarians in close association with medical doctors and dentists. It is pleasing to know that satisfactory progress has already been made so that we can expect the training programme to start soon. The decision to found a faculty for Bantu veterinary education was preceded by an investigation by a committee on which your Association was represented.

A matter of grave concern is the dearth of suitably qualified teaching staff to man the existing Faculty when enlarged and the non-european faculty to be created soon. Although this seems a universal problem, judging by the situation in the United States of America which has 30 000 veterinarians and still has problems in finding suitable teaching staff, everything possible will have to be done to overcome the shortage.

The establishment of any additional faculty of veterinary science will have to be thoroughly considered at the appropriate time.

The world is experiencing an explosion of scientific knowledge and the increased information filters through to the man in the street. Also the modern farmer is a much more knowledgeable person than ever before and he expects of his veterinarian a level of competence far above what he can gain for himself by reading the available literature. While this is true for farmers in all the different subdivisions of the animal industry, one is justified in questioning the applicability of veterinary education in its traditional form to the present-day situation. Any radical change could, however, only be brought about after careful study and deliberation but I am convinced that the time has arrived for the veterinary profession to consider what scope and level of knowledge it requires of the different categories of its members. The educationalists would then have to do everything possible to adjust the curriculum to meet the requirements. The medical profession has recently se an example of positive action by rationalising th medical curriculum and shortening the duration c the academic part of the course from six years to five

Adjustments to the veterinary curriculum will hav to take into account the following likely futur developments:

The food producing animals will be kept un der systems of husbandry of increasing size and inten sity which means that veterinarians in practice will b called upon to develop the use of preventive medicin techniques.

The demand for specialisation in differen branches of veterinary science will increase analogou to the situation in medical practice.

It is certain that veterinarians will be in volved in public health activities to a greater exten than at present.

On account of their broadly based biologica training the contributions of veterinarians are likel to increase in quality and quantity both in pure an applied biological science. Faculties of veterinar science will in future be called upon to contribute in creasingly to basic knowledge in the life sciences Only by developing post-graduate training an research to a high level will this be possible.

I notice from the programme, Mr President, tha the organizers of the Congress have provided for wide range of subjects to be introduced by appropriat experts. I trust that they will stimulate discussion b all interested delegates and that veterinary science i this country will benefit from your deliberations. I a also pleased to see that you have distinguished gue speakers from abroad to address the Congress o matters vital to the members of your Associatio Although they have been welcomed by the President wish to extend on behalf of the Government of th Republic of South Africa a most cordial welcome t them and trust that apart from the proceedings of th Congress, they will enjoy their sojourn in our country

In conclusion I wish you a very successful Congres and declare it open.

Ek verklaar hiermee die kongres as geopen.

BOOK REVIEW

BOEKRESENSI

THE HUSBANDRY AND HEALTH OF THE DOMESTIC BUFFALO

W. ROSS COCKRILL, EDITOR

Food and Agriculture Organisation, United Nations, Rome 1974.
Pp. xiv 993, Figs 223, Tabs 137.
Price: not stated.

This book deals in great detail with numerous subjects related to the health and disease of the various types of domestic buffalo encountered in so many parts of the world where they play a most important part in providing labour, meat, milk and animal fibre. The publication forms part of a project sponsored by the Australian Freedom from Hunger Campaign.

The book is beautifully printed, well bound and extensively illustrated with photographs of which many are in full colour. The contents deal with Species, Types and Breeds, Observations on Skin Colour and Hair Patterns, Genetics, Blood Groups and Polymorphisms, Environmental Physiology, Reproduction and its Physiology, Nutrition Aspects of Disease, Parasites and Parasitic Disease Management and Use, The Working Buffalo, Milk and Mil Production, Meat and Meat Production. A further sectio covers the buffaloes of various parts of the world.

The book contains an extensive list of references at th end of each chapter and a bibliography which extends ov 123 pages. The publication is well indexed, and in a represents a magnificent collection of data which should b available in every library.

L W v d

Jl. S. Afr. vet. Ass. 47(1) 7-9 1976

PRESIDENTIAL ADDRESS: BIENNIAL NATIONAL VETERINARY CONGRESS, DURBAN SEPTEMBER 1975

PRESIDENTSREDE: TWEEJAARLIKSE NASIONALE VETERINÊRE KONGRES, DURBAN, SEPTEMBER 1975

DR B.H. PAPPIN

Geagte Minister, Mnr die Onder-burgemeester, Geërde Gaste en Kollegas:

Ter aanvang is dit my aangename voorreg om dank en waardering uit te spreek teenoor die Minister van Nasionale Opvoeding, Sy Edele Senator J.P. van der Spuy vir sy teenwoordigheid en bereidwilligheid om ons Kongres te open. Ons is baie bly om ook Mev. van der Spuy hier in ons midde te hê en ons hoop dat beide van u die aand by ons sal geniet. U teenwoordigheid verleen luister aan ons professie en vereniging en alhoewel ons voel dat ons hierdie erkenning in 'n groot mate verdien, is u ondersteuning op hierdie tydstip, waar uitbreiding aan opvoedkundige geleenthede vir ons uiters noodsaaklik geword het, besonder gepas en word dit baie hoog waardeer. I am conscious too, of the presence here tonight of our two distinguished guests from England both of whom have been intimately involved with veterinary education. We know that their contribution, from what we have heard today, will make a lasting impact.

Dit het vir ons reeds baie vroeg in hierdie jaar duidelik geword dat ons professie, soos ander in Suid-Afrika, 'n krisis bereik het wat beskikbare mannekrag betref. Verslae van ons verskillende funksionele groepe oor hulle beskikbare en toekomstige vereistes wat veeartspotensiaal betref was sorgwekkend.

Ons professie moet nie net hulp verleen met die wel en die weë van die gemeenskap en sy huisdiere nie, maar is ook belas met die welstand en die nood van die veenywerheid en gesondheidsdienste, ingnemende ook die menigte probleme van veeartsenykundige aard wat ons landbou-ekonomie raak veral met betrekking tot die voedselproduserende diere.

In 'n kort oorsig van die uitwerking van hierdie tekort aan veeartse op ons professie, en daardeur op pasiënte, die produksie van dierlike proteïen en die gesondheid van ons mense, moet ek eerstens verwys na ons Fakulteit, die bakermat van ons professie.

Die Fakulteit van Veeartsenykunde, Universiteit van Pretoria, en geleë te Onderstepoort was nog besig om aan te pas na die onlangse uitlywing uit die Departement van Landbou Tegniese Dienste met 'n gevolglike verlies van doserende en tegniese personeel. Skielik is dit belas met inname van 90 studente per jaar. Dit behoort tot 'n verdubbeling van personeel te lei indien die intieme kontak met die student, wat veterinêre kliniese opleiding en navorsing vereis, gehandhaaf moet word teen die optimale verhouding van 10 studente per dosent. Dit is ondenkbaar en onaanvaarbaar dat 'n toestand sal ontstaan waar 'n tekort aan dosente relatief tot studentegetalle sal lei tot 'n laer standaard van opleiding en gegradueerdes. Dit sou ons aansien en respek plaaslik en oorsee noodwendig skaad.

Om poste vir professionele personeel te vul moet die Fakulteit meeding met ander vertakkings van veeartsenykunde wat, in sekere gevalle, groter finansiele voordele inhou. Ongelukkig het die Fakulteit ook nie net probleme gehad om die poste te vul nie maar ook om personeel te behou. Alles inaggeneem was daar in 1974 'n werklike tekort van 8 uit 'n totale Fakulteitpersoneel van ongeveer 50. Ons is inderdaad dankbaar teenoor die Universiteit van Pretoria vir die voorsiening van toerusting soos 'n elektronmikroskoop en ander fasiliteite wat sal meehelp as aantrekking om vakante poste te vul. Ons is ook beïndruk deur al die pogings van die Dekaan en sy personeel om hierdie probleem die hoof te bied.

Die Departement van Bantoe Onderwys het ons in kennis gestel van die beoogde oprigting van 'n nuwe inter-etniese Universiteit naby Ga-Rankuwa waar daar onder andere Bantoe veeartse opgelei sal word en waar akademiese hulp met die opleiding vanaf die Fakulteit gegee sal moet word; 'n verdere opoffering van ons akademici op hulle tydsbesteding. Desnieteenstaande is ons oortuig van die waarde van hierdie regte stap van die kant van die Regering om Bantoeveeartse op te lei; dit sal 'n wesenlike leemte in die tuislande vul. Een van die erkende metodes om die behoeftes aan veeartse te bepaal is om 'n formule saam te stel sodat die beskikbare veeartsenypersoneel met die werklike diereenhede in verband gebring word. Volgens die V.L.O./W.G.O. word die syfer vir ontwikkelende lande op 1:30 000 gestel. In KwaZulu, met twee beskikbare veeartse, is hierdie verhouding 1:403 000 en in die Transkei met sy drie veeartsposte is hierdie syfer 1:367 000.

In 'n onlangse memorandum aan die Rektor van die Universiteit van Pretoria het ons aanbeveel dat 'n onafhanklike Ambulatoriese en Buitepasiënte-diens by die kampus geskep word en 'n Kliniese Sentrum opgerig word in 'n gebied nie te ver van Onderstepoort af nie — waar dit as 'n hospitaal vir groot- en kleinvee kan dien. Hierdie diens en die noodsaaklikheid van kliniese assistente in elke departement sal nog 'n groter aanvraag vir dosente tot gevolg hê.

By ons wêreldbekende Navorsingsinstituut te Onderstepoort was daar verlede jaar 'n tekort van 10 veeartse — en hier verwys ek na hoog gespesialiseerde persone. Vir die volle bearbeiding van die narvorsingstaak moet 62 poste gevul wees in plaas van die huidige 43. Daar is 'n dringende tekort aan meer veeartse om die duisende patologiese monsters jaarliks te hanteer, om navorsing voort te sit ten opsigte van die lae kalwingspersentasies en hoë kalwermortaliteite, om as viroloë en bakterioloë opgelei te word, om verdere navorsing te doen oor insekoorgedraagde siektes soos bloutong, perdesiekte, Slenkdalkoors, Wesselbronsiekte en 3-daestywesiekte en die probleme en komplikasies ge-

assosieer met Chlamidia-infeksies. Die Instituut produseer jaarliks meer as 152 miljoen dosisse van 36 verskillende tipes entstowwe. Perinatale mortaliteit in kalwers, die probleme en toestande verwant aan die intensifisering van die veebedryf by varke en pluimvee, en die noodsaaklikheid van nuwe en beter entstowwe benodig almal een waardevolle persoon, naamlik die hoogsopgeleide veearts in navorsing. Dit is sekerlik vir my onnodig om te beklemtoon dat enige personeeltekort aan ons navorsingsinstituut kan lei tot ernstige ekonomiese verliese vir ons land.

In die Departement Veeartsenydiens het dieselfde kritieke situasie ontstaan. Hier is daar 34 poste vakant uit 'n totaal van 130 met die gevolg dat die posisie ontstaan het waar groot gebiede sonder 'n Staatsveearts moet klaarkom. Hierdie manne het die taak en die verantwoordelikheid om ons kwarantynstasies te beheer en te voorkom dat dieresiektes ingebring word, om ook alle aanmeldbare siektes soos tering, besmetlike misgeboorte, Newcastlesiekte, hondsdolheid, brandsiekte, varkpes, nagana, bek en klouseer en baie meer, te beheer en uit te roei. Hulle beheer ook vleisinspeksiedienste by baie abattoirs, dra die verantwoordelikheid vir die in- en uitvoer van vleis en die verskaffing van dienste by diagnostiese sentra. Laasgenoemde is 'n baie waardevolle diens vir boere en omliggende veeartse wat sonder enige twyfel uitgebrei moet word indien siektebeheer meer ekstensief en doeltreffend toegepas moet word. Om al hierdie noodsaaklike dienste in nasionale belang te kan onderneem moes die Staatsveeartsenydienste hulle mannekrag optimaal benut, en was hy genoodsaak om ook nog senior veeinspekteurs te gebruik om take te verrig wat essensieel die van veeartse is bv: tuberkulintoetse.

Veeartse in munisipale diens is betrokke by beheer van higiëne van melk, wat beheer by melkplase en -depots behels, en ook in sommige gevalle by vleishigiëne en algemene voedselhigiëne. Hier is die mannekragtekort nie so kritiek nie. Veeartse in die nywerheid is hoofsaaklik in 'n tegniese, adviserende, besturende of in navorsingskapasiteit almal geassosieer met die handel (hoofsaaklik die ontwikkeling en verskaffing van veemiddels aan die boer) en dit sal betreurenswaardig wees indien die veeartsprofessie nie hierdie senior poste sou kan vul nie as gevolg van die gebrek aan mannekrag. Die welbekende en sorgwekkende tekort van plattelandse praktisyns duur onverpoosd voort en hulle werksaamhede word daarby bemoeilik deur spoedbeperkings. In die afwesigheid van lokums en assistente moet hierdie mense diksels besoeke wegwys en telefoniese advies bedien. Nog 'n ontwikkeling is die oprigting van grootdierhospitale (en meer sal gebou kan word indien finansiële hulp van georganiseerde Landbou of die Landbank beskikbaar was) waarheen siek of beseerde diere gebring kan word om sodoende vermorsing van die veearts se waardevolle tyd op die pad te bespaar en derhalwe 'n meer doeltreffende, gesofistikeerde en ekonomiese diens te lewer. Die rol wat die plattelandse veearts speel in voorkomende geneeskunde, die hulp wat hy die Staatsdiens verleen in die vorm van teringtoetse, sy betrekking in die probleme van intensiewe boerdery en sy nuwe verantwoordelikheid met die beheer van antimikrobiese stowwe verleen 'n nuwe aansig tot sy klassieke rol van grootdierpraktisyn. Hy is bewus van die feit dat ons jaarliks honderde miljoene Rand verloor, hoofsaaklik as gevolg van voorkombare veeverliese, en dit veral in 'n tydstip wanneer ons 'n besliste rol en verantwoordelikheid het om die groeiende bevolkingsmassa te help voed.

The urban private practitioners and the equine practitioners, coping mostly with companion and working animals, have entered into a new sphere of service to the public and their patients over the last 10 or 15 years. Sophisticated modern hospitals and clinics have been built throughout our country and to match these excellent facilities, these veterinarians have updated their knowledge at many outstanding continuing education courses. The practitioners have defined the standards these hospitals have to meet and these are being revised to make provision for modern aseptic theatres, x-ray and laboratory facilities, a 24 hour service, intensive care units, heated bird rooms etc.

With the increasing expansion of our race horse industry and also the increase in the number of riding horses, the work of the equine practitioner has so advanced that specific equine hospitals are now being built.

The South African public are great pet owners and have become very conscious and appreciative of the professional help that is available and the demand is growing.

There are some 350 private practitioners with a current list of vacancies of about 78. On linear projection there will be a need for about 1 000 men in 10 years time. The manpower shortage is considerably retarding the development of this sector of our profession and those involved have to work long hours and rely heavily on para-veterinary help to cope with their burden of work.

Animal welfare societies, pet food manufacturers, zoological gardens, the national and provincial wild life conservation departments and the Defence Force all compete for the limited supply of Veterinarians.

This brief survey of our veterinary field, Mr Minister, Ladies and Gentlemen, and the rough statistical analysis of our manpower problem, forms the background to our major endeavours in this past year. It was our first duty to inform the Minister of Agriculture, in a detailed memorandum and at a subsequent interview, of the critical position in the Veterinary Services falling within his Department. We felt that these sections of our profession were particularly vulnerable in this stress period and, as I have indicated, could have far reaching consequences on our national interest. He appreciated the problem fully and has undertaken to do what he can to alleviate the situation.

When we realised our precarious position we also contacted our Minister of National Education to enquire when the Cabinet's decision to admit more students to our present Faculty would be implemented. After an exchange of letters our Minister, with commendable urgency, informed us that he intended to approach the Cabinet without delay for funds to enable the University of Pretoria to double the student intake next year in temporary accommodation and to plan the extension of the Faculty's buildings in such a way that 120 students could be admitted to the second year of the B.V.Sc. course.

In the light of our knowledge of the urgent need for more veterinarians we were indeed grateful that the Department of National Education had realised that doubling the present student intake alone would not meet our deficiency. It could also be implied that the importation of veterinarians from countries where an excess now exists was not contemplated as we have to

contend with conditions in many fields which are peculiar to Africa.

It was also pleasing that the Minister in realising the needs of the profession, was prepared to invest R15 000 000 to further veterinary education.

Our problem was that this bold venture contradicted our firm belief that nowhere in South Africa was there sufficient large animal material, in the environs of a University, to meet the needs of clinical material for such large classes. We feel that the intake of students at any single school must be limited to that number which can adequately be supplied with the necessary quality, quantity and variety of clinical teaching material which the readily accessible environment can supply. As in the case of Australia and other countries, South Africa has an essentially extensive livestock industry. It cannot hope to provide, at any one centre, the concentration of teaching material for the large numbers of students equivalent to those accommodated by schools in the intensively developed countries of Western Europe and North America.

We have motivated our desire for a second faculty for Whites once more in a memorandum to the Minister in the earnest belief that it will be in the best interests of our country to spread veterinary education in two widely separate schools. The other advantages of a second faculty have been enumerated many times but we feel that the two further considerations have recently come strongly to the fore, viz. the need for many more veterinarians and the astronomical financial cost of stock losses the country has to bear.

I fully appreciate the Minister's contention that the present faculty must not be prejudiced by the competition for academicians which a second faculty would bring. To this end we shall endeavour to the best of our ability to assist the University of Pretoria to find sufficient staff to adequately teach a student intake of 90 per year, even though we still have reservations at this level. We have sent a memorandum to the Rector stressing the need for better salaries and promotion possibilities, facilities and equipment to attract more staff. We have also stressed that there is a largely untapped source of part-time teachers at the neighbouring Institute and in clinical practice. The Dean too, through the medium of our newsletter to all members, has detailed the advantages of an academic career. I feel that it is our duty, as individuals, to canvass for more academicians amongst our colleagues in all our diverse interests.

Our attention had been drawn to the fact that there exists in South Africa today, at another University, facilities and staff probably sufficient to immediately accept 30-35 veterinary students through to their third year and that there is sufficient large animal material in the vicinity of this school to accommodate this number of students.

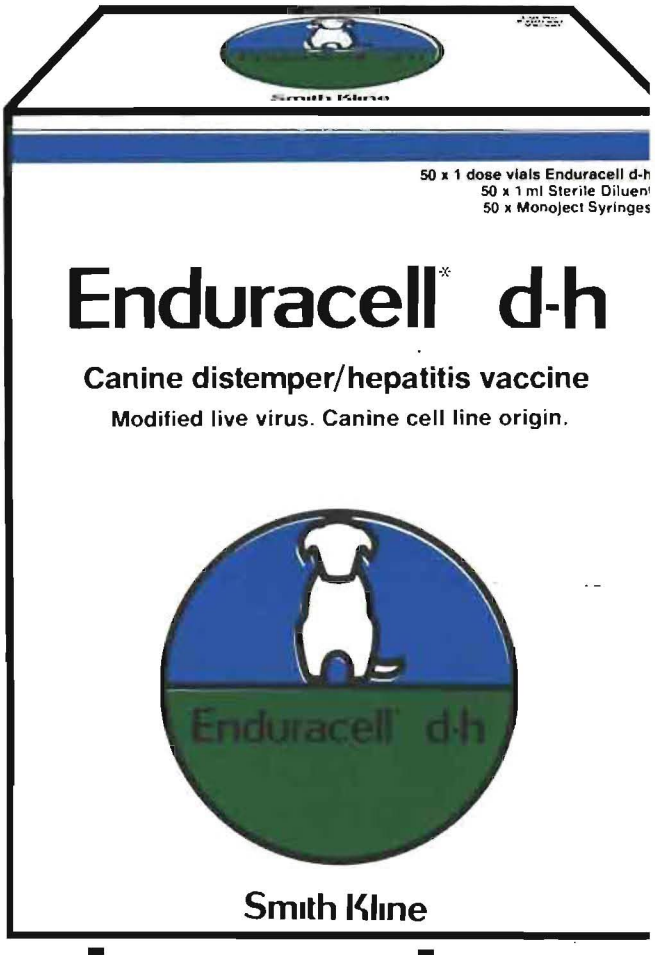
However these are matters for the Minister and his Department in their wisdom and in their knowledge of the overall financial position, to determine. We get so little opportunity to communicate in the complex lives we lead today that I have confined my Presidential address to the critical veterinary manpower shortage and the duty that has devolved on our Association to suggest ways that it may be overcome.

His best friend looks even better...







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INTRODUCTION OF THE RE-ELECTED PRESIDENT OF THE SOUTH AFRICAN VETERINARY ASSOCIATION:

VOORSTELLING: HERVERKOSE PRESIDENT VAN DIE SUID-AFRIKAANSE VETERINÊRE VERENIGING:

DR B.H. PAPPIN

For various reasons it was impossible for me to introduce our President on his election to office in 1974. He has now been unanimously re-elected for a further term of office, and I welcome this belated opportunity. To many who know him, or have recently got to know him because of his active and leading role in Association affairs, this may be superfluous. Nevertheless, a record of his past achievements is not out of place for those to whom he is less well known.

Basil Henry Pappin was born in Johannesburg on the 12th November 1924. After matriculating at the Pretoria Boys High School he proceeded to the University of Pretoria where he was awarded the degree of BVSc in 1946. He also won both the Sir Arnold Theiler (SA Biological Society) and the Clinical (Witwatersrand Branch, SAVA) medals.

After two years as assistant in practices in Johannesburg he established his own practice in Germiston in 1949. This has grown into a most successful practice with six partners and its own modern hospital complex as the result of the dedication, initiative, drive and hard work of himself and his partners.

He has been an active member of the Witwatersrand Branch of the SAVA since 1947 and served as its Chairman from 1970 to 1972. He became a founder of the Clinicians' Group of the Branch in 1965 and has served on its National Executive since 1972. He has served on The Council of the SAVA since 1970 and on numerous of its standing (Disciplinary and Fees) and *ad hoc* committees. He is also a member of the British Small Animal Veterinary Association and a practitioner affiliate member of the American Animal Hospital Association.

But not only as a veterinarian did he excel and make his mark. He also took upon himself some of the civic or community responsibilities, which we all have, by becoming most active in welfare work, i.a. as a member and president (1964-65 and 1966-1967) of the Germiston Rotary Club. He is also a member of the SA Wild Life Society and a founder of its Germiston centre.

He married Agnes Phillips and they have five teenage children. Whenever possible they "retire" for short periods to enjoy their sub-tropical fruit farm in the Eastern Transvaal.

This Association and the profession is without doubt extremely privileged to have as its president and head a man of such integrity, knowledge, experience, enthusiasm and dedication. His term of office must surely be successful and fruitful and I wish him all the best. His wife and his partners in practice are also making a magnificent contribution to the well-being and advancement of the profession by

Om verskeie redes was dit vir my onmoontlik om ons nuwe President aan u bekend te stel toe hy in 1974 verkies is. Sedertdien is hy onbestrede herverkies en ek verwelkom die geleentheid om dit nou te doen. Vir die wat hom reeds ken, of wat hom in die afgelope jaar leer ken het as gevolg van sy aktiewe optrede in die sake van die SAVV en sy takke, is voorstelling sekerlik oorbodig. Nietemin sou dit van pas wees ter wille van diegene wat hom minder goed ken.

Basil Henry Pappin is op 12 November 1924 in Johannesburg gebore en het aan die Pretoria Boys' High School gematrikuleer. Na 'n jaar aan die Universiteit van Natal het hy na die Universiteit van Pretoria gegaan om in 1946 die graad BVSc aldaar te verwerf. Ten tye daarvan is beide die Sir Arnold Theiler (SA Biologiese Vereniging) en die Kliniese (Tak Witwatersrand, SAVV) medaljes aan hom toegeken.

Na twee jaar as assistent in praktyke in Johannesburg het hy in 1949 sy eie praktyk te Germiston begin. Dit het ontwikkel in een van die grootste en mees suksesvolle praktyke in die Republiek met ses vennote en 'n moderne hospitaalkompleks. Die toewyding, inisiatief, dryfkrag en harde werk van hom en sy vennote is daarvoor verantwoordelik.

Hy is sedert 1947 'n aktiewe lid van die Tak Witwatersrand van die SAVV, wat hy ook vanaf 1970 tot 1972 as Voorsitter gedien het. Hy was 'n stigter van die Kliniese Groep van die Tak in 1965 en dien sedert 1972 op die Nasionale Bestuur van die Groep. Hy is sedert 1970 'n lid van die Raad van die SAVV en dien op verskeie komitees, waaronder Dissiplinêr, Professionele Gelde, en ander. Hy is ook 'n lid van die British Small Animal Veterinary Association en 'n praktisyn-geaffilieerde lid van die American Animal Hospital Association.

Maar dis nie slegs as veearts wat hy presteer het nie. Hy het ook omgesien na die verantwoordelikhede wat ons almal ten opsigte van die gemeenskap behoort te dra deur aktief in welsynsaangeleenthede op te tree, onder andere deur lidmaatskap en presidentskap (1964-65 en 1966-67) van die Rotary Club van Germiston. Hy is ook 'n lid van die SA Natuurlewevereniging en 'n stigter van die Germistonse sentrum daarvan.

Hy is getroud met Agnes Phillips en hulle het vyf tienderjarige kinders. Wanneer moontlik bring hulle 'n rustydjie deur op hulle vrugteplaas in die Oostelike Transvaal.

Sonder twyfel is die Vereniging en die beroep bevoorreg om so 'n persoon as President en leier te kan betrek. Sy integriteit, bekwaamheid, kennis, ondervinding, entoesiasme en toegewydheid moet noodwen-

making it possible for him to undertake what has become a most demanding honour.

A.B. LA GRANGE
(IMMEDIATE PAST PRESIDENT)

dig lei tot 'n baie suksesvolle en vrugbare termyn, en daartoe wens ek hom alles van die beste. Hy word bekwaam bygestaan deur sy vrou. Deur hom in staat te stel om sy veeleisende taak met eer uit te voer dra sy vennote ook pragtig by tot die welsyn en vooruitgang van die professie.

A.B. LA GRANGE
(UITGETREDE PRESIDENT)

Dr Basil Henry PAPPIN
President, S.A.V.A./S.A.V.V.
1974-1976

Jl. S. Afr. vet. Ass. 47(1) 13-16 1976

ADDRESS **VOORDRAG**

VETERINARY REMEDIES: THE VETERINARIAN'S RIGHTS AND OBLIGATIONS*

T.W. NAUDÉ†

In the Republic of South Africa veterinary remedies are divided into two main groups viz. *stock remedies* (registered under the Fertilizers Farm Feeds, Agricultural Remedies and Stock Remedies Act, 1947) and *prescription or ethical remedies* (controlled under the Medicines and Related Substances Control Act, 1965). These two groups will be discussed separately.

I. STOCK REMEDIES

According to the amendment to the Act which is to be considered by Parliament in 1976, *"stock remedy"* means a substance intended or offered to be used in connection with domestic animals, livestock, poultry, fish or wild animals (including wild birds) for the prevention, treatment, diagnosis or cure of any disease, infection or other unhealthy condition, or for the maintenance or improvement of health, growth, production or working capacity, but excluding a chemical substance, biological substance or other substance in as much as it is controlled by the Medicines Control Act, 1965 (Act No. 101 of 1965) or the Hazardous Substances Act, 1973 (Act No. 15 of 1973). (In the present definition of a *stock remedy* the reference to prescription remedies is phrased as follows: "...but excluding any preparation dispensed on the prescription of a veterinarian for a particular patient or group of patients"). A *stock remedy* is therefore:

(a) Registered under Act No. 36 of 1947 (the Fertilizers, Farm Feeds, Agricultural Remedies and Stock Remedies Act).
(b) Freely available over-the-counter to the farmer, pet owner or the public.
(c) Controlled as regards labelling, efficacy, safety and quality;
(d) Recognisable by the phrases "For animal use only" and "Reg. No. G ... Act 36/1947" in both official languages on the main panel of the label.

It includes i.a. most vaccines, anthelmintics, dips, certain mastitis preparations, udder disinfectants, certain antibiotics and a number of diverse medicines e.g. Vitamin A preparations, babesicides and milk fever remedies.

A proviso of registration is that except where the product is used prophylactically, the average farmer must be able to diagnose the condition for which the remedy is intended with a reasonable degree of certainty. Due to the extensive nature of our country and the lack of professional veterinary services in many districts, this group of remedies has been left in the hands of the farmer for emergency animal treatment in an endeavour to fulfil the farmer's immediate requirements as far as possible.

By control of labelling and advertising of all *stock remedies* the farmer is, furthermore, protected against undue commercial exploitation.

Certain *stock remedies* (some dips especially) are classed Group I poisons (oral LD50 of the active substance for the rat is <50 mg/kg) and the requirements for the safe storage and handling of these products are prescribed. These products may be recognised by the skull and crossbones and the phrase "Poison-extremely toxic" in both official languages on the main label. Such products must be kept under lock and key, must be accurately recorded as to stock and sales and the signatures of the purchasers thereof must be obtained.

