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# TYDSKRIF VAN DIE SUID-AFRIKAANSE VETERINÊRE VERENIGING

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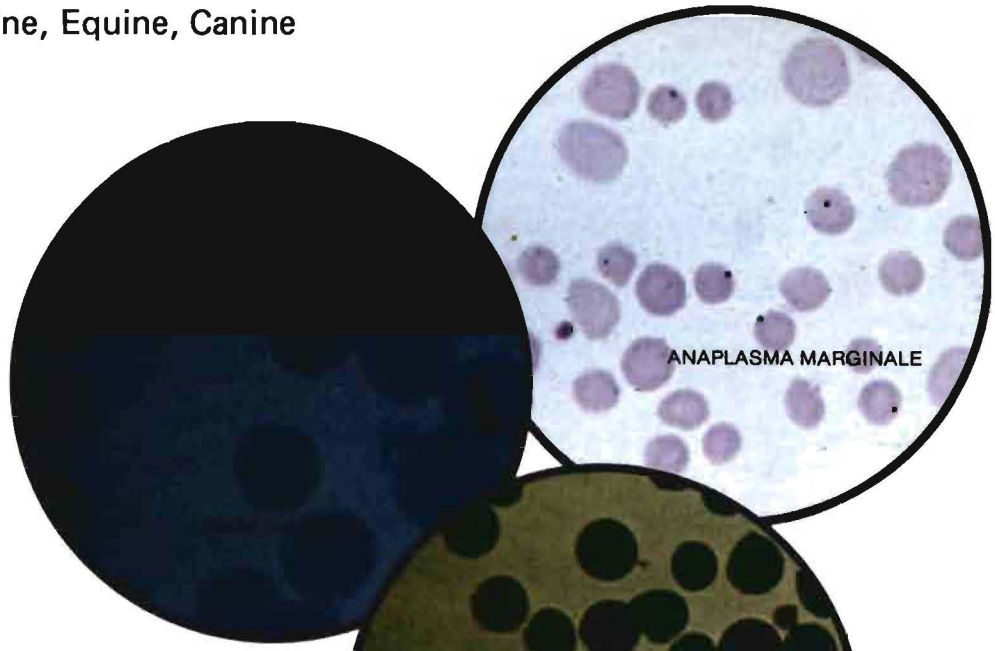


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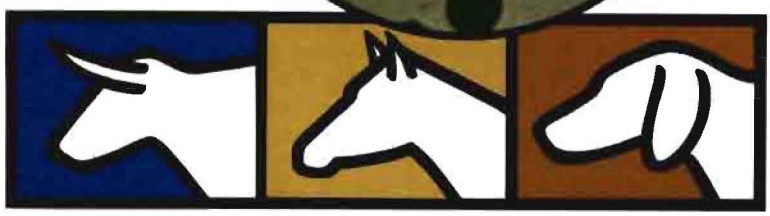

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In the article on EFFECT OF DIETARY LASALOCID ON COCCIDIAL OOCYST NUMBERS, FEEDLOT PERFORMANCE AND WOOL GROWTH OF LAMBS by S. Couvaras, H.P. van Niekerk and Shan E. Thomas in paragraph 5 on page 112 of Volume 51, No. 2 (June 1980 issue) for "60%" improved feed conversion rate please read "6%".

**Index to Advertisers** **Advertensie-Opgaaf**

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**EDITORIAL** **VAN DIE REDAKSIE**

# SWANN VINDICATED – BUT IS CONTROL OVER ANTIMICROBIALS ADEQUATE?

In 1969 Great Britain's Swann Committee reported on its findings relative to the use of Antibiotics in Animal Husbandry and Veterinary Medicine. Its concern over the very real danger of the rapid emergence of multiple drug resistance in *Salmonella* and other organisms and the eventual spread of such resistant bacteria to man lead to the promulgation of a set of regulations to control agricultural use of antimicrobial substances. Antimicrobial feed additives were available without prescription to farmers and the feed industry on the basis that these had no or little therapeutic use. Therapeutic antimicrobials could only be prescribed by a medical or veterinary practitioner for animals under his care.

At about the same time the authorities in South Africa and the S A V A took note of the situation and the potential threat to animal and human health. This resulted in the withdrawal of many antibiotics previously registered as 'stock remedies' under Act 36/1947 and consequently these were no longer available without veterinary prescription. These moves were hotly opposed by some sectors of the pharmaceutical and feed industries and by organised agriculture. At the same time the veterinary profession was given and accepted the task of rational and disciplined use of antimicrobials.

In the U S A the Food and Drug Administration looked into the matter and sponsored various investigations into the likelyhood that widespread use of antimicrobials in the food animal industry would jeopardise animal and human health. Recent reports indicate that these investigations have not been able to prove conclusively that such use of antibiotics is either harmful or not harmful to health. Further investigations are to be conducted.

The development of multiple drug resistance in *Salmonella* and other enterobacteria was never doubted but many disputed that the phenomenon would ever result in actual health problems. The fact that intensive investigation follows the outbreak of foodborne infection in England and Wales made it a foregone conclusion that if multiple drug resistant *Salmonella* of animal origin were to spread to man, this would be recorded in Britain. The British Medical Journal's editorial on "Why has Swann failed?" (17 May 1980), is reproduced below because it contains information and comment which is equally applicable in the Republic of South Africa. It behoves organised agriculture, pharmaceutical and feed firms, livestock owners, the Registrars of Acts 101/1965 and 36/1947 and, above all, members of the veterinary profession to take note of what has happened in Britain and what may well happen in South Africa. In fact, in the absence of intensive investigations, such resistance may well already have occurred and spread from animals to man in the Republic.

There is certainly no room for complacency and both the responsible authorities and the veterinarians in this country need to review the situation and take steps to ensure that they fully meet their responsibilities. The widespread use of tetracyclines in this country and the possible implications relative to increasing resistance to chloramphenicol are perhaps the most important matter to be considered. As far as the veterinary profession is concerned, the kind of adverse publicity which appeared in "The Times" of 28 May 80 (see below) needs to be avoided at all costs.

## WHY HAS SWANN FAILED?*

Current regulations on the use of antibiotics in animals bred for food have failed to prevent the rapid emergence of multiple drug resistance in *Salmonella typhimurium* in bovines in Britain. Presenting convincing evidence for this, Threlfall and his colleagues (p1210) show that two types of multiresistant salmonellas that first appeared in bovines in 1977 were identified in 290 cases of human salmonellosis in Britain up to the end of 1979. During 1979 a new variant appeared and affected calves on at least 50 farms in the south and south-west of England, spreading to Cambridgeshire and Yorkshire through the distribution of infected animals. This latest multiply-resistant strain was induced by the new use of trimethoprim in bovines. Twenty human infections with this strain had been identified by the end of 1979. These developments must be a cause of real concern to doctors: the latest variant is resistant to ampicillin, chloramphenicol, kanamycin, streptomycin, sulphonamides, tetracyclines, trimethoprim, and furazolidone. If such a strain causes septicaemia or other extraintestinal lesions in a patient most antibiotics will be ineffective. In human salmonellosis caused by the first two of the multiresistant types most of the patients suffered only mild-to-moderate enteritis, but some had severe diarrhoea persisting for several weeks; one elderly patient died, seven had invasion of the blood stream, and one 3-year-old infant died of septicaemia. Obviously we cannot dismiss the problems as unimportant, particularly as cases have not been confined to farming families and the multiresistant strains have evidently entered the food chain.

Legislation was enacted, as a result of the Swann Report,[1] precisely to prevent such developments. Antimicrobial substances (mainly antibiotics) were classi-

*British Medical Journal, 17 May 1980, pages 1195–1196. Reproduced with the permission of the Publishing Manager.


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fied into two categories, "feed" and "therapeutic." Those in the feed category had either no therapeutic role or very little, and were available for use in animal feeds without prescription. Therapeutic antimicrobials could be prescribed only by a medical or a veterinary practitioner, and the regulations emphasised that the veterinarian was to prescribe a therapeutic antimicrobial only if he had the animals under his care. Suggestions that chloramphenicol might be prohibited from veterinary use were resisted; they were not recommended by the Swann Report on the grounds that strict limitations on its use were to be set out and that these would be observed by a responsible profession anxious to maintain good ethical standards. In an earlier paper[2] Threlfall and his colleagues suggested that the veterinary profession ought to show more prudence in its prescribing habits; and in their present paper they say that "the responsibility to prevent or control drug resistance in S *typhimurium* in bovines in Britain lies with the veterinary profession." But this oversimplifies the matter: members of the medical profession also must accept reproach for their uncritical prescribing of antibiotics. Richmond and Linton[3] conclude that the medical as opposed to the veterinary use of tetracycline seems to have created the main selective pressure for the high incidence of tetracycline-resistant organisms in the human population.

Apart from incautious prescribing, we must look at other factors in seeking to discover why the Swann recommendations have failed in their main objective – namely, preventing the spread of resistant micro-organisms from farm animals to man. The Swann Commitee laid considerable emphasis on preserving the therapeutic usefulness of chloramphenicol, and the great increase in chloramphenicol resistance in the agricultural environment is particularly worrying. There are several reasons for the failure. Firsly, the use of other drugs – such as tetracycline, trimethoprim, and probably others used in animals – may encourage the spread of resistance to chloramphenicol as part of the multiple resistance carried by some of the organisms the antibiotics are directed against. Secondly, over-enthusiastic representatives of pharmaceutical firms as well as black market operators may find farmers, including poultry producers, all too ready to sidetrack their veterinarians and to bid for any supplies of prescription-only antibiotics that may become available through irregular channels. Prosecutions may close that door if evidence is forthcoming, and farmers need to be educated out of attempting to diagnose and treat or prevent enteritis by using antibiotics without veterinary help. Thirdly, advertisements in farming papers encourage such attitudes among farmers; this trend needs to be reversed, however delicately and indirectly the hint is conveyed. Fourthly, the long-incubated protein-processing order should have been introduced well before now and made law to reduce salmonella-infected feeds given to animals.

We need an administrative and political reaction to the regrettable discovery that – for whatever reason – the limited regulations based on the Swann Report have failed. Exactly why they failed, and when, may not be easy to discover; but clearly the present state of affairs is unsatisfactory and dangerous and more stringent regulations are needed.

## VETS BLAMED FOR SPREAD OF BACTERIA†

By Our Medical Correspondent

Misuse of antibiotics by veterinary surgeons and farmers is blamed in a report published today in the *British Medical Journal* for the spread of new strains of antibiotic-resistant bacteria.

In the past 3 years the Central Public Health Laboratory in London has traced 310 patients with intestinal infections caused by those drug-resistant bacteria, 2 patients (one elderly and one aged 3) died.

The resistance has arisen as farmers have found that cattle given antibiotics gain weight faster and have fewer illnesses. Routine use of such antibiotics, however, leads to the bacteria developing resistance.

Cattle are the main source of a bacterium, *Salmonella typhimurium,* which is also an important cause of human intestinal infections. New strains of that bacterium resistant to 6 of the most commonly used antibiotics were first identified in calves in 1977.

Human cases were reported in the same year; most infections took the form of diarrhoea, but there, were 7 cases of *septicaemia.*

Detailed laboratory studies suggest, the reports says, that the emergence of those strains is attributable to the continued widespread use of antibiotics for the treatment and prevention of infections in farm cattle.

Commenting on the report in a leading article, the *British Medical Journal* criticizes "over-enthusiastic representatives of pharmaceutical firms" and farmers who are prepared to side-track their veterinarians to obtain antibiotics from black market sources.

The continued growth of the resistance of the bacteria found in cattle, the journal says, is a cause of real concern for doctors: if those strains cause *septicaemia* in their patients, most antibiotics will be ineffective.

†The Times (England), 28 May 1980


1. *Joint Committee on the Use of Antibiotics in Animal Husbandry and Veterinary Medicine (Swann Report).* Cmmd 4190. London: HMSO, 1969.
2. Threlfall B.J., Ward L.R., Rowe B. Spread of multiresistant strains of *Salmonella typhimurium* phage types 204 and 193 in Britain. *Br Med J* 1978; ii: 997.
3. Richmond M.H., Linton K.B. The use of tetracycline in the community and its possible relation to the excretion of tetracycline-resistant bacteria. *J Antimicrob Chemother* 1980; 6:33–41.