Although many *stock remedies* contain chemical substances listed in the schedules of the Medicines and Related Substances Control Act (see below), registered *stock remedies* are specifically excluded from control under this act. It is, therefore, quite evident that the labelling of, for instance, a specific tetracycline formulation as a *stock remedy* would differ completely from the same formulation as a prescription remedy.

II. VETERINARY PREPARATIONS CONTROLLED UNDER THE MEDICINES AND RELATED SUBSTANCES CONTROL ACT, 1965 (ACT 101 OF 1965)

A. *Veterinary Prescription Medicines*

This entails those preparations (or so-called "ethical medicines") which are available to the veterinarian and pharmacist. The public can only obtain them on a veterinary prescription through a pharmacist. (The term "ethical remedy" should rather not be used for this group as it could conceivably include medicines which could be registered as over-the-counter *stock remedies* but which the firm, according to its own policy, only sells via the veterinarian and/or pharmacist).

Until relatively recently the acquisition of these drugs by the veterinarian was controlled by Act 13 of 1928 (the Medical Dental and Pharmacy Act). This Act has now been repealed and has been replaced by several other acts of which the Medicines and Related Substances Control Act, 1965, is of particular importance to the veterinarian. There are a number of significant changes related to the control of medicines of which the veterinarian should be aware.

Act 101 of 1965 was specifically intended to control medicines for human use — veterinary medicines are, at this stage, only controlled as substances. It makes provision for a statutory body, the Medicines Control Council, to control these medicines and substances

* From an address presented in Afrikaans at the A.G.M. of the S.W.A. Branch of the S.A.V.A., Windhoek, 13th of October 1975.

† Technical Adviser to the Registering Officer, Act 36 of 1947, Veterinary Research Institute, 0110 Onderstepoort.

with the aid of eight committees, amongst others the committees on safety, efficacy, quality, scheduling, advertisement and information. Members for these expert committees are drawn from all over the country and include persons from the medical, pharmaceutical, veterinary and allied professions, both from within and without the civil service. The administrative aspects of the council are the responsibility of the Registrar of the Medicines Control Council and his staff. They are attached to the Department of Health.

All medicines for human use must be registered prior to sale. All new preparations are subjected to stringent efficacy, safety and quality requirements and placed in specific schedules prior to registration. All those medicines which were on the market prior to 5/7/68 are being called up in pharmacological groups and systematically evaluated in a similar fashion. It is anticipated that several years will have passed before all medicines have been evaluated.

As far as veterinary prescription remedies are concerned present control is confined to the availability to the veterinary, medical, dental and pharmaceutical professions by virtue of the fact that the active principles are listed as scheduled substances. No control whatsoever is exercised over the efficacy, safety and quality of such medicines. These aspects are left entirely to the discretion of the veterinarian prescribing or using such medicine and he has to rely on the integrity of the manufacturer involved.

B. *Schedule 1 and 2 substances sold by pharmacist for veterinary use*

Under the Medicines and Related Substances Control Act, 1965, the pharmacist may sell preparations, falling into these two categories, at his own discretion. From a veterinary viewpoint this is a very minor group. The groups listed under these two schedules are detailed below.

III. VETERINARY PRESCRIPTION REMEDIES CONTAINING SUBSTANCES WHICH ARE NOT CONTROLLED UNDER THE MEDICINES CONTROL ACT

This group consists of a relatively small group of products. At present they fall in a grey area from a legal viewpoint.

Apart from a few diverse substances which only have veterinary application and which have not been scheduled to date, it comprises mainly ectoparasiticides which the relevant firms make available only to veterinarians or on a veterinary prescription.

Realising these deficiencies in the control of veterinary prescription medicines, the Veterinary Board made representations to the Medicines Control Council during 1975 to provide for control, in terms of the Medicines and Related Substances Control Act of the quality, efficacy and safety of those medicines not registered as *stock remedies* under Act 36 of 1947. The Medicines Control Council has acceded to this request. It is expected that the necessary amendments to the Act will be effected during this year. It is envisaged that a veterinary committee, similar to the eight existing committees of the Council, will be formed to attend to veterinary aspects.

It will probably take several years before effective control over all aspects of veterinary prescription remedies will have been obtained. The present position of the veterinarian in respect to these preparations after the repeal of Act 13 of 1928 is as follows:

A. SCHEDULING

All medicines, including veterinary preparations, are subject to scheduling according to the active substance(s) that they contain in order to control the acquisition and usage of such substances. After thorough consideration by the Scheduling Committee of the Medicines Control Council the scheduling status of each substance is determined and it is placed into one of the following 10 schedules. The availability, the prescription requirements, etc., become increasingly more difficult as the number of the schedule increases and it ranges from freely available over the counter of any shop to completely prohibited. (The examples quoted below in each schedule are mainly those of veterinary interest. For the full list please refer to Government Notice No. R2244 of 28/11/75.)

Unscheduled *(Over-the-counter medicines)*

This includes vitamin formulations (other than certain Vitamin A and/or Vitamin D preparations), trace elements, most purgatives, aspirin, etc. As a medical preparation each medicine in this category is subject to registration although freely available at shops. All such substances for animal use must, however, be registered as *stock remedies* under Act 36 of 1947. (Note: a substance or remedy that has been declared Unscheduled ("Ongeskeduleerd") must not be confused with a substance or remedy that is not scheduled ("nie-geskeduleerd") simply because it has not yet been subjected to scheduling).

Schedule 1 *(Pharmacy remedies)*

This includes the milder analgesics like phenacitin and preparations containing limited concentrations of codeine, chlorodyne, morphine and opium; chloroform and ether; certain antibiotics, sulphonamides and local anaesthetics for external application; antihistamines when intended specifically for topical application and for travel sickness; and all preparations for injection unless otherwise scheduled.

Schedule 2 *(Pharmacy remedies)*

Antihistamines, arsenic and arsenical preparations containing more than 0,01% arsenic, apomorphine, atropine, furazolidone and nitrofurazone, strychnine and preparations containing more than 0,2% strychnine are of veterinary importance.

Schedule 3 *(Prescription remedies frequently repeated)*

This includes i.a. digitalis and related substances, insulin, isoniazid (INH), primidone and certain other anti-epileptic substances and thyroid preparations.

Schedule 4 *(The main group of prescription drugs)*

Examples here include antibiotics, sulphonamides, corticosteroids, diuretics, adrenergic blocking and stimulating agents, hormones and local anaesthetics.

Schedule 5 *(The "minor tranquillizers" and other psychotropic drugs and anaesthetics)*

The phenothiazine derivatives e.g. acetyl- and chlorpromazine and promethazine, the buterophenones like azaperone, most of the barbiturates, chloral hydrate, halothane and related substances are included here.

Schedule 6 *(The "major tranquillizers" and possibly habit forming drugs)*

In this group only pentobarbital (e.g. "Sagatal"

and phencyclidine are of general veterinary importance.

Schedule 7 *(Therapeutic narcotics or habit forming drugs)*

The drugs in general veterinary use in this schedule are chlorodyne, codeine, etorphine (M99), morphine, opium and all the related substances and derivatives, cocaine, diethylthiambutene ("Themalon"), fentanyl, pethidine and related substances.

Schedule 8 *(Prohibited substances)*

Except for amphetamine and related substances which were used as analeptics in the past there are no substances of veterinary therapeutic importance in this group which includes heroin, coca leaves, dagga and tetrahydrocannabinol, lysergic acid (LSD) and mescaline.

Schedule 9 *(Exempted prohibited substances)*

Only two substances, amphetamine and dextroamphetamine fall into this schedule.

The scheduling procedure of Act 101 places medicines in four groups:

(a) **The Unscheduled group**

(b) **The Schedule 1 and 2 group**

These are more or less equivalent to the 4th Schedule Division I and II poisons under the now repealed Act 13 of 1928 and contain those substances that the pharmacist may sell at his discretion without prescription.

(c) **The Schedule 3 to 7 group**

These are the prescription substances. Under Act 13 of 1928, Schedule 3 to 6 were known as the 6th Schedule or Potentially Harmful Drugs (P.H.D.) whereas Schedule 7 was known as the Habit Forming Drugs (H.F.D.).

(d) **The Schedule 8 and 9 group**

These are the prohibited groups which are very strictly controlled. Schedule 8 substances can only be obtained on permit from the Secretary of Health for analytical or research purposes under rigidly controlled conditions. Amphetamine and dextroamphetamine (Schedule 9) are the two exempted prohibited substances which may be acquired only by the Secretary of Health for the purpose of providing it to a medical practitioner for the treatment of a patient subject to specified conditions.

B. ACQUISITION AND SALE OF VETERINARY THERAPEUTIC SUBSTANCES

(a) *Unscheduled substances*

Provided these products are registered as *stock remedies* under Act 36 of 1947 and the provisos of this Act are complied with, they are freely available from general dealers and also from pharmacists. The veterinarian may also sell these to clients for the treatment of animals under his care but he may not keep an "open shop".

(b) *Schedule 1 and 2 substances*

These may only be sold by pharmacists. However, under Section 22A(15) the veterinarian, medical practitioner or dentist, is also allowed to sell Schedule 1-7 substances "...in the course of lawfully carrying on his professional activities as such to or for any patient or animal under his care or treatment". He may, however, not keep an "open shop" or pharmacy. A prescription is not required for these substances but the person to whom Schedule 2 substances is sold must be over 16 years of age. If not, a written order from a person known to the seller is required.

(c) *Schedule 3 to 7 substances*

As above, these may be acquired by the veterinarian on order but are only available to the public and animal owner on a veterinary prescription through a pharmacist. Prescription requirements become progressively more rigorous; the following apply to different schedules:

Schedule 3: This may be obtained on a written prescription or a verbal (e.g. telephonic) instruction. The pharmacist may repeat the prescription without the prescriber's instructions for 6 months.

Schedule 4: Only a written prescription is acceptable and a verbal instruction must be confirmed within 7 days. The prescription may only be repeated if the number of times and intervals at which it may be dispensed, is specifically stated. If this is not done the prescription will be dispensed once only.

Schedule 5: Only a written prescription may be dispensed and no verbal (telephonic) prescriptions are allowed. It may be repeated as for Schedule 4.

Schedules 6 and 7: Only a written prescription is valid and it may not be repeated. It is only valid for 30 days from the date of issue. The quantity prescribed may not exceed that required for a period of 30 days. The quantities must be written in figures as well as words. For a veterinarian to obtain these substances a written order is required by law.

C. REGISTERS AND RECORDS TO BE KEPT BY THE VETERINARIAN

Whereas the pharmacist must keep special registers of all transactions of substances in Schedule 2 to 7, the veterinarian must keep a permanent record, in his own handwriting, of all medicines listed in these schedules which he has sold or used. Such records must state: The date of sale or use, the form of preparation and quantity, and the name and address of the person supplied. Such a record must be retained for at least 3 years from the date of the last entry.

For substances listed in Schedule 5 and 6 the veterinarian must, in addition, keep a record of all acquisitions in the form of the invoices of purchase and retain these for at least 3 years.

Every veterinarian who handles Schedule 7 substances must keep the Schedule 7 Substances Register* in which the receipts, issues and usage of all these products must be noted as they occur and must be balanced quarterly.

D. PRESCRIPTION REQUIREMENTS

According to the Medicines Control Act a veterinary prescription must comply with the following requirements. It must contain:

(a) The name, qualifications and address of the veterinarian. (This may be in print but all other details listed below must be in his own handwriting.)

(b) The date.

(c) The name and address of the person to whom the scheduled substance is to be sold. (Although not required by Regulation, the animal(s) for which the medicine is intended, should be specified.)

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* Obtainable from A.P.S. (Pty) Ltd, Box 31360. 2017 Braamfontein at R6.25 each.

(d) The name and quantity of the substance or medicine. For Schedule 6 and 7 substances the quantity must be given in words as well as in figures.
(e) The prescription must be signed by the veterinarian.

Note that the words "For veterinary use only/Sleg vir veeartsenykundige gebruik", as required under Act 13 of 1928, is no longer necessary on a prescription.

Furthermore it is important to remember that if the prescription is not specifically endorsed "non nomen" and initialled to indicate that the name of the drug may not be mentioned on the label, the pharmacist is compelled to label the container with the name of the drug prescribed.

E. LABELLING OF VETERINARY PRESCRIPTION REMEDIES

At this stage, where these products are only controlled as substances, the following are the label requirements by law:
(a) It must be in both official languages.
(b) The name and the approved name(s) of the active principle(s) and composition by mass, volume or units.
(c) The name and business address of the seller.
(d) Storage and preservation requirements, if any.
(e) Name and percentage preservative, if any.
(f) Batch number and expiry date (where applicable).
(g) Dosage, if practicable.
(h) The letter "S" and the schedule no. e.g. S4.

F. SALE OF PRODUCTS WHEN NOT IN THE ORIGINAL CONTAINER

When a veterinarian sells scheduled substances to a client in a container other than the original (e.g. when a few tablets are dispensed from a stock bottle) the following must appear on his label:
(a) The name and address of the veterinarian.
(b) Directions for use.
(c) Name of the person to whom the drug is sold (and the animal(s) for which it is intended).

It would appear logical that the veterinarian should put the name of the drug on to this label as well.

G. REQUIREMENTS REGARDING PHARMACEUTICAL REPRESENTATIVES

Only Schedule 1 to 4 substances may be handled by an unqualified pharmaceutical representative for a manufacturer or wholesale dealer in pharmaceutical products and then only under specified conditions:

He must have a document of authorisation from such manufacturer or wholesaler on which his name and address and the names and quantities of the substances he may have, as well as the period of authorisation, is specified. This document shall be produced, on request, to any person to whom such substances are sold. The sale of these substances may only take place on a written order issued and signed by the veterinarian. On this the latter's name and address, the name and quantity of the substance sold and the date on which the sale took place must appear.

In conclusion, it is essential to point out that the privilege and right of the veterinarian to handle and sell scheduled substances in the course of lawfully carrying out his duties is bestowed on him personally and that he may not employ any other person to do it for him. If he does this he may be criminally prosecuted.

The definition of a *stock remedy* under Act 36 of 1947 has been changed in order to comply with the new Medicine and Related Substance Control Act. The grey area in relation to veterinary prescription remedies that existed in the past, particularly in the minds of the pharmaceutical profession, has thus been clearly defined. It is trusted that some of the existing and past problems and misunderstanding between the two professions in this regard will now be cleared up. It is also quite clear that veterinarians must take full cognizance of the legal changes that have been brought about. We will have to adapt our prescription habits and our ways of handling prescription remedies accordingly. As members of a responsible profession we must at all times act according to the provisions of the law.

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THE CONTROL OF TICKS, FLEAS AND LICE ON DOGS BY MEANS OF A SENDRAN* — IMPREGNATED COLLAR

I.G. HORAK**

ABSTRACT

Horak I.G. **The control of ticks, fleas and lice on dogs by means of a Sendran-impregnated collar.** *Journal of the South African Veterinary Association* (1976) **47** No. 1, 17-18 (En) Faculty of Veterinary Science, Onderstepoort, 0110, South Africa.

Plastic collars impregnated with 9,4% Sendran effectively controlled adult *Rhipicephalus sanguineus* for a period of 49 days and immature ticks for a period possibly in excess of 70 days when fitted to four dogs. Four uncollared dogs served as controls.

Although flea burdens were extremely low the collars were apparently effective for a period in excess of 70 days.

A medicated collar killed all the ticks, fleas and lice on a severely parasited dog within a period of 48 hours.

INTRODUCTION

The use of insecticide impregnated collars for the control of ticks and fleas on dogs and cats has been described by various authors[1 2 3 4]. The mode of action of these collars is by the slow release of small amounts of insecticide in either powder, crystal or gaseous form.

The present paper describes a trial utilising a plastic collar impregnated with θ-Isopropoxyphenyl methylcarbamate (Sendran).

MATERIALS AND METHODS

EXPERIMENT 1

Dogs

Eight dogs, consisting of 3 Dachshunds (short-coated, one dog, two bitches), 1 mongrel (wire-haired, dog), 1 mongrel (silky-coated, dog), 1 Cocker Spaniel (silky-coated, dog), 1 Keeshond (long-haired, dog) and 1 Labrador cross (short-coated, bitch) were housed in separate pens in a kennel.

Kennel

The kennel consisted of 12 separate pens, divided by a wall into two rows of six pens each. Portion of each pen was roofed and these portions were separated by walls that did not reach the roof. The remainder of the pen was open and separated from adjacent pens by wire-mesh.

The bedding in the roofed portion consisted of sacking and the whole kennel had a concrete floor.

Management

The majority of dogs was rotated through the various pens at 7-10 day intervals. The exceptions being the Keeshond and Labrador cross which were not rotated.

The roofed portion of each pen was not cleaned out nor was the bedding replaced. The open portion was cleaned twice daily.

Ecto-parasites

Tick and flea counts were done on each dog at approximately seven day intervals and in addition on each of the two days immediately after fitting the collars. The whole dog was examined and with the exception of the first three counts the ticks were identified as immature or adult. At various occasions adult ticks were removed for specific identification.

Collars

After the first two ecto-parasite counts, collars impregnated with 9,4% Sendran* were fitted to the Dachshund male and one bitch, the Cocker Spaniel and the Labrador cross.

EXPERIMENT 2

A mongrel dog, which had been caught in a snare, was presented for treatment and was found to be heavily infested with lice, ticks and fleas. The dog was fitted with a Sendran-impregnated collar* and housed in a cage.

The cage floor was swept 16, 48 and 72 hours after fitting

Table 1: **ECTO-PARASITE RECOVERIES ON COLLARED AND CONTROL DOGS**

Day	Mean number and range of immature and adult ticks recovered from				Fleas	
	Collared Dogs		Controls		No. of Dogs infested	
	Immature	Adult	Immature	Adult	Collared	Controls
- 8	15 (2-31)		22 (4-40)		0	1
0	33 (8-65)		26 (3-44)		0	1
	Collars fitted	—	—		Collars fitted	—
+ 1	19 (2-37)		31 (5-47)		0	0
+ 2	1 (0-3)	8 (0-19)	11 (0-29)	0 (3-21)	0	0
+ 7	0	0	2 (0-5)	7 (0-16)	0	0
+14	0*	0*	20 (0-47)	4 (0-9)	0*	1
+22	20 (0-46)	1 (0-1)	96 (8-180)	8 (1-13)	0	0
+28	4 (1-11)	1 (0-3)	44 (14-82)	7 (2-12)	0	2
+35	1 (0-3)	1 (0-4)	29 (6-84)	8 (5-10)	0	1
+42	0	2 (0-6)	35 (8-70)	8 (6-9)	0	3
+49	1 (0-2)*	4 (2-7)*	46 (6-106)	11 (6-17)	0*	1
+56	0	4 (2-6)	25 (5-56)	9 (6-13)	0	1
+63	1 (0-4)	5 (2-9)	18 (0-52)*	14 (8-21)*	0	1*
+70	1 (0-1)	6 (0-15)	4 (0-12)*	13 (6-20)*	0	1*

* Only three dogs examined

* Thuron Industries, Inc.

** Faculty of Veterinary Science, University of Pretoria, Box 12580, Onderstepoort 0110.

* VET-KEM Tick and Flea Collar : I. LOPIS & SONS

the collar and the sweepings collected and examined, the dog was also examined for parasites at each occasion.

RESULTS

EXPERIMENT 1

All the adult ticks examined were identified as *Rhipicephalus sanguineus*. The results of the ecto-parasite counts are summarised in Table 1.

Collared Dogs

The fitting of the collars on Day 0, resulted in a marked reduction in tick numbers during the following two days and no ticks were recovered seven and 14 days after fitting. Twenty-two days after collaring, these dogs harboured a mean number of 20 immature and one adult ticks. Thereafter, the immature tick burdens fell markedly while the adult tick numbers rose gradually but constantly until the termination of the trial.

The adult tick numbers on the Labrador cross rose more rapidly than on the other collared dogs.

No fleas were encountered on these dogs throughout the experimental period.

Controls

Tick numbers declined on these dogs during the seven days after collaring the other dogs, this decline was largely due to a drop in immature tick burdens. The latter burdens rose rapidly thereafter to reach a peak of 96 immature ticks on Day + 22, declining again subsequently. After reaching the lowest level in the trial on Day + 14, the adult tick burdens rose erratically to reach a peak on Day + 63.

Fleas were not always present, but one or more dogs were infested from Day + 28 onwards.

EXPERIMENT 2

The numbers of ecto-parasites recovered from the floor of the cage are summarised in Table 2.

Table 2: NUMBER OF DEAD ECTO-PARASITES RECOVERED AFTER FITTING A SENDRAN-IMPREGNATED COLLAR

Period after collaring	Numbers of dead ecto-parasites recovered				
	Heterodoxus sp.		*R. sanguineus*		*Ctenocephalides canis*
	Nymphs	Adults	Im-matures	Adults	Adults
16 hours	4 983	2 057	82	4	21
16-72 hours	430	227	10	4	3
Total Nos. recovered	5 413	2 284	92	8	24

The majority of ecto-parasites was recovered from the floor of the kennel within 16 hours of fitting the collar. All these parasites were dead and no living lice, fleas or ticks were observed on the dog when it was examined on this occasion; dead lice and ticks were, however, present. When the dog was examined 48 and 72 hours after collaring only dead ticks and no other parasites were observed.

DISCUSSION

EXPERIMENT 1

The collars effectively reduced the numbers of ticks on the collared dogs when compared with the controls, and it would appear that the immature stages are particularly susceptible to the acaricide.

The apparent reduction in efficacy that occurred 22 days after collaring must be viewed in the light of the fairly severe challenge with immature ticks which was occurring at that time. Thus although the collars were not keeping the dogs tick-free, they considerably reduced the numbers of ticks under conditions of severe challenge. Similar observations have been made by Polk in the United States[4].

Efficacy in excess of 60% against adult ticks lasted approximately 49 days, while that against immatures was possibly considerably longer.

The reduction in tick numbers on the control dogs shortly after collaring the other dogs was probably due to the amount of acaricide present in the kennels from the freshly fitted collars, thus reducing the amount of infestation available.

Although the number of dogs infested with fleas was disappointingly low, it can be assumed that the collars were effective against fleas, for not one of the collared dogs became infested, whereas one or more of the control dogs were usually infested. In trials in the United States the efficacy of the collars against fleas lasted for more than 91 days, thus considerably exceeding that against ticks[3].

EXPERIMENT 2

The lice were highly susceptible to the effects of Sendran and were observed to be dropping from the dog within two hours of fitting the collar. As no untreated control dogs were available no estimation can be made as to the duration of efficacy of the collar against lice.

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THE "HERD APPROACH" TO BOVINE REPRODUCTIVE DISORDERS

PART I: THE DIAGNOSIS AND MANAGEMENT OF INFECTIOUS INFERTILITY

G.P. RETIEF* A.P. SCHUTTE** AND R.I. COUBROUGH***

ABSTRACT

Retief, G.P.; Schutte, A.P.; Coubrough, R.I. **The "herd approach" to bovine reproductive disorders. Part I: The diagnosis and management of infectious infertility.** *Journal of the South African Veterinary Association* (1976) **47** No. 1, 19-22 (En) Division of Veterinary Services, Onderstepoort, 0110 South Africa.

The authors describe the methods employed by the Artificial Insemination Section of the Division of Veterinary Services in collaboration with the Reproduction and Bacteriology Section of the Veterinary Research Institute, Onderstepoort when herds are examined for infertility. A "herd" rather than an "individual animal" approach is advocated and certain infectious causes of infertility are discussed.

INTRODUCTION

The diagnosis and treatment of certain reproductive disorders, particularly those of an infectious nature, in individual cattle serves very little purpose unless the basic problem in the herd has been identified and if possible rectified [11 31 36 38].

It is often difficult to convince cattle owners of the wisdom of such an approach. The veterinarian is usually merely called to attend to one or more "problem" animals. This may hamper diagnosis of the problem since in most cases, to arrive at a diagnosis, bulls as well as a selection of cows should be examined [12 36]. As the selection process may be incomprehensible to him (the selected animals need not show any obvious symptoms) selection should not be left to the farmer's discretion.

A full herd examination takes time and the owner may baulk at the extra expense, but with state assistance any progressive farmer should be easy to convince that it is justified. Once the basic problem is solved, the farmer should be happy to follow instructions regarding management, nutrition and recording. Periodic routine visits would then follow as a matter of course.

The methods described in this article have been employed on many herds by the AI Section of the Division of Veterinary Services in collaboration with the Reproduction and Bacteriology Section of the Veterinary Research Institute Onderstepoort with gratifying and sometimes marked improvement in reproductive performance in certain herds.

METHOD OF INVESTIGATION

PRELIMINARY INFORMATION

Advance arrangements are made with the diagnostic laboratory regarding the number and type of specimens to be taken, as well as the expected date and time of arrival.

A detailed herd history is obtained from the owner (Table 1). Few farmers are able to supply full details. Those available indicate how well records have been kept.

EXAMINATION

Cows

Where the calving rate is poor, and the pregnancy state of the herd is unknown, examination for pregnancy is done, using plastic shoulder length gloves.

About 20 cows in each of the following categories are then selected for further examination:

1. Cows with poor breeding history e.g. repeat breeders, cows showing anoestrum, a discharge or having aborted.

Table 1: RECORD SHEET USED IN HERD EXAMINATION

1.	Name and address of farmer and private veterinarian.
2.	Breed: Stud/Grade
3.	Breeding policy: AI /handservice/bull running with cows? If the latter: breeding season or throughout the year?
4.	Number of cows: In calf: Dry, not in calf: Recent calvers: Heifers : In calf: Not pregnant: Calves : Unweaned: Weaned: Bulls : Used: Immature:
5.	Origin of the herd: Where and from whom were most of the animals bought. Out of hand/by auction.
6.	Closed/Open herd?
7.	Type and quality of records kept:
8.	Contact with animals of different species on the farm and with cattle on neighbouring farms?
9.	Abortions: Number, early/late?
10.	Retained placenta — after abortion/normal calving. Is it a problem in the herd? Yes/no
11.	Abnormal oestrous cycles?
12.	Discharge especially 10-21 days after service?
13.	Previous reproductive history of the herd/the basic complaint of the owner/when did the trouble start?
14.	Management and nutrition: Stocking rate: Camp system: Supplementary feeding: Heifers first bred by weight or age? From this history we try to ascertain whether the problem is (a) Infectious or (b) Functional.

2. Cows and heifers served 10-21 days prior to the examination; and
3. Recent calvers.

The cows in these groups are all examined per vagina and rectum. The state of the vagina, uterus and ovaries is described. In each case mucous is collected from the cervix and vagina. A plastic insemination pipette is attached to a syringe, introduced into the cervix, and negative pressure is produced and maintained while the pipette is withdrawn. If possible the mucous is forcibly ejected into a sterile collecting bottle, or otherwise the ends of the pipette are heat sealed.

Finally 100 ml of blood is collected from each cow into sterile bottles (without preservative).

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Bulls

Bulls are most important animals as far as examination and sampling are concerned, as the chances of a positive diagnosis is far greater than in cows. Preferably examine all the bulls in the herd, or a selection of the older bulls, immediately after the breeding season, or while the bulls are running with the cows.

Sheath-washings are collected before genital examination of the bulls. To avoid urinary contamination, the cranial portion of the prepuce and the opening is stimulated manually. This usually induces micturition. Fifty ml of a buffer solution obtained from the laboratory is run into the preputial cavity via a sterilised glass or plastic funnel and rubber tube.

With the tube still *in situ,* the preputial opening is held firmly around it, while the prepuce is vigorously massaged for at least 100 strokes. The funnel is then rapidly lowered to syphon out the sheath-washing. The end of the tube is then pinched closed, removed from the prepuce and inserted into the original bottle to collect the fluid.

To avoid the spread of disease and contamination of samples, a separate sterile tube and funnel is used for each bull.

Correctly obtained sheath-washings will be milky or cloudy with preputial debris.

The testes are then palpated, accessory glands examined per rectum and details recorded.

Semen is obtained by rectal massage of the accessory sex glands, or otherwise by electrical stimulation. For microbiological examination the samples of semen should be taken as aseptically as possible, avoiding contamination with preputial material. The latter is achieved by relaxing the penis with an ataractic drug such as "Combelen*", and exterior fixation by means of a bandage prior to collection of semen in sterile bottles. The samples are dispatched to the laboratory immediately after an aliquot has been examined for motility and viability.

Poor motility and viability under these conditions are usually regarded as insignificant, as these factors may easily have been influenced by the collection methods. In addition two semen smears are prepared; one being stained by the Karras method[13], or with India Ink[4], and examined for abnormal sperm, and the other with nigrosin/eosin.

Semen samples showing sperm abnormalities higher than 20% and especially where there is a large number of head abnormalities are considered to indicate a possible lowered fertility of the bull[24]. In evaluating the semen sample both the immediate and past breeding history of the bull must be taken into account[8].

HANDLING OF SPECIMENS

Rapid transportation of the specimens from the farm to the laboratory is essential. The owner may himself arrange for transport by road or air. Every effort is made to ensure that they reach the laboratory before 15h00 on the day of collection. If this is not possible, mucous from the cows is collected on the first afternoon and refrigerated overnight. (If dry ice is available this period may be extended even further.) This procedure is not acceptable for sheath-washings.

Sheath-washings are then collected on the morning of the second day and dispatched immediately to reach the laboratory within 8 hours of collection[7 8 32]. Blood samples are allowed to coagulate overnight after which the serum is decanted into sterile 10 ml bottles for various tests.

If Infectious Pustular Vulvo-vaginitis (IPV) or chlamydiosis is suspected, the serum is kept under refrigeration, until duplicate serum samples are collected a month later. The latter are sent to the laboratory with the former.

FARM OR SURGERY DIAGNOSTIC METHODS FOR TRICHOMONIASIS

Trichomonas foetus is fragile outside the host and only remains viable for a period of 8-12 hours after collection[8 29 32]. Therefore where circumstances prohibit the timeous dispatch of samples to the laboratory, the following procedure may be adopted in the follow-up work after treatment[25]. This should only be done if the diagnosis of trichomoniasis alone is at issue.

1. The sheath-washing is centrifuged for 10 minutes at 2 000 r.p.m.
2. Three separate drops of the sediment are microscopically examined for the characteristically moving organisms, using a 40× objective lens.
3. About three drops of the sediment are then added to a medium prepared as follows[26]:
   (a) Heat separated milk in a double boiler for 20 minutes.
   (b) Cool and fill sterile 10 ml bottles.
   (c) Add 1 mg/ml streptomycin sulphate and 1 000 IU penicillin g/ml prior to use.
4. Incubate at 35°C (an egg incubator may be used or the sealed bottles may be placed in a vacuum flask water-bath at 37°C).
5. After 2 to 3 days a drop of the medium is placed on a microscope slide and examined under a 40× objective lens. A positive culture invariably separates out into a clear fluid and milk solids. The organisms are usually easily located in the clear fluid[5]. About 20% of positive cases are missed when resorting only to direct microscopic examination. Therefore inoculation and incubation of the specimens is essential. Some samples are positive on direct microscopic examination but fail to grow on the medium[28].

A I HERDS

Where conception rates are low, technique and timing of insemination, oestrous observation and general management are checked. One or more of these factors are usually responsible, but non-specific infection and functional infertility are often found in such herds[3 11 36]. (See Part II.)

Few farmers make exclusive use of A I. Bulls are frequently used on dairy heifers or after the A I breeding season in beef cattle to pick up any cows missed during the season. Venereal diseases can therefore not be disregarded when seeking the causes of infertility in such herds. In one of two recently examined herds, vibriosis was spread by the use of an infected vasectomized teaser bull, while in the other, transmission occurred when infected semen collected from a bull on the farm was used.

INTERPRETATION OF RESULTS AND DISCUSSION OF SPECIFIC DISEASES

VIBRIOSIS

If sheath-washings or mucous specimens from any bull or cow in a herd are positive for vibriosis, all cows, mature heifers and bulls should be inoculated with vibriosis vaccine[5 7 9]. The monovalent vaccine used by earlier workers[5 7 9] apparently freed bulls of the infection. The trivalent vaccine now available in limited quantities in South Africa has a strong curative effect on bulls and cows and confers a local immunity which protects them from infection[34]. The conception rate of many infected herds has improved to a remarkable extent by using the vaccine on all adult animals[28 33].

A positive diagnosis often depends upon the stage of the cycle relative to sampling, when infective mating took place, and the stage of the breeding season. The best samples from cows are taken at the oestrum following an infective mating, and from bulls during the breeding season. Where accurate selection of probably infected animals is difficult because of inadequate records, as many animals as possible should be sampled. Where a single positive sample is found, the entire herd is presumed infected.

A I still remains the safest way of combating the spread of this disease.

TRICHOMONIASIS

All positive bulls are treated if trichomoniasis is diagnos-

* Bayer.

ed in sheath-washings or mucous. Excellent results have been achieved in more than 80 bulls treated with Dimetridazole ("Emtryl"*) intravenously[6 18 27]. More soluble formulations of Dimetridazole**, Tinidazole***, Ipronidazole**** and other imidazole derivatives are also giving good results[27].

The problem of re-infection of the 'cured' bulls by carrier cows remains a vexing one, since there is as yet no sure way of eliminating the carrier state[29]. Most cows develop an immunity and "cleanse" themselves after two or three oestrus cycles, but anoestrus cows and pyometra cases may remain carriers[29]. Cases of pyometra are encountered in infected herds but they constitute less than 1% of all the cases examined, and must therefore not be regarded as typical of the disease in a herd[28]. Cows may carry the infection throughout a normal pregnancy[21], and although this observation has not been substantiated in recent years, it must be borne in mind. It is therefore safer to rest cows for at least two cycles after calving. Treated and clean bulls should be used only on virgin heifers, and cows in their third cycle after two rest cycles. The bulls are tested periodically and treated if necessary. Young bulls act merely as mechanical transmitters of the disease and may be used in lieu of a rest period.

As is the case in vibriosis the spread of this disease is also limited by the use of A I.

"EPIVAG" (Infectious epididymitis of bulls and vaginitis of cows).

The aetiology of this syndrome is still not fully understood. Diagnosis depends on clinical signs in the herd. On speculum examination cows will show copious lemon-yellow, stringy, odourless mucous adhering to the walls of the reddened vagina.

In chronically infected herds a high percentage of cows may have cystic ovaries, chronic salpyngitis and/or perioophoritis.

A large number of the latter may have chronic to subacute peritonitis and even pleuritis without manifesting clinical signs[35]. This fact must be remembered when advising slaughter of these cows, as many of them may be condemned as unfit for human consumption for this reason.

The disease in bulls often commences as an acute to subacute inflammation of the seminal vesicles. When palpated rectally, these glands are diffusely swollen and have indistinct lobulations. Pressure on them induces pain.

The more chronic lesion becomes localised in one or more of the lobules, and is usually palpable as a hard painless swelling.

The infection usually spreads to one of the epididymides after a time varying from weeks to months. The lesion usually remains unilateral and starts as a painful diffuse swelling of either the head or the tail. In chronic cases the lesion is a hard, well-defined, painless swelling varying from 2 to 10 cm in size, or the entire epididymis may be affected, which may become so large as to cause pressure atrophy of the testis[35].

The aetiology of this disease is unknown. Various investigations have, however, revealed the following:

(a) Because of the failure to isolate pathogenic bacteria from the vaginal pus of infected cows, viruses have long been thought to be the cause of the disease. A number of viruses were in fact isolated, and investigations are continuing. At present their true role in the aetiology is not clear.

(b) Almost invariably *Mycoplasma bovigenitalium* and or a Uroplasm can be isolated in large numbers from the pure seminal plasma of bulls showing lesions or from bulls in an infected herd not showing lesions. *Mycoplasma bovimastitides* produces the typical lesions in cows. A variety of mycoplasms have been isolated from the prepuce of clinically "normal" bulls from "clean" herds. It is therefore important to eliminate preputial material from a semen sample if a correct diagnosis of pathogenic mycoplasmas is to be made[32].

(c) *Chlamydia* have been isolated from the accessory sex glands of infected bulls.

(d) *Vibrio fetus* and/or *Trichomonas foetus* are often encountered in these herds.

(e) The serum of many bulls and a large number of cows from an infected herd contains antibodies to IPV virus but this can also be said of many other apparently healthy herds. Approximately 70% of all cattle tested in South Africa show positive IPV titres[32].

(f) The disease is not necessarily associated with unhygienic conditions and poor management practices.

The only worthwhile form of control is achieved by using A I after elimination of the cows with the ovarian pathology. It is imperative that culled animals be slaughtered and not auctioned.

Under conditions where A I is not practical, replacement of bulls showing lesions is the only way to manage the disease to a certain extent. Regular examinations of the entire herd should be carried out to eliminate cows with ovarian pathology.

IPV (INFECTIOUS PUSTULAR VULVOVAGINITIS)

This disease is probably of minor importance in the causation of infertility in South Africa because of its transient nature and the lack of a susceptible population as most herds tested show positive IPV titres and are probably immune.

In a susceptible herd, the virus spreads rapidly through virtually all the cows causing an acute pustular vulvovaginitis which is transient; no trace of the lesion remaining after 14 days[14].

Bulls may show acute pustular balano-posthitis[30], which may require treatment with antibiotic ointments to control secondary bacteria which aggravate the lesion and thus enhance adhesion formation.

Diagnosis depends upon the demonstration of the lesions and a rise in antibody titre in two serum samples from the same animals taken at a month's interval. The initial serum sample is stored under refrigeration and forwarded one month later with the second sample.

IBR/IPV virus is rarely isolated from aborted foetuses in South Africa[15 32].

CHLAMYDIOSIS

A history of abortions and weak calves suffering from arthritis, pneumonia, diarrhoea and/or nervous symptoms is often indicative. In paired serum samples taken as in the case of IPV a rise in titre is considered positive. In order to diagnose the disease by demonstration of the causative organism, fresh foetuses, (preferable), calves or specimens from all internal organs, including brain and cotyledons, should be brought to the laboratory as soon as possible. If this is not possible, organ specimens may be frozen and submitted packed on ice, ensuring that they reach the laboratory in a refrigerated state.

Should chlamydiosis be diagnosed in a herd, all the mature females may be inoculated with 2 ml of inactivated ovine Enzootic abortion vaccine produced by the Veterinary Research Institute, and a booster dose one month later. In our experience vaccination does appear to assist in the control of the disease. Immunity develops in about 2 months[16 17]. Antibodies may not pass through the placental barrier.

If the problem centres in the newborn, a combination of chloramphenicol and tetracycline may be administered to all calves for 2-3 days immediately after birth. This seems to control the clinical manifestations of the disease since it has been shown to have an effect on the elementary bodies[32].

Mixed infections are often encountered in fertility work and especially in the new-born. This is particularly true of chlamydia which is often isolated in conjunction with *Escherischia coli, Salmonella* and viruses[32]. This should be borne in mind when diagnosis, prophylaxis and treatment are considered.

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* "Emtryl soluble powder" May Baker.

** "Emtryl Injectable" May Baker.

*** "Fasigyn" Pfizer.

**** "I P Z soluble powder" May Baker.

BRUCELLOSIS

With the increasing trend towards intensification of animal production, new problems have arisen concerning the control of this disease. Immunity to the disease in herds where heifers are regularly inoculated is never so strong that a sufficient challenge will not break it down. Thus under intensive conditions where a massive build-up of infection may take place due to the congestion of animals, the disease may strike even in inoculated herds[22] [23].

Where there is a history of abortions, serum samples from a representative number of cows (especially cows having aborted more than a month prior to the test) will show a positive agglutination test. An aliquot of the positive sera should also be submitted for the complement fixation test. If these tests are also positive, and the cattle were not inoculated in the previous six months, a diagnosis of brucellosis may be made in the herd.

Often a series of different tests like serum agglutination, complement fixation, and Rose Bengal tests is necessary in order to distinguish between the different types of immunoglobulins produced in response to vaccination or infection[19] [20].

To combat the disease in a herd, the farmer may choose to eradicate the disease from the herd, or to limit its spread. Eradictaion is achieved by slaughter of all positive reactors. Limiting the spread is achieved by vaccination of the entire herd (previously unvaccinated), after identification of the positive reactors, with the warning that 2-3% of cows in the last trimester of pregnancy may abort[29].

Positive cows which abort or calve down normally may shed the bacteria for about 3 weeks after calving. An attempt should therefore be made to isolate these animals for at least a month after parturition[37]. Where practical, sleeping and feeding quarters are moved and cleaned.

CONCLUSION

The control of infectious genital diseases is possible if a herd approach is adopted. Much work remains to be done, but by correct use of available diagnostic and advisory services the shockingly low calving and calf rearing percentage in South Africa can be vastly improved.

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STUDIES ON THE LIFE CYCLE OF THE LUNGWORM, *PNEUMOSTRONGYLUS CALCARATUS*, MÖNNIG, 1932

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ABSTRACT

Anderson Irmgard G.H. **Studies on the life cycle of the lungworm,** *Pneumostrongylus calcaratus,* **Mönnig, 1932.** *Journal of the South African Veterinary Association* (1976) **47** No. 1, 23 - 27 (En) University of Zululand, Empangeni, 3880 South Africa.

The morphology of the first, second and third stage larvae of *Pneumostrongylus calcaratus* is described. Greater numbers of larvae were present in experimentally than in naturally infested slugs. An attempt to transmit infestation to impala was unsuccessful.

INTRODUCTION

In a previous publication, preliminary observations on the life cycle of the lungworm, *Pneumostrongylus calcaratus,* Mönnig, 1932 were reported.[7] The slug, *Urocyclus (Elisolimax) flavescens* (Keferstein, 1866) was experimentally infested with *P. calcaratus* to recover the larval stages. Descriptions of the larval stages in the previous report were based on fixed specimens; while the present descriptions are based on fresh material only. Naturally infested slugs were collected and the incidence and degree of infestation compared with those infested experimentally.

In an attempt to transmit infestation to the definitive host, slugs were infested and the larvae obtained from them fed to handreared impala without success.

MATERIALS AND METHODS

Slugs, *Urocyclus (Elisolimax) flavescens,* were infested and examined for first, second and third stage larvae of *P. calcaratus* as described previously[7]. The slugs were also examined microscopically while infestation was taking place.

At least 10 freshly killed, unfixed larvae of each stage were examined to determine the range of variation, mean and standard deviation of the various structures; when less than 10 readings were available the standard deviation was not calculated. The larvae were drawn with the aid of a camera lucida.

The number of *P. calcaratus* larvae were calculated in 30 experimentally infested and 35 naturally infested slugs. The foot of the slug was measured when it started to move. The slug was then killed with a scalpel and 3/5th of the foot was examined for larvae as described by Rose[15].

The 3/5th included one piece each of the anterior, middle and posterior parts. The estimated total number of larvae in the whole foot was then calculated. The naturally infested slugs were collected at 5 different stations on the Nyala game ranch[6] throughout the year. No collections were made during October, December and January.

A few of the impala shot on the Nyala game ranch[6] were also examined for larvae in the spinal cord.

Two of four small impala, were successfully handreared. These two impala, when 4 months old, were infested with third stage larvae recovered from both naturally and experimentally infested slugs. The impala were killed 7 and 18 days after infestation and all their internal organs as well as their spinal cords were examined for worms.

A number of first stage larvae were stored in water in order to determine their longevity.

Table 1: DIMENSIONS (μm) OF DEVELOPMENTAL STAGES OF *P. CALCARATUS*

Stages of development	First stage larvae		Second stage larvae		Third stage larvae	
	Range	Mean ± s.d.	Range	Mean ± s.d.	Range	Mean ± s.d.
*Total length	314-341	325,6±8,9	520-807	707,17±87,1	604-780	699,3±52,0
Width: Anterior end	5-10	7,4±1,6	5-18	12,9±3,7	8-27	16,5±6,7
Maximum	15-19	17,2±1,3	39-57	47,5±6,0	30-92	50,8±23,2
Anus	9-12	11,0	23-37	29,8±4,5	20-54	33,7
Oesophagus: Length	141-163	152,3±6,3	176-289	224,8±43,1	169-260	212,8±27,1
Width	11-14	12,5	20-34	25,3±5,0	20-32	22,9
Distance of excretory pore from anterior end	72-95	87,6±7,5	98-176	121,5±44,7	100-157	126,5±16,3
Distance of nerve ring from anterior end	74-108	94,5±9,0	118-182	147,5±23,9	125-189	162,0±25,7
Genital primordium: Length	—	—	11-16	14,2±1,8	9-49	22,5±11,9
Width	—	—	7-11	8,4±1,3	6-20	11,4
Distance from genital primordium to tail	—	—	221-322	279,2±28,5	163-329	235,0
Distance of anus to tail (with sheath if present)	19-39	30,1±6,9	46-72	55,8±8,2	39-113	62,7±25,3
Distance from tail to sheath	No sheath		4-27	10,2±9,0	1,52	20,2±24,3
Distance of sheath from anterior end	*No sheath*		11-49	17,8	7-22	51,0

*The total length includes the sheath if present.

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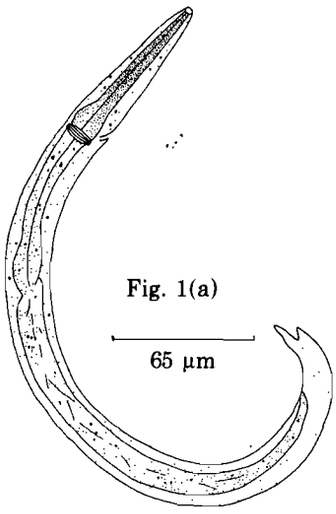

Fig. 1(a) First stage larva of *Pneumostrongylus calcaratus*
(b) Enlargement of tail with hook

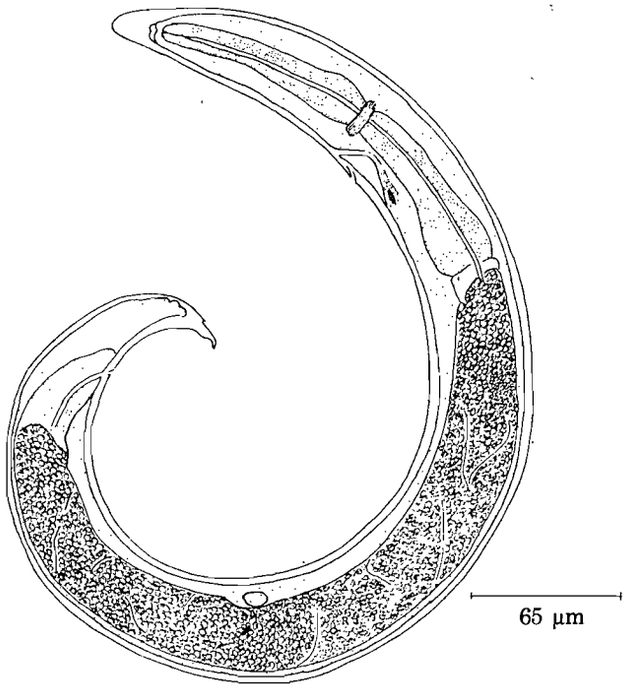

Fig. 2 Second stage larva of *P. calcaratus* within cuticle of first stage.

Table 2: **NUMBER OF *P. CALCARATUS* LARVAE OBTAINED FROM EXPERIMENTALLY INFESTED SLUGS**

Stage of development	Days after infestation	Size of foot (mm)	No. of larvae in different parts of foot			Total counted in three parts of foot	Estimated total of larvae in slug
			Anterior	Middle	Posterior		
*L_1	5	30 x 5	11	1	6	18	30
	"	25 x 6	1	2	1	4	7
L_1; **L_2	6	50 x 10	6	3	3	12	20
	"	45 x 10	12	13	20	45	75
	"	40 x 10	5	9	15	29	48
	7	22 x 5	1	1	3	5	8
	"	30 x 8	3	4	4	11	18
L_2	8	60 x 15	20	19	32	71	118
	"	40 x 10	1	2	0	3	5
	9	60 x 8	0	0	1	1	2
	11	45 x 13	36	34	21	91	152
	"	35 x 8	28	50	58	136	227
	12	50 x 10	>200	> 200	> 200	> 600	>1000
	"	45 x 10	10	3	1	14	23
	"	40 x 8	11	8	13	32	53
	"	30 x 5	0	0	1	1	2
	"	30 x 6	8	1	4	13	22
L_2; ***L_3	13	40 x 10	10	6	23	39	65
L_3	14	60 x 10	> 200	> 200	> 200	> 600	> 1000
	"	35 x 8	2	13	4	19	32
	15	60 x 15	50	100	100	250	417
	"	30 x 4	1	4	4	9	15
	16	35 x 5	6	1	5	12	20
	"	35 x 6	6	4	0	10	17
	20	60 x 8	28	31	8	67	112
	"	35 x 8	4	1	2	7	12
	21	60 x 15	5	4	4	13	22
	30	55 x 7	13	6	9	28	47
	41	45 x 8	7	3	0	10	17
	87	60 x 15	15	14	17	46	77

*L_1 — first stage larvae
**L_2 — second stage larvae
***L_3 — third stage larvae

Table 3: **NUMBER OF *P. CALCARATUS* LARVAE OBTAINED FROM NATURALLY INFESTED SLUGS**

Month	No. of slugs		Stage of development	Size of foot (mm)	No. of larvae in different parts of foot			Total counted in 3 parts	Estimated total number of larvae in slug
	Collected	Infested			Anterior	Middle	Posterior		
February	44	15	L_2	45 x 10	4	0	0	4	7
			L_3	35 x 8	3	1	0	4	7
				35 x 9	1	1	0	2	3
				40 x 9	3	1	0	4	7
				35 x 8	0	0	1	1	2
				35 x 10	2	1	0	3	5
				35 x 8	2	2	0	4	7
				40 x 10	5	2	0	7	12
				35 x 10	1	0	2	3	5
				35 x 8	1	1	0	2	3
				30 x 8	8	0	0	8	13
				40 x 12	3	0	0	3	5
				45 x 10	0	1	0	1	2
				40 x 10	4	1	0	5	8
				45 x 11	0	0	1	1	2
March	5	2	L_2	40 x 10	10	16	30	56	93
			L_3	35 x 10	5	5	1	11	18
April	1	0							
May	2	1	L_3	30 x 8	17	10	9	36	60
August	12	2	L_2	25 x 5	0	0	1	1	2
			L_3	20 x 5	1	0	0	1	2
September	25	1	L_3	15 x 4	0	1	0	1	2
November	42	14	L_3	40 x 7	1	0	0	1	2
				40 x 9	4	0	0	4	7
				30 x 6	1	0	0	1	2
				40 x 8	1	2	0	3	5
				30 x 6	0	1	0	1	2
				40 x 10	1	1	1	3	5
				30 x 6	1	0	0	1	2
				35 x 7	4	1	0	5	8
				40 x 8	6	0	0	6	10
				30 x 8	0	4	1	5	8
				40 x 10	1	2	1	4	7
				40 x 9	1	0	0	1	2
				40 x 8	1	0	0	1	2
				30 x 6	0	1	0	1	2

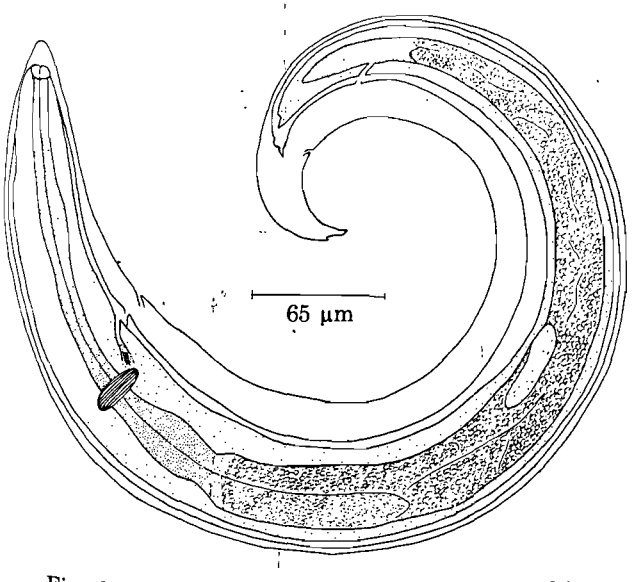

Fig. 3 Third stage larva of *P. calcaratus* within cuticle of first and second stage (after 20 days of infection)

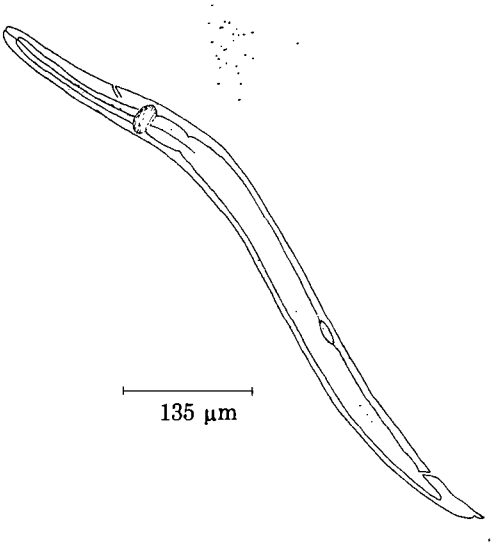

Fig. 4 Third stage larva of *P. calcaratus* without sheaths (after 21 days of infection)

RESULTS

Measurements of various structures of the first, second and third stage larvae of *P. calcaratus* are summarized in Table 1. These show that growth took place from first to second stage, but that from second to third stage the larvae did not get much longer.

These three larval stages are drawn in Figures 1, 2, 3 and 4.

(i) The first stage larva (fig. 1(a)) with its characteristic hook on the tail, was described earlier[7]. Figure 1(b) shows an enlargement of the hook on the tail. First stage larvae were able to survive in water for 8-10 weeks. It was also observed that first stage larvae moved fairly slowly, but as soon as they came in contact with mucous released by the slug, they started to move and twist their bodies rapidly.

(ii) After about 6 to 7 days of infestation, the second stage larva (fig. 2) was found in the foot of the slug. This stage has a dense, dark mass of food granules in all the cells of the gut. The second stage larva retains the sheath of the first stage and also has two small indentations on its tail. These indentations, the sheath of the first stage and the dense granulation make them easily distinguishable from the other stages. The dark granulation also makes them easy to find in the tissue of the slug.

(iii) After 13 - 14 days the third larval stage was found, often still surrounded by the sheaths of the first and second stage. In the early third stage the granules in the gut are still fairly dark (fig. 3) but they gradually become lighter and after 20 days of infestation, the parasite becomes almost transparent (fig. 4). The third stage larva can also be distinguished by the small, pointed appendage on its tail.

The experimentally infested slugs were examined on several days after infestation and the number of larvae obtained are given in Table 2. On day 6 to 7 the ratio of $L_1 : L_2$ was 82:20 and on day 13 the ratio of $L_2 : L_3$ was 36:3.

Table 3 lists the number of larvae found in naturally infested slugs. These slugs were collected throughout the year from the Nyala game ranch. Very few slugs were found during April and May and none during June and July. This table also shows that the naturally infested slugs are less heavily infested with lungworm larvae, than the experimentally infested slugs. In the experimental slugs two were still alive after 12 days with over a thousand larvae in their feet. Of the 100 slugs, however, which were to be used for infestation of small impalas, only 17 survived for 15 days. Slugs seem to die sooner when heavily infested with lungworm larvae. Amongst the naturally infested slugs, the greatest, estimated individual total burden was 93 larvae. Only 5 of the 35 naturally infested slugs had more than 10 estimated larvae in their feet. Amonst the experimentally infested slugs the greatest number was over a thousand and of the 30 slugs examined 25 had more than 10 larvae.

No lungworm larvae were found in the spinal cords of the impala shot on the Nyala game ranch. In some animals, however, little nodules of recent infestations were found on the outside of the lungs. When these nodules were opened one or two lungworms were found in each. One complete male was extracted and measured 35 mm in length.

No lungworm larvae were recovered from the two small impala which had been infested artificially.

DISCUSSION

Gerichter[5] found that the first stage larvae of *Cystocaulus ocreatus, Muellerius capillaris* and *Protostrongylus rufescens* could survive several months in water. Rose[15] kept *M. capillaris* alive in a faecal pellet for several months. These investigations showed that *P. calcaratus* can also survive in water for more than two months.

Gerichter[5] found that first stage larvae of *C. ocreatus* "makes vigorous efforts to penetrate the tissue" of the slug. In these experiments it was shown that contact with mucous excreted by the snails served as a stimulant to greater activity by the first stage larvae of *P. calcaratus*, thus enhan-

Table 4: **COMPARISON OF THE RATES OF DEVELOPMENT OF *P. CALCARATUS* AND OTHER LUNGWORMS IN THEIR INTERMEDIATE HOSTS**

Parasitic Species	Mollusc Species	Author	Developmental stage	Age in days	Temperature (°C)
Cystocaulus ocreatus	—	Gerichter[5]	L_2	13-14	20-30
Muellerius capillaris	*Angriolimax agrostis* & *A. reticulatus*	Rose[15]		8	20
Pneumostrongylus calcaratus	*Urocyclus (Elisolimax) flavescens*			6-7	20-30
Pneumostrongylus tenuis	*Zonitoides arboreus* & *Discus cronkhitei*	Anderson[1]		11	20-30
C. ocreatus	—	Davtjan[2]	L_3	30-40	19-24
	—	Gerichter[5]		18-19	20-30
	Helicella & *Helix* spp.	Kassai[12]		30-46	20-21
	Zelrina, Theba & *Cepaea* spp.	Kassai[12]		20-30	20-21
Elaphostrongylus rangiferi	*Trichida hispida* & *Succinea putris*	Mitskevitch[14]		27-35	—
M. capillaris	—	Gerichter[4]		35	30
	—	Hobmaiers[8]		12-14	—
	Zonitoides arboreus	Mapes[13]		27	—
	A. agrostis & *A. reticulatus*	Rose[15]		10-13	20
	Hyalina cellaria & *Milax sowerbyi*	Williams[16]		14	—
Parelaphostrongylus odocoilei	*A. agrostis, A. linne, A. campestris, Helix aspersa* & *Epigramophora arrosa*	Hobmaiers[10]		35	—
P. tenuis	*Z. arboreus* & *D. cronkhitei*	Anderson[1]		15-16	20-30
		Anderson[1]		27	18
P. calcaratus	*U. (E) flavescens*			13-14	20-30
Protostrongylus rufescens	—	Davtjan[3]		35-42	—
		Hobmaiers[9]		12-14	—
		Joyeux & Gaud[11]		15-20	—

cing their chances of infesting the intermediate host.