# *IN VIVO* NUCLEAR TECHNIQUES IN VETERINARY MEDICINE

D.J. GOOSEN*, I. DORMEHL†, M. MAREE† and J.J. VAN DER WATT†

ABSTRACT: Goosen D.J.; Dormehl I.; Maree M.; van der Watt J.J. **In vivo nuclear techniques in veterinary medicine.** *Journal of the South African Veterinary Association* (1980) **51** No 4 209–212 (En) H.A. Grové Animal Research Centre, H.F. Verwoerd Hospital, Private Bag X169, 0001 Pretoria.

A description is given of the more relevant procedures of diagnostic nuclear medicine as well as of future trends in this field, especially with regard to *in vivo* research and clinical applications at the biochemical level. The part played by animal models in the past and present is emphasised. The inclusion of a nuclear medicine facility, as equipped and run by the Atomic Energy Board at the H.A. Grové Animal Research Centre is put into perspective.

## INTRODUCTION

Radioisotopic investigations in veterinary medicine are presently largely limited to *in vitro* radioimmunoassays. *In vivo* tracer techniques are rarely applied in nutritional and metabolic studies and in haemopoietic and gastro-enterological investigations. The use of radioisotopes for the study of normal or pathological structure or function of organs in animals is virtually unknown to veterinarians, mainly due to the high cost and unavailability of equipment. Notwithstanding these deterrents, nuclear techniques may become important diagnostic aids in veterinary schools and research centres in the foreseeable future. An initial investigation was conducted at the H.A. Grové Animal Research Centre in an attempt to illustrate the value of these techniques.

## TECHNIQUES AND APPLICATIONS

*In vivo* nuclear diagnostic procedures involve the oral or intravenous administration of radionuclides to the patient for the purpose of obtaining an organ image or data on organ structure or metabolic function by means of specialised instrumentation and radiopharmaceutical substances selected for their physical, physiological and biochemical specificity.

The radionuclides administered to the patient in diagnostic nuclear medicine are all gamma emitters, i.e., they radiate gamma rays in their decay to stability. These isotopes are incorporated into radiopharmaceutical substances designed to localise in a particular organ or to trace a particular organic function or metabolic process. These *"in vitro"* gamma rays are subsequently detected externally by using extremely sensitive detectors. The range of radiopharmaceutical substances has advanced rapidly since the time of $^{131}I$ to the presently fashionable short-lived positron emitters, which permit the use of labels like C, N, and O which are natural to the body rather than analogues[8 12]. These isotopes have the additional advantage of very short half-lives which make the use of possible larger dosages for greatly improved imaging and without the unnecessary radiation burden to the patient. Furthermore the double gamma rays which they emit permit tomographic sectional visualisation of an organ (slicing). This, however, is only possible with very sophisticated, expensive and still largely experimental equipment. The most extensively used radioisotope in nuclear medicine, using conventional equipment, is 99m Technetium ($^{99m}Tc$) which is in a metastable state and decays with a half life of $T_{½}$=6,06 h to the more stable $^{99}Tc$ by emitting a gamma guantum of energy E = Kev[9]. This is a convenient energy for detection outside the body and also well-suited for practical detection equipment. Another advantage is the short half-life which can furthermore be effectively modified by biological removal to reduce radiation dose to the body.

The detecting device is a gamma camera and consists of a NaI-scintillation crystal of up to 400 mm in diameter and 12 mm thick which is surveyed by a hexagonal array of photomultiplier tubes, usually 37 in number, each with its own electronic circuit consisting of a preamplifier, amplifier and pulse height analyzer whereby the original gamma ray is converted into an electric pulse which is eventually energy selected for display on an oscilloscope screen[1]. A polaroid photograph can be taken of the display but X-ray-type transparencies are also possible. The camera is interfaced to a data processor where the analogue signals are converted to digital information for storage and data manipulations.

**Fig. 1.** Normal baboon liver and spleen – (ventral) 15 min after intravenous injection of 600$\mu$Ci $^{99m}Tc$--Sn-colloid and trapped by the healthy reticuloendothelial cells.

The content of information in any information processing system, such as the gamma camera, can be expressed as the ability to recognise a signal in the presence of "noise". The detectability of a small lesion in a nuclear medicine image (e.g., a small volume with

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reduced radioactivity surrounded by a region filled with radioactivity) depends on the so-called signal-to-noise ratio. This ratio is not great in nuclear medicine imaging systems – usually 2 – 3 in the best of images. In liver and brain scintigrams current systems cannot resolve lesions smaller than 20 mm in diameter. For the liver scan a $^{99m}$Tc-labelled colloid is administered to the patient and trapped by the healthy reticulo-endothelial cells of which the majority are to be found in the liver[6]. There the colloid remains and a good static anatomic image of the liver will be available depending on the physical half-life of the label. Non-functioning cells will present as areas of low concentration of radioactivity or "cold spots" (Fig 1).

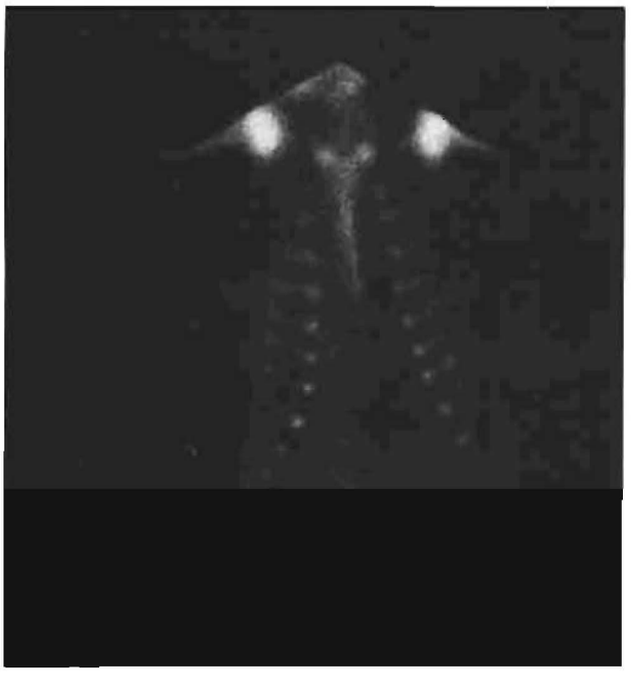

**Fig. 2.** Normal baboon thorax (ventral) 4 h after intravenous-injection of 6mCi $^{99m}$Tc-labelled methylene diphosphonate (Tc-MDP). Note that the areas of active growth (epiphyses of long bones) normally exhibit increased activity.

The rationale of brain scanning is that most intracranial lesions alter the blood-brain barrier so that an administered radiopharmaceutical substance will localise in and/or around the lesion[3]. The procedure is largely non-specific since neoplasms, vascular accidents or arteriovenous malformations are all presented as areas of increased radioactivity. Some differentiation becomes possible by sequential follow-up scintigrams of the patient or by a dynamic study of, e.g., one picture every 3 s to obtain a cerebral flow pattern.

An exceptionally high degree of lesion resolution (as little as 10mm in diameter) is obtained with "hot-spot" bone imaging because of the high level of radioactivity in the lesion[4]. In this case bone seekers such as Tc-labelled phosphates are intravenously administered and chemoabsorption and incorporation into the immature collagen occurs within 2–4 h (Fig. 2 & 3).

A brief review of the limitations of medical radionuclide imaging techniques in competition with X-ray Computerised Axial Tomography (CAT-scanning)[11] and ultrasound suggests that the emphasis in this area should be on the measurement of physiological uptake of tracer materials. This calls for adequate data handling equipment to permit rapid dynamic studies. As digital technology has progressed explosively during the past few years, the approach to data storage and display has become progressively refined. For example, image processing has proceeded from simple smoothing techniques to sophisticated noise filtering and edge detection procedures such as those applied to cardiac ejection fraction studies where it is essential to determine with absolute accuracy the left ventricular boundaries at end-systole and end-diastole[5].

Fast analogue – to – digital conversion allows up to 100 images per second in data acquisition. This is of great advantage in dynamic cardiac studies including cinematic representation of cardiac motion, the evaluation of cardiac transit times and left vetricular ejection fraction from first pass studies where $^{99m}$Tc diethylene triamine pentaacetic acid (DTPA) is followed in its passage through the heart and lungs[13] (Fig. 4).

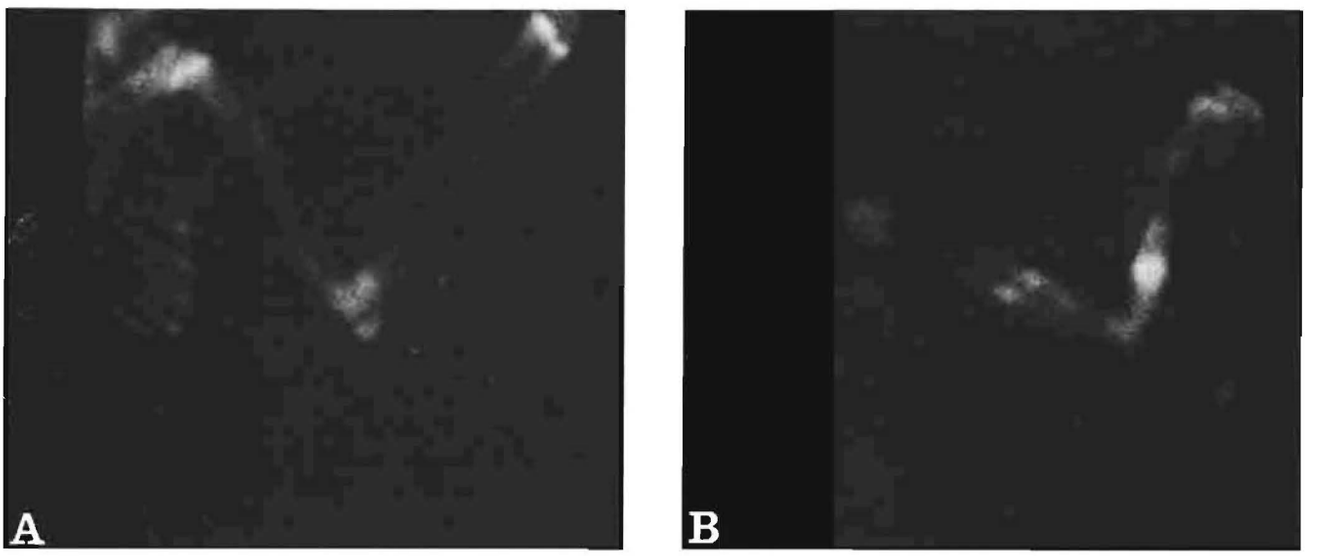


**Fig. 3.** Normal baboon (humerus, radius and ulna) (A) versus fractures humerus, radius and ulna (B) where the lesions are demonstrated as "hotspots" due to the chemoabsorption and incorporation of the Tc-MDP into immature collagen.

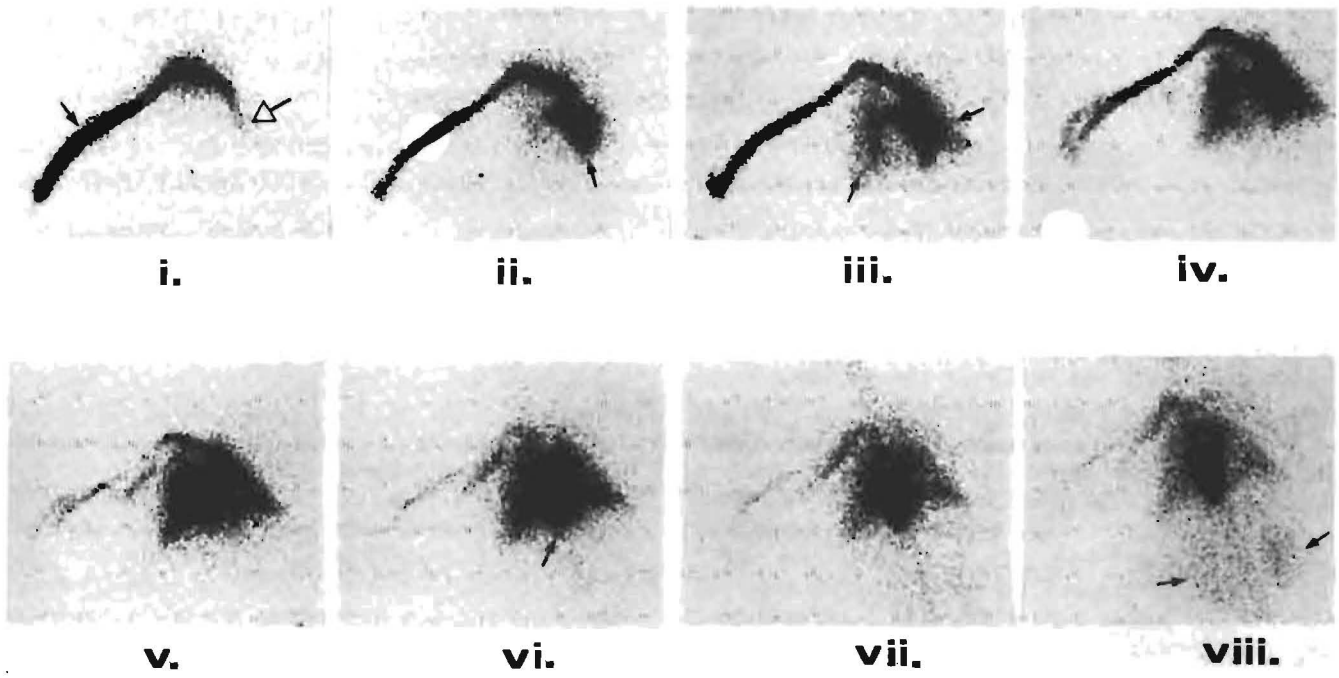


**Fig. 4.** First passage cardiac study (left Ventral anterior Oblique) sequential frames at 1/s after intravenous bolus injection of 6mCi $^{99m}$Tc – DTPA. Note how the isotope is transported via the vena cava superior (i) (→) into the different cardiac chambers i.e. right atrium (i) (→), right ventricle (ii) (→), through the lungs (iii→vi) (→) and into the left cardiac (vi→viii) chambers with final distribution via the aorta to the remainder of the body. Note the early appearance (7–8s post injection) in the kidneys (vii–viii) (→) of the baboon.

Another example of dynamic data acquisition is the evaluation of uptake and washout of a radioisotope by the kidneys[7]. The scanning agent, $^{131}$I-orthodiodohippurate of which 80 % is excreted by tubular secretion, is ideal for dynamic studies for the determination of tubular function. $^{99m}$Tc-DTPA, in contrast, indicates the efficiency of glomerular filtration[10].

Probably the major technical development in recent years is emission computerized tomography[14]. This has become an important factor in the quantitative evaluation of cardiac and brain metabolic studies. Quantitation of the uptake of the tracer places an emphasis on the removal of scattered radiation from overlying and underlying activity. A good example here is scanning of the myocardium in order to identify infarcted and ischaemic areas. 201 Thallium ($^{201}$T$\ell$) behaves physiologically as a potassium analogue in as far as it is indicative of perfusion and metabolic defects in the myocardium[2]. The quantitation of the $^{201}$T$\ell$ images is, however, problematic: it is a single gamma emitter and therefore does not automatically lend itself to tomographic slicing. In addition its primary emission, which is a low energy X-ray (E=80 KeV), can hardly be separated from scattered radiation. Recently a special collimator, which acts as a system of lenses, has become available. This device can be fitted to the face of a large field of view gamma camera. In conjunction with a data processor, 7 non-overlapping images (slices) through an organ can be obtained using this configuration. Thus emission tomography and its advantages could, to some extent, become available to those institutes limited to conventional equipment.

## CONCLUSIONS

The procedures discussed above constitute only some of the interesting and very relevant features of diagnostic nuclear medicine. It is needless to say that in the development of sophisticated instrumentation and radiopharmaceuticals, the animal model continues to play a major role. The inclusion of an adequate nuclear medicine facility in any well-equipped modern animal research and professional post-graduate training centre is therefore fully justified. As this need has never been adequately fulfilled in South Africa, the Division of Life Sciences of the Atomic Energy Board in close collaboration with the Department of Hospital Services of the Transvaal Provincial Administration has recently established such a research facility. It is equipped with the most modern detecting and data processing instrumentation and radiopharmacy laboratories with access to the National Isotope Production Centre of the Atomic Energy Board. The viability of the nuclear medicine facility is assured as it forms an integral part of the H. A. Grové Animal Research Centre at the H.F. Verwoerd Hospital in Pretoria. This centre complies with the most stringent staff and equipment requirements of the International Committee on Laboratory Animals and the Codes of Practice of all other national and international regulatory bodies governing animal experimentation.

All investigations were performed on male or female baboons (*Papio ursinus orientalis*). Prior to the administration of the radiopharmaceutical, anaesthesia was induced by means of ketamine hydrochloride (Ketalar, Parke Davis, Isando) and maintained with 1 – 2 % Halothane (Fluothane, I.C.I., Johannesburg).

The creation of animal models of a variety of human diseases is therefore possible and clinically applicable nuclear medicine procedures can now be developed and evaluated in South Africa by means of static and dynamic investigations in various experimental animals with non-invasive, non-lethal and painless procedures.

## ACKNOWLEDGEMENTS

We wish to extend our sincere gratitude to Mr N. Hugo for preparation of photographs and to staff of the H.A. Grové Animal Centre for their conscientious assistance.

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## BOOK REVIEW — BOEKRESENSIE

### AN ATLAS OF VETERINARY SURGERY


J. HICKMAN and R.G. WALKER
John Wright & Sons Ltd, Bristol 1980 Second edition, pp 244, numerous illustrations. Price UK £18,00
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Met hierdie boek word gepoog om chirurgiese prosedures op 'n aanskoulike wyse voor te stel deur hoofsaaklik van noukeurige sketse gebruik te maak en die geskrewe instruksies tot die minimum te beperk.

Die boek is verdeel in twaalf seksies waarin die chirurgiese prosedures voorgestel word volgens sisteme of streke.

In die eerste hoofstuk word algemene chirurgiese prosedures behandel waar-onder die teaterroetine, voorbereiding van die pasiënt, algemene chirurgiese instrumentasie en naatmateriaal.

In die tweede hoofstuk word die mees algemene chirurgie van die kop en nek van kleindiere sowel as grootdiere, kortliks behandel.

Abdomale en uro-genitale sisteme word in aparte seksies behandel met die kleindier as hoofkomponent.

Die hoofstuk oor die uier van die teef, en enkele afwykings van die spene van koei, beslaan slegs vier bladsye.

In die hoofstuk oor breuke (hernias) word die mees algemene breuke van die hond met "sprekende" sketse behandel.

Torakale -, oftalmiese – en neurochirurgie is met enkele uitsonderings bedoel as nie-gespesialiseerde werk, en om die algemene chirurg hiertoe aan te moedig.

Bykans vyf en twintig persent van die boek word aan ortopediese chirurgie gewy.

Met behulp van beskrywing en vele sketse word 'n besondere tegniek baie gouer en makliker verduidelik. Dit is opvallend hoedanig 'n teorie deur middel van sketse 'n werklikheid word.

In die laaste seksies van die boek work amputasies en algemene chirurgie fragmentaries aangebied.

Die sketse en kort beskrywings van die chirurgiese prosedures, maak die boek aantreklik en heelwat nuttige handleiding word verskaf. Ongelukkig is daar, veral wat die voorgraadse student betref, erge tekortkominge. Baie van die gebreke in die boek kan daaraan toegeskryf word dat die skrywers 'n te wye veld van chirurgie probeer bestryk.

Onder algemene en teaterprosedures word uitsluitlik na die kleindier verwys. Ander opvallende weglatings is:- die gastro-intestinale chirurgie vir koliek in die perd, penis en skede chirurgie van grootdiere, keratektomie operasies, intra-medullêre pen en figuur agt ortopediese draad fiksasie van fraktuur van die olecranon.

Aanvegbare stellings wat gemaak word is onder andere dat die laspunte van monofilament nylon knope inwendige irritasie en weefselreaksie veroorsaak, dat in jong wyfie katte met ovariohisterektomie die ovariese bloedvate sonder afbinding afgeskeur kan word, dat die m. sphincter ani internus betrokke is met perineale herniorafie, die wegskeur van speenslymvlies vir obstruksie van koeispene.

Dit is veral die algemene praktisyn wat met so 'n chirurgiese atlas gestimuleer sal word om 'n wyer veld van chirurgie te verken.

A.M. Lubbbe

# CHEMOTHERAPEUTIC OESTRUS CONTROL IN THE BITCH AND LATEST ADVANCES

D.G. CATTON

ABSTRACT: Catton D.G. **Chemotherapeutic oestrus control in the bitch and latest advances.** *Journal of the South African Veterinary Association* (1980) **51** No. 4 213–218 (En) Upjohn (Pty) Ltd, Box 246, 1600 Isando, Rep. of South Africa.

The control of oestrus in the intact bitch through the use of chemotherapeutics is briefly reviewed. Particular attention is paid to the recently introduced product mibolerone and a summary of currently available products is presented.

## INTRODUCTION

Concern for an ever increasing and, in some ways, burdensome pet population has resulted in widespread attention being given to the development of effective methods to control pet fertility. Investigative attempts have focussed on a variety of possible contraceptive methods. A vast amount of research has already taken place and more is in progress, which has already produced effective methods and will hopefully lead to improvements in the future. This presentation is concerned with an evaluation of various chemotherapeutic reproduction control agents that have been reported for the dog, and the current position in South Africa. It is also intended to briefly introduce Upjohn's new product mibolerone* The evaluation of the chemotherapeutics is based on the definition of an ideal contraceptive as reported by the National Conference on the Ecology of the Surplus Dog and Cat Problem[8]. On this basis, the effectiveness, safety, acceptability, availability, ease of administration, cost and reversibility were of major concern.

### Surgical sterilisation

Although this presentation by its title is not intended to review surgical sterilisation, one is obliged to refer to it because it is the principal method utilised. Ovariohysterectomy (spaying) is the most effective method of inducing sterility in the bitch. Spaying not only controls reproductive function and eliminates the nuisance of oestrus, but also provides a health advantage by virtually eliminating common uterine diseases, including mucometra and pyometra. Because of pet owners' psychological and economic resistance, ovariohysterectomy has not been the answer in controlling animal overpopulation. Emotional considerations of owners for their pets often results in opposition to irreversible surgical sterilization.

### General history

Chemical control of pet reproduction has received wide attention over the past 25 years. However, it was not until the late 1960's and early 1970's that detailed information about the physiology of reproduction, such as hormone levels, began to appear in scientific literature. A review of literature up to 1959[2, 49] shows us that the dog has an oestrous cycle which differs from that of most animals. The long interval between oestrous periods is not determined by season of the year, as happens in a number of species, nor does the dog return to heat shortly after regression of the corpus luteum, as is typical of other species. There is an interval of about 3–6 months following regression of the corpus luteum in the dog before another oestrous cycle begins. This phenomenon is still not understood, but may be suggestive of a need for recovery from progestational stimulation of the uterus and/or may reflect some sort of natural selection against continuous progestational exposure of the dog.

The late 1950's and early 1960's were the era of human oral contraceptive study and development. Two aspects of the human oral contraceptive development had an impact on dog reproductive information. For some reason, dogs were chosen as one of the species for safety studies of oral contraceptive drug candidates. There were 2 rather confusing aspects to this decision. First of all, was the fact that there was little knowledge of any important aspects of normal reproduction in the dog. Secondly, this decision failed to account for species differences. The dog is now known to be a very poor model for human reproduction because of the unusual uterine sensitivity to progesterone both in terms of dosage required and biological effects. Although fertility can be abolished in the bitch by administering various progestational steroids, pathological changes in the uterus may be induced which subsequently necessitate ovariohysterectomy.

### Progestational era

Following investigations with progesterone [11, 17, 18, 27, 43] in the late 1950's and early 1960's, two related products were introduced by the Upjohn Company to the veterinary profession to control reproduction in the dog. The first product, 17 alpha-acetoxyprogesterone† (Fig. 1), which was introduced in 1960, was designed for oral use and was not a marketing success. One predominant reason was the high cost, which was approximately US $90 for a year's treatment. In 1963 Upjohn introduced injectable medroxyprogesterone acetate‡[10] (Fig. 1). Injections were to be made every 6 months, and this procedure was well accepted by both veterinarians and pet owners. However, medroxyprogesterone acetate sales were discontinued in April, 1966 in the USA for basically 2 reasons. First was a prolonged and unpredictable return to oestrus. This appeared to be due to very slow and variable absorption from the injection site. The second reason for the removal of the product was because of an increased incidence (15–20%) of uterine effects, primarily cystic endometrial hyperplasia (CEH). When this situation occurred, the practising veterinarian usually diagnosed pyometra. Although naturally occurring pyometra usually produces a toxic

*Matenon, The Upjohn Co., containing mibolerone 100 μg/ml.