A comparison of the rates of development of the second and third stage larvae are listed in Table 4. This shows that the rate of development of *P. calcaratus* is similar to that of *M. capillaris* as found by Hobmaier[8], Rose[15] and Williams[16]. The second stage of *Pneumostrongylus tenuis*[1] takes about 4 days longer and that of the third stage only one to two days longer than *P. calcaratus*. In the other lungworms mentioned in the table, the rate of development is more than three days longer than in *P. calcaratus*.

Measurements of *P. calcaratus* and *P. tenuis* have been compared in a previous publication[7] where some fixed material was used. The anatomy of *P. calcaratus* as shown in figures 1-4 is similar to that of *P. tenuis* as described by Anderson[1]. The only observed difference is the indentations on the tail of the second larval stage. The third stage found in the tissue of the slug, is also often still within the sheaths of the first and second stages.

No feeding probably takes place in the second and third stage, due to the fact that sheaths from the previous stages surround the larvae. The food granules that are present in the intestine probably serve as a source of nutriment.

Since no lungworm larvae were found in the little impala which had been artificially infested, further investigations will be carried out to determine the mode of infestation in impala.

ACKNOWLEDGEMENTS

Grateful appreciation is expressed to Messrs. I. and R. Scott-Barnes and G. Meintjies for placing their ranches at my disposal and for helping me to obtain the material for this survey. My sincerest thanks go to Dr A. Verster for her assistance and advice and to Mrs M. Lotz for assisting in the preparation of the figures. Grateful thanks also go to the University of Zululand for the facilities provided and to the C.S.I.R. for their financial support.

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BOOK REVIEW | BOEKRESENSIE

PROCEEDINGS OF THE IDF SEMINAR ON MASTITIS CONTROL 1975

F.H. DODD, T.K. GRIFFIN AND R.G. KINGWILL

International Dairy Federation, Secretary General, Square Vergote 41, 1040 Bruxelles, Belgium. Pp X + 512, Tabs. 146, Figs. 80, price 500 Blg. Fr. (R11,32) including packing and postage.

The proceedings are an accurate record of some 64 papers presented and the discussions held at a seminar on mastitis control, attended by about 150 delegates from 24 countries at Reading University from 9-14 April 1975.

The major aims of the seminar were to bring together people with a direct interest in the control of mastitis either in research, the organization of control schemes, extension services or in the dairy industry, to review briefly the factors that influence the prevention and elimination of infection, and to concentrate attention on how such available knowledge can be used to control the disease in bovines. Its effective control could reduce losses in production by at least 10%, an enormous quantity of some 38 000 million kg of milk per year on a world-wide basis.

The contents of the proceedings closely follow the programme of the seminar. Included are an introductory paper, six main sessions pertaining to the diagnosis of mastitis and intramammary infection, somatic cell counting, prevention of infection, elimination of infection, mastitis control systems and implementation of control techniques respectively, the reviewing session and conclusions.

As with the highly efficient organization of the successful seminar, the editors can also be congratulated on its Proceedings. Participants in the seminar are still ruminating on the wide range of very interesting and stimulating data offered. The Proceedings constitute a veritable mine of information to all associated with the hygienically preventive supervision of milk supplies and dairy cattle in particular. Though not necessarily providing the ready-made answers possibly expected, the bound copy of the Proceedings is a comprehensive source of information worth its price even in these inflation-ridden times.

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THE DIAGNOSIS AND TREATMENT OF ACID-BASE DERANGED DOGS INFECTED WITH *BABESIA CANIS*

W.D. MALHERBE*, A. IMMELMAN**, W.H. HAUPT* AND H.J. WALZL***

ABSTRACT

Malherbe, W.D.; Immelman, A.; Haupt, W.H.; Walzl, H.J. **The diagnosis and treatment of acid-base deranged dogs infected with** *Babesia canis*. *Journal of the South African Veterinary Association.* (1976) **47** No. 1, 29 - 33 (En) Veterinary Research Institute, Onderstepoort, 0110, South Africa.

A study was made of the acid-base status of *Babesia canis* infected dogs judged unlikely to recover after specific babesicidal drug therapy despite the use of blood transfusion and other conventional supportive measures. Such cases were invariably acidotic and responded well and often dramatically to supportive intravenous sodium bicarbonate administration. Elevated blood urea nitrogen, also responded gratifyingly to this procedure. The rationale is discussed in some detail.

INTRODUCTION

Specific drug treatment of *Babesia canis* in dogs has been largely feasible since about 1909 when trypan blue dye was found[12] to be effective. As drug resistance developed to both this drug and its successors, new ones were synthesized. These have proved very efficient in their parasiticidal effect. The usual supportive procedures in severely affected cases, notably blood transfusion, administration of vitamin B complex preparations and liver protectants, proved to be of some use. A certain proportion of cases remain refractory to all treatment, and these can be recognized by experienced clinicians as being unlikely to recover.

Laboratory tests[4-9] were carried out to determine the organic changes in such animals which made their recovery uncertain. It was concluded at the time[9] that "infected dogs could die from various causes: medical shock in peracute cases (particularly young dogs), liver failure, kidney failure, heart failure and occasionally pulmonary failure. Different combinations of these could be operative, and they could play an important part in morbidity and mortality even after elimination of the causal parasites by appropriate specific chemotherapy"

Despite some improvements in the supportive treatment some dogs failed to respond and died. These included those with:

a. the peracute form, usually young dogs very heavily parasitized in spite of a history of very short illness and frequently in a state of medical shock;
b. severe icterus;
c. a history of having received specific treatment several days previously but remaining anorectus and lethargic;
d. haemoglobinuria; and
e. severe hyperpnoea associated with the infection.

Blood transfusion, aimed at correcting the anaemia and therefore expected to reverse organic involvement, proved disappointing. Lactated Ringers' solution similarly only occasionally appeared to be of any material benefit. Empirical attempts were made to correct suspected acidosis by intravenous administration of a saline drip containing an arbitrary amount of chemically pure sodium bicarbonate. Vomiting, however, proved troublesome and the procedure was abandoned.

A definitive study was clearly required to establish reasons for the poor results obtained in the categories of cases mentioned above.

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DETERMINATION OF ACID-BASE STATUS OF "POOR-PROGNOSIS" CASES

The acid-base status of these animals was investigated during the summer of 1972-1973.

Cases of canine babesiosis presented at the Outpatients Clinic of the Faculty of Veterinary Science were selected for the purpose of this study on the basis of a clinical conviction of slender chances of successful treatment.

On presentation blood was collected anaerobically from the jugular vein by means of "Venoject" * evacuated glass specimen tubes containing sodium heparin as anticoagulant. This provided for expeditious sampling with a minimum of trauma to red cells and no exposure to air.

The acid-base studies were carried out by means of a Radiometer pH meter Model 27, in conjunction with the Astrup Micro Tonometer for the equilibration of the blood as described by Astrup[1] at two different and known levels of carbon dioxide (CO_2) in oxygen.

The pH meter has an expanded scale with scale divisions of 0,01 pH units and has a suction microglass electrode with a capillary tube to contain a micro-quantity of blood of the order of 20 μl. The apparatus was kept warm with circulating water at 38°C so that specimens could be processed without delay. This arrangement, moreover, obviates to a large extent hysteretic ("memory") effects and allows for automatic and immediate adjustment of the specimen to the correct temperature.

By means of the Siggaard-Andersen Curve Nomogram[13 14] the acid-base status was characterized from the "actual" pH of the blood and the two pH readings at the known CO_2 levels after equilibration.

Blood urea nitrogen (BUN) was determined by the titrimetric method of Hench and Aldrich[3], conjugated and unconjugated plasma bilirubin by a modification of the classical Malloy and Evelyn method[10], and routine haematology by use of the Coulter Counter.

For reference a "normal" range of acid-base parameters of relevance to this study was determined by constructing percentage ogives (or cumulative relative frequency polygons)[15] and reading off the 80% frequency range for each of the parameters. The work was done at an altitude of about 1 450 m above sea level. The values regarded as "normal" are given in Table 1.

Table 1: NORMAL ACID-BASE VALUES FOR JUGULAR BLOOD OF DOGS AT ONDERSTEPOORT

Parameter	Mean	Range
"Actual" pH	7,36	7,32 - 7,42
Base excess (BE) (mEq/l)	-6	-3 to -8
P_{CO_2} (mm Hg)	33	30 - 35
"Total CO_2 content" (mM/l)	18,8	16,6 - 21,5

* Jintan Terumo Co., Ltd., Tokyo

When the "actual" pH of the blood was within the range given, the status was regarded as being either normal or compensated. Above the range it was alkalaemic (uncompensated alkalosis), and below, acidaemic (uncompensated acidosis).

A numerically higher figure for negative base excess (BE) was interpreted as indicating *metabolic acidosis* and a lower or a positive one as a *metabolic alkalosis* regardless of pH. The CO_2 tension ($P\ CO_2$) as the "respiratory component" reflected *respiratory acidosis* when higher than the range or *respiratory alkalosis* if it fell below the normal range. A metabolic acidosis could possibly be compensated (through hyperventilation of the lungs) by a respiratory alkalosis resulting in a normal or near normal "actual" pH.

"Total CO_2 content", figures representing the sum of dissolved CO_2 in the plasma and the CO_2 liberated by the dissolved plasma bicarbonate on addition of acid in the laboratory, are included in the table for comparison with figures which can be obtained by Van Slyke type methods such as the Natelson Microgasometer. Since nearly all the "total CO_2" is contributed by bicarbonate the figure obtained is often loosely called the bicarbonate level.

Upon clinical identification of cases classified as having a prognosis of poor-to-hopeless, blood was collected anaerobically and the acid-base status immediately determined. A solution containing sodium bicarbonate was then administered intravenously at a dosage rate which varied somewhat during the course of the summer. The following formula was used most frequently:

$$X \times \frac{\text{mass of patient (kg)}}{5} = \text{mEq sodium bicarbonate}$$

required where X represented the negative "base excess" figure, numerically decreased by about 4 mEq. For example, if the BE figure was –19 mEq/*l* the requirement would be calculated as about 15 mEq per kg of extracellular fluid. The figure 5 represented very roughly the fraction of body mass comprising extracellular fluid.

In this work use was made of commercial solutions presumably sterilized by means other than autoclaving:

a. Baxter's Plasmalyte B (a balanced electrolyte solution containing 28 mEq of sodium bicarbonate per litre).
b. 8,5% sodium bicarbonate in water (i.e. 1 mEq/ml). (Saphar)
c. 4,2% sodium bicarbonate in water (0,5 mEq/ml). (Saphar)

In shocked cases Plasmalyte B proved suitable for restoration of effective circulation blood volume but in most cases it was considered too bulky and had to be supplemented. The 8,5% solution proved to be more flexible as it could be injected as supplied or added to a simple glucose-saline or plain isotonic glucose solution, or even to transfusion blood directly, and was well tolerated.

After this infusion or transfusion all patients were treated with a babesicidal drug, either phenamidine (May & Baker) or imidocarb (Wellcome) as these could then cause little distress.

When blood transfusion was decided upon the dose used in the later part of the study was based on the formula

$$\frac{\text{Desired PCV} - \text{Patient's PCV}}{\text{Desired PCV}} \times 80 \text{ ml}$$

Table 2: **SUMMARY OF RELEVANT DETAILS OF POOR-RISK DOGS WITH BABESIOSIS**

Dog No.	Dog 1		Dog 2		
Description	Alsatian dog, 9 m., 19 kg.		Keeshond bitch, 5 m., 8 kg.		
Clinical comment	Very weak, icteric, dyspnoeic		Very weak, icteric, haemoglobinuric		
"Astrups"	Day 1	Day 2	Day 1		
pH	7,39	7,49	7,38		
BE	–14,8	–9,8	–9,9		
P_{CO_2}	15,6	17,6	25,2		
T_{CO_2}	9,5	13,5	15,1		
BUN	—	104,9	68,1		
Additional treatment	None		None		
Outcome and Remarks	Died Day 3 of nephritis		Died Day 1 of shock		

Dog 3	Dog 4		Dog 5		
Alsatian dog, 7 m., 28 kg	Alsatian dog, 10 m., 20 kg		Mongrel dog, 2 y., 7,5 kg		
Had to be carried, icteric	Icteric almost comatose		Collapsed, icteric		
	Day 1	Day 2	Day 1	Day 2	Day 3
—	—	7,30	7,27	7,37	
	—	–10,5	–23,5	–4,4	
	—	30,2	13,3	35	
	—	15,2	9,8	20,8	
—	—	—	97	55,2	42,3
Included B.T., Pl.B. 150 ml on Day 1, 25 mEq bicarb. and B.T. on Day 2	Day 1 B.T. only Day 2 Pl.B. 220 ml = 6 mEq Day 3 Pl.B. 280 ml = 8 mEq Day 4 Pl.B. 350 ml = 10 mEq Day 5 Pl.B. 300 ml = 8,4 mEq		Day 1 30 mEq bicarb. and 200 ml of blood		
No improvement Day 1 Prompt recovery after treatment on Day 2	Improvement present but tardy after Pl.B. initiated. Inadequate bicarb. content.		Very acidotic. Made good recovery after bicarb. treatment.		

Dog 6				Dog 7		Dog 8			Dog 9	
Rottweiler x bitch 13,5 kg				Mongrel dog, 1,5 y. 25 kg		Collie dog, 4 y., 24 kg			Poodle bitch, 2 y., 6 kg	
Peracute, very anaemic, comatose				Very anaemic, collapsed, comatose		Very icteric			Very severely anaemic and slightly icteric	
Day 1 a.m.	p.m.	Day 2	Day 5	Day 1	Day 2	Day 1	Day 2	Day 3	Day 1	Day 4
7,14	7,34	7,44	—	7,21	7,45	7,40	7,38	—	7,42	7,42
−24	− 9,4	− 5	—	−14,5	− 2,4	− 5,5	− 7,8	—	−15,2	− 5
10	28,5	26	—	31,5	32	30	26,4	—	13,2	27,7
5,8	15,5	18	—	9,9	21,7	19,2	15,9	—	8,6	18,5
62,5	—	26	20	81	—	—	68,1	49,7	70	33,1
Day 1 a.m.: 30 mEq bicarb and 150 ml Pl.B. After second "Astrup": 750 ml Pl.B and 18 mEq bicarb.				50 mEq bicarb and 400 ml blood on Day 1 150 ml Pl.B. on Day 2		15 mEq bicarb given on Day 2			Day 1: 12 mEq bicarb and 250 ml blood	
"Remarkable" clinical response after first infusion				"Spectacular" recovery from shocked condition		Improvement started after bicarb. on Day 2.			Good recovery.	

Dog 10		Dog 11	Dog 12	Dog 13		
Boxer bitch, 2 y. 17 kg		Gt. Dane dog, 1 y. 38 kg	Husky dog, 4 m., 17 kg	Alsatian dog, 7 m., 26,5 kg		
Collapsed, very anaemic, severe respiratory distress		Collapsed, dyspnoeic, icteric, anaemic	Terminal shock 6 hrs after specific treatment	Peracute, haemoglobinuric, severely dyspnoeic and shocked		
Day 1	Day 4	Day 1	Day 1	Day 1	Day 2	Day 3
7,21	7,43	7,49	6,97	7,40	7,46	7,48
−21	− 1	− 8,2	−29	− 9	− 3,2	− 2,6
15	35	18,7	11,5	23,5	28,3	27
6	24,1	14,7	4,1	14,7	20,5	20,3
92	18,4	57	79,1	82,8	—	20,2
Day 1: 5 mEq bicarb. and 350 ml blood		Blood transfusion only	None	25 mEq bicarb. on Day 1		
Immediate clinical improvement and and good recovery		Died overnight	Died just after specimen taken	Uninterrupted recovery after bicarb.		

Dog 14		Dog 15	Dog 16		
Collie dog, 9 m., 22 kg		Chihuahua bitch, 5 m., 2,5 kg	Labrador X Alsatian dog, 3 m., 6,5 kg		
Practically dying day after trypan blue treatment		Collapsed, peracute, dyspnoeic, haemoglobinuric	Collapsed, icteric, deep abdominal breathing		
Before bicarb.	4,5 h later		Day 1	Day 2	2 h after treatment
7,43	7,48	7,30	7,46	7,33	7,44
−10,6	+ 1,4	−19,6	− 6,7	− 9,3	− 2,1
20,3	33	13,0	24,4	30,5	31
13,6	24	6,9	17,6	17	21,5
59,4	—	55,2	36,8	—	—
40 mEq bicarb. + 500 ml blood		12 mEq bicarb. + 50 ml blood	Day 1: 200 ml blood Day 2: 7 mEq bicarb and 120 ml blood		
Recovered from time of infusion		Recovered rapidly	Habitus improved within 2 h after bicarb.		

Dog 17			Dog 18			Dog 19		
Alsatian bitch, 6 m., 18 kg			Schipperke bitch, 4 m., 2,25 kg			Mongrel dog, 3 m., 7 kg		
Very anaemic, slightly icteric, collapsed, haemoglobinuric			Collapsed, dyspnoeic, very anaemic			Anaemic, haemoglobinuric, listless		
Before treatment	+ 4 h	+ 24 h	Before treatment	+ 4 h	+ 24 h	Before treatment	+ 4 h	+ 24 h
7,07	7,36	7,45	7,21	7,49	7,49	7,38	7,42	7,44
−22	− 8,2	− 5,5	−18,8	− 2	0	−11,5	−7,3	−7,2
14	28	25,5	19	29	30,5	23	23,5	23
4,4	16,0	17,9	17,8	22,9	23,4	13,4	15,3	16,1
64,4	—	—	62,7	—	—	—	—	—
50 mEq bicarb and 750 ml blood			6,6 mEq bicarb and 100 ml blood			7 mEq bicarb and 250 ml blood		
Manifestly on way to recovery 4 h after bicarb.			As Case 17			As Case 17		

Abbreviations

BE:	base excess (mEq/l)	m.:	months	P_{CO_2}:	Carbon dioxide tension (mm Hg)
Pl.B:	Plasmalyte B (Baxter)	y.:	years	T_{CO_2}:	"total CO_2 content" (mM/l)
bicarb:	sodium bicarbonate solution (mEq)	h.:	hour(s)	BUN:	blood urea nitrogen (mg/100 ml)
		min:	minute(s)	BT:	blood transfusion

per kg body mass of the patient. The abbreviation PCV represents packed cell volume (or haematocrit figure) and the 80 ml roughly the volume of blood per kg body mass.

Follow-up acid-base determinations were done as judged desirable and the BUN was monitored in some cases.

CASE REPORTS

The descriptions, clinical status, relevant laboratory findings, supportive treatment and outcome in respect of 19 dogs are presented in Table 2 in condensed form.

Acidosis was regularly present in the "poor prognosis" cases, frequently in a very severe degree, as was also urea retention.

The pH of jugular blood was sometimes within the normal range as in Dogs 1, 2, 8, 9, 13, 16 (day 2) and 19. In Dogs 11, 14 and 16 (Day 1) it was above the range and in the remaining cases (Dogs 4, 5, 6, 7, 10, 12, 15, 17 and 18) it was below. All, however, gave "base-excess" figures numerically greater to the negative side than about −8 mEq/l indicating a varying but often severe degree of metabolic acidosis.

The BUN figures, (normal animals up to about 25 mg/100 ml) were invariably elevated to well above this figure, and nearly always responded well to correction of the metabolic acidosis. This was facilitated by the fact that the great majority of dogs were young.

In the beginning, when the concept of "protection of pH" as the criterion of necessity for treatment of acidosis was held, several dogs died. Dogs 1 and 2 with full compensation on presentation, but acidotic, nevertheless died for lack of alkalinization. Dog 8 started clinical recovery only after bicarbonate was given on the second day. Dog 3, (23 kg) without acid-base data, received quite inadequate amounts of bicarbonate (150 ml Plasmalyte B, containing just over 4 mEq.). Clinical improvement was immediate after a 25 mEq dose.

Blood transfusion alone or combined with quite inadequate bicarbonate gave poor results in Dogs 4, 11 and 16, while the clinical effect was excellent with adequate bicarbonate administration as seen in Dogs 5, 7, 9, 10, 14, 15, 17, 18 and 19. Dog 12 illustrated the sort of figures for acidaemia which may be found in terminal cases but which may possibly still have recovered if there had been a little more time.

The severely anaemic patients showing severe acidosis and which responded well after receiving only bicarbonate as supportive treatment, are represented by Dog 6.

DISCUSSION

The 19 cases in this report are considered to be representative of the somewhat larger group selected for study by experienced clinicians as animals which had very little or no prospect for recovery using the methods previously in vogue.

For supportive treatment the most important procedure had been blood transfusion in order to correct the anaemia resulting from the disease. The often disappointing results obtained could be readily understood when it was realized that these poor-prognosis cases were regularly acidotic and that the transfusion blood contributed more acid, so aggravating the existing acidosis. Moss[11] has drawn attention to this fact, quoting work that gave the level of acid in bank blood as high as 5-8 mEq/l. This would necessitate the infusion of bicarbonate in similar amount just to counteract the introduction of more acid. For a look at this aspect in the context of the present study, blood was collected from a healthy dog into a heparinized "Venoject" tube as well as into a standard transfusion vacuum bottle containing the requisite amount of ACD mixture.

Immediately after collection the pH was 7,48 and 6,88 respectively. After two days storage under refrigeration the "bank" blood's pH was 6,80; after seven days 6,63; after 14 days 6,55 and after 24 days it was 6,45. The lesson was plain.

Lactated Ringer's solution was tried as nearly physiological with indifferent results. It is now realized that sodium lactate, though producing bicarbonate in a body with good liver and kidney function, does not do this in severely acidotic patients. Presumably there is an excessive concentration of lactate ions in the blood as a result of tissue hypoxia and consequent defective oxidation of lactate to pyruvate. It has also been pointed out[2] that the last stages of intermediary metabolism (citric acid cycle) following the production of carbon dioxide, water and energy could be written as follows:

$$CO_2 + H_2O \rightleftharpoons H_2CO_3 \rightleftharpoons H^+ + HCO_3$$

The consequent formation of bicarbonate from lactate or indeed any other carbon compound fully oxidized to CO_2 must be accompanied by the production of an equivalent number of hydrogen (H^+) ions. So, unless or until the distal tubular cells of the kidneys are able to exchange H^+ for Na^+ or any other cations there can be no net gain of bicarbonate ions. Since in acidosis there are already too many H^+ ions, this is not possible. Moreover, the regular finding of elevated BUN levels is an indication of impaired renal perfusion and function which would further reduce the renal capacity for dealing with H^+ ions.

The acidosis of babesiosis results primarly from the anaemia and anoxaemia produced by the destruction of red blood cells by the parasites and immune mechanisms. This

reduced oxygen carrying capacity of the circulating blood results in reduced oxidation of lactate to pyruvate in the glycolytic pathway and an accumulation of lactate and depletion of bicarbonate in the tissues. Metabolic acidosis may lead[11] to a decrease of effective circulating blood volume through relaxation of the precapillary sphincters with resultant pooling and engorgement of the capillaries. Myocardial contractility is reduced and cardiac output decreased. This results in lowered perfusion of all tissues and hence formation of more lactic acid, a rise of PCO_2 from less efficient pulmonary function and retention of fixed acids, sulphuric and phosphoric, (products of normal metabolism), as a consequence of reduced functioning of the kidneys. Potassium levels would tend to rise and the vicious cycle leads ultimately to fibrillation and heart failure. When this whole process proceeds precipitately enough the picture presented is that of shock.

Clearly this process must be terminated and sodium bicarbonate is the logical substance to do this by being immediately available to bind H^+ ions.

The "actual" pH of the blood has been found to be of less importance than the extent of base deficit (or negative "base excess"). A compensated pH (within normal limits) is accomplished in an acidotic animal at the expense of great respiratory exertion. Where these hyperventilating animals were treated with bicarbonate they were greatly relieved within a few hours, resulting in major saving of energy. Dog 5, for instance, with a respiratory rate of 46/min before bicarbonate was given, had a rate of 30/min forty minutes after infusion was commenced; 27/min after 60 min and 22/min after 85 min.

In addition to this reduction of energy consumption, correction of the base deficit has other effects as illustrated by Dog 6 which received no blood transfusion though severely anaemic. Here the administration of bicarbonate alone, resulted in immediate and lasting clinical improvement. Clearly the oxygen carrying capacity of the blood was enhanced with the assumption of improved oxygen saturation at the higher pH making more oxygen available per unit mass of haemoglobin in the more acid milieu of the tissues. Moreover, with the restoration of the effective circulating blood volume and cardiac output, and hence of adequate tissue perfusion, the oxidative processes could be restored to the benefit of the overall functioning of tissue cells.

Alkalaemia following treatment in some cases has been explained as resulting from the administration of too much bicarbonate or from hyperventilation to compensate for the anaemia. Citrate ions from transfusion blood could in these circumstances also become available for conversion to bicarbonate. This, however, seemed unimportant and clinical recovery did not seem to be affected in any way.

Van der Horst[16] has stated in connection with bicarbonate treatment in humans that complete restoration of neutrality and normality is not mandatory and need not to be attempted. The intravenous administration of bicarbonate may produce complete or only temporary and short-acting benefit, depending on the ability of the kidneys (or lungs) to promote homeostasis and the presence or absence of a continuing primary cause for the acidosis. In the case of babesiosis, elimination of the primary cause is generally easily accomplished by specific babesicidal chemotherapy. The dosage therefore does not appear to be very critical though it can be anticipated that the best and quickest results would follow a correctly computed dose, based on exact acid-base characterization. The degrees of sophistication, dictated by the equipment available, as a basis for dosage, could be given as follows:

1. The Astrup procedure as used in this study: dosage as given above.
2. The Slyke type of procedure: the formula would be something like

   $$X \times \frac{\text{mass of patient}}{5} = \text{mEq bicarbonate required}$$

   where X is the difference between 19 and the "total CO_2" figure obtained.
3. No apparatus. Here clinical judgment would be important mainly of the degree of depression of the sensorium (drowsiness, coma) and the nature of respiration. The dosage would range from 1-3 mEq per kg body mass. A similar recommendation has been given by van der Horst[16] as a guide in paediatric practice.

The 8,5% sodium bicarbonate solution mainly used in this investigation was usually incorporated in a glucose-saline or plain isotonic glucose drip or in transfusion blood. It can also be injected directly into a vein in cases of emergency. Usually, however, fluid therapy is advisable to assist in restoring effective circulating blood volume. A point which should be mentioned is that the 8,5% solution has six times the concentration of an isotonic one and is very irritant in subcutaneous and perivenous tissues. The amount of fluid to be administered depends on clinical evaluation and varies from about 10 - 30 ml/kg.

There are dangers associated with over-enthusiastic treatment with sodium bicarbonate[2]. Important enzyme systems can be directly affected by metabolic alkalosis and potassium depletion in the body may seriously affect carbohydrate metabolism. In the present series, however, possibly as a result of acid-base monitoring, this was no problem and homeostasis was rapidly established.

In conclusion, the words of Winters, Engel and Dell[17] are worth quoting:

"Laboratory data, clinical acumen and physiological orientation are interdependent in the management of sick patients with acid-base disorders — as they are in every other area of clinical medicine."

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PRELIMINARY REPORT ON THE INTERMANDIBULAR CUTANEOUS GLANDULAR AREA AND THE INFRAORBITAL GLAND OF THE STEENBOK

M. COHEN* AND W.H. GERNEKE**

ABSTRACT

Cohen M.; Gerneke W.H. **Preliminary report on the intermandibular cutaneous glandular area and the infraorbital gland of the steenbok.** *Journal of the South African Veterinary Association* (1976) **47** No. 1, 35—37 (En) Eugene Marais Chair of Wildlife Management, University of Pretoria, Pretoria 0002.

An intermandibular glandular area, never before described in any other ungulate has been found in the steenbok *(Raphicerus campestris)*. It consists of enlarged sebaceous and apocrine sweat glands situated in the upper two-thirds of the dermis. They produce a dirty white almost flaky odoriferous substance which clings to the hairs of the area and is easily rubbed off for marking territorial areas as well as for marking females during mating.

The infraorbital gland produces a black secretion which is the combined secretion of melanaceous, branched alveolar, sebaceous and enlarged apocrine sweat glands. The function of this secretion is not known as it is not used for any obvious marking purposes.

INTRODUCTION

Most mammals have a well-developed olfactory sense and extensively use chemical information in silent communication. The structures and modes of behaviour that are involved are many. Any animal utilizing the environment in a normal manner leaves tracks, urine and faeces in its path. These may serve as signals to other animals which pass by later. Many species are not, however, dependent on urine and faeces, but have evolved specialized integumentary scent glands. The deposition of the odoriferous products are often accompanied by characteristic patterns of movements and postures which serve to enhance the mark.

Steenbok *(Raphicerus campestris)* mark their environment by deposits of urine and faeces and, as has been assumed up to now, scent marks from various specialized cutaneous glands. Both males and females possess pairs of infraorbital (antorbital) glands, a single cutaneous intermandibular glandular area and interdigital pouches in fore- and hindfeet. The skin glands in these pouches are, however, comparable in size to those of the skin and may therefore not be actively used for marking. Microscopic examination of an adult male duiker *(Sylvicapra grimmia)* and an adult female oribi *(Ourebia ourebia)* showed the intermandibular glandular area to be absent in these two species.

This article is confined to:

a) a description of the intermandibular glandular area, which as far as can be ascertained has not been described in the steenbok before, nor in any other ungulate, and
b) a brief account of the infraorbital gland which has a remarkable melanaceous secretion.

MATERIALS AND METHODS

The observations reported here were made during a long-term field study on the biology and behaviour of the steenbok in the Kruger National Park by the first author. Observations were made using 8 x 30 Nikon binoculars from a Land Cruiser. All aspects of behaviour including the marking behaviour were photographed using a 500mm lense and Super 8mm movie film. Skin samples from the intermandibular area and other parts of two adult males and one adult female were collected and fixed in 10% formalin. These were processed using routine histological techniques. Paraffin sections cut at 5 microns were stained with acid hematoxylin and eosin.

GLAND TOPOGRAPHY AND ANATOMY

In the steenbok the intermandibular area is of a lighter colour and is covered with shorter coarser hairs (Fig. 1). Its central area, extending roughly from the chin to the region of the epiglottis contains the intermandibular glandular area as a palpable thickened glandular patch. In a verticai section it can be distinctly seen as a thickened glandular layer in the upper two-thirds of the dermis. In the living animal the glandular patch can only be distinguished from the adjacent areas by the presence of a dirty white, almost flaky secretion which easily attaches to any object which is rubbed over the area. The exact measurements and location of the gland will be published elsewhere (Gerneke and Cohen, in preparation).

The intermandibular glandular area was found to be present in all 40 adult animals examined during immobilization with a mixture of 10 mg Fentanyl* and 10 mg Rompun** for the purposes of attaching ear tags.

Microscopic investigation of the unspecialized skin of the steenbok revealed the usual small sebaceous and apocrine sweat glands associated with the hair follicles (Fig. 2). In the intermandibular glandular area of the female both sebaceous and sweat glands were enlarged (Fig. 3) but the greatest development was seen in the intermandibular glandular area of the males (Fig. 4). Apart from being more extensively coiled, the apocrine sweat glands revealed a high columnar epithelium indicating greatly increased activity which one would expect to occur in such an enlarged gland. Its activity is possibly further increased during mating seasons. This, however, remains to be determined. The sebaceous glands revealed larger cells and increased branching of the alveoli.

The infraorbital gland situated below the eyes was also found to consist of branched tubulo-alveolar sebaceous and enlarged apocrine sweat glands (Fig. 5). The combined secretion is black due to the melanaceous secretion of the sebaceous glands. Melanoblasts situated above the basal

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*Fentanyl, Ethnor Laboratories (Pty) Ltd. Box 1934, 2000 Johannesburg.

**Rompun, Bayer Pharmaceuticals, Box 10233, 2000 Johannesburg.

Fig. 1 The intermandibular area of the steenbok can clearly be distinguished by its covering with lighter coloured and coarser hairs. The intermandibular glandular area is an oval, more centrally situated palpable area containing in the living animal a dirty white almost flakelike glandular secretion on the hairs. (Photograph: H. Braack).

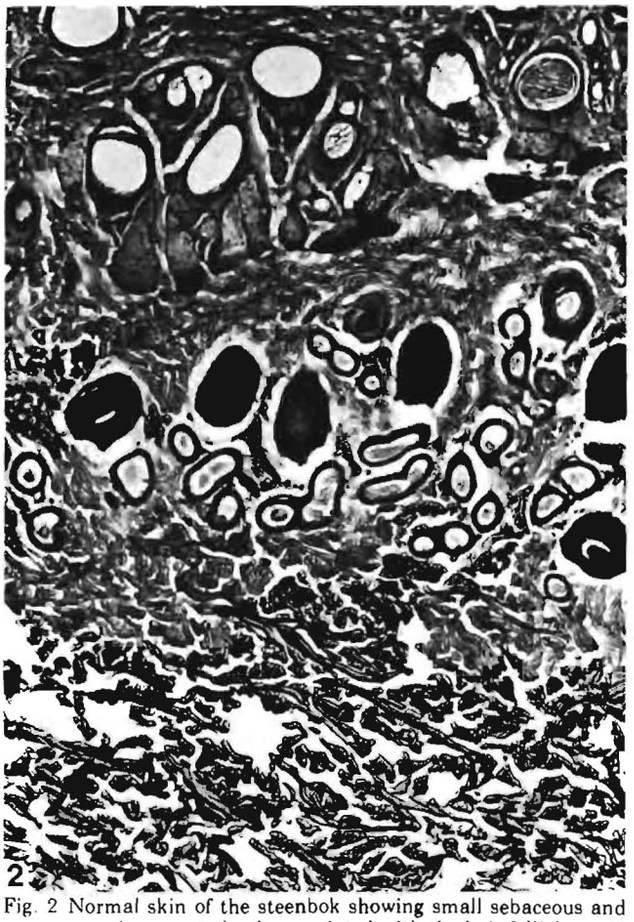

Fig. 2 Normal skin of the steenbok showing small sebaceous and apocrine sweat glands associated with the hair follicles. ×40

Fig. 3 The intermandibular glandular area of the female steenbok with both sebaceous and apocrine sweat glands increased in size. ×40

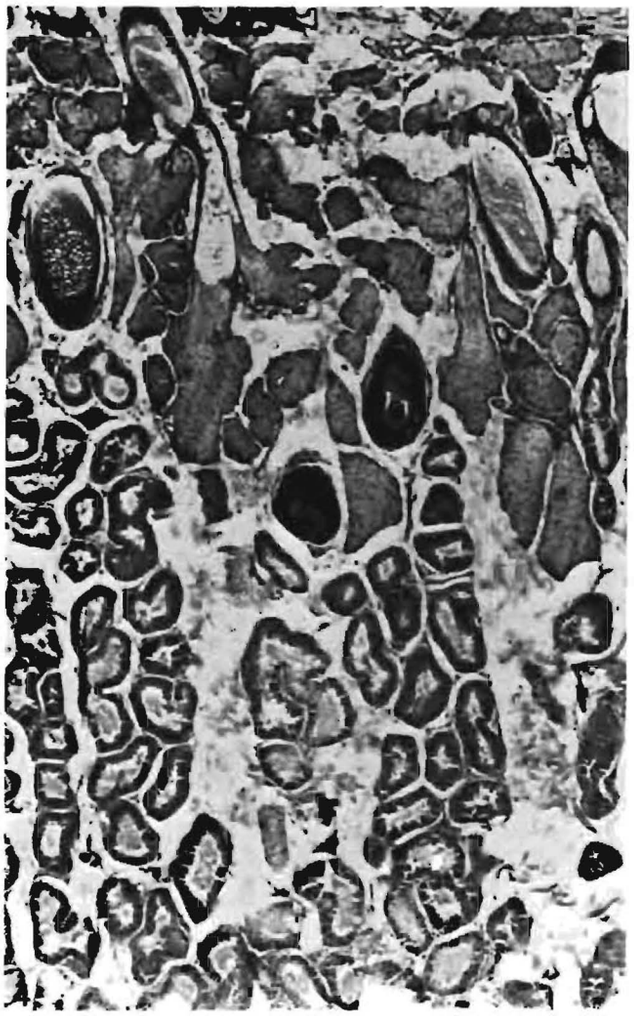

Fig. 4 The intermandibular glandular area of the male steenbok with greatly increased sebaceous and apocrine sweat glands. Only about two-thirds of the latter are shown at this magnification. ×40

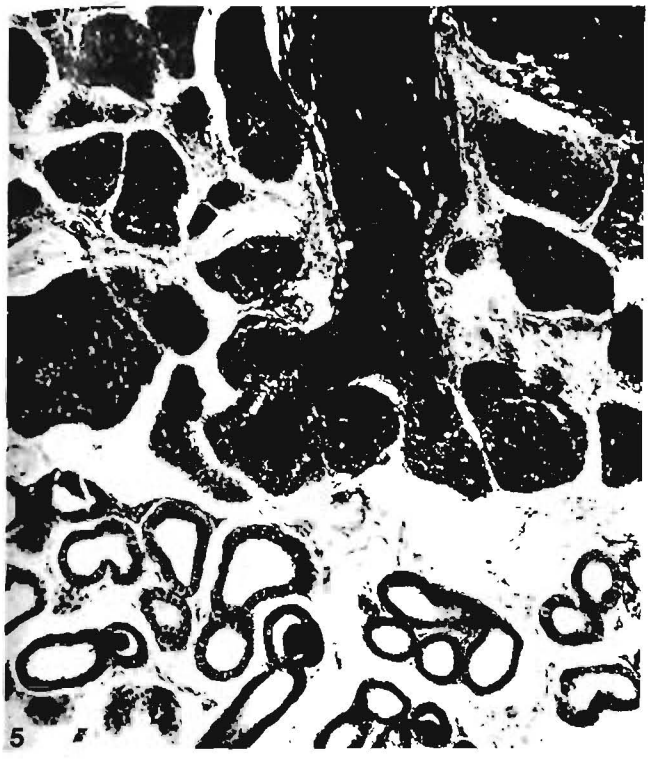

Fig. 5 Part of the infraorbital gland revealing the melanaceous branched sebaceous glands and enlarged apocrine sweat glands deeper in the dermis. ×64

Fig. 6 Two adjoining melanaceous alveoli of the infraorbital gland are shown separated by a thin layer of connective tissue. In the proximal layers of the alveoli branching melanoblasts (arrow) or their processes can be seen. By means of cytocrine secretion the melanin granules are passed from the processes to the sebaceous gland cells which are then seen filled with black melanin granules (†). ×800

laminae of the alveoli (Fig. 6) pass melanin granules into the sebaceous gland cells. These cells with melanin content and their own lipoidal globules are passed off (holocrine secretion) as a black mass and mix with the secretion of the apocrine sweat glands. Smaller granular masses apparently separated from the main mass can occasionally be seen at the opening of the gland. No obvious action on the part of the steenbok, causing their formation or deposition in the environment has yet been noted.

Such a melanaceous secretion of sebaceous glands is a unique feature and as far as can be ascertained has not been described before in animals.

The specimens for the above preliminary investigation were collected from animals which were in the solitary phase. Greater histological details of the glands will be reported elsewhere (Gerneke and Cohen, in preparation).

The function of the intermandibular glandular area corresponds to that of most of the other specialized cutaneous glands in secreting odoriferous substances contributary to marking or mating. It is inactive in very young steenbok as was indicated by the absence of secretion from the glandular area of a 16-day-old steenbok.

The function of the infraorbital gland, especially of its melanaceous secretion, has not yet been determined. Preliminary observations indicate that it is not used for obvious marking activity.

ETHOLOGY

Both male and female steenbok are solitary in habit. Solitary behaviour does not imply the absence of social organization and steenbok are in olfactory and optical contact with each other. Adult males and to a lesser extent adult females maintain a spacing system[1]. Several types of behaviour are employed in the maintenance of separate areas. One such activity is marking with the intermandibular glandular area.

There are two principal ways by which animals deposit odoriferous products in their environment:

1. Intentional marking; e.g. the secretion of the infraorbital gland in the oribi[2] and the gazelle *(Gazella thomsoni)*, is wiped off on to a marking post.
2. Incidental marking; e.g. the secretion of the interdigital gland in those species where it is present is deposited as the animals move about their activity zones.

Observations of steenbok have indicated that the intermandibular glandular area is used for both types of scent marking. The animals approach a marking site and mark it intentionally by placing this area on the "post" and rubbing off some secretion. Incidental marking occurs during normal feeding activity when animals pass the chin over a foliage in an attempt to reach more distal parts of the plant. In performing this action they inadvertently wipe some of the secretion on to the leaves and twigs.

Beside the territorial function the intermandibular glandular area has a reproductive function. Males have been observed intentionally to mark females, during courtship, or younger animals to establish their dominance. When in the vicinity of females, the frequency of intentional marking by males is higher than when they are alone.

ACKNOWLEDGEMENTS

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CLINICAL TRIALS WITH ORGOTEIN (PALOSEIN*)

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INTRODUCTION

Orgotein as the active principle of the drug Palosein, became available for clinical testing in horses in South Africa in 1975. It had previously been evaluated for the treatment of equine lameness in the United States[3,4] and the purpose of the trial reported below was to confirm its activity and define its benefits in equine practice in South Africa.

Orgotein is a water-soluble metalloprotein which occurs as an intra-cellular component of the tissues of several species of animal. When used therapeutically it acts extracellularly and possesses a variety of interesting pharmacological properties as described by Huber[6]. It has a potent modifying effect on the inflammatory process which unlike steroids does not delay healing, nor does it produce the side-effects associated with steroids.

The mode of action of orgotein is thought to be based on its chemotactic and stabilising effect on polymorpho-nuclear leucocytes (PMN's) together with its superoxide dismutase enzymatic activity. By increasing the concentration of PMN's in the circulation after its injection[5] orgotein enhances the phagocytosis of debris which accompanies or occurs as a result of inflammation. Secondly, orgotein prevents the release of lysosomal enzymes from the phagocytosing PMN's at the site of injury. This activity inhibits the exacerbation of the inflammation which these irritant substances normally produce.

One of the normal intra-cellular metabolites of tissue is known to be the highly toxic superoxide radical. Following injury this substance occurs extracellularly and it has been shown that phagocytosing PMN's release large amounts of the superoxide radical and a sizeable proportion of this escapes into the extracellular environment[1]. It is in this situation that the superoxide dismutase activity of therapeutically administered orgotein is thought to exert its effect. The function of the superoxide dismutase enzyme is to neutralise the toxic effects of the superoxide radical and this has been shown to occur both intra- and extracellularly[7].

Orgotein has been shown to be remarkably safe[2] in toxicological, teratological and immunological studies. It has been administered to horses by the intramuscular, sub-cutaneous, sub-conjunctival, intra-articular and intra-synovial routes with no adverse effects except for the occasional transient local reaction at the site of injection.

MATERIALS AND METHODS

This paper concerns a multicentric investigation involving 68 horses, almost all Thoroughbreds with only a few Crossbreds. The lesions treated were all caused by direct or indirect trauma and were classified according to the type of tissue primarily involved i.e. bone, articular and synovial structures or soft tissues.

The therapeutic regimen was variable. However, the pattern was intramuscular injection (occasionally locally into the lesion) of 5 mg Palosein at 1-3 days interval from 2-10 times depending on the severity of the injury. When a long series of treatments was prescribed the interval was usually lengthened. Conventional non-specific supportive treatment was given when indicated. In the majority of cases where local treatment was applied this was done only once or twice to be followed by intramuscular injection. Local infiltration was done in seven cases of soft tissue traumas, seven cases of bone involvement and in 20 cases intra-articular injection was given.

RESULTS

The results are shown in the following tables.

Table 1a: **BONE**

Response	
Excellent & Good	Poor & None
9	1

Table 1b: **COMPARISON WITH CONVENTIONAL TREATMENT**

Much better than expected	Better than expected	As expected	Worse than expected
3	4	2	1

Table 2a: **SOFT TISSUE**

Response	
Excellent & Good	Poor & None
22	7

Table 2b: **COMPARISON WITH CONVENTIONAL TREATMENT**

Much better than expected	Better than expected	As expected	Worse than expected	Assessment difficult
4	13	9	1	2

Table 3a: **ARTICULAR**

Response	
Excellent & Good	Poor and None
19	10

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Table 3b: **COMPARISON WITH CONVENTIONAL TREATMENT**

Much better than expected	Better than expected	As expected	Worse than expected	Assessment difficult
2	14	7	4	2

Table 4a: **ALL INJURIES (68 CASES): RESPONSE**

	Excellent & Good	Poor & None (including cases difficult to assess)
TOTAL	50	18
%	73,5	26,5

Table 4b: **COMPARISON WITH CONVENTIONAL TREATMENT**

Much better than expected	Better than expected	As expected	Worse than expected	Cases difficult to assess	Total
9	31	18	6	4	68
13%	46%	27%	9%	5%	

Statistical analyses was based on the Kolmogorov-Smirnov one sample test:

	Group 1 Better and much better than expected	Group 2 As expected and worse
I BONE		
Observed	7	3
Theoretically	0	10
Gives $\hat{D} = 0,7$; for n = 10 →	p <0,01	
II SOFT TISSUE		
Observed	17	10
Theoretically	0	27
Gives $\hat{D} = 0,63$; for n = 27 →	p<0,01	
III ARTICULAR RESPONSE		
Observed	16	11
Theoretically	0	27
Gives $\hat{D} = 0,59$; n = 27 →	p<0,01	
IV ALL INJURIES		
Observed	40	28
Theoretically	0	68
Gives $\hat{D} = 0,588$; n = 68 →	p<0,01	

No systemic side-effects were seen with injection into muscle, traumatised tissue, around bone or into joints. Swelling and tenderness were occasionally evinced but this passed off usually in a matter of days.

Only one definite case of recurrence of the injury was noted. It was generally felt that clinically many cases tended to recover so rapidly that the trainer would be tempted to put the horse to work too soon.

DISCUSSION

The anti-inflammatory action of a drug is not necessarily beneficial, if inflammation did not have survival value, it would have been eliminated in the process of evolution. It is generally accepted however, that certain aspects of the inflammatory process may have an adverse effect e.g. excessive oedema retards the movement of humoral and cellular defence mechanisms, or oxygen towards the injured area while the carrying away of noxious and catabolic substances is inhibited. It is therefore of great interest that the action of Palosein as far as has been established is its chemotactic effect on polymorphonuclear leucocytes, its stabilising effect on the walls of these cells thereby resisting rupturing and release of inflammatory lysosomes and the enzymatic activity as a detoxicant of the superoxide radicle.

The response of individual cases, as reflected in the tables is an indication of the improvement which followed and includes those cases which would have recovered if left to themselves — *vix medicatrix naturae*. The favourable and excellent responses obtained were 90% for bone, 76% for soft tissues and 66% for joints and synovial structures.

Although influenced to a great degree by subjective judgment and therefore exposed to greater error the recorded judgment of the various clinicians as to the benefit obtained by the use of Palosein over and above the expected improvement is illuminating.

In bone this enhanced improvement occurred in 70% of cases, in soft tissue in 59% and in joints and synovial structures in 55%.

These two sets of figures which result from using somewhat different criteria show an interesting correlation which tends to increase the validity of both sets of observations.

CONCLUSIONS

Palosein has been shown in this clinical trial to be a good addition to the therapeutic armamentatium of the clinician for treating traumatic injuries, particularly the acute. The statistical findings are impressive. So far no important adverse side-effects have been found. The full range of clinical indication and application will only be clarified when experience has been gained over a long time, covering a very much larger number of cases.

Palosein does not conflict with any of the usual supportive manoeuvres and its use is aesthetically and from the humane standpoint eminently acceptable.

The warning against a too short convalescent period must be repeated on general principles.

A follow-up of cases a year or two after treatment will provide important information as to whether conditions treated with Palosein have a low recurrence rate comparable to those treated according to conventional methods.

ACKNOWLEDGEMENT

The assistance of Mr G. Arabin with the statistical analysis of our results is recorded with appreciation.

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OUT OF THE PAST — UIT DIE VERLEDE

RICKETS AND OTHER DEFICIENCY DISEASES OF THE OSSEOUS SYSTEM

(*Last Lecture given by Sir Arnold Theiler to B V Sc V Students, Onderstepoort, 1936)

The causes of these diseases may be divided into 3 main groups, namely

1. Malnutrition — protein deficiency
2. Mineral deficiency or abnormalities
3. Vitamin deficiency esp. Vit. D.

They will thus be considered accordingly.

Malnutrition

In this case it is mainly a protein deficiency or even an amino acid deficiency e.g. the protein of maize is deficient in certain amino acids. In plain words one may say that if the osteoblasts are not "fed" properly they cannot produce bone. Instances of this may be seen in Dr Quin's physiological experiments in connection with the Alimentary System where improper or insufficient absorption takes place due to fistulae, anastomoses and other derangements.

Again, in Mönnig's *Oesophagostomum* experiments the same conditions may be seen with heavy worm infestations — in this case probably due to toxin formation, but not impossibly due to digestive and absorptive derangement. Probably in practice the same will be seen in cattle in certain areas of South Africa in winter or during periods of drought.

Mineral Abnormalities

1. **Phosphorus deficiency**

(a) *Rickets*

Definition:- A superabundance of osteoid tissue is formed which transgresses the normal limits due to a phosphorus deficiency or perhaps an excess of Ca and normal amount of phosphorus (experiments still in progress), in *young animals* before the epiphyseal line is closed. Whereas normally the width of the epiphyseal line is 0,1 cm, in rickets it becomes wider, perhaps 1 cm, not due to increased formation of bone but due to decreased absorption.

Produced in pigs experimentally: Clinically the animal's growth is greatly retarded, the joints are abnormal in shape and size and the animal has pain due to the fact that the newly formed osteoid matrix does not ossify and consequently the nerves are not protected against pressure etc.

(b) *Osteomalacia* this is the so-called rickets in adult animals especially cattle. The cause, pathology, etc. is exactly the same as in young animals except that it occurs after the epiphyseal line has closed. It is the so-called "stywesiekte" (stiffsickness) of cattle in South Africa — "True Stywesiekte" (Crotalariosis of cattle is not true stywesiekte — a laminitis) is a phosphorus deficiency in old animals. The condition may be cured by giving the animals phosphorus in the form of bone meal — that is in *cattle* and *sheep.*

In *pigs* — feed a normal ration with correct Ca-P ratio (1:1) — cure. Vitamin deficiency almost impossible to produce in South Africa, due to its abundance of sunlight, except under extremely artificial conditions, where all the food is boiled etc.

In *horses* only one case of rickets is on record in a foal. It cannot be produced in the horse, or perhaps only with extreme difficulty.

NB A Ca deficiency does not produce rickets.

2. **Calcium Deficiency**

Osteoporosis is produced where there is an insufficiency of calcium with a sufficiency of phosphorus.

Definition:- It is a resorptive atrophy of the osseous tissue i.e. the bone deposited is reabsorbed leaving a porous, easily fractured bone, with an absence of osteoid tissue.

In pigs in this condition one sees a paralysis of the back.

3. **Abnormal Calcium-Phosphorus Ratio**

If an excess of P over Ca is fed a condition of *Osteodystrophia fibrosa* develops.

Definition:- Excessive breakdown of bone replaced by fibrous bone tissue.

This condition can be produced in pigs and horses. At one time it was thought to be an infectious disease in horses. Often seen in "old days" in horses in cities and towns (Johannesburg) due to the fact that the animals were fed exclusively on a bran ration which is rich in P but poor in Ca. When fed on hay as well, the condition did not appear.

It affects the whole skeletal system, but is clinically only seen in those parts where continuous and great mechanical stress (or trauma) is applied such as in the jaw bones. These swell up to a great extent, giving the face a puffed appearance, due to excessive formation of fibrous bone, to combat the continual strain placed upon it. The animals become progressively weaker until they are unable to stand any longer and have to be placed in slings until death supervenes. The condition responds readily to feeding with a normal Ca:P ratio and complete recovery takes place. Whether the swelling of the jaw bones will disappear

This is probably the last lecture Sir Arnold ever gave, as he died in London soon afterwards.
*Kindly made available by Dr M. de Lange, who graduated in 1936.

completely, perhaps with contraction or absorption, is not known but indications are present to show that it may perhaps reduce to some extent.

4. **Calcium and Phosphorus Deficiency**

Experiments in pigs show that the animal grows well if both minerals are deficient as long as they are given in a normal ratio.

Osteodystrophia Fibrosa in Human beings.

This disease in humans is always associated with a tumour in the parathyroids which causes a derangement in the Ca metabolism etc. This is never seen in animals.

Vitamin deficiency

This is commonly known to be the cause of rickets in humans in Europe. The theory is that Vitamin D is necessary to fix the Ca and P in the bones. However, in S.A. with our abundance of sunlight the animal is able to produce its own Vit. D as well as the plants on which such an animal is fed. This is therefore of little practical importance except perhaps in instances where animals are kept under extremely artificial conditions such as where all foods are boiled etc.

In all above-named conditions the skeletal system is weakened to such an extent that deformities and fractures are frequent. In rickets with fractures a superabundance of osteoid tissue is formed — no callus is formed due to P deficiency and the absence of Vit. D.

BOOK REVIEW BOEKRESENSIE

CIH KEYS TO THE NEMATODE PARASITES OF VERTEBRATES

Edited by R.C. ANDERSON, A.G. CHITS and Sheila WILLMOTT
Commonwealth Agricultural Bureaux, Farnham Royal, 1974.

No. 1: General Introduction, Glossary of Terms and Keys to subclasses, orders, and super families pp. iv & 17, Figs XIV & 68.

No. 2: Keys to genera of the Ascaridoidea pp. iv & 15, Figs 46 Price £2.00 each.

These two publications are the first of a series to revise and bring up to date the well-known standard work on vertebrate nematode taxonomy, viz. Yorke & Maplestone's *Nematode Parasites of Vertebrates* published in 1926. These keys at long last promise to be worthy successors to the venerable Yorke & Maplestone which rendered excellent service for close to 50 years.

This series differs from that of Yorke & Maplestone in that the species of the various genera are not listed. This omission is understandable if one takes into consideration the rate at which the number of species have proliferated in recent years. The illustrations add greatly to the value of these publications as they are clear and of adequate size. Moreover, in some instances, the structures referred to in the keys are indicated with arrows.

The editors have certainly provided "a working tool" for those concerned with the taxonomy of helminths and there is no doubt that the CIH keys will be much used for many years to come.

A V

BOOK REVIEW BOEKRESENSIE

ANTIBIOTICS IN MILK

HARRY MOL

A.A. Balkema, Rotterdam 1975

Pp viii + 206, Figs. 4, Graphs 7, Tabs 46. Publ. Price: unstated

This is the first comprehensive publication to appear on this matter of considerable importance to the public health and the dairy industry. It provides authoritative data on antimicrobial residues in milk resulting from the therapeutic and other uses of these substances. After classifying these "antibiotics", the author systematically deals with the public health hazards of antibiotics in food and the technical and economic problems they cause. Special attention is accorded to the veterinary aspects of residues in milk and the occurrence of such residues. The legal aspects are covered in a separate chapter, and the methods for antibiotic assay are fully recorded, discussed and criticised where necessary, with special attention being given to factors affecting the sensitivity, accuracy and reliability of such tests. Methods of correction of inherent faults are suggested, and extensive attention is given to the quantitative methods and their mathematical interpretation. The list of references does not quite cover world literature but nearly all modern research and views are included.

The book is well printed and clearly set out. It is perhaps warranted to criticise the numbering of graphs, tables and figures, as the method leads to some confusion at first glance.

The book is highly recommended to those who are directly and indirectly concerned with the problem of residues in milk, and this most certainly includes practising as well as public health veterinarians.