†Prodox, The Upjohn Co. containing 17α-acetoxy-progesterone 25 or 50 mg/mℓ.

‡Promone, The Upjohn Co. containing medroxyprogesterone acetate 50 mg/mℓ.



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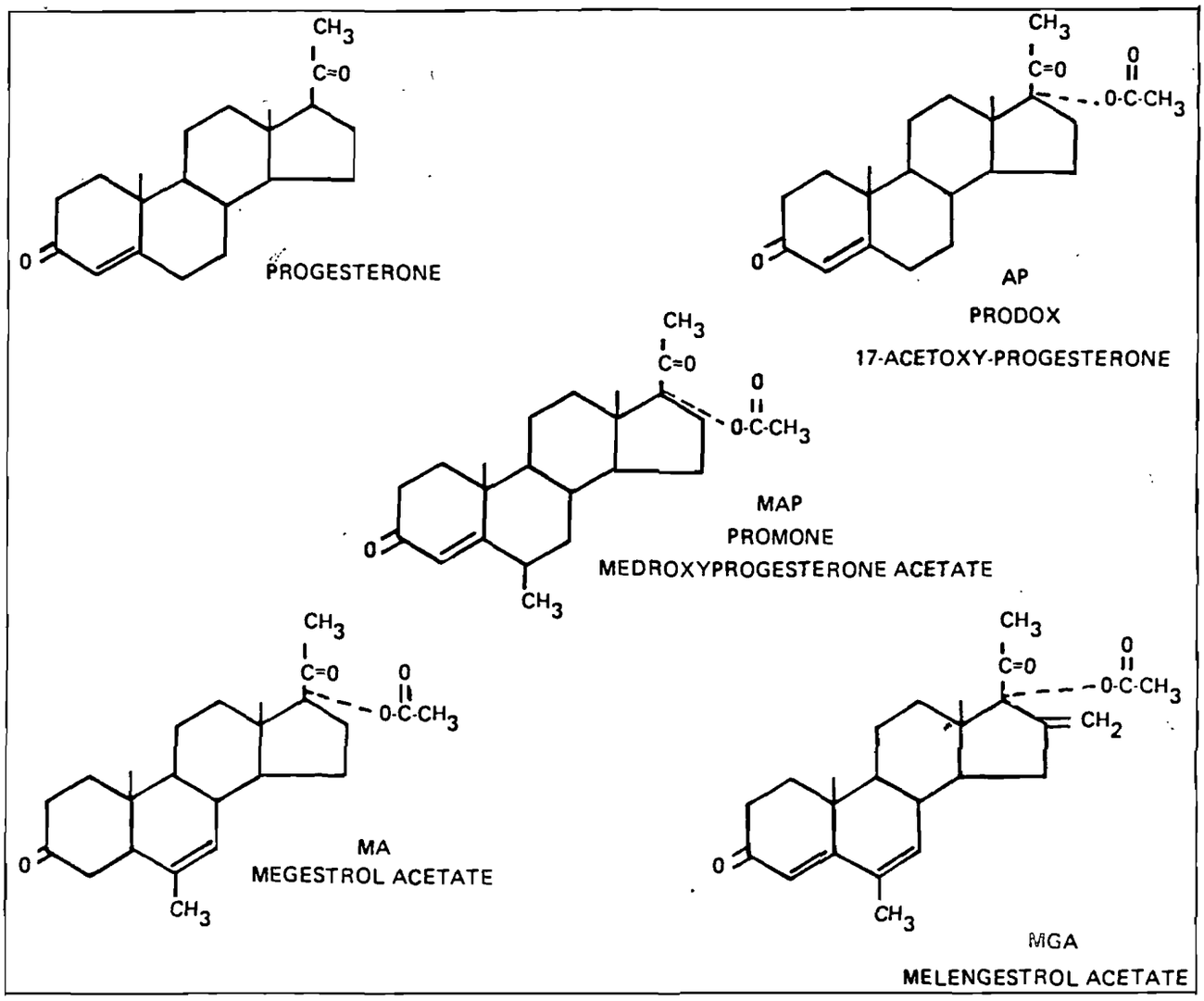


**Fig. 1.** Progestogens used in dogs.

effect and rapid action is usually needed to save the life of the dog, the "uterine effects" following medroxyprogesterone acetate were not typically of this nature.

In 1974, Schering marketed megestrol acetate § (Fig. 1). This product was available outside the USA earlier than 1974. Megestrol acetate is not recommended for continuous use, but rather has 2 treatment regimes. These are 8 days at a high dose or 32 days at a low dose. The high dose regime is for taking dogs out of heat that already show signs of oestrus, and the low dose treatment is to prevent oestrus in a dog that has not yet demonstrated signs of oestrus and in which an oestrus is not desired for a particular 32 day period. The onset of the next oestrus following the pro-oestrus or anoestrus treatments is variable (2–9 months) with most bitches becoming sexually active in 4 to 6 months.[31] Fertility at this oestrus is normal as are subsequent litter size and sex ratio.

Varous progestational hormone administration schedules and side effects observed are illustrated in Table 1. Biological effects that have been associated with the use of progestational steroids in the bitch are reflected in Table 2. In general, progestogens administered in low dosages and initiated during anoestrus are highly effective in preventing oestrous activity. Improperly used, hoewever, these steroids can produce deleterious effects in the bitch.

§Ovaban, Schering Corp. containing megestrol acetate 5 or 20mg tablet.

### Other pharmacological methods

#### *Androgen implants*

Testosterone administered as a subcutaneous implant, has been tested as a contraceptive[13]. The bitch is anaesthetised and the implants are inserted in the flank region. Implanted bitches are maintained in anoestrus for 420–840 days. The exact mechanism of action of testosterone is unknown. Few deleterious effects were experienced by the bitch during the implantation period. It was claimed that there were neither obvious alterations in the animal's behaviour nor apparent discomfort due to the implants. Testosterone implantation results in increased masculinization (enlarged clitoris). The interval between removal and onset of oestrus varied considerably (34–291 days). Conception rate at this first oestrus and subsequent litter size, birth rates, sex ratios and maternal behaviour were normal. The androgen implant contraceptive seems promising. This method is relatively simple, requiring one or two clinical visits,

Table 1: PROGESTOGENS USED FOR REPRODUCTION CONTROL IN THE BITCH

| Progestogen | Route of Administration | Advantages | Disadvantages | References |
|---|---|---|---|---|
| Progesterone in oil suspension | injection | successfully delays oestrus | frequent injections required, cystic endometrial hyperplasia (CEH), mammary hyperplasia, endometritis, obesity, metabolic disorders | 6, 13, 14, 15, 35, 43 |
| Medroxyprogesterone acetate (MAP) | injection or oral | successfully delays oestrus | CEH, pyometra, mucometra, mammary hyperplasia, metabolic disorders, vomiting, anorexia | 3, 6, 8, 10, 14, 15, 22, 41, 42, 45, 46, 47 |
| 17 alpha-acetoxy-progesterone | oral | effective and safety claimed, normal fertility after withdrawal | dose required varies with frequency of administration | 6, 9 |
| Norethisterone acetate | oral | safety claimed | only used for temporary postponement of oestrus | 15, 21, 29, 30 |
| Delmadinone acetate | injection | safety and efficacy claimed when administered during anoestrus | no report | 24, 42 |
| Melengestrol acetate | implant or oral | suppressed & prevented oestrus and ovulation | uterine pathology reported | 48 |
| Chlormadinone | oral | safety claimed | Pyometra, endometrial calcification, myometrial fibrosis, mammary hyperplasia and neoplasm, metabolic disorders | 28, 44 |
| Megestrol acetate (Ovaban) | oral | safe with proper dosing and accurate timing | timing is critical, long-term high dosing causes CEH | 6, 15, 31, 35 |

Table 2: BIOLOGICAL EFFECTS THAT HAVE BEEN ASSOCIATED WITH THE USE OF PROGESTATIONAL STEROIDS IN THE BITCH[35]

| | |
|---|---|
| Oestrous Inhibition | Stimulation of Mammary Nodules |
| Oestrous Prevention | Elevated Blood Glucose |
| Oestrous Suppression | Altered Glucose Tolerance |
| Endometrial Stimulation | Hypercholesterolemia |
| Endometritis | Renal Glomerular Lesions |
| Metritis | Masculinization of Female Foetus |
| Mucometra | Cystic Hyperplasia of Gallbladder |
| Pyometritis | Clitoral Enlargement |

long lasting and probably cheaper than other reversible methods.

*Chemical inducement of asexuality*

The injection of androgens into very young female mice and rats has been shown to inhibit sexual maturation and induce sterility[4, 5]. All available data suggest that androgens as well as progestin derivatives do not produce this effect in the bitch. Newborn female puppies within 48 hours of birth were injected with testosterone propionate, testosterone cyclopentylpropionate, or medroxyprogesterone acetate[47]. Enlarged clitorises were noted in all treated bitches. Age at first oestrus and subsequent fertility were not affected and were similar to controls. In contrast to the rodent, hypothalamic differentiation has occurred at birth in the dog, making the administration of steroids ineffective in inducing sterility.

*Chemosterilants in the male*

Although this too is not covered under the title of this paper, I believe it is interesting to briefly review some of the research that has been done in male dogs. There is little data available on reducing fertility in the male dog through administration of drugs. There is evidence indicating that chronic administration of an alkylating agent (alphachlorohydrin) inhibited canine spermatogenesis and caused a gradual loss of spermatogonia[16]. In addition, recent experiments have shown that calcium choride ($CaCl_2$) injected directly into the testicle is a convenient technique for inducing sterilization in the male dog[25]. Other preliminary studies in the dog indicated that a single epididymal injection of specific sclerosing agents will decrease sperm numbers to zero within 70–90 days with no concomitant effect on ejaculate volume.

Immunological methods

Another area which has been investigated is immunological methods. Neutralising endogenous hormones through immunisation is a promising nonsurgical method of inhibiting fertility. Research studies currently being performed in the dog are based upon earlier work which indicated that rabbits injected with a purified bovine pituitary gonadotropin, luteinizing hormone (LH), became actively immunized.[1, 19, 20] Injected rabbits produced antibodies which cross-reacted with endogenous circulating LH, causing genital atrophy and infertility. In canine studies[19, 20] male dogs received a series of bovine LH injections over a 3 month period.

The sera of immunized dogs contained antibodies to the foreign hormone. Treated males were unable to ejaculate approximately 6 weeks after immunisation and reproductive function was impaired for as long as one year after injections were initiated. Atrophy of the reproductive organs occurred with significant decreases in testicular, epididymal and prostate weight in comparison to controls. No other adverse physical or behavioural effects were observed. The usefulness of the immunological technique in the dog is limited since a highly purified antigen source such as bovine LH is not currently commercially available and repeated antigen injections are required. Theoretically it could be safe, simple and economical. There appears to be declining interest in the future feasibility of immunologic reproduction control methods in the dog, at least until a more highly purified source of gonadotropin antigen becomes available.

### Mibolerone

The latest advance in other pharmacological methods utilised in the bitch, is the development by The Upjohn Company of the product nibolerone[49]. This is a chemical contraceptive which has been tested extensively in the bitch and is a non-progestational steroid which has anabolic, androgenic and anti-gonadotropic activity[12, 26, 32]. Although a structural kinship exists between the steroids progesterone, testosterone and mibolerone (Fig. 2), tests in the bitch have shown mibolerone to have neither oestrogenic nor progestational properties. Under the influence of mibolerone, there is primary and secondary follicular development on the ovary, but follicles never mature to ovulatory size. This is because the LH surge is blocked, thus preventing ovulation. Thus oestrus, ovulation and corpus luteum formation are prevented for as long as mibolerone is administered[32, 36]. However, it must be borne in mind that this drug is ineffective in inhibiting oestrus when initiated after the onset of pro-oestrus.