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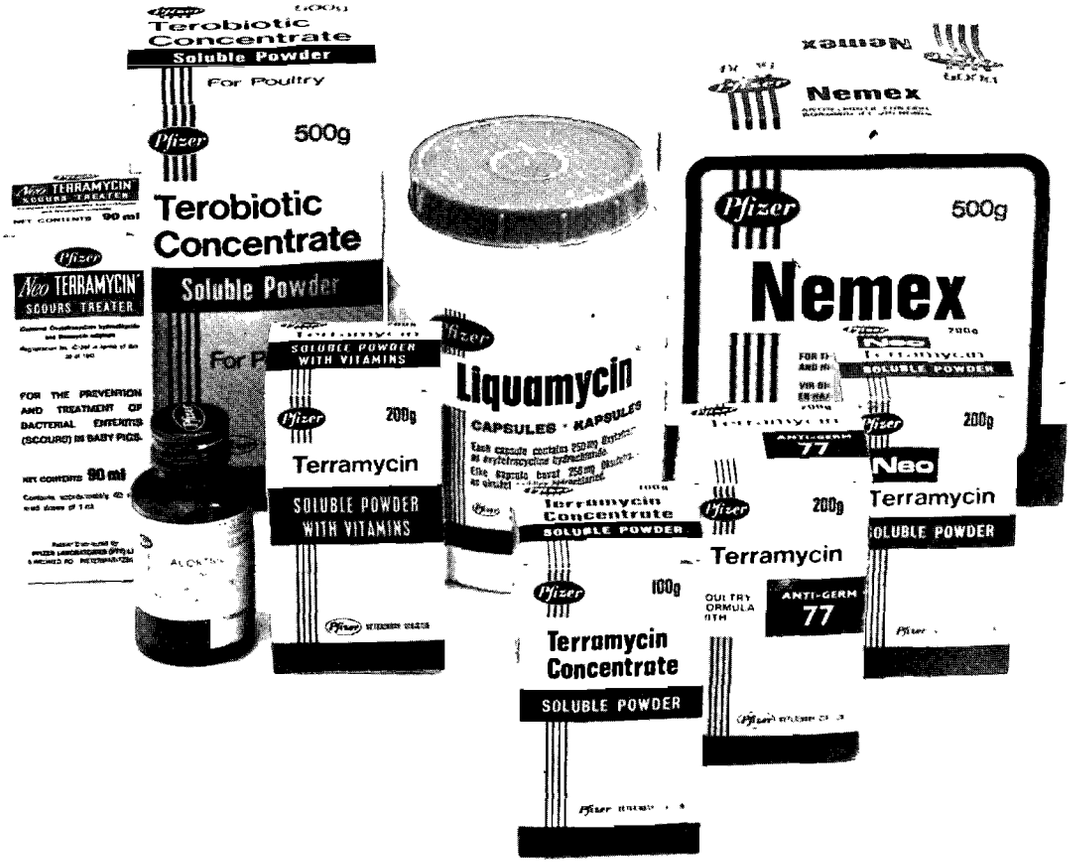

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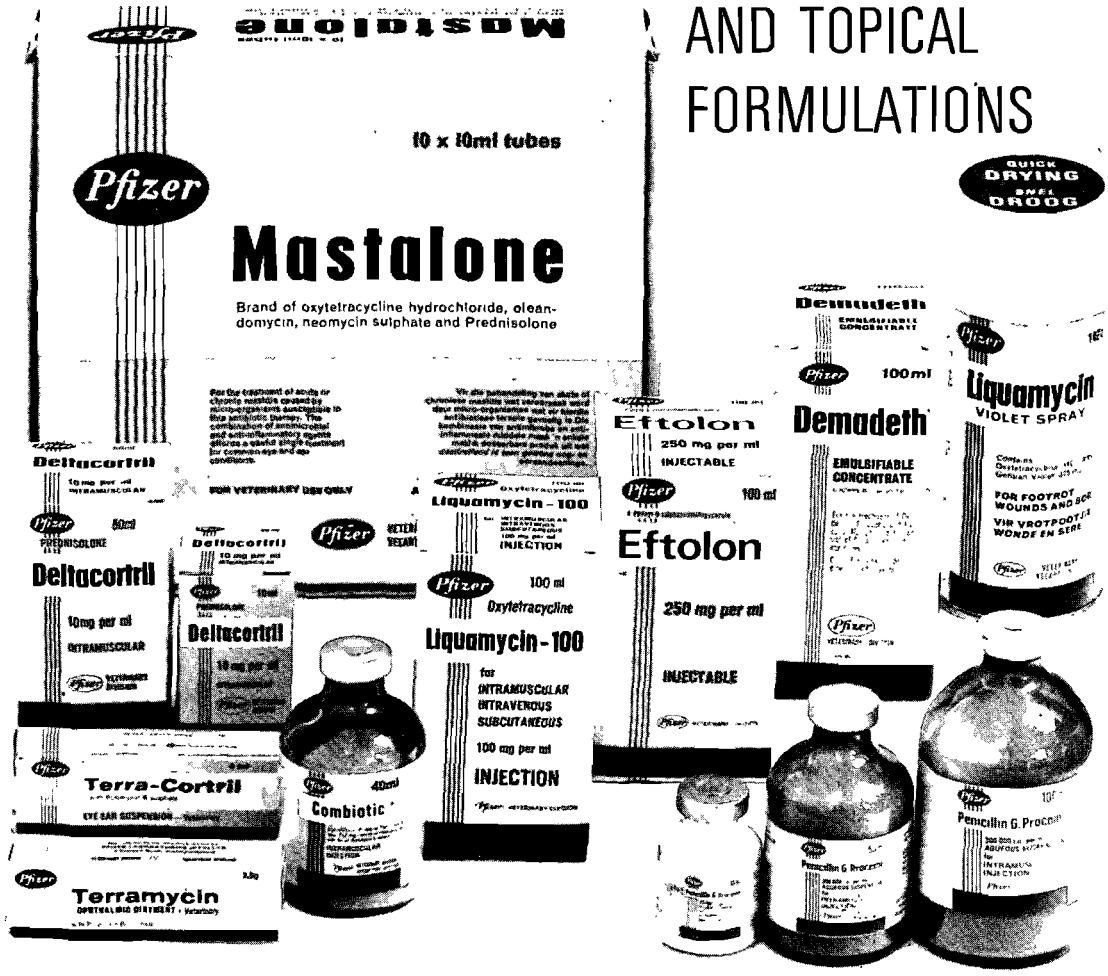

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CASE REPORT **GEVALVERSLAG**

GASTRIC MUCORMYCOSIS AND MONILIASIS IN AN UNIMMUNOSUPPRESSED PIG FOLLOWING RENAL TRANSPLANTATION

S.E. ABBOTT, J. VAN DEN ENDE, R. HICKMAN AND J. TERBLANCHE*

ABSTRACT

Abbott, S.E.; van den Ende, J.; Hickman, R.; Terblanche, J. **Gastric mucormycosis and moniliasis in an unimmunosuppressed pig following renal transplantation.** *Journal of the South African Veterinary Association* (1976) **47** No. 1, 47 - 48 (En) University of Cape Town Medical School, Observatory, 7935 South Africa.

Gastric ulcers frequently complicate experimental surgery in the pig. This report concerns a super-infection of such an ulcer with fungi of the Mucor and Candida groups.

INTRODUCTION

The rare fungal disease, gastric mucormycosis has been reported only occasionally to occur with concurrent moniliasis in the pig. In 1959, Gitter and Austwick[1] described mucormycosis and moniliasis (candidiasis) in a litter of suckling pigs. Seven cases of gastric mucormycosis were reported by Tscherniak[2] in 8- and 6-month-old pigs. The lesions were large ulcers of the cardiac and pyloric mucosa.

The Mucoraceae, members of the class Phycomycetes (true fungi), include three genera, namely *Mucor, Rhizopus* and *Absidia* which are soil saprophytes and common laboratory contaminants. They have for many years been known to be potentially pathogenic to animal and man and may complicate diabetes mellitus, prolonged steroid and antibiotic therapy, malnutrition and renal disease[3].

Case Report

The pig, a 2-month-old Landrace boar, was one of a series on which a large study was performed of unimmunosuppressed renal auto- and allografts.[4] As part of the routine transplant procedure, a gastroduodenostomy was performed to reduce the incidence of gastric ulceration (Spilg, unpublished observations). Postoperatively, the animals were given intravenous 10% Invert sugar in Ringer's lactate for 2 days and thereafter fed *ad lib* with commercial 'Creep' Meal (Epol Company, South Africa). Chloromycetin (500 mg) and crystalline penicillin (600 mg) were given daily in divided doses intravenously for 5 days. The pig died on the 16th postoperative day. The serum creatinine level was 8,9 mg/100 ml one day before death.

At autopsy (performed 2 hours after death), a gastric ulcer, about 20 mm in diameter, with raised haemorrhagic margins, was present in the pars oesophagea. Representative formalin-fixed tissue sections were stained with haematoxylin and eosin, Gomori's methenamine silver and the periodic acid Schiff method. Microscopic examination revealed subacute ulceration of the gastric mucosa with marked inflammatory reaction with proliferation of granulation tissue. The inflammatory process extended deep into the submucosal layer. Several non-septate, branching hyphae about 15 µ in diameter were present in the surface exudate and within the submucosal tissue. These fungal elements were also present in the lumina and walls of small submucosal vessels and some of these vessels were occluded by apparent thrombus in which hyphae were present (Fig. 1). In addition, numerous thin filaments (less

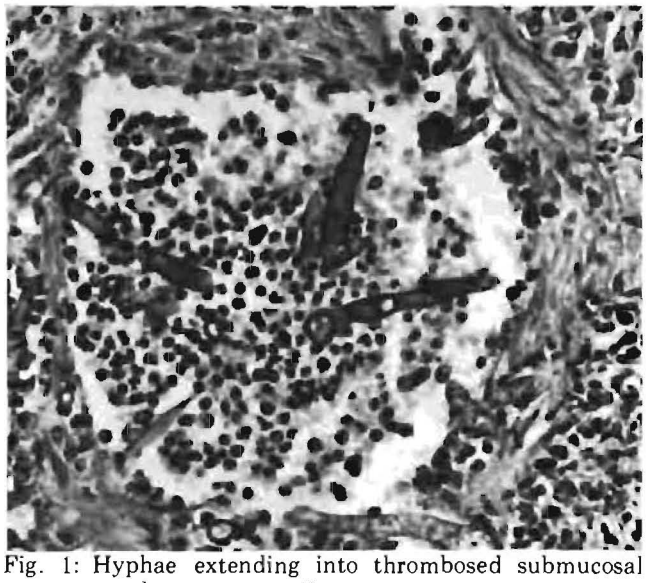

Fig. 1: Hyphae extending into thrombosed submucosal vessel (P.A.S. × 800).

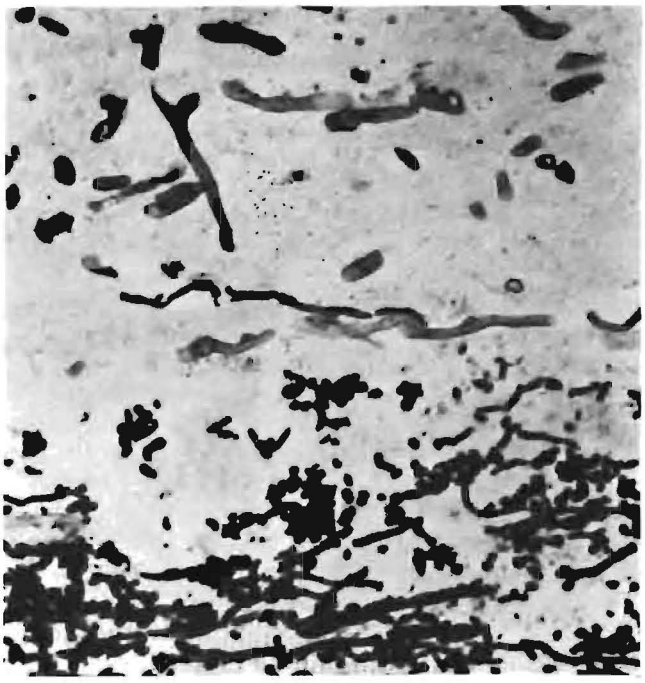

Fig. 2: Numerous darkly staining filaments and yeast cells (bottom). Large non-septate hyphae (above). Methenamine silver × 800.

* Departments of Surgery and Pathology, University of Cape Town Medical School, 7935 Observatory, Cape.

than 5 μ in diameter) and yeast cells were seen within the surface exudate and especially concentrated in one area. These pseudo-mycelial fragments and yeast cells stained more darkly and uniformly than the larger non-septate hyphae, in the silver impregnated sections (Fig. 2).

The kidneys were haemorrhagic and swollen and histologically showed mononuclear cell infiltrates and severe glomerular and tubular destruction. These changes were in keeping with transplant rejection of histological category 2 (significant) as described by Terblanche *et al.*[4]

Sections of the heart showed a thin fibrinous pericarditis with little evidence of organisation. No aetiological agent was evident but the features were consistent with terminal uraemia.

The lungs, liver, thyroid, spleen and thymus were histologically unremarkable and there was no evidence of dissemination of fungal elements in any of the organs examined.

DISCUSSION

Although no specimens from the infected ulcer were submitted for bacteriological examination, the two organisms described were morphologically consistent with a *Mucor* and a *Candida* species. The presence of many coarse branching non-septate hyphae with obvious invasion of blood vessels is characteristic of mucormycosis. Secondly, the features of the pseudomycelia and yeast cells seen on the surface exudate are those of a *Candida* species.

Ulceration of the stomach in swine has particular significance because it is often fatal. The abattoir incidence of porcine gastric ulceration varies. Curtin [5] reported an incidence of 19,46% and O'Brien[6] reported 25,8%. The mortality from this disease has been estimated at 4,59%[5]. A high incidence of gastric ulceration after experimental liver transplantation in the pig has been reported by Dent *et al.*[7] These workers found the incidence to be 80% with a mortality rate of 60% from haemorrhage or perforation.

Porcine gastric ulcers are of two varieties, one associated with fungal infections involving the glandular mucosa and one of dietary origin involving the pars oesophagea.[8] The mucormycotic ulcers occur in pigs under conditions of low resistance, debilitating infections, disturbance in metabolism and artificial feeding. The lesions in the stomach are usually raised, circular, haemorrhagic areas varying in size. Histologically the mucosa is entirely lacking and is replaced by a granulomatous process extending into the submucosa. The blood vessels are often thrombosed and invaded by hyphae. The predilection of this organism for growing in the walls of blood vessels is a remarkable feature.

Candida species frequently complicate gastro-intestinal lesions caused by other agents not only in swine but also in other domestic animals.[9] In the United States, the common practice of using antibiotics in the feed of young pigs as a growth promoting factor and for therapeutic purposes enhances the growth of this organism which appears to be a secondary, not a primary offender in mucosal ulceration of the stomach. Mehnert[10] has shown that any of the *Candida* species can be regarded as pathogenic when these fungi are localised in the alimentary tract of young pigs.

In the case presented above, there was obvious fungal involvement by the two organisms described but it appears that the role of these opportunistic fungi was of a secondary nature. This is to date the only case of gastric ulceration with fungal superinfection which we have encountered in our laboratory pigs, in which we generally observe a high incidence of gastric ulceration.

Postoperative debility in association with uraemia due to allograft rejection, and prophylactic antibiotic administration probably favoured the opportunistic infection by fungi.

ACKNOWLEDGEMENT

We wish to acknowledge the continued advice and support of Professor J.H. Louw, Head of the Department of Surgery, University of Cape Town.

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Jl. S. Afr. vet. Ass. 47(1) 49-52 1976

CASE REPORT **GEVALVERSLAG**

CRYPTOCOCCOSIS IN A DOG

J.A.W. COETZER*, G.D. IMES** AND C. IRVINE-SMITH***

ABSTRACT

Coetzer, J.A.W.; Imes, G.D.; Irvine-Smith, C. **Cryptococcosis in a dog.** *Journal of the South African Veterinary Association* (1976) **47** No. 1, 49-52 (En) Section Pathology, Vet. Res. Inst., Onderstepoort, 0110 Rep. South Africa.

A report is presented of a fatal systemic *Cryptococcus neoformans* infection involving the central nervous system of a dog and complicated by a concurrent infection of the brain and spleen by an unidentified filamentous fungus resembling a *Paecilomyces* spp. The literature on cryptococcosis in domestic animals is briefly reviewed.

INTRODUCTION

Usually saphrophytic[22], *Cryptococcus neoformans* is an ubiquitous organism found in soil, dust and manure[21]; pathogenic strains of the yeast have been recovered from the skin and mucous membrane of healthy persons[3]. Powell *et al*[24] cultured pigeon excreta from old roosting areas and 84% of samples contained the pathogenic yeast.

Cryptococcosis usually develops during the course of some other disease, particularly malignant disease of the reticuloendothelial system in man[22]. Cases have been reported in association with leukemia[11], and systemic lupus erythematosus[6] and as a cause of pleural effusion in man[7].

In animals this organism has been incriminated in nasal granuloma in the horse[25 29], lymphadenitis in cats[5], intraocular cryptococcosis in cats[10], arthritis in dogs[19], a granulomatous chorioretinitis with detachment of the retina in dogs[12 20 21] and as a cause of bovine mastitis[16 27].

Cryptococcus neoformans has a predilection for the central nervous system. Even in cases beginning with cutaneous lesions the CNS is invaded sooner or later[4 13]. Lesions may be in the CNS only, or associated with a systemic infection in the dog[2 19], cat[5 14], horse[8 17] and cheetah[15 30].

Cryptococcosis is rare in animals in South Africa. The case reported here is remarkable because of a concurrent infection of the brain and spleen by an unidentified fungus.

HISTORY AND CLINICAL FINDINGS

A Dachshund male of 18 months was presented with a history of recent convulsions; temperature 39,5°C, noticeably enlarged submandibular and sublingual lymph nodes, disorientation, slight ataxia to the left, and apparently blind with marked mydriasis but a good appetite.

Haematologic examination yielded the following information: packed cell volume 43%, total white cell count 22 650, neutrophils 67% (15 175), monocytes 2% (453), lymphocytes 22% (4 983), eosinophiles 4% (906).

After a tentative diagnosis of meningitis and oral administration of 250 mg chloramphenicol and vitamins three times per day the temperature dropped and the patient showed no further nervous signs; the lymph nodes remained enlarged.

After 5 days the patient was discharged with a diagnosis of lymphosarcoma and a poor prognosis.

Ten days later, pentabarbitone became necessary to control another episode of violent convulsions; a deep coma ensued and the dog died soon afterwards.

NECROPSY FINDINGS

Noticeable was emaciation, marked enlargement of all lymph nodes, and a slightly enlarged spleen with focal white nodules 3-4 mm in diameter, throughout its substance. The lungs looked normal but the mediastinal lymph nodes were enlarged. The meninges were thickened and gelatinous in appearance. Greyish raised plaques of about 0,5 mm in diameter were seen in the conjunctiva and over the third eyelid.

MATERIAL AND METHODS

Paraffin embedded formalin fixed tissue sections of 5 microns were stained with haematoxylin and eosin (H & E), the periodic acid-Schiff reaction (PAS), Mayer's mucicarmine (MM), cresyl echt violet (CEV) and Gomori's methenamine silver nitrate (GMS)[1].

MICROSCOPIC FINDINGS

Microscopic examination revealed a marked granulomatous meningoencephalitis characterized by two types of lesions:

1) Colonies or aggregations of fungal organisms which were spherical to ellipsoidal in shape and measured from 5-15 microns (Fig. 1). Many of these organisms showed budding and all were surrounded by a clear space of 5-10 microns between the organism cell wall and the surrounding tissue (Fig. 2) containing small numbers of epithelioid cells and occasional plasma cells and lymphocytes.

   The fungi stained well with PAS, MM, CEV and GMS. Morphologically and histochemically the organisms were identified as *C. neoformans*.

2) The other more striking lesion consisted of numerous discrete often confluent tubercle-like

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The views expressed by the author** do not necessarily reflect the views of the U.S. Air Force of the Department of Defence.

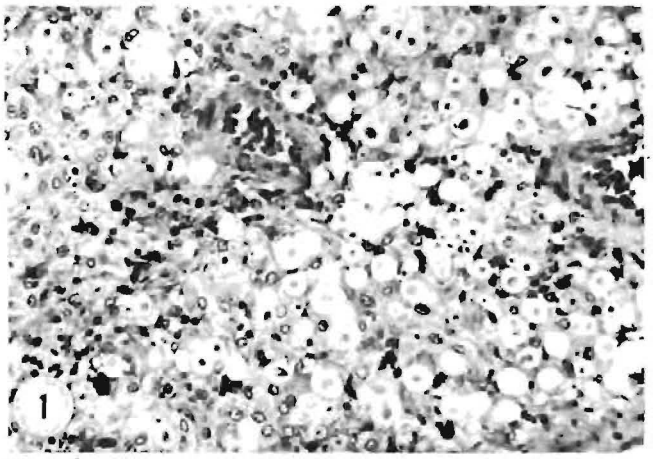

Fig. 1 *C.neoformans*
dog brain
Aggregations of organisms with minimal inflammatory reaction HE × 500.

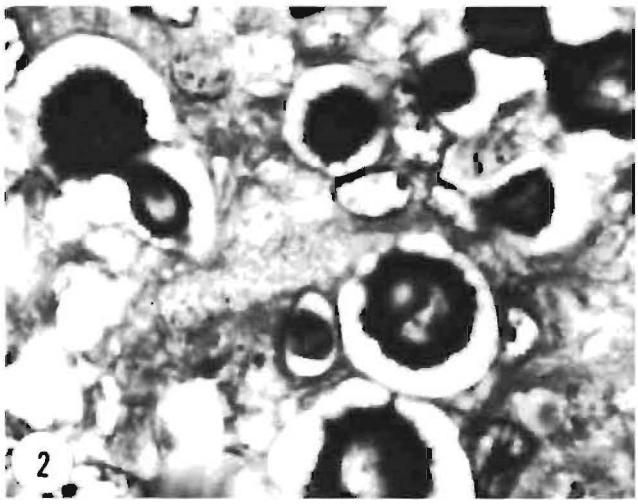

Fig. 2 Budding forms of *C.neoformans* surrounded by a clear spac PAS × 1200.

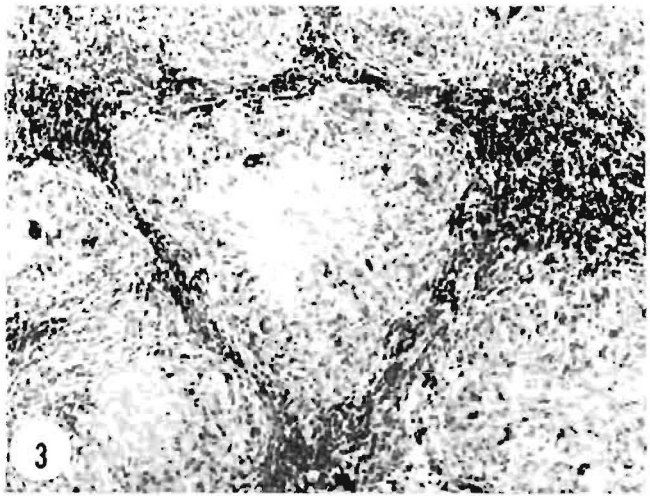

Fig. 3 Tuberculoid granulomas in brain with no organisms visible HE × 200.

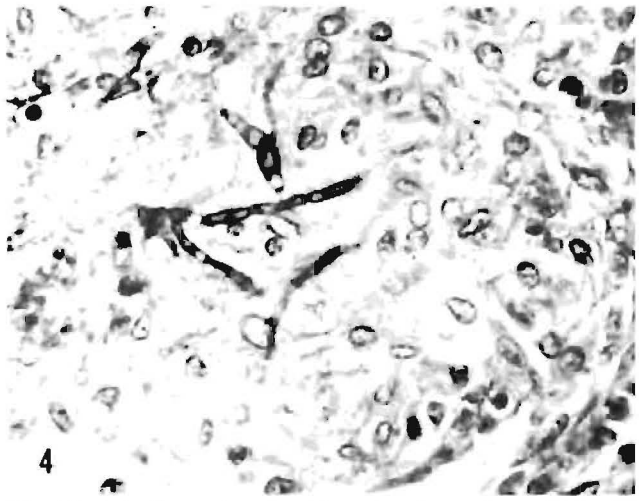

Fig. 4 Septate filamentous fungi in centre of granuloma. PAS × 50

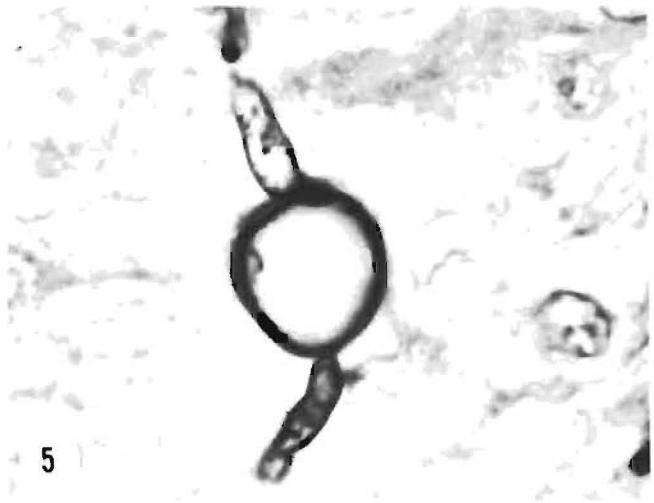

Fig. 5 Ballooned in filamentous fungus PAS × 1200.

Fig. 6 Filamentous fungus with dichotomous branching PAS × 1200.

granulomas, having centres (Fig. 3) of caseation necrosis and containing occasional giant cells of both the foreign-body and Langhans type. Surrounding the necrotic centre was a thick collar of epithelioid cells and occasional giant cells of both types. Some of the smaller granulomas consisted chiefly of epithelioid cells without necrotic centres (Fig. 4). All granulomas were encompassed by a thin band of connective tissue. Plasma cells and lymphocytes surrounded most of the granulomas with some perivascular mononuclear cuffing of nearby vessels. Only the PAS reaction and GMS stain revealed a fungus which formed septate branching hyphae about 4-7 microns in diameter with sides essentially parallel but occasionally greatly ballooned and dilated (Fig. 5). Branching was predominantly at angles greater than 45°. Dichotomous branching was rare (Fig. 6).

The meninges were markedly thickened with numerous organisms in a deeply eosinophilic oedematous fluid. A slight, mononuclear cellular reaction was seen in the meninges and around the colonies

or cysts of cryptococcal organisms scattered throughout the brain.

Two types of lesions were also found in the spleen:

1) Colonies of cryptococci surrounded by a slight inflammatory reaction of a few histiocytes, plasma cells, lymphocytes and Langhans giant cells surrounded by slight fibroplasia. Some giant cells contained organisms.
2) Granulomas scattered throughout the red and white pulp consisting of histiocytes, plasma cells, lymphocytes and Langhans giant cells with a few neutrophils and a slight peripheral fibroplasia. Some granulomas had a necrotic centre containing septate branching fungi identical to those present in the brain.

Similar lesions were seen in the lymph nodes but none showed central necrosis and no branching fungi could be identified. Numerous colonies of cryptococcal organisms were situated throughout the node.

The membrana nictitans showed cysts of cryptococcal organisms with the same surrounding reaction as described above.

IDENTIFICATION OF THE FUNGI

The morphology and staining characteristics of the cryptococci were typical. The filamentous fungus could not be positively identified by light microscopy because no hyphae were observed in the H & E sections, the near absence of dichotomous branching and the presence of numerous ballooned hyphal segments. Of the known pathogenic fungi *Aspergillus* and *Candida* spp and the filamentous form of *C. neoformans* were considered.

A wax block of formalin fixed brain tissue was sent to the Department of Health, Education and Welfare, Public Health Service, Atlanta, Georgia, USA where our diagnosis of *C. neoformans* was confirmed by the fluorescent antibody technique. The filamentous fungus was not stained by the fluorescent screening conjugate for *Aspergillus* spp or *C. neoformans*.

DISCUSSION

Cryptococcosis of the CNS in man usually results from haematogenous dissemination after primary pulmonary disease[10]. But according to Lurie & Shadomy[23] only airborne particles less than 5 microns in diameter can reach the alveoli. *C. neoformans* varies from 5-20 microns in diameter and some authors[5] have suggested that even in a heavily contaminated environment the number of viable organisms reaching the alveoli is quite small, making infection via the respiratory route unlikely. The prosector in this case found nothing grossly wrong with the lungs although the mediastinal lymph nodes were enlarged.

The brain lesions in the area of the acoustic and optic nerves strongly suggest extension of infection from an otitis or opthalmitis. Wagner, Pick & Krigman[28] reported cryptococcal meningoencephalitis in a dog originating from an otitis externa.

Shadomy & Lurie[26] found that the inflammatory response of mice to the Coward strain (hyphal form) of *C. neoformans* was of 3 types: a histiocytic granulomatous reaction was seen in the lung and kidney. A tuberculoid granulomatous reaction with central necrosis and epithelioid cells in the lung and spleen; and micro-abscesses in the lung and liver. They expressed the opinion that the "caseous" lesion in the centre of the granuloma may be due to hypersensitivity that develops after the protecting capsular material has disappeared.

The unidentified fungus in this case is probably not normally a pathogen. Of the fungi not usually thought to be systemic pathogens, *Paecilomyces* spp was considered. Jang *et al*[18], reported this condition in a dog and their histological description and photomicrographs were rather similar to the organism seen in the material examined by us.

Emmons *et al*[10]., state that untreated CNS cryptococcosis in man is invariably fatal. Amphotericin B, a highly nephrotoxic substance, is the only effective drug known. The possibility of making a definitive diagnosis of CNS cryptococcosis in a live animal is remote. Because of the toxicity of amphotericin B, its use for suspected systemic fungal infection in animals would not be warranted.

ACKNOWLEDGEMENT

The authors express their appreciation to the following:

1) Dr William Kaplan of the Department of Health, Education and Welfare, Public Health Service, Mycology Division, Atlanta, Georgia, USA.
2) Mr A.M. du Bruyn and Mr J.L. de B. van der Merwe and Technicians, Veterinary Research Institute, Onderstepoort.

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LETTER TO THE EDITOR

30th January 1976

Sir,

I hope very much that your Journal might be able to carry a note of our 150th Anniversary as we believe that our books have contributed significantly to the training of many of your veterinary students and post graduates, and U.K. trained veterinarians who are currently practising in your country.

Baillière Tindall has a long and interesting history which began 150 years ago with the establishment near Bedford Square, London, of the English branch of a French publishing house which had been founded in Paris in 1818 by a Huguenot from Beauvais named Jean-Baptiste Baillière and which specialised in the publication of books on all aspects of medicine.

The firm prospered, started to publish English medical books, and moved to larger premises in Regent Street where its conduct was placed in the hand of Hippolyte Baillière, a younger brother of the founder. On his death in 1867 his widow carried on the business for a time and was joined in it by Albert Alfred Tindall who was already experienced in the field of medical publishing. In 1870 he was joined by William Cox and the firm adopted the style of Baillière Tindall & Cox under which name it became one of the leading medical publishers in the country.

AAN DIE REDAKSIE

In 1965 the firm entered into association with Cassell & Co. Ltd and took over the medical books formerly published by that company. In 1971 it became the Baillière Tindall division of Cassell and Collier Macmillan Publishers Ltd who had acquired the Cassell group of companies in the previous year. From the very beginning the firm has been active in the publication of books and journals in human medicine and ancillary subjects. It later became one of the pioneers of veterinary publishing, in which field it remains one of the principal firms publishing in the English language.

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Yours sincerely

NEVILLE MENDELSON
Baillière Tindall
7 & 8 Henrietta Street
London WC 2E 8QE
U.K.

TO THE EDITOR — AAN DIE REDAKSIE

A MICRO-ORGANISM IN TICKS

Dear Sir,

I noticed that Dr G. Theiler made a statement concerning my work on ticks in her address on "Past-Workers on Tick and Tick-borne Diseases in Southern Africa", published in your Journal Vol. 46, No. 4, p. 303-310, 1975, which is not quite correct.

Dr Theiler states on page 308, Predators and parasites, Fungi: "O.G.H. Fiedler's work on *Beauveria thuringiensis* remains unpublished".

The results of the study actually were presented as a paper at a "Symposium on the Biology and Control of Ticks in Southern Africa", held from the 1st to 3rd July, 1969, at Rhodes University, Grahamstown, under the title "The Occurrence of an Acaricidal Micro-organism in South African Ticks". The paper was published in the Proceedings by the Rhodes University, p. 170-174.

I would be pleased if you could publish a correction, as the micro-organism may play a role in the biological control of ticks in future.

Yours sincerely,
Dr O.G.H. Fiedler.

11 Arcadia Galleries
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Pretoria 0002
South Africa

15 January, 1976.

HUMAN RABIES VACCINES

Sir,

The human rabies vaccines at present in use are crude extracts of either brain or duck embryo extract. The material at best is only partially purified and considering the number of doses which are injected it is surprising that there have not been more reports of neuroparalytic or other complications developing with the use of these vaccines.

As early as 1938 Kligler and Bernkopf cultivated the rabies virus in the allantois of the developing chick embryo. Peck *et al* (1955) grew the virus in duck embryo and the inactivated virus suspension prepared in this manner is still used extensively to-day in the United States and in this country for both pre- and post-exposure vaccination. The duck embryo vaccine has proved to be efficacious with a low incidence of side reactions, but as with the more immunopotent neural vaccines the high percentage of impurities are antibody inducing themselves. Therefore the immune mechanism of the animal is hard pressed to deal not only with the inactivated virus but with a far higher concentration of impurities.

In the last ten years research has increased our knowledge regarding the rabies virus (Hummeler and Koprowski 1969) and with the development of more sophisticated virological techniques the solution to the rabies vaccine problem appears to be in sight.

Both Kissling (1958) and Fenje (1960) adapted rabies virus to growth in baby hamster kidney cells. This initial work led ultimately to the development of highly purified and antigenic tissue culture vaccines which protected laboratory animals more effectively than the vaccines at present used for man (Koprowski, 1967; Wiktor *et al*, 1969).

The rabies virus was also adapted to growth in a human diploid cell strain (HDCS) by Wiktor and Koprowski (1965) and it is this adapted virus known as PM/WI-38 which has been tested extensively as a vaccine strain over the last five years, and which is at present being sold on the world markets for use in man.

The virus is grown in WI-38 cells, concentrated by ultrafiltration (Strohmaier, 1967) and finally inactivated with β - propiolactone. The recommended dosage (Merieux Institute, Lyon) is as follows:

For preventative vaccination two sub-cutaneous injections are given one month apart with booster injections 1, 3 and 5 years later.

For post-bite therapy four injections are given on day 0, 3, 7 and 14 with boosters on days 30 and 90. The usual precautionary procedures are followed regarding wounds.

One of the advantages of this vaccine will be obvious from the schedule in that the number of injections has been radically reduced and also the side-reactions, if any, are very mild. Most important is that high antibody titres are induced very rapidly by this new vaccine. The efficacy of this vaccine as a potent immunogen has been proved in a number of experiments (Sikes *et al*, 1971; Wiktor *et al*, 1973) but its role in post-bite vaccination has still to be substantiated.

In this country rabies vaccines for human use are prepared from suckling rat brain material infected with the Pitman-Moore strain of virus. However it is the intention of the State Vaccine Laboratories to investigate the possibilities of preparing a tissue culture vaccine much along the same lines as that described above.

(Dr) W. Katz
State Vaccine Institute
Alexandra Road
7405 Pinelands

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CASE REPORT **GEVALVERSLAG**

CONGENITAL DIAPHRAGMATIC HIATUS IN A DOG

G.F. BATH*

ABSTRACT

Bath, G.F. **Congenital diaphragmatic hiatus in a dog.** *Journal of the South African Veterinary Association* (1976) **47** No. 1, 55 — 56 (En) Regional Veterinary Lab., Middelburg, 5900 South Africa.

The symptoms and post mortem findings of a case of diaphragmatic hiatus in a dog are described. At necropsy the caudate and right lateral lobes of the liver, the pyloric portion of the stomach, most of the small intestine and large intestines, the spleen, pancreas and omentum were found in the thoracic cavity.

SYMPTOMATOLOGY

The dog, a 3 year old male Schipperke, had been normal until shortly before presentation, and had no history of previous illness or difficulty in breathing. The most prominent symptoms were dyspnoea and hyperpnoea; hypothermia; pale, dirty-coloured mucous membranes; rapid, weak pulse and very poor habitus. Within minutes of admission, the dog died.

POST MORTEM FINDINGS

The diaphragm contained an oval hiatus dorsolaterally on the right side, about 10 cm across and representing approximately one-fifth of the total diaphragmatic area (Fig. 1). Between the 10th and 12th rib, the edge of the hiatus against the body wall was represented by a band of tissue about 1 mm thick. The edge was rounded and showed no signs of adhesions or inflammation. The *hiatus oesophagicus*, *hiatus aorticus* and *foramen venae cavae* were normal and not involved in the pathological hiatus. The *trigonium lumbocostale* was also normal.

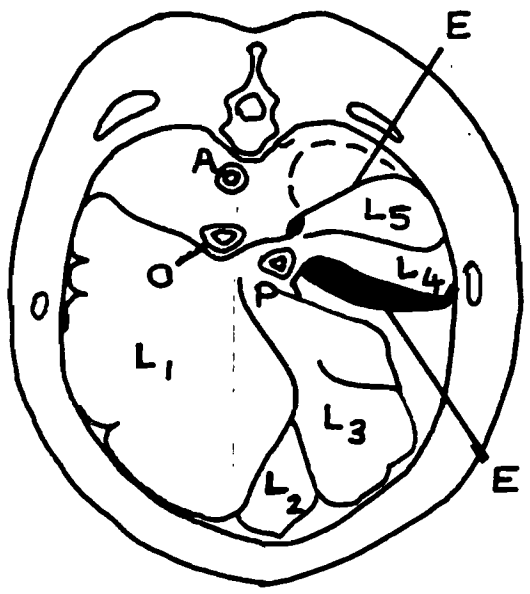

Fig 1: Reconstruction of Diaphragm and Liver, Posterior View

A	=	Aorta
O	=	Oesophagus
P	=	Posterior vena cava
L	=	Liver
L1	=	Left lateral lobe
L2	=	Right medial lobe
L3	=	Papillary lobe
L4	=	Right lateral lobe
L5	=	Caudate lobe
E	=	Edge of diaphragmatic hiatus

In the thorax, the heart and lungs were displaced to the left, the lungs were severely atelectatic and only one third to one quarter of their normal volume. The caudate and right lateral lobes of the liver were situated within the thorax between the right diaphragmatic lobe of the lung and the dorsal part of the diaphragm. They were slightly hypoplastic and their shape corresponded very closely with that of the space occupied. They were connected via the hiatus by a thin cord of tissue to the other lobes of the liver. These two lobes were thus partly covered on their dorsal aspect by the lung (Fig. 2). The rest of the thoracic cavity was occupied by the pyloric portion of the stomach, most of the small intestine, spleen, pancreas, omentum and large intestine (Fig. 2).

All that the abdominal cavity contained was the cardiac part of the greatly distended stomach, the kidneys, the other five lobes of the liver, the descending colon and part of the duodenum and small intestine. The stomach lay with the greater curvature dorsolaterally on the right side, and was greatly distended by gas. Solid and liquid contents were scanty. An acute flexure of the cardia and the proximal part of the duodenum (which lay within the thorax) was caused by this malpositioning, with a further acute flexure at the *flexura secunda* which was anchored within the abdomen. Most of the abdominal lobes of the liver projected beyond the costal arch, and appeared enlarged.

No transudate, exudate or adhesions were found in the thoracic or abdominal cavities. No signs of inflammation or hyperaemia or strangulation of any organs were evident.

DISCUSSION

Whatever the original cause of the hiatus, it is apparent that the dog had adapted very well to its presence, and was able to breathe without difficulty. The two hepatic lobes which lay within the thorax appear to have acted as a partial or complete barrier to herniation when supported by the fully expanded lungs (see reconstruction in Fig. 1).

Lack of support by the right lateral and caudate lobes of the liver as well as hypertrophy and displace-

* Regional Veterinary Laboratory, 5900 Middelburg.

ment of the other lobes resulted in the stomach being more mobile to the right than normally, and made it possible for the stomach to rotate from the normal position of the greater curvature lying ventral and lateral on the left to one in which the greater curvature lay dorsal and lateral on the right (Fig. 2). This would entail kinking of both cardia and pylorus. Once this had occurred, any gaseous distension of the stomach would increase the kinking, and tend to prevent deflation from either end of the stomach. This organ in dogs is capable of tremendous enlargement and could occupy virtually the entire abdominal cavity. Such enlargement would bring about a significant rise in abdominal pressure as compared to that in the thorax. Under these conditions, a barrier across the diaphragmatic hiatus formed by intrathoracic liver lobes would be pushed aside and abdominal contents would enter the thorax, causing collapse of the lungs. After this had begun, the condition would have been self perpetuating, terminating with a grossly distended and malpositioned stomach, herniation of abdominal contents into the thoracic cavity, and atelectasis of the lungs. Death was apparently due to shock and anoxia.

To allow for the development of organs and adaption of respiration as outlined above, the hiatus and intrathoracic malpositioning of the liver was either congenital or acquired very soon after birth. On the balance of evidence it seems more likely that the hiatus was congenital, and due to failure of closure of the pleuro-peritoneal membrane.

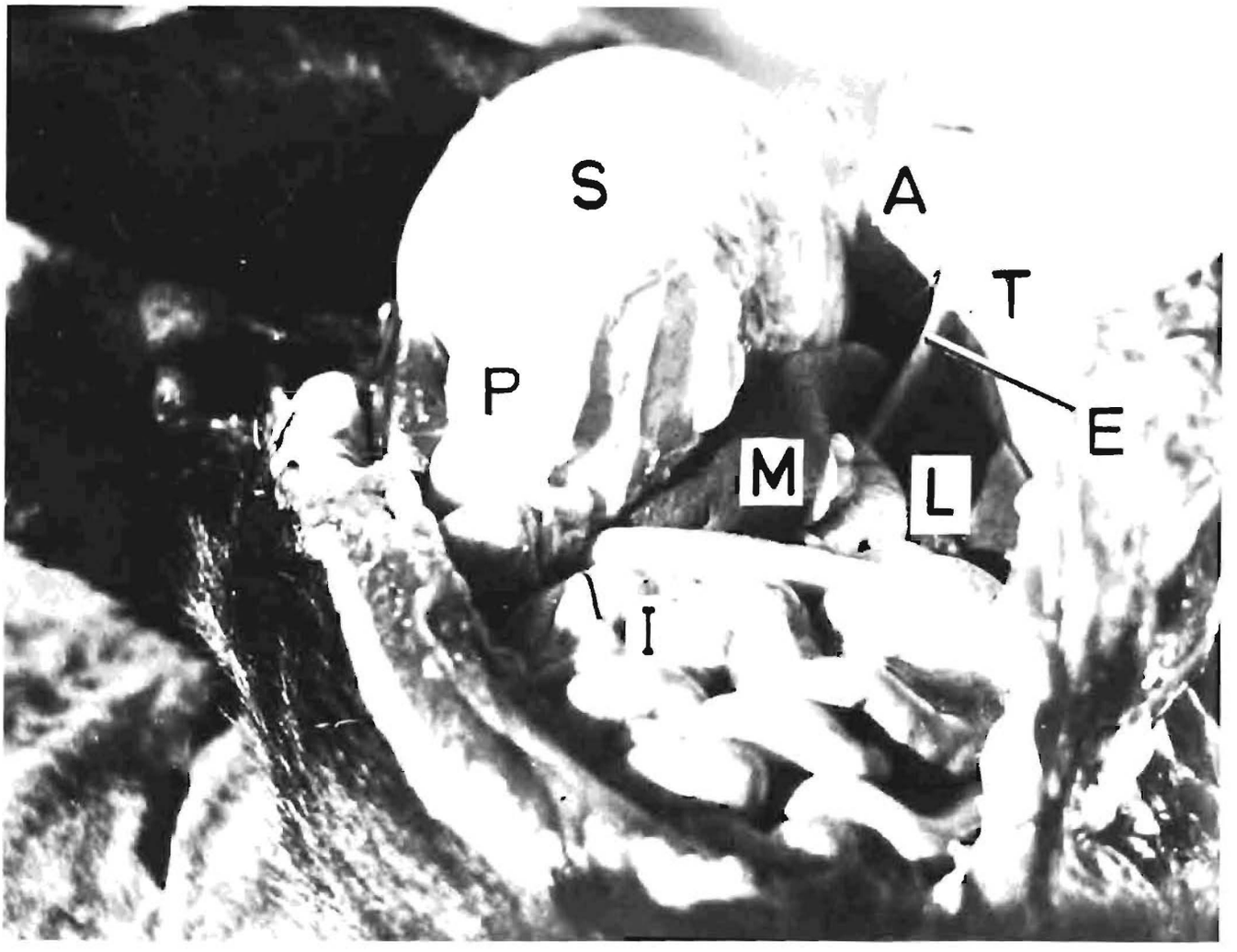

Fig. 2: Antero-Lateral view of Opened Thorax and Abdomen

A	=	Abdominal cavity	M	=	Spleen
T	=	Thoracic cavity	I	=	Small Intestines
E	=	Edge of diaphragmatic hiatus	P		Pylorus
L	=	Liver (intrathoracic portion)	S	=	Stomach (greater curvature)

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CASE REPORT **GEVALVERSLAG**

CORDOPHILOSIS AND FATAL GASTRO-INTESTINAL VERMINOSIS IN ELAND

E. YOUNG* AND P.A. BASSON**

ABSTRACT

Young, E.; Basson, P.A. **Cordophilosis and fatal gastro-intestinal verminosis in eland.** *Journal of the South African Veterinary Association* (1976) **47** No. 1, 57 (En) Kruger National Park, Skukuza, 1350 South Africa.

Fatal cases of cordophilosis and gastro-intestinal verminosis in eland are reported. Some of these cases were complicated with heartwater or cytauxzoonosis. Cordophilosis resulted in acute cardiac arrest. Infestations with gastro-intestinal nematodes were responsible for extreme emaciation. Clinically affected eland responded dramatically to systemic treatment with levamisole hydrochloride.

Cordophilosis is a common parasitic condition of kudu, *Tragelaphus strepsiceros*, and bushbuck, *Tragelaphus scriptus*, in the Kruger National Park[1,2]. It is suspected that animals of these species may, under certain circumstances, succumb to infestation but this supposition has not yet been substantiated.

Recently it was found that eland also become infested with *Cordophilus sagittus*, and may die as a result of it. Nearly half of 33 eland, translocated from the Addo Elephant National Park to the Kruger National Park, died suddenly with clinical signs of acute cardiac arrest. Post mortem examinations invariably confirmed the clinical diagnoses.

Prominent heart lesions caused by this filarid parasite, were consistently found in association with other signs of heart failure. The most striking lesions were sub-epicardial aneurysms, up to 3 cm in diameter, which contained curled up *C. sagittus*. The aneurysms gave the affected heart an irregular and somewhat distorted appearance (Fig. 1). This is the first record of *C. sagittus* and fatal cordophilosis in eland. Some eland were simultaneously suffering from cordophilosis and heartwater or cytauxzoonosis and in these animals the other pathogens could also have contributed to mortality.

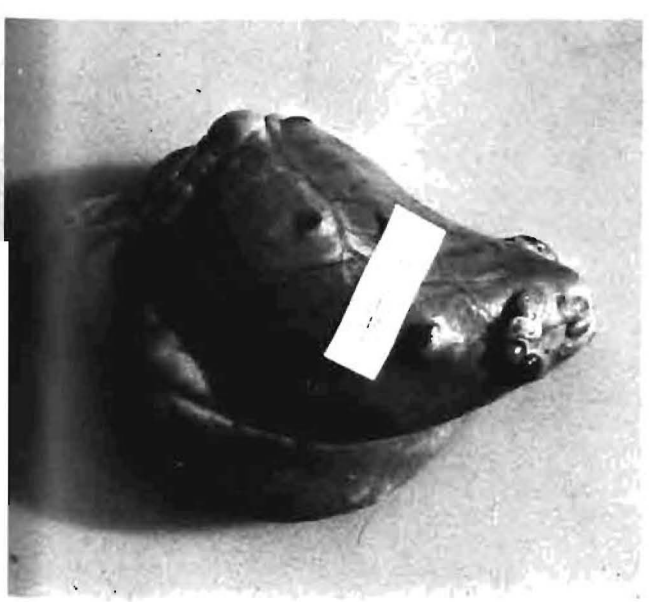

Fig. 1: Lesions of cordophilosis in the heart of an eland.

Other eland became extremely emaciated and died. Post mortem examinations revealed very heavy infestations of gastro-intestinal nematodes, especially *Haemonchus* spp. Wet and marshy conditions in their camp apparently contributed to the severe parasitic infestations.

All emaciated and clinically affected animals were subsequently captured with nets or immobilized with oripavine hydrochloride (M99 or Etorphine, Reckitt) and flupyridol (Azaperone, Janssen Pharm.) and treated intramuscularly with 6.6 mg/kg levamisole hydrochloride (Ripercol-L, Janssen Pharm.). Clinical improvement was dramatic and fecal egg counts, averaging 1 200 eggs per gram prior to treatment, became negative. The effect of treatment on the gastro-intestinal parasites was clearly successful but its effect on *Cordophilus* could not be assessed.

As in the case of domestic animals, translocation of wild animals may be followed by outbreaks of disease and parasitism. Pathogens may be latent in animals and only cause clinical signs under the influence of stress. Furthermore, susceptible animals, such as these eland, may be exposed to parasites to which they have no resistance. Parasites may also be introduced into regions where they did not previously occur. Parasite and disease control should therefore form an integral part of all wildlife translocation projects.

ACKNOWLEDGEMENTS

We have pleasure in thanking Dr A. Verster for the identification of parasites, Messrs Thys Mostert, Paul Venter and Mike Landman, as well as other members of the National Parks Board and the Division of Veterinary Services, Skukuza, for their help with the capture and treatment of sick animals and Mr Piet Burger for laboratory assistance.

Dr A. Verster, Dr U. de V. Pienaar and Dr D.V. Gradwell are thanked for reading the original manuscript and the Director of Veterinary Services for permission to publish.

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* Division of Veterinary Services, Kruger National Park, 1350 Skukuza.

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Jl. S. Afr. vet. Ass. 47(1) 59-62 1976

CASE REPORT **GEVALVERSLAG**

MYASTHENIA GRAVIS IN A FOX TERRIER LITTER

W.L. JENKINS*, ENETTE VAN DYK** AND C.B. MCDONALD†

ABSTRACT: Jenkins, W.L.; van Dyk, Enette; McDonald, C.B. **Myaesthenia gravis in a Fox Terrier litter** *Journal of the South African Veterinary Association* (1976) **47** No. 1,59-62 (En) Dept. Phys., Pharm. & Tox., Fac. Vet. Science, Univ. Pretoria, Box 12580, Onderstepoort, 0110 Rep. of South Africa.

A report of a myasthenia gravis-like syndrome, which occurred in two Fox Terrier litter mates, is presented. The clinical signs which appeared within 5 weeks of birth, and the subsequent course of the disease are described. Treatment with neostigmine and atropine initially provided control. Some recognised features of the condition in dogs are briefly reviewed.

INTRODUCTION

Myasthenia gravis has been recognised as a clinical entity in man for about 300 years. It is only in the last 40 years, however, that any substantial progress has been made in the understanding and treatment of the condition[16].

The major clinical features of the syndrome include weakness, greater susceptibility to fatigue and rapid exhaustion of skeletal muscles during activity[21]. Complete or partial recovery may follow a sufficient period of rest. Muscle groups may be affected symmetrically or assymmetrically and the clinical signs may remain localized or become generalized[12]. Atrophy of the muscles occurs rather infrequently and there is no obvious disturbance of sensory or reflex function. Myasthenia gravis is a chronic disease and exacerbations and remissions often occur unpredictably. Another characteristic is the high incidence of thymoma in affected individuals[20]. The diagnosis is generally based on the swift and dramatic improvement of the muscle weakness following the administration of neostigmine or other suitable anticholinesterase agents[5 9].

The site of the physiological defect in myasthenia gravis is the neuromuscular junction, but there is still no agreement regarding the cause or actual nature of the interference with neuromuscular transmission[8]. Several possibilities have been explored and there is some evidence of a presynaptic disturbance leading either to a diminished acetylcholine synthesis or a reduced postsynaptic sensitivity to acetylcholine [6 24]. Postsynaptic aberrations have also been implicated and ultramicroscopic changes in the motor endplate have been described[2]. These alterations include poor differentiation of the end-plate, axonal degeneration and thickening of the basement membrane. Although an increase in acetylcholinesterase activity would seem to be another possible cause of myasthenia gravis, this could not be demonstrated by histochemical methods[4]. It has also been suggested that an endogenous circulating neuromuscular blocking agent may be present [1 25], but the presence of such a substance has yet to be conclusively proved. Finally, it must be noted that there is considerable evidence at the present time to suggest that myasthenia gravis may well be a form of auto-immune disease in which an antibody is formed against the motor end-plate receptor proteins[3 22].

The incidence of myasthenia gravis varies between 1 in 20 000 to 1 in 50 000 in most European communities[23], and until fairly recently the disease was regarded as being unique to man. Rare cases of what would seem to be a similar, or very closely related, clinical syndrome have, however, been described in the dog[10 13 14 15 17 18 19 26] and the cat[7].

It is the purpose of this case report to record another instance of the myasthenic syndrome in the dog. The disease has not previously been confirmed in litter mates although there is strong circumstantial evidence that it has indeed occurred[10 19].

CASE HISTORY

Two brown and white, 5 week old, pedigreed, smooth-haired Fox Terrier male puppies from a litter of three (the third puppy apparently suffocated soon after birth) were presented to one of the authors for examination. The owner's complaint was that the puppies appeared to be normal on awakening every morning, but tired very quickly thereafter. They then assumed sitting positions and after variable periods of rest, were able to regain their feet and stagger about once more.

These observations were confirmed and it was noted that the clinical signs commenced within 5-10 minutes of the puppies being encouraged to move about. Besides weakness in the hindquarters, they showed a markedly stilted gait in their forelimbs. The appetite of both animals was good although some difficulty was experienced in prehension when they were fed from floor level.

About a year previously a similar case in a Fox Terrier pup from the same kennels had been presented for examination. This animal died suddenly at 4 months of age without showing any signs of improvement after symptomatic therapy. Further enquiries revealed that both the affected litters had a common sire. A related pup in another litter developed similar clinical signs at a very young age and died within a few months. This latter puppy's pedigree could not be traced but there was strong evidence that the sire of the cases described earlier was also closely related to this animal.

Further detailed clinical and radiological examination revealed no other abnormalities and the puppies were treated on general principles for several days.

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They were then referred to the Department of Medicine, Faculty of Veterinary Science, University of Pretoria.

CLINICAL EXAMINATION

The habitus of the puppies was very good. They were lively and playful and took great interest in their surroundings. Even when in a paretic state they reacted positively to their environment.

In the early morning they moved quite freely and their postural positions were normal, but within an hour of awakening or following a short period of exercise they became incapable of adopting natural postures and locomotion was abnormal. Neither animal was ever heard to bark and they were never seen to scratch themselves.

The appetite of both puppies was excellent, but they did experience difficulty when feeding from floor level and often tended to lose balance as they ate. Raising the dishes greatly facilitated prehension. Emesis was not observed at any time.

The rectal temperatures at the time of examination were 38,0°C and 37,5°C and varied little throughout the course of the disease.

The various muscle groups of both animals were not particularly well developed but no gross abnormality of the musculoskeletal system was detected. The locomotor derangement was very characteristic. When allowed to run or move about, the puppies did so quite easily and with perfectly rhythmic movements. After a variable period of time, but usually within 10 minutes, their strides became shorter and shorter especially in the forelimbs. The hindquarters then commenced to sway from side to side with increasing intensity. The puppies would then sit down in an almost upright position with their forelimbs held beside their bodies and often barely touching the ground. If they were encouraged or forced to move about, the forelimbs folded up and they dragged themselves along on the anterior carpal surface. Eventually they would collapse into sternal or lateral recumbency and were then unable to move although they would make periodic tentative efforts to drag themselves along the ground. After a variable period of rest, but never less than 10-15 minutes, the puppies would recover partially and the whole process could be repeated. Locomotory ability was directly related to the period of rest.

Fine muscular fasciculations were observed in the masseter and temporal muscles, but only after eating. Moreover, the mandibles of both animals tended to hang about a centimetre open almost continuously.

Full clinical examinations carried out after prolonged periods of rest failed to reveal any additional significant aberrations in any other organ system.

The only abnormality revealed by laboratory investigations was a reversal of the urinary creatine to creatinine ratio. These were 1,3 and 1,75 respectively whereas a normal value of 0,7 is accepted by our laboratory. There was no significant deviation in the plasma or erythrocyte cholinesterase activity in either animal.

DIAGNOSIS

The anamnesis and the result of the clinical examinations suggested a diagnosis of myasthenia gravis. To confirm this premise a reversible cholinesterase inhibitor was administered parenterally and the response observed[5, 17].

A dose of 0,175 mg (0,05 mg/kg) neostigmine methylsulphate ("Prostigmin", Roche) was injected subcutaneously and 0,3 mg (0,08 mg/kg) atropine sulphate was given intramuscularly at the same time to minimise the muscarinic effects of the neostigmine. This procedure was carried out after the puppies had been exercised and had become paretic.

The response to the administration of the cholinesterase inhibitor was dramatic. Within five to ten minutes the puppies were able to rise and were soon fully mobile. The effect lasted for about 4-5 hours during which time they were able to run freely without any impediment whatsoever. A gradual deterioration then occurred and their condition returned to the pre-treatment state.

It was felt that the dramatic response to neostigmine substantiated the diagnosis of myasthenia gravis.

TREATMENT AND COURSE

The puppies were treated with neostigmine and atropine daily for about 6 weeks. Towards the end of this period, however, the neostigmine was given orally rather than subcutaneously. The animals responded satisfactorily initially and thrived under the circumstances. Supportive therapy included vitamin and mineral supplementation and de-worming when required.

Following this period of apparently successful control of the condition, the puppies' response to treatment became very erratic and both animals suddenly died within two days of one another.

The gross necropsy findings and histological examination of the muscles and other tissues were negative. Unfortunately ultramicroscopic investigations were not carried out.

DISCUSSION

Sixteen cases of myasthenia in the dog have previously been reported in the literature. The clinical and pathological results encountered in 10 of these animals have recently been reviewed by Palmer and Barker[19]. These authors have also compared the features of the disease observed in the dog with those described in man.

A synopsis of the suspected cases of myasthenia recorded in dogs, together with the most notable clinical findings, is presented in Table 1. The two puppies seen by us are included in the series.

There appears to be no breed predisposition although the majority of cases have been encountered in larger breeds.