Mibolerone has also been extensively tested concurrently with other classes of drugs to determine whether there were any apparent drug interactions. The other classes of drugs included general and local anaesthetics, analgesic/antipyretics, anthelmintics, parasiticides, antibacterials, fungicides, corticosteroids and biologicals[23]. From all trials completed, concurrent therapy at this point in time has not resulted in any drug interactions. The following side effects have been associated with the use of mibolerone:

1. Clitoral enlargement – In clinical studies, 20% had clitoral enlargement 'detectable' and 1% were considered 'objectionable'.
2. Vaginal discharge – Although this side effect was not frequently noted in the clinical field tests, 10% of the kennel dogs at the Upjohn Research facility showed some degree of vaginal discharge when examined closely. Overall 1% were considered 'objectionable'. This appears to result from irritation of the clitoral fossa and vaginal vestibule caused by clitoral enlargement. It normally takes the form of a white viscid liquid.
3. Riding behaviour – This appears to be associated with irritation of the clitoral fossa and vaginal vestibule. It appeared in 1,6% of the dogs and all cases were considered 'objectionable'.
4. Epiphora – This was reported in 5,6% of the animals receiving mibolerone, however, this included all dogs that exhibited the condition prior to their exposure to the drug.
5. Musky body odour – 4,3%. This percent included those with musky odour at the onset of the study.

Immature females have shown an increased sensitivity to mibolerone, expressed primarily as clitoral enlargement and vaginal discharge (1%). When comparing the side effects in the bitch which result from the use of progestational agents or mibolerone, it can be seen that mibolerone does not cause any life threatening consequences. A comparative list of the biological effects of the two classes of compounds is included in Table 3.

Throughout safety and toxicity studies, mibolerone was found to have a wide margin of safety and a minimum number of undesirable side effects in the bitch. The oestrous prevention efficacy of mibolerone was extensively evaluated in kennel dogs, in university studies and in field tests conducted by veterinary practitioners[37, 38, 39, 40]. These studies demonstrated that while efficacy is not 100%, with proper dosage and routine treatment, oestrus prevention of at least 95% can be expected.

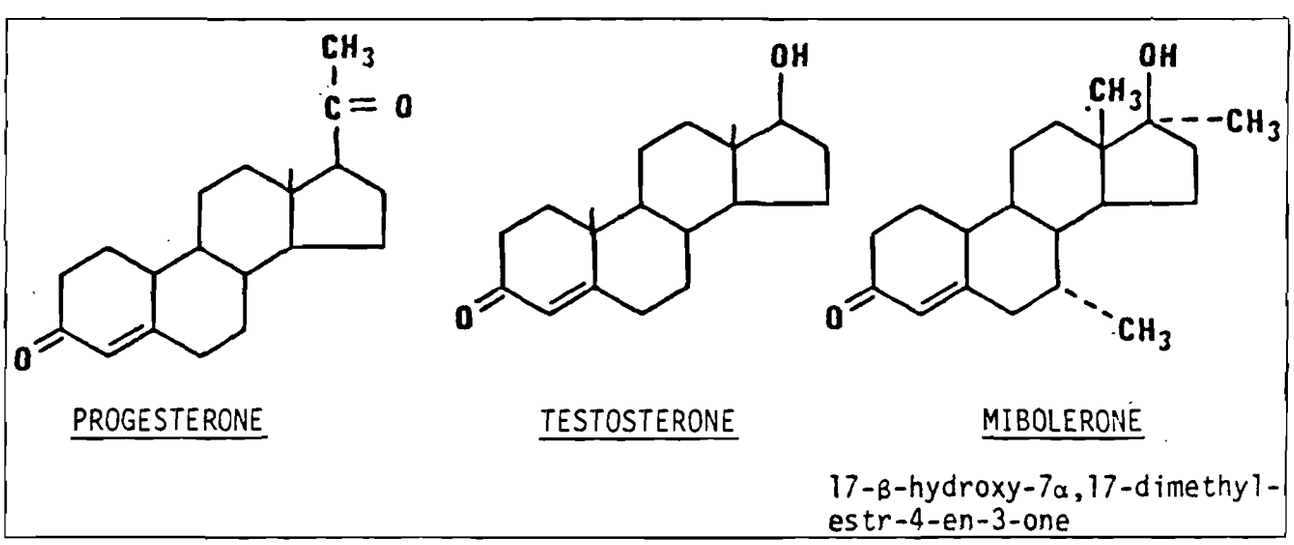


**Fig. 2.** Structural kinship between progestrone, testoterone and mibolerone.

Table 3: COMPARISON OF THE BIOLOGICAL EFFECTS OF PROGESTOGENS AND MIBOLERONE[36]

| Item | Progestogens* | Mibolerone** |
|---|---|---|
| Oestrous Prevention | Yes | Yes |
| Oestrous Suppression | Yes | No |
| Endometrial Hyperplasia | Yes | No |
| Metritis | Increased incidence | No increased incidence |
| Mucometra | Increased incidence | No increased incidence |
| Endometritis | Increased incidence | No increased incidence |
| Pyometritis | Increased incidence | No increased incidence |
| Stimulated Mammary Nodules | Yes | No |
| Elevated Blood Glucose | Yes | No |
| Altered Glucose Tolerance | Yes | No |
| Hypercholesterolaemia | Yes | No |
| Renal Glomerular Lesions | Yes | No |
| Cystic Hyperplasia of Gallbladder | Yes | No |
| Masculinization of Female Fetus | Yes | Yes |
| Clitoral Enlargement | Yes | Yes |
| Hypocholesterolaemia | No | Yes |
| Elevated SGPT | No | Yes |

*Biological effects reported in scientific literature.
**Biological effects noted in mibolerone research animals and reported in the literature.

### Position in South Africa

The products that are currently commercially available in South Africa for reproduction control are listed in Table 4.

Table 4: CHEMOTHERAPEUTICS FOR REPRODUCTION CONTROL IN THE BITCH

| Trade Name | |
|---|---|
| Injectable products | Active Ingredient |
| Depo Provera 50mg/mℓ 5 mℓ | Medroxyprogesterone Acetate |
| Depo Provera 100 mg/mℓ 5 mℓ | Medroxyprogesterone Acetate |
| Depo Provera 150 mg/mℓ 1 mℓ | Medrosyprogesterone Acetate |
| Tablets | |
| Hormovet 2,5 mg | Melengestrol Acetate |
| Megestrate 5 mg | Melengestrol Acetate |
| Ovarid 5 mg | Melengestrol Acetate |
| Ovarid 20 mg | Melengestrol Acetate |
| Perlutex 5 mg | Medroxyprogesterone Acetate |
| Drops | |
| Matenon 100 μg/mℓ | Mibolerone |

### *Future areas of research*

Biomedical studies concerned with controlling human fertility will provide insight into other possible methods for controlling reproduction in the dog. Intravaginal rings, intracervical or intrauterine devices, or biodegradable delivery systems, all of which can be utilised to release pharmacological agents, are being tested in humans and may be eventually useful as pet contraceptives.

Other areas of current investigation in dog reproduction control include development of new sclerosing agents, thermocontraceptive methods, microwaves or ultrasonic techniques and injectable abortifacients[33].

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## BOOK REVIEW

## BOEKRESENSIE

### MILLER'S GUIDE TO THE DISSECTION OF THE DOG

H.E. EVANS and A. DE LAHUNTA
W.B. Saunders Company Philadelphia 2nd Ed. 1980. pp vi 305 Fig. 217 Tabs. 4 Publ. Price £9.50

Miller's Guide to the Dissection of the dog is a revised *and updated version of the 1st Edition. Some of the* dissection procedures have been altered and new descriptive material and illustrations added. The purpose of the book is to facilitate a thorough dissection of the dog. With aid of 217 illustrations, clear and concise dissection instructions and proper descriptions, the student of canine anatomy can gain essential anatomical knowledge. Because the book is a dissection guide and not a textbook, the student interested in a more detailed description of the structures under consideration, will have to consult a textbook. The student will greatly benefit from the added 14 illustrations as well as the improvement of some of the previous illustrations. The inclusion of the innervation of muscles under the muscular system is a great improvement.

In the reviewer's opinion this is a well written book and it is presented in a way which will facilitate the dissection and study of the dog's anatomy.

A.J. Bezuidenhout

# AN UNIDENTIFIED *BABESIA* OF THE DOMESTIC CAT (*FELIS DOMESTICUS*)

C.G. STEWART*, K.J.W. HACKETT† and M.G. COLLETT‡

ABSTRACT: Stewart C.G.; Hackett K.; Collett M. **An unidentified *Babesia* of the domestic cat *(Felis domesticus).*** *Journal of the South African Veterinary Association* (1980) **51** No. 4 219–221(En) Dept. Infec. Dis., Fac. Vet. Science, Univ. Pretoria, Box 12580, 0110 Onderstepoort, Republic of South Africa.

An unidentified *Babesia* was seen in a blood smear from a cat showing signs of anaemia. The cat responded to treatment with diminazine (Berenil§). The morphology of the parasite is described and a comparison is made with other *Babesia* which have been described from the domestic cat and wild felids. This parasite most closely resembled *B. herpailuri* described from a jaguarundi in South America.

## INTRODUCTION

Babesiosis in the domestic cat due to *Babesia felis* is an important disease of cats in the Republic of South Africa, particularly in the coastal areas[8, 13]. Natural infection in cats has not been reported from any other country[6]. *B. felis* has also been reported from the wild cat (*Felis hybica*), the puma (*F. concolor*), the American lynx (*Lynx rufus*), the leopard *(Panthera pardus syn F. pardus)* and the lion (*F. leo*)[10].

A number of authors have listed *B. felis* as the only species causing natural infection in domestic cats[9, 10, 14] whereas others[6] consider *B. cati* isolated in India from a wild cat[12] to be similar to a *Babesia* reported by Mangrulkar[11] and seen in smears obtained at autopsy from a single domestic cat.

Several *Babesia* species obtained from wild Felidae have been established as experimental infections in the domestic cat. A strain of *B. felis* obtained from a wild cat in the Sudan was established experimentally in domestic cats[3]. *B. pantherae* isolated from a leopard (*Panthera pardus*) in Kenya[2, 6] and *B. herpailuri* obtained from a jaguarundi (*Felis yagouaroundi,* syn. *Herpailurus jaguarundi*) in South America[4] were also established experimentally in cats[5].

## CASE HISTORY

An 8 year old neutered Siamese cat showing signs of fever, malaise, anorexia, muddy mucous membranes and mild constipation was submitted for examination. The cat had been obtained as a kitten by the owner and had been reared in Salisbury, Zimbabwe.The cat was treated with sulphadoxine and trimethoprim** and vitamin B 12 for 3 days by which time sufficient improvement had occurred to allow the animal to return home. Two days later the cat was returned showing signs of anorexia, pale muddy mucous membranes and a temperature of 40°C. The cat was treated with amoxycillin†† and a blood smear was taken which was positive for *Babesia.* The cat was treated with diminazine§ (5 mg/kg) and a multi-vitamin mixture‡‡. The next day the temperature had dropped to 39,2°C and the cat had started to eat and was clinically normal 2 days later. When seen 9 days later it was eating well and the colour of the mucous membranes had returned to normal.

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§Hoechst Pharmaceuticals (Pty) Ltd.
**Borgal V, Hoechst Pharmaceuticals.
††Clamoxyl, Beecham Research Labs.
‡‡Parenterovite, Beecham Research Labs.

## MATERIAL AND METHODS

Parasites were measured by means of an adjustable occular micrometer§§ and drawings were made with the aid of a drawing tube¶¶. Blood was collected on 3 different days and the haematological examinations as shown in Table 1 were performed. Blood smears were stained with Giemsa.

## RESULTS

Babesia parasites were seen in the red blood cells. The parasitaemia was very low with approximately one parasite being seen per 30 microscopic fields examined (approximately 400 erythrocytes per field). Signs of anaemia were present which included polychromasia of red blood cells. A very noticeable feature of the smear was the extensive erythrophagocytosis of both parasitized and non-parasitized erythrocytes.

Table 1: HAEMATOLOGICAL RESULTS

| | Day after Treatment | | |
|---|---|---|---|
| | 0 | 1 | 13 |
| Hb g/ℓ | 72 | 70 | 120 |
| Haematocrit | 0,25 | 0,25 | 0,37 |
| MCHC | 0,29 | 0,28 | 0,32 |
| WBC × $10^9$/ℓ | 7900 | 12000 | 4900 |
| Neutrophils | 92 | 84 | 79 |
| Lymphocytes | 6 | 16 | 17 |
| Monocytes | – | – | 2 |
| Eosinophils | 2 | – | 2 |
| Basophils | – | – | – |
| *Parasites* | + | – | – |

The results of haematological examination are shown in Table I. These results show that the cat had a moderate anaemia which returned to normal following treatment. Both the haemoglobin level and the haematocrit were below normal. There was a neutrophilia, probably associated with stress.

## DESCRIPTION OF THE PARASITE

The morphology of the parasites is shown in Figs. 1 and 2. Single forms, paired forms and budding forms were seen within the erythrocytes and occurred mainly in the centre of the cell. Extra-cellular forms were rarely seen. The maximum number of parasites seen in a single cell was 8. At no time was there any evidence

§§Okular 15 x 5 K Wild.
¶¶Wild.


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that parasites had divided by quarternary division with the formation of cross forms which are typical of *B. felis*. Most of the dividing forms were arranged at acute angles (80%) and more rarely at right angles (7%) or obtuse angles (13%).

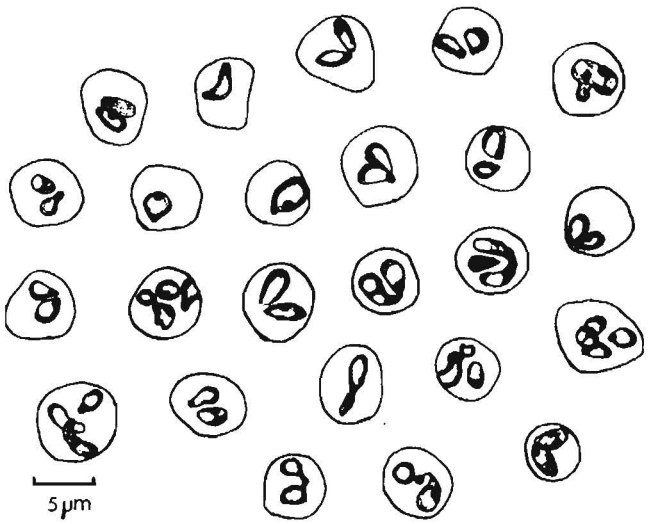


**Fig. 1.** Drawings of infected red blood cells.

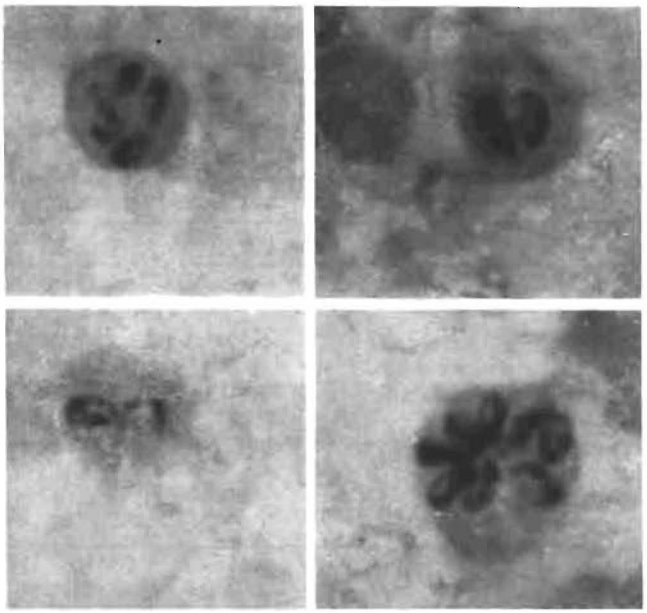

**Fig. 2.** Different forms of *Babesia* species from a cat.

Dividing forms measured from 1,57–3,49 μm (mean 2,65 μm) in length and from 0,86–1,72 μm (mean 1,31 μm) in width (30 parasites measured). Single forms varied from 1,84–3,53 μm in length (mean 2,04 μm) and from 1,22–2,50 μm (mean 1,92 μm) in width (10 parasites measured).

## DISCUSSION

The relevant measurements comparing the parasite under discussion with *B. pantheri, B. herpailuri, B. cati* and *B. felis* are shown in Table 2. This parasite was larger than *B. felis* and cross forms typical of *B. felis* were not seen. *B. pantheri* is smaller and also differs in the fact that single forms are situated on the periphery of the host cells and the majority of pairs form an obtuse angle. *B. cati* is also smaller with a maximum size of 1,5 μm for single parasites and a maximum measurement of 2,5 μm for all types of parasite. In the present study the maximum size was 3,49 μm in length. The measurements of *B. herpailuri* are very similar to the parasite examined in this study, as are the drawings and photograph[4, 5]. For this reason the parasite has been tentatively identified as *B. herpailuri*. This parasite has not previously been reported from Africa, and Jaguarundi can be excluded as the natural host in this case as they do not occur in Zimbabwe. Other wild Felidae or wild species may have been the source of infection but as this cat was reared in a built-up suburb of Salisbury, the larger wild Felidae can be excluded.

Table 2: A COMPARISON OF THE UNIDENTIFIED CAT *BABESIA* AND OTHER *BABESIA* OF FELIDS

| Form | The Unidentified Cat *Babesia* | *B. herpailuri*[5] | *B. pantheri*[5] | *B. cati*[11] | *B. felis*[5] |
|---|---|---|---|---|---|
| Length Pairs μm | 2,65 | 2,7 | 2,2 | | 0,3 |
| Width Pairs μm | 1,31 | 1,3 | 1,2 | 2,5–0,5 | 0,1 |
| Length Single μm | 2,04 | 2,7 | 2,0 | | 0,9 |
| Width Single μm | 1,92 | 2,2 | 1,8 | | 0,7 |
| % Obtuse angle | 13% | 3% | 60% | | |
| % $L^S$ | 7% | 41% | 25% | | |
| % Acute | 80% | 56% | 15% | | |
| Position | Central | Central | Peripheral | | Central |

Generally *Babesia* spp are fairly host-specific. *B. microti* of rodents, and *B. divergens* and *B. bovis* of cattle, however, have been recorded in man[15] and it would appear that the genus *Babesia* are less host-specific than was previously thought. The possibility exists, therefore, that this *Babesia* may occur naturally in some other host and that this infection in a domestic cat was merely a chance phenomenon. *B. canis* which commonly occurs in Zimbabwe, can be excluded as all attempts to transmit this parasite to cats have been unsuccessful[8, 16] and, in addition, this parasite is smaller than *B. canis*. Levine[10] gives the size of *B. canis* as 4,5 μm x 2,4 μm. The other possibility is that this is a specific infection of domestic cats which has been missed in the past. This would only be possible if the parasite normally occurred at very low parasitaemia as blood smears are frequently examined by practising veterinarians and even low parasitaemias as seen in this case are unlikely to be missed.

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## BOOK REVIEW — BOEKRESENSIE

### SURGERY OF TRAUMATIZED SKIN: MANAGEMENT AND RECONSTRUCTION IN THE DOG AND CAT

STEVEN F. SWAIM
W.B. Saunders Company, Philadelphia pp 585, figs 493, colour plates 30. Price £22–75
(ISBN 0 7216 8688 8)

Hierdie boek is meer omvattend as wat die titel aandui. In die eerste seksie word 'n oorsig gegee van die anatomie en fisiologie van die vel met 37 verwysingsbronne. In die hoofstuk waarin velbeserings en wondgenesing behandel word, word 'n deeglike samevatting van die betrokke feite gegee.

In die tweede seksie word die hantering van besoedelde en besmette wonde, en termiese trouma, metodies gedoen met soveel as 91 verwysingsbronne. Die korrekte hantering en versorging van wonde vorm 'n belangrike deel van velchirurgie.

In die tweede helfte van die boek word die spesifieke chirurgie van velwonde behandel met aanskoulike meegaande sketse en foto's.

In die hoofstuk wat handel oor algemene beginsels van wondchirurgie en hegting, word onder andere 'n gedetailleerde beskrywing gegee van hegtingstegnieke en verskeidenheid van naatmateriaal.

Die tegnieke van rekonstruksie van die vel word volledig behandel.

In afsonderlike hoofstukke word die spesifieke chirurgie van die ooglede, oor en sommige velafwykings voorgestel.

Die basiese feite van hantering en chirurgie van die vel, word uiters aanskoulik aangebied in hierdie boek.

Op 'n oortuigende wyse word die teorie en die praktiese werklikheid met mekaar versoen. Hierdie boek is 'n handige verwysingsbron vir die leerlingchirurg, en 'n praktiese handboek vir die toegewyde praktisyn. Hierdie boek kan baie sterk aanbeveel word, omdat die voorgestelde chirurgiese prosedures gerugsteun word deur basiese feitekennis.

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# ANTHELMINTIC EFFICIENCY OF FENBENDAZOLE IN EQUINES

F.S. MALAN* and R.K. REINECKE†

ABSTRACT: Malan F.S.; Reinecke R.K. **Anthelmintic efficiency of fenbendazole in equines** *Journal of the South African Veterinary Association* (1980) **51** No 4 223–226 (En) Hoechst Research Station,P.O. Box 124, 1320 Malelane, Republic of South Africa.

A single oral dose of fenbendazole (FBZ) at 10mg/kg body mass was given to 5 donkeys. A further 5 donkeys were dosed with a medicated lick (1mg FBZ/g lick) until the oral consumption was 10mg/kg body mass.

In both trials FBZ was highly effective against adults of the following genera: *Cyathostomum, Cylicocyelus, Cylicostephanus, Cylicodontophorus, Poteriostomum, Cabellonema, Craterostomum* and *Triodontophorus;* similarly high efficiency was obtained against the following species: *Habronema majus, Habronema musca, Strongylus vulgaris* and *Oxyuris equi* and worms identified as belonging to the subfamily Cyathostostominae.

These results were confirmed in horses and in addition FBZ at 10mg/kg was highly effective against *Gyalocephalus capitatus, Oesophagodontos robustus* and *Parascaris equorum.*

## INTRODUCTION

The efficacy of fenbendazole (FBZ) for treatment of adult gastrointestinal nematode infections in horses has been demonstrated. At a dose level of 5mg/kg FBZ is 100% effective in the removal of adult *Strongylus vulgaris, 99% for adult Strongylus edentatus,* 92% for small strongyles, 100% for mature *Oxyuris equi* and 80% for mature *Parascaris equorum*[1]. Treatment of horses with FBZ at dose levels of 7,5–60 mg/kg produced negative faecal egg counts for a minimum period of 6 to 8 weeks[5].

This paper reports on critical anthelmintic tests in donkeys and modified critical anthelmintic tests on horses.

## CRITICAL ANTHELMINTIC TESTS

### MATERIALS AND METHODS

Two groups of 5 donkeys were kept on a cement floor each one in its own pen. Hay and water were supplied *ad libitum.* Body mass of donkeys were determined before treatment and worm egg counts were done before and after treatment.

One group was treated with 10% FBZ suspension (Panacur: Hoechst) at a dosage rate of 10mg/kg body mass *per os* with a stomach tube.

Another group was given FBZ mixed in a lick (National Chemical Products). Each gram of the lick contained 1 mg FBZ and the total amount of FBZ consumed was 10mg/kg body mass.

Every morning and afternoon for 7 days after treatment the total faecal output was collected from each donkey. Faecal worm egg counts were done every day.

Thereafter the faeces were broken between the fingers and mixed before being weighed. In each specimen two aliquots of 1/10 and 1/100 by mass respectively were sieved with water on 150 μm sieves and the residue on the sieve surface collected and formalin added. Worms were counted macroscopically in the larger specimen (1/10) and microscopically in the smaller specimen (1/100).

Animals were slaughtered and methods used for worm recovery were the same as those previously described by Reinecke and Le Roux[6].

After the donkeys were shot, the throats were cut and the thoracic and abdominal cavities were opened by making an incision along the ventral line of the animal and the left half of the thorax and the abdominal wall was removed. The organs from the thoracic and abdominal cavities were removed from the carcass.

The different organs of the gastro-intestinal tract were then isolated by tying double ligatures around the gut to separate it into the stomach, small intestine, caecum, ventral colon and dorsal colon.

The contents of the small intestine and stomach were removed, iodine added and then sieved through a 150 μm sieve on top of a 38 μm sieve to obtain residue and filtrate samples.

The entire ingesta of the caecum; ¼ by mass of ingesta in the ventral colon and ½ by mass of the dorsal colon contents were placed in the modified Baermann apparatus as described[6].

These specimens were left in the waterbath at 40°C for two hours. Two fractions: a residue and filtrate were obtained. Iodine was added to each fraction and sieved. The residue was sieved on 150 μm and filtrate on 38 μm sieves and the residues collected and preserved in 10% formalin.

Digests were prepared by removing the mucosa and muscularis layers off the serosa of the gut. After mincing the mucosa and muscularis it was digested by adding 2% pepsin m/v and 10 N HCL 3% v/v in a glass jar. The jars were then placed in a waterbath at a temperature of 40°C for 3 hours. The whole stomach mucosa was digested, and after determining the mass of the gut wall ½ the mucosa of the small intestine and caecum, ¼ of the ventral colon and ½ of the dorsal colon were digested in separated jars in the waterbath. The digested mucosae were then sieved on a 38 μm sieve, the residue on the sieve surface collected and 10% formalin added. Nematodes were recovered microscopically.

Residue samples of ingesta were examined macroscopically and filtrate sample microscopically with a stereo microscope. Nematodes present were placed in a specimen bottle containing 10% formalin. Helminths were identified at a later stage by placing them on glass slides, clearing them with lactophenol, examining them microscopically and classifying them using the descriptions of Lichtenfels[3]. Cyathostomes were identified to genus level, but the species were determined in the other helminths.

### RESULTS

Data are summarized in Tables 1 and 2.

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Table 1: DONKEYS GIVEN A SINGLE DOSE FBZ AT 10 mg/kg LIVE MASS. WORMS RECOVERED FROM FAECES AND AT NECROPSY

| Donkey Number | Site | Cyatho-stominae | | *Triodontophorus* | *S. vulgaris* | *S. equinus* | *Craterostomum* | H. majus | | *H. musca* | D. megastoma | | *T. axei* | *P. vivipara* | *G. nasalis* | *G. intestinalis* |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | $L_4$ | Adult | | | | | $L_4$ | Adult | | $L_4$ | Adult | | | $L_3$ | $L_3$ |
| | Faeces | 7 344 | 28 021 | 2 944 | 10 | 0 | 0 | 0 | 270 | 10 | 0 | 3 | 0 | 0 | 0 | 0 |
| 0934 | Necropsy | 2 208 | 188 | 0 | 8 | 0 | 0 | 0 | 60 | 11 | 4 | 77 | 0 | 0 | 31 | 4 |
| | Reduction | 76,9% | 99,3% | 100% | 55,5% | — | — | — | 81,8% | 47,6% | 0% | 3,8% | — | — | 0% | 0% |
| | Faeces | 1 100 | 11 992 | 200 | 10 | 10 | 10 | 0 | 10 | 0 | 0 | 0 | 0 | 36 200 | 0 | 0 |
| 0935 | Necropsy | 1 618 | 4 | 0 | 0 | 0 | 0 | 8 | 0 | 0 | 34 | 31 | 12 | 0 | 1 | 0 |
| | Reduction | 40,5% | 99,9% | 100% | 100% | 100% | 100% | 0% | 100% | — | 0% | 0% | 0% | 100% | 0% | — |
| | Faeces | 13 262 | 110 482 | 360 | 10 | 0 | 2 164 | 0 | 130 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0948 | Necropsy | 1 782 | 0 | 0 | 0 | 0 | 0 | 0 | 44 | 0 | 323 | 86 | 54 | 0 | 5 | 4 |
| | Reduction | 88,2% | 100% | 100% | 100% | — | 100% | — | 74,7% | — | 0% | 0% | 0% | — | 0% | 0% |
| | Faeces | 9 709 | 29 242 | 300 | 380 | 0 | 3 462 | 0 | 150 | 10 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0949 | Necropsy | 5 639[1] | 74 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 12 | 0 |
| | Reduction | 63,3% | 99,7% | 100% | 100% | — | 100% | — | 100% | 100% | — | — | — | — | 0% | — |
| | Faeces | 1 706 | 725 | 20 | 120 | 0 | 12 | 0 | 70 | 0 | 0 | 0 | 8 | 0 | 0 | 0 |
| 0950 | Necropsy | 7 147[2] | 28 | 0 | 10 | 0 | 0 | 0 | 10 | 0 | 122 | 84 | 20 | 0 | 0 | 2 |
| | Reduction | 19,2% | 96,5% | 100% | 92,3% | — | 100% | — | 87,3% | — | 0% | 0% | 28,6% | — | — | 0% |

1 Plus 10 $L_3$ of Cyathostominae at necropsy
2 Plus 138 $L_3$ of Cyathostominae at necropsy

Table 2: Group 2: DONKEYS HAD ACCESS TO A BLOCK CONTAINING 1 mg FBZ/g AND WERE FED UNTIL THEY HAD CONSUMED 10 mg/kg LIVE MASS. WORMS RECOVERED FROM FAECES AND AT NECROPSY

| Donkey | Site | Cyatho-stominae | | *Triodontophorus* | *S. vulgaris* | *S. equinus* | *Craterostomum* | *H. majus* | | *H. musca* | *D. mega-stoma* | Number | | | *T. axei* | *P. vivipara* | *G. nasalis* | *G. intestinalis* |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | $L_4$ | Adult | | | | | $L_4$ | Adult | | $L_4$ | Adult | $L_4$ | Adult | | | $L_3$ | $L_3$ |
| | Faeces | 3 267 | 3 075 | 0 | 10 | 0 | 0 | 0 | 120 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0943 | Necropsy | 8 144[1] | 2 | 0 | 4 | 0 | 0 | 0 | 0 | 0 | 98 | 20 | 0 | 0 | 0 | 0 | 10 | 2 |
| | Reduction | 28,6% | 99,9% | — | 71,4% | — | — | — | 100% | — | 0% | 0% | — | — | — | — | 0% | 0% |
| | Faeces | 1 525 | 10 236 | 130 | 130 | 0 | 125 | 0 | 100 | 0 | 202 | 0 | 0 | 0 | 0 | 28 670 | 0 | 0 |
| 0944 | Necropsy | 3 386 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 87 | 3 | 0 | 0 | 76 | 0 | 0 | 0 |
| | Reduction | 31,1% | 100% | 100% | 100% | — | 100% | — | 99% | — | 69,8% | 0% | — | — | 0% | 100% | — | — |
| | Faeces | 6 949 | 7 860 | 240 | 40 | 0 | 11 | 0 | 40 | 10 | 0 | 1 | 230 | 10 | 0 | 0 | 0 | 0 |
| 0945 | Necropsy | 4 182[2] | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 140 | 41 | 0 | 0 | 16 | 0 | 0 | 1 |
| | Reduction | 62,4% | 100% | 100% | 100% | — | 100% | — | 100% | 100% | 0% | 2,4% | 100% | 100% | 0% | — | — | 0% |
| | Faeces | 3 505 | 5 971 | 120 | 180 | 10 | 325 | 0 | 100 | 0 | 0 | 0 | 10 | 0 | 0 | 0 | 0 | 0 |
| 0946 | Necropsy | 5 095[3] | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 10 | 2 | 0 | 0 | 0 | 0 | 42 | 1 |
| | Reduction | 40,8% | 100% | 100% | 99,4% | 100% | 100% | — | 100% | — | 0% | 0% | 100% | — | — | — | 0% | 0% |
| | Faeces | 523 | 3 188 | 0 | 0 | 0 | 115 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0953 | Necropsy | 214 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 141 | 1 | 0 | 0 | 66 | 0 | 5 | 0 |
| | Reduction | 71,4% | 100% | — | — | — | 100% | 0% | — | — | 0% | 0% | — | — | 0% | — | 0% | — |

1 Plus 804 $L_3$ of Cyathostominae recovered at necropsy
2 Plus 572 $L_3$ of Cyathostominae recovered at necropsy
3 Plus 404 $L_3$ of Cyathostominae recovered at necropsy

The genera of Cyathostominae are presented in these tables under the heading of the subfamily. Details of the efficacy of FBZ at 10mg/kg against the different genera of Cyathostominae are:

1. *Cyathostomum, Cylicostephanus, Cylicodontophorus, Gyalocephalus, Poteriostomum* and *Cabellonema* 100%.
2. *Cylicocyclus:* in Group 1 it ranged from 93 – 100%

and in Group 2 from 99,9 – 100%.

3. *Cylindropharynx* – ineffective

The incidence of the various genera differed. *Cylicocylus, Cyathostomum* and *Cylicostephanus* were always present and *Cylicocyclus* the most prevalent in 8 of the 10 faecal specimens. *Cylicodontophorus* was less numerous and present in the faeces of 8 donkeys. *Poteriostomum* was present in 2 and *Gyalocephalus* and *Cabellonema* in the faeces of 1 donkey only. At necropsy 8 *Cylindropharynx* were recovered from 1 donkey but none were present in the faeces.

The efficiency against other adult stronyles varied as follows: *Triodontophorus, Craterostomum* and *Strongylus equinus* 100% and *Strongylus vulgaris* 55,5–100%.

Fenbendazole efficiency varied against adults of other species as follows: *Habronema majus* 81,8–100%, *Habronema musca* 47,6–100%, *Probstmayria vivipara* 100%, $L_4$ and adult *Oxyuris equi* 100% but it was ineffective against adult *Trichostrongylus axei*, $L_4$ and adult *Draschia megastoma*, 3rd instar larvae of *Gasterophilus nasalis* and *Gasterophilus intestinalis* and against $L_4$ of Cyathostominae ranged from 19,2–88,1%.

## MODIFIED CRITICAL ANTHELMINTIC TEST IN HORSES

The term modified critical anthelmintic test is used to denote a combination of faecal worm egg counts and counts of worms expelled in the faeces. The host is not killed after treatment.

### MATERIALS AND METHODS

Seventeen horses from a Boerperd stud breeder were selected. Faecal worm egg counts were high and some had *Parascaris equorum* eggs.

The mass of each horse was determined and the animals were treated with a 10% suspension of fenbendazole at a dosage rate of 10mg/kg body mass administered through a stomach tube. The horses were individually kept in loose boxes and they were fed sugar cane tops *ad libitum* and water was supplied.

Faeces were collected for a week every morning and afternoon. A 1/10 and 1/100 aliquot by mass of each sample was collected and washed on a 150 $\mu$m sieve. The residues were poured into glass jars and formalin was added.

Due to the large numbers of worms the 1/10 aliquots were used and they were examined microscopically with a stero microscope.

The larger nematodes, i.e. *P. equorum* and *O. equi* were removed from the total mass of faeces.

Egg counts and faecal cultures were made very 7 days for the three weeks following treatment.

### RESULTS

Results of egg counts and worm recoveries are presented in Table 3. The following genera and species were recovered from faeces after treatment.

| *Species* | *No. of horses infected* |
|---|---|
| *P. equorum* | 6 |
| *O. equi* | 14 |
| *S. equinus* | 3 |
| *S. edentatus* | 1 |
| *S. vulgaris* | 5 |
| $L_4$ | 12 |
| Cyathostominae | 13 |
| *Cylicocyclus* | 14 |
| *Cyathostomum* | 14 |
| *Cylicostephanus* | 14 |
| *Cylicodontophorus* | 10 |
| *Poteriostomum* | 3 |
| *Triodontophorus* | 3 |
| *Craterostomum* | 1 |
| *P. vivipara* | 7 |
| *Oesophagodontus robustus* | 3 |
| *Gyalocephalus capitatus* | 1 |

Table 3: MODIFIED CRITICAL TEST IN HORSES. WORMS EXPELLED IN FAECES AFTER TREATMENT WITH A SINGLE DOSE OF FENBENDAZOLE AT 10 mg/kg LIVE MASS

| | Bruintjie | Breker | Brunet | Busuko | Calvyn | Ds Wit | Engela | Girlie | Hessie | Marissa | Mosjes | Nerine | Nettie | Pieter | Slapoor | Vossie | Vossies foal |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Worms expelled in faeces | | | | | | | | | | | | | | | | |
| *P. equorum* | 26 | 1 | 0 | 0 | 0 | 2 | 1 | 0 | 0 | 5 | 0 | 1 | 0 | 0 | 0 | 0 | 0 |
| *O. equi* | 381 | 15 | 1 023 | 0 | 0 | 2 353 | 2 376 | 4 813 | 5 | 501 | 3 | 0 | 1 761 | 5 | 498 | 11 | 37 |
| *S. equinus* | 0 | 0 | 10 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 18 | 0 | 0 | 11 | 0 |
| *S. edentatus* | 10 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| *S. vulgaris* | 135 | 0 | 0 | 10 | 43 | 10 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 10 | 0 | 0 |
| *Triodontophorus* | 0 | 0 | 0 | 0 | 0 | 273 | 2 161 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 13 | 0 | 0 |
| *Craterostomum* | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 210 | 0 | 0 | 0 | 0 | 0 | 0 |
| $L_4$ of *H. musca* | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 200 | 0 | 0 | 0 | 0 | 0 |
| *P. vivipara* | 0 | 0 | 100 | 98 | 100 | 0 | 0 | 100 | 200 | 100 | 8 | 0 | 0 | 0 | 0 | 0 | 0 |
| *O. robustus* | 0 | 0 | 0 | 0 | 0 | 0 | 234 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| $L_4$ Cyathostominae | 1 168 | 0 | 2 942 | 1 | 0 | 1 722 | 41 223 | 950 | 2 066 | 1 266 | 436 | 1 021 | 1 969 | 0 | 1 826 | 0 | 0 |
| Adult Cyathostominae | 37 882 | 0 | 31 780 | 92 | 141 | 127 997 | 28 161 | 47 556 | 67 667 | 219 847 | 40 603 | 12 484 | 25 679 | 72 543 | 0 | 25 269 | 79 478 | 0 |
| | Faecal worm egg counts on day of treatment (Day 0) and every 7 days thereafter. | | | | | | | | | | | | | | | | |
| Day 0 | 2 800 | 66 | 4 300 | 3 000 | 1 200 | 2 267 | 2 000 | 2 000 | 1 800 | 1 400 | 2 000 | 1 100 | 2 900 | 0 | 3 133 | 2 067 | 333 |
| Day +7 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Day +14 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Day +21 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |

The worms were expelled within 5 days of treatment and eggs of both *P.equorum* and strongyles were absent in all the horses for at least 3 weeks after treatment. All the faecal cultures were negative for infective larvae.

In addition to the favourable results in the previous trials, FBZ at 10mg/kg was highly affective against *P. equorum, Strongylus edentatus* and *Oesophagodontus robustus*. It is interesting to note that *O.equi* was present in faeces of 14 horses ranging in numbers from 5–4 813 and 4 940 *Gyalocephalus capitatus* were expelled in the faeces of one horse compared with a count of only 171 of this species in the faeces of a donkey.

## DISCUSSION

These trials confirmed the results of other workers on adult strongyles[1,2]. We were able to confirm results[2] on adult *H.majus* and *H.musca* but FBZ had no effect on adult or $L_4$ of *D. megastoma* nor $L_4$ of *H.majus*.

Most of the $L_4$ of Cyathostominae recovered at necropsy in our trials were present in the digested caecal and colonic wall and very few in the ingesta of these organs. Although critical anthelmintic tests were not a suitable method of assessing anthelmintic efficiency of either $L_3$ (third stage larvae) or $L_4$ of *Oesophagostomum columbianum* in sheep, because these larvae are digested in their passage down the intestinal tract[7], it would seem from the present results that this is not the case with $L_4$ of cyathostominae in the caecum or colon of donkeys. Possibly the enzymes of the caecum and colon capable of digesting these worms are absent in equines accounting for their presence in faeces in a relatively undamaged state.

Third stage larvae and $L_4$ of *O.columbianum* of sheep are either in the intestinal wall or $L_4$ in the lumen of the small intestine initially, before they migrate to the colon. Apparently enzyme action in the small intestine on dead $L_4$ of *O. columbianum* accounts for their absence in faeces after treatment.

We confirmed the observations of Duncan *et al.*[2] on $L_4$ of cyathostominae in the lumen and apparently showed that even at a dose of 10mg/kg FBZ was 19,2–88% effective against these larvae. This is an assumption only, because we had no method in a critical anthelmintic test of determining whether $L^4$ were present either in the ingesta or the caecal and colonic wall when the donkeys were treated with FBZ.

Subsequently, however, Malan[4] carried out a controlled anthelmintic test on donkeys. Most of the treated group were dosed with FBZ at 60mg/kg while others were given higher doses from 120mg/kg to a maximum of 800mg/kg. He was unable to confirm the highly favourable results of FBZ at 60mg/kg against $L_4$ in the intestinal wall[2] and even at higher doses the efficiency of FBZ varied.

In our trials we used aged donkeys while Duncan *et al.*[2] worked with 6–12 month old ponies. Fenbendazole may not be as efficient in old donkeys as it is in young ponies.

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ABSTRACT: B. Sutherland, 1980. **Physiological age determination in female Stomoxys calcitrans Linnaeus (Diptera: Muscidae).** *Onderstepoort Journal of Veterinary Research,* 47, 83–88 (1980).

The primary sex organs of female *Stomoxys calcitrans* consist of a single pair of ovaries, each containing 80–100 polytrophic ovarioles. A natural population of these females can be grouped according to age into different reproductive categories. The technique described defines these categories and enables one to distinguish between newly-emerged, nulliparous and uniparous females and females that thave reproduced twice (biparous) or more (pauciparous).

ABSTRACT: J.J. van Staden, H.N. van der Made & Eileen Jordan, 1980 **The Control of bacterial contamination in carcass meal with propionic acid.** *Onderstepoort Journal of Veterinary Research,* 47, 77–82 (1980).

The effect of the addition of 2,3,5,7 and 10% respectively of propionic acid on known numbers of bacteria added to or contained in carcass meal was examined. *Escherichia coli* was totally inhibited by 2% propionic acid, while 5% of the acid both inhibited *Salmonella typhimurium* and brought about a 74,7% reduction in the total aerobic bacterial count.

ABSTRACT: B.J.H. Barnard, S.B. Buys, J.H. du Preez, S.P. Greyling & H.J. Venter, 1980. **Turkey meningo-encephalitis in South Africa.** *Onderstepoort Journal of Veterinary Research,* 47, 89–94 (1980).

Turkey meningo-encephalitis is a neuroparalytic disease of turkeys first described and shown to be caused by a flavivirus in Israel. During 1978 a similar disease was observed in South Africa. In addition to the lesions described in Israel, myocarditis, regression of the ovary and egg peritonitis were constant findings. The similarity in host range, symptoms and pathological changes produced by the virus isolated locally and in Israel and the serological cross-reaction between the 2 virus isolates indicate that they are identical.

# *ANATRICHOSOMA* SP. INFESTATION IN THE FOOTPADS OF A CAT

A. LUCIA LANGE*, ANNA VERSTER†, S.R. VAN AMSTEL‡ R. DE LA REY§

ABSTRACT: Lange A.L.; Verster A.; Van Amstel S.R.; De la Rey R. **Anatrichosoma sp. infestation in the footpads of a cat.** *Journal of the South African Veterinary Association.* (1980) **51** No. 4 227–229 (En) Department of Pathology, Faculty of Veterinary Science, University of Pretoria, Box 12580, 0110 Onderstepoort, Republic of South Africa.

A nematode of the genus *Anatrichosoma* caused extensive necrosis and sloughing of the footpads of a domestic cat.

No male worms were found but a description of the female is given. This is the first report of *Anatrichosoma* sp. in the Republic of S.A. as well as the first report of it in a cat.

## INTRODUCTION

In 1922 Swift, Boots & Miller described a nematode which caused blisters on the palms of the hands and the soles of the feet of rhesus monkeys, *Mucaca mulatta*[7]. This nematode was provisionally named *Trichosoma cutaneum*, but subsequently Chitwood & Smith reclassified it as *Anatrichosoma cutaneum*[2]. *A. cutaneum* and other *Anatrichosoma* spp. have been found to parasitise the nasal cavity of the rhesus and other monkeys[1 3 6]. Parasites similar to these have been found in man where they caused cutaneous larval migrans (creeping eruption)[4 5].

This is the first report of *Anatrichosoma* in the Republic of South Africa as well as the first report of it in a cat.

## HISTORY

Late in January 1980 the owners of a 13 year old male domestic cat noticed that the cat was lame in one leg. On investigation it was found that some of the footpads of the affected limb were raw. The owners concluded that the cat had jumped onto a hot plate of a stove and had burned itself. After 2 days of unsuccessful treatment for burns by the owners, the cat was taken to a veterinary surgeon. The cat was treated for about 2 weeks for a suspected fungal infection, but no improvement was evident; in fact, the other feet started showing similar lesions. He was then referred to the Department of Medicine, Faculty of Veterinary Science at Onderstepoort.

On arrival at the Department of Medicine the cat appeared listless and depressed. He was disinclined to move due to complete or partial sloughing of the epidermis of the footpads of all 4 feet. The lesions closely resembled burn wounds with necrosis of the epidermis and exudation which caused caking of the hair on the feet and between the toes. Where there was total sloughing of the epidermis of a particular footpad the underlying tissue appeared soft and red. The dorsal surface of both carpal joints had pressure sores characterised by alopecia, scab formation and a thickened skin. It was presumed that the cat had "walked" on these joints due to the feet lesions. He was severely dehydrated and slightly anaemic. On palpation of the abdomen it was found that both kidneys were small and hard, and that their surfaces were irregular. The blood urea nitrogen value was 192 mg% and a diagnosis of chronic interstitial nephritis was made. Symptomatic treatment was instituted but as the prognosis was considered to be poor, he was killed by the administration of an overdosage of a barbiturate, after consultation with the owners.

The owners of the cat live in Brits, Transvaal, and their property borders on the Magaliesberg range. A number of baboons (*Papio ursinus*) and vervet or African green monkeys (*Cercopithecus aethiops*) live on the slopes of the mountain and often come down near the house to feed. The cat frequently went hunting on the mountain.

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## POST MORTEM EXAMINATION

The animal was dehydrated, slightly anaemic and in a poor condition. The most striking lesions were those associated with the feet. All 5 pads of the left front paw had no epidermis. The centre pad of the right front paw showed almost complete detachment of the epithelium; only a small area of the epidermis being still attached. The detached part was dry, discoloured and hard. Both hind feet showed similar lesions of the centre pads and the lateral toe pads. Pressure sores as described in the history were found on the dorsal carpal surfaces.

The only other significant lesions were in the kidneys. These were shrunken and fibrotic with an irregular surface. A small number of healed infarcts (2mm diameter) were seen in both kidneys.

Specimens of the feet and kidneys were fixed in 10% buffered formalin for histopathological examination. In addition, some of the footpads were frozen for later investigation.

### Stereo-microscopic examination

Sloughed portions of formalin-fixed and frozen footpads were examined. The inner surface of the epidermis contained long, slender, slightly coiled worms projecting from the surface, as well as brown structures arranged in a zig-zag fashion, which were embedded in the tissue. These structures were tubes filled with brown eggs. One complete female worm and fragments of 3 others were dissected from the fixed and frozen tissue. These were identified as belonging to the genus *Anatrichosoma* (vide infra).

### Histopathological examination

Examination of haematoxylin and eosin stained tissue sections revealed the presence of several worms in the epidermal layers of the footpads (Fig. 1) and numerous migratory tracts in which there was complete destruc-


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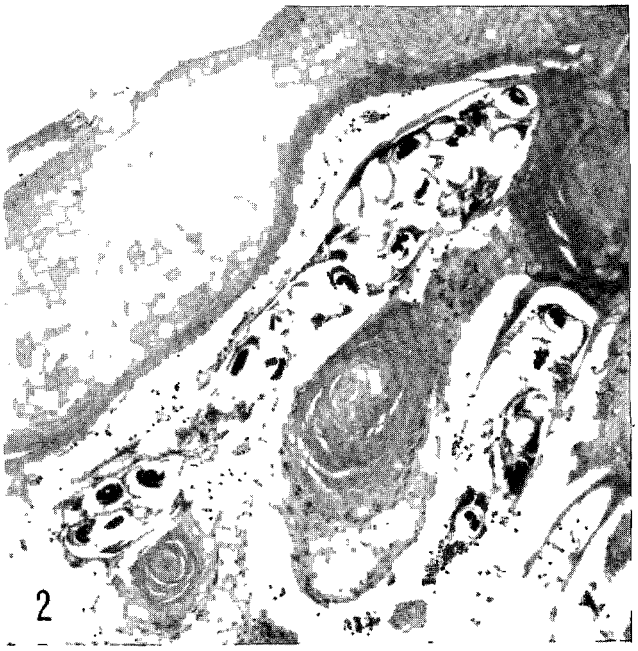