With only 18 reported instances of the condition, it is difficult to suggest any sex differences. However, there have been 13 cases in male dogs and only five in females recorded to date.

The syndrome has occurred most frequently in young adult dogs from 9 months to 2 years old (10 cases), but has also been described in dogs from 4-11 years old (5 cases). The present paper describes three cases in young puppies of a condition that may be similar to congenital myasthenia in man.

The clinical signs may vary considerably, as they do in man, but the onset of muscular weakness following exercise, followed by virtually complete recovery after a period of rest, is highly suggestive of the myasthenic syndrome. The dramatic response to the administration of an anticholinesterase agent, such as neostigmine, pyridostigmine, edrophonium, or even

an organic phosphorus compound, confirms the diagnosis[5 9 17].

In a number of the described cases, oesophageal dilatation has been a noteworthy pathological finding and is probably responsible for the ptyalism, dysphagia and regurgitation which have been observed in certain instances. Inhalation pneumonia would also seem to be a potential complication. It is interesting to note, however, that dilatation of the oesophagus was not seen in our cases. Thymomata have been described in only two of the 18 cases (Table 1). This is a lower incidence than that seen in man[20]. Fine structural abnormalities of the motor end-plates involving both pre- and postsynaptic elements of the junctional regions also have been described[2 26]. The usefulness of such diagnostic procedures as electromyographic studies and ultrastructural examination following muscle biopsy have been discussed by various authors[10 11 26]. There is generally a reduction in the magnitude of induced action potentials in muscle following repeated nerve stimulation. This effect may be limited by the administration of an inhibitor of cholinesterase.

The initial response to parenteral neostigmine therapy in the Fox Terrier puppies was very satisfactory indeed. The reaction to oral administration of the drug was much less reliable and ultimately the response seen in the animals became very erratic. Immediately prior to their deaths, therapeutic management of the condition was extremely tenuous. At no stage did any signs of remission or recovery become evident.

On reviewing the reactions to treatment of the cases reported in the literature, it would seem that these are rather unpredictable. Some dogs have recovered rapidly (within a week) and completely, others have responded less quickly (within 2 months), and a few have shown periodic remissions. Finally there have been cases, as with the puppies, which have initially responded satisfactorily, but which after a period of time have been difficult to control.

There do appear to be general similarities between the myasthenic syndrome in the dog and myasthenia gravis in man. Until further clarification is obtained, however, with respect to aetiology, physiopathology, immunology and ultrastructural changes, no definite comparisons can be ventured.

Table 1: **SUMMARY OF MYASTHENIA CASES DESCRIBED IN THE DOG**

Case	Reference	Breed	Sex	Age	Course	Pathological Findings
1	18	Cocker Spaniel	M	14 months	Remission following neostigmine administration	—
2	14	Chow	F	2 years	Remission following neostigmine administration	—
3	26	Mixed	M	8 months	Remission following pyridostigmine administration	—
4	15	Pointer	M	4 years	Recurrent attacks controlled with fenchlorphos	—
5	10 19	Alsatian	F	6 years	Apparent recovery following neostigmine administration	Died 19 months later Thymoma
6	10 19	Golden Retriever	M	10 months	Recovery following neostigmine administration	—
7	10 19	Labrador	M	8½ months	Positive response to neostigmine but euthanasia due to secondary complications	Oesophageal dilatation and inhalation pneumonia
8	10 19	Alsatian	M	16 months	Remission following neostigmine therapy	Oesophageal dilatation
9	17	English Coonhound	M	6 years	Remission following neostigmine therapy	Oesophageal dilatation and inhalation pneumonia
10	13	Boxer x Labrador	M	6 years	Positive response to edrophonium, Euthanasia	Thymoma and oesophageal dilatation
11	19	Dalmatian	F	2 years	Positive response to neostigmine	Oesophageal dilatation
12	19	Retriever	M	11 years	Positive response to neostigmine	Oesophageal dilatation (?)
13	19	Collie	M	2 years	Recovery following neostigmine administration	Oesophageal dilatation
14	19	Alsatian	M	2 years	Recovery following neostigmine administration	Oesophageal dilatation
15	19	Jack Russel Terrier	M	2 months	Recovery following neostigmine administration	—
16	19	Pointer	F	18 months	Positive response to neostigmine	Oesophageal dilatation and severe pneumonia
17	*	Fox Terrier	M	5 weeks	Positive response to neostigmine	None
18	*	Fox Terrier	M	5 weeks	Positive response to neostigmine	None

*This report.

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Photo/Foto: Martin Gibb

NUWE GRADUANDI

ONS NUWE KOLLEGAS — GRADUANDI van die FAKULTEIT VEEARTSENYKUNDE, UNIVERSITEIT VAN PRETORIA

Op die oorkantse bladsy verskyn 'n klasfoto van die Finalejaar-klas van 1975. Tydens 'n gradeplegtigheid op 15 November 1975 is die graad B.V.Sc. aan een-en-dertig nuwe veeartse toegeken; een hiervan is met lof deur mej Jeanne de Wet verwerf. Na voldoening aan al die vereistes vir die graad kan die ander kandidate in die eerskomende maande ook verwag om te gradueer.

By 'n afskeidsfunksie wat die lede van die Fakulteitspersoneel vir die klas aangebied het is die volgende toekennings deur die genoemde instansies gemaak:

1. S.A. Biologiese Vereniging : Die Sir Arnold Theiler-medalje aan Mej Jeanne de Wet.
2. Die Tak Witwatersrand, S.A.V.V.: Kliniese Medalje aan Mej J. de Wet
3. Agricura Laboratoria Beperk : Prys vir Patologie aan Mej J. de Wet
4. Maybaker (SA) (Edms) Beperk : Kliniese Prys aan Alfred Benjamin Kidd
5. Pfizer Laboratorium (Edms) Beperk : Pfizer-prys aan Alan Buchanan King
6. I.C.I. South Africa (Pharmaceuticals) Beperk :
   1) Prys vir Geneeskunde en Infeksiesiektes aan Mej Elizabeth Judith Dalton
   2) Prys vir Chirurgie en Geslagskunde aan Jacob Jacobus Pretorius
7. Elanco Landbou- & Veeartsenyprodukte : Lilly Laboratories-prys vir
   1) Pluimveesiektes aan Gert Louis Coetzee;
   2) Varksiektes aan Anna Pienaar de Vos
8. Leeubrug-voere (Edms) Beperk : Pryse vir Kliniese Kundigheid aan
   1) Christo Petrus v.d. Merwe en
   2) Andrew Peter van Zyl

Namens die S.A.V.V. sê ons aan hulle:
"VEELS GELUK en BAIE WELKOM IN DIE PROFESSIE"

NEW GRADUATES

OUR NEW COLLEAGUES — GRADUATES of the FACULTY OF VETERINARY SCIENCE, UNIVERSITY OF PRETORIA

A photograph of the Final Year Class of 1975 appears on the opposite page. At a graduation ceremony on the 15th of November 1975 the degree of B.V.Sc. was awarded to 31 new veterinarians; Miss Jeanne de Wet received her degree with honours. After meeting all the requirements for the degree the other candidates can expect to graduate early in 1976.

At a farewell function arranged by Faculty Staff for the class, the following awards were made by the donors mentioned :

1. S.A. Biological Society : The Sir Arnold Theiler Medal to Miss J. de Wet
2. The Witwatersrand Branch, S.A.V.A. : Clinical Medal to Miss J. de Wet
3. Agricura Laboratories (Ltd) : Prize for Pathology to Miss Jeanne de Wet
4. Maybaker (SA) (Pty) Limited : Clinical Prize to Alfred Benjamin Kidd
5. Pfizer Laboratories (Pty) Limited : Pfizer prize to Alan Buchanan King
6. I.C.I. South Africa (Pharmaceuticals) Limited :
   1) Prize for Medicine and Infectious Diseases to Miss Elizabeth Judith Dalton;
   2) Prize for Surgery and Genesiology to Jacob Jacobus Pretorius
7. Elanco Agricultural & Veterinary Products : Lilly Laboratories prize
   1) Poultry Diseases to Gert Louis Coetzee
   2) Porcine Diseases to Anna Pienaar de Vos
8. Lion Bridge Feeds (Pty) Limited : Prizes for Clinical Sciences to
   1) Christo Petrus v.d. Merwe and
   2) Andrew Peter van Zyl

On behalf of the S.A.V.A. we say :
"CONGRATULATIONS AND WELCOME TO THE PROFESSION"

Photo opposite

1st Row *(sitting) (l to r)*/1ste Ry *(sittende) (l na r)*: J.A. van Rooyen, J.D. Brand, W.J. Wiese, Mev. J.P.C. Sandenbergh, J.J. Pretorius, Mej. E.J. Dalton, G.L. Coetzee, Mej. A.P. de Vos, A.I.L. Fraser, A. du Plessis, A.B. Kidd, S.P. Greyling.

2nd Row *(l to r)*/2de Ry *(l na r)*: G.P. Moelich, E.E. Martin, T.C. van Wyk, A.P.J. Fourie, H.A. van Rooyen, D.R. Waterman, W.J. Krüger, A.D. Fisher, P.J. Schiff, M.B. Walsh, C.P. van der Merwe, A. Beytell, M.J. Caldicott, A.B. King.

3rd Row *(l to r)*/3de Ry *(l na r)*: J.V. Marais, M.M. Rubensohn, R.W.J. van Rensburg, Z.A. Rossouw, P.C. Burger, A.P. van Zyl, P.M. du Preez, B.A.J. van Wyk, M.G. Collett, S.R. Seaman, D.J. Christie, C.C. Aitken, J. Jooste. Inset: Mej. J. de Wet

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In Memoriam

OTTO HELMUTH JEROME MEHNERT
3/5/1914-15/2/75

Born in Jamestown, C.P., he successfully undertook the Staatsexamen for Veterinarians in Leipzig in 1938 and obtained the degree of Dr. Med. Vet. from the same university in 1939. He was engaged in private practice and meat inspection for a year until 1940, when he entered the Army. In 1945 he resumed private practice and municipal meat hygiene work in Germany until 1957, when he returned to South Africa to take up a position as State Veterinarian, eventually becoming Senior State Veterinarian in Greytown, Natal in 1963 until 1966, when he returned to Germany.

To his wife Louise and their children we extend our sincere sympathy on the death of our colleague.

HARALD ECKARD GUSTAV HOLTZ
11.4.10 - 23.8.75

Harald Holtz was born in Usona near Okahandja in South West Africa.

During a holiday in Germany the family was prevented from returning to South Africa due to the outbreak of the first World War, and so he spent his childhood in Germany.

In 1920 he returned to S.W.A. with his parents. He went to school in Windhoek and was confirmed in 1924. After matriculating he returned to Germany where he studied at the Veterinary School of the University of Giessen. He obtained the Dr. Med. Vet. in 1937.

After graduating he came to Pretoria and was later also stationed in East Africa. He returned to S.W.A. in 1939 where he carried out his duties as State Veterinarian at various centres for 19 years. He spent some time in Cape Town in consequence of a lung ailment for which he had to receive medical treatment.

In 1958 he became State Veterinarian in Port Shepstone. As a widower he remarried in 1965. He is survived by his wife Etna and two daughters of his first marriage, to whom we extend our sincere sympathy.

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The Association's Constitution provides for non-veterinarians to be awarded honorary membership for service to veterinary science. Members who have rendered special services can be made Honorary Life Vice Presidents. In 1971 the Association recognised the need for another way of giving due credit and recognition to veterinarians "FOR DISTINGUISHED SERVICE TO THE PROFESSION".

At the S.A.V.A.'s Biennial National Veterinary Congress held in East London in September 1971 the then president, Dr L.W. van den Heever awarded the Association's first gold medal on its behalf to

PROF. DR WILLEM OTTO NEITZ

In doing so he gave the following brief summary of Prof. Neitz's achievements:

Prof. Neitz was awarded the BVSc degree by the University of Pretoria in 1929 and the DVSc degree in 1948. He also studied tropical diseases at the Tropeninstitut (Bernhard-Nocht Institut) at Hamburg and the London School of Hygiene and Tropical Medicine.

A brilliant research career followed on his appointment at the Veterinary Research Institute, Onderstepoort in 1930. This was marked by many notable scientific achievements, particularly in the fields of infectious diseases. Prof. Neitz has more than 119 scientific papers to his credit; several are still in press. They deal primarily with his research work on veterinary protozoology, virology, bacteriology and entomology, the latter as applied to the transmission of infectious diseases and tick toxicoses.

Highlights of his studies are his elucidation of the aetiology of sweating sickness and corridor disease, the discovery of the schizonticidal effect of tetracyclines on *Theileria parva* (the cause of East Coast fever), the chemotherapy of heartwater with sulphonamides and the discovery of the multiplicity of strains in the aetiology of bluetongue.

For this work he has achieved not only local but world recognition, as indicated by the following awards:

the Senior Captain Scott Medal by the S.A. Biological Society in 1954;

the "Havengaprys vir Geneeskunde" by the S.A. Akademie vir Wetenskap en Kuns in 1957;

the degree Dr med. vet. *honoris causa* by the Tierärtzliche Hochschule, Hannover, Germany in 1963; and

the South Africa Medal for 1970 by the S.A. Association for the Advancement of Science.

He has served on several international bodies and societies, for instance: the expert panel on ticks and tick-borne diseases of FAO/OIE; as consultant to FAO on African swine fever; the Committee for the compilation of the List of Animal Diseases of the International Veterinary Congress; leader of discussions on ovine and caprine rickettsial diseases at an OIE

TOEKENNING

DIE SUID-AFRIKAANSE VETERINÊRE VERENIGING SE GOUE MEDALJE

Die Konstitusie van die Vereniging maak daarvoor voorsiening dat ere-lidmaatskap aan nie-veeartse vir uitstaande bydraes tot die veeartsenykundige wetenskap toegeken kan word. Lede kan, omrede besondere dienste aan die Vereniging, Lewenslange Vise-presidentskap aangebied word. Hiervan is ten volle gebruik gemaak.

In 1971 het die Vereniging besef dat daar ook 'n ander manier gevind moet word om waardering uit te spreek en erkenning te gee aan veeartse "VIR UITMUNTENDE DIENS AAN DIE PROFESSIE".

Tydens die S.A.V.V. se Tweejaarlikse Nasionale Veterinêre Kongres in September 1971 te Oos-Londen het die toenmalige president Dr L.W. v.d. Heever die Vereniging se eerste goue medalje namens die Vereniging toegeken aan

PROF. DR WILLEM OTTO NEITZ

By die geleentheid is die volgende kort opsomming van Prof. Neitz se prestasies voorgelees:

Die Universiteit van Pretoria het die BVSc-graad in 1929 en die DVSc-graad in 1948 aan prof Neitz toegeken. Nagraadse studie is aan die Tropeninstitut (Bernhard-Nocht Institut) te Hamburg en die London School of Hygiene and Tropical Medicine onderneem.

'n Briljante loopbaan het gevolg op sy aanstelling aan die Navorsingsinstituut vir Veeartsenykunde te Onderstepoort in 1930. Dit was gekenmerk deur verskeie noemenswaardige wetenskaplike prestasies veral op die gebied van infeksiesiektes. Vandag het Prof. Neitz meer as 119 wetenskaplike publikasies op sy kerfstok, en verskeie is nog in die pers. Hulle handel in die eerste plek oor sy navorsing op die gebied van veterinêre protosoölogie, virologie, bakteriologie en entomologie; laasgenoemde het veral toepassing op die oordrag van infeksiesiektes en bosluistoksikose.

Die hoogtepunte van sy studies was die werk wat gelei het tot bepaling van die etiologie van sweetsiekte en korridorsiekte, die ontdekking van die schizontdodende uitwerking van die tetrasiklines op *Theileria parva* (oorsaak van Ooskuskoors), die chemoterapie van hartwater met sulfonamides en die veelvoudighede van stamme in die veroorsaking van bloutong.

Vir hierdie werk het hy beide plaaslike- en wêrelderkenning ontvang, soos aangedui deur die volgende toekennings:

die Senior Captain Scott-medalje van die S.A. Biologiese Vereniging;

die Havengaprys vir Geneeskunde van die S.A. Akademie vir Wetenskap en Kuns in 1957.

die graad Dr Med. Vet. *honoris causa* van die Tierärtzliche Hochschule, Hannover, Duitsland in 1963;

die Suid-Afrika-medalje in 1970 van die S.A. Vereniging vir die Bevordering van die Wetenskap.

Meeting, Paris, 1968; he has been invited to serve as chairman of the subsection Trypanosomiasis at the World Veterinary Congress, Mexico in 1971.

Prof. Neitz has also made a tremendous contribution to veterinary education in S.A. by serving as Professor of Protozoology and Virology and as Professor of Protozoology in the Faculty of Veterinary Science from 1948 to 1967.

He has on several occasions on invitation conducted seminars on tropical diseases at the Free University of West Berlin.

Hy het op verskeie internasionale liggame en genootskappe gedien, bv. die paneel van deskundiges op bosluise en bosluisgedraagde siektes van FAO/OIE; as konsultant vir FAO ten opsigte van Afrikaanse varkpes; die Komitee vir opstelling van die Lys van Dieresiektes van die Internasionale Veeartsenykundige Kongres; besprekingsleier oor rickettsiale siektes van skape en bokke; voorsitter, onderafdeling Tripanosomiase, Wêreld Veterinêre Kongres, Mexico, 1971.

Prof. Neitz se bydrae tot veeartsenykundige opleiding is ook iets groots. Hy was Professor in Protosoölogie en Virologie en later Professor in Protosoïese siektes aan die Fakulteit van Veeartsenykunde vanaf 1948 tot 1967. Op verskeie geleenthede het hy op uitnodiging van die Wes-Berlynse Vry-universiteit seminare oor tropiese siektes aangebied.

Prof. Dr W.O. Neitz (Photo: A de Bruyn)

ANNOUNCEMENT

BEKENDSTELLING

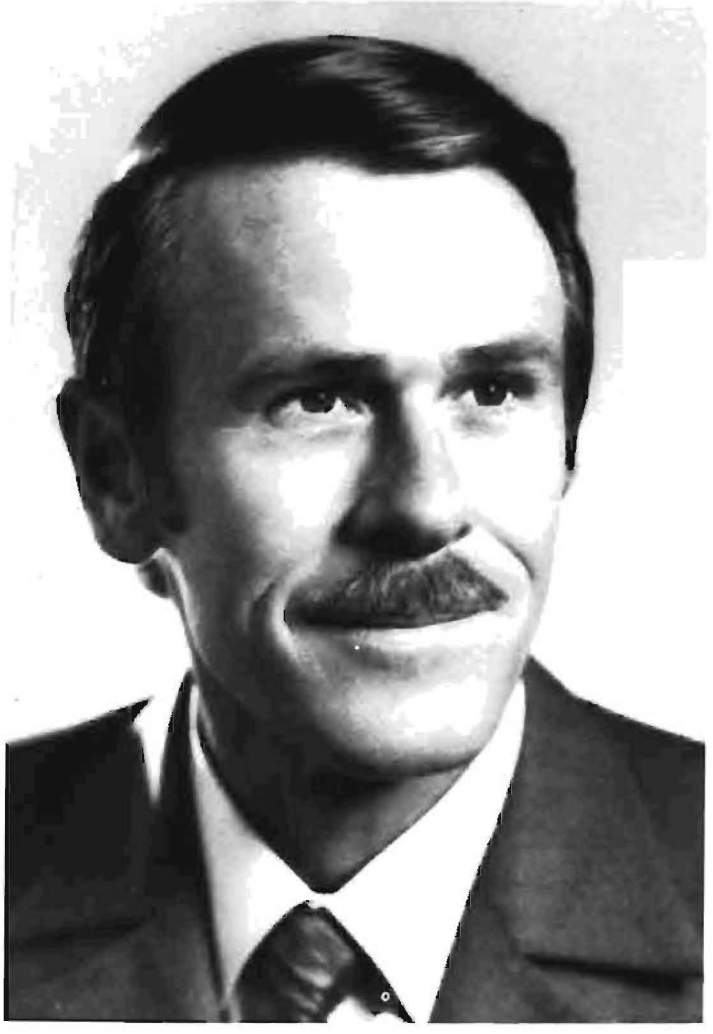
SAVA DIRECTOR/SAVV DIREKTEUR

Mr M.C. (Tienie) Roos has been appointed as our Director as from April 1 1976. He was born in February 1929 in Stellenbosch, and is the son of the late Mr Hennie Roos, a former headmaster in Wellington and a former chairman of the Boland Rugby Union. He matriculated in 1945 at the Hoër Jongenskool in Wellington, later attaining the B.Comm degree from the University of Stellenbosch. Thereafter he joined the KWV in Paarl. In 1963 he moved to Pretoria where he was appointed as head of the Mealie Board's section of Advertising and Information, as well as being the Board's liaison officer. In 1972 he established his own company producing and marketing plastic products, but has recently relinquished his interest in the Company. Mr Roos is married and has five children. His eldest daughter was married to a young colleague, Gerhard Harmse, in January of this year. For relaxation Mr Roos regularly plays tennis and enjoys a game of golf.

Mr Roos will be directly responsible to the executive committee and in particular to the president. His duties will be manifold, but will include the management of our office, the control of our finances and liaison both within and outside our profession. On behalf of the SAVA we heartily welcome Mr Roos in our midst, and wish him every success in the years which lie ahead.

Mnr M.C. (Tienie) Roos is vanaf 1 April 1976 as ons Direkteur aangestel. Hy is in Februarie 1929 op Stellenbosch gebore en is die seun van mnr Hennie Roos, in lewe skoolhoof op Wellington en voorsitter van die Bolandse Rugby-unie. Hy het in 1945 aan die Hoër Jongenskool op Wellington gematrikuleer en toe die B. Comm graad op Stellenbosch verwerf. Daarna het hy in diens getree by die KWV in die Paarl en in 1963 na Pretoria verhuis waar hy aangestel is as hoof van die Mielieraad se Afdeling Reklame en Inligting en ook die Raad se skakelbeampte was. In 1972 het hy 'n eie firma gestig vir die vervaardiging en bemarking van plastiek produkte, maar het pas sy belange van die hand gesit. Mnr Roos is getroud en het vyf kinders — sy oudste dogter is in Januarie met 'n jong kollega, Gerhard Harmse getroud. Om te ontspan speel mnr Roos gereeld tennis en dikwels gholf.

Mnr Roos sal aanspreeklik wees aan die uitvoerende komitee en in besonder aan die president. Sy take sal veelsydig wees onder andere die bestuur van ons kantoor, die kontrole van ons finansies, en skakelwerk binne en buite ons professie. Namens die SAVV wil ons Mnr Roos hartlik in ons midde verwelkom, en hom alle sukses towens in die jare wat voorlê.



Mr M.C. (Tienie) Roos

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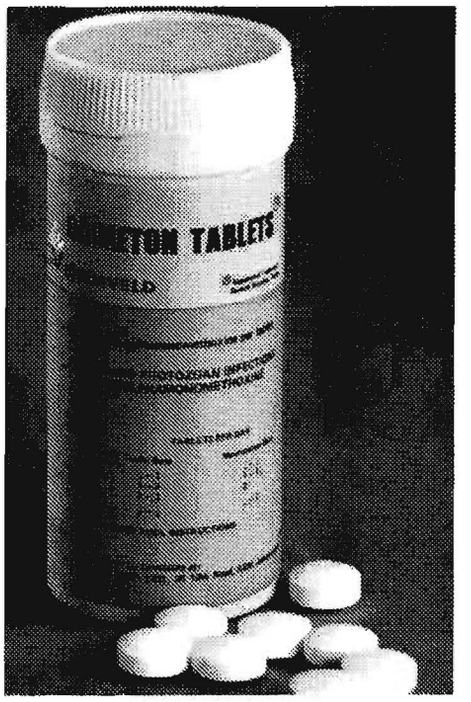

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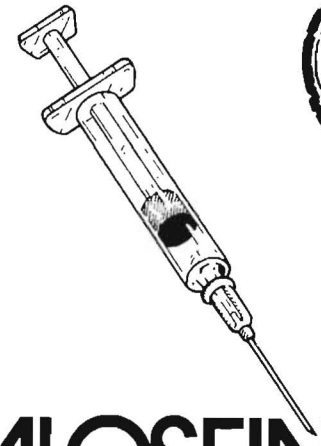

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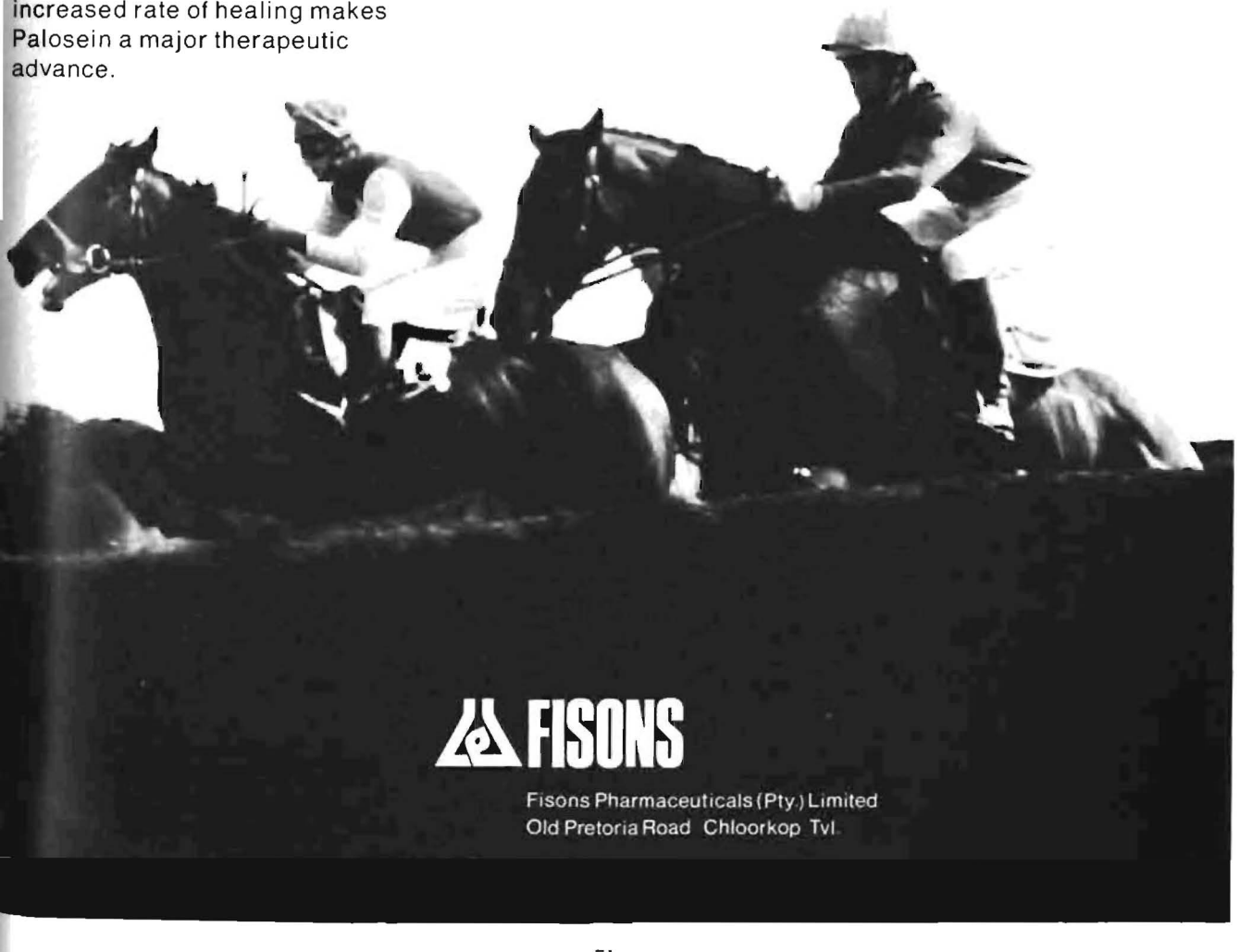

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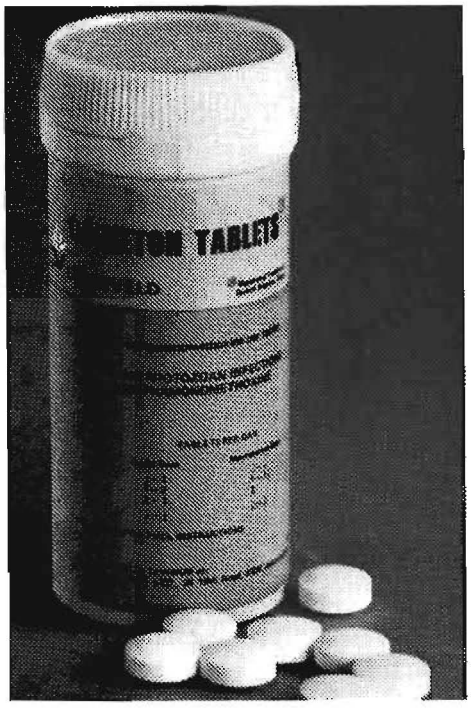

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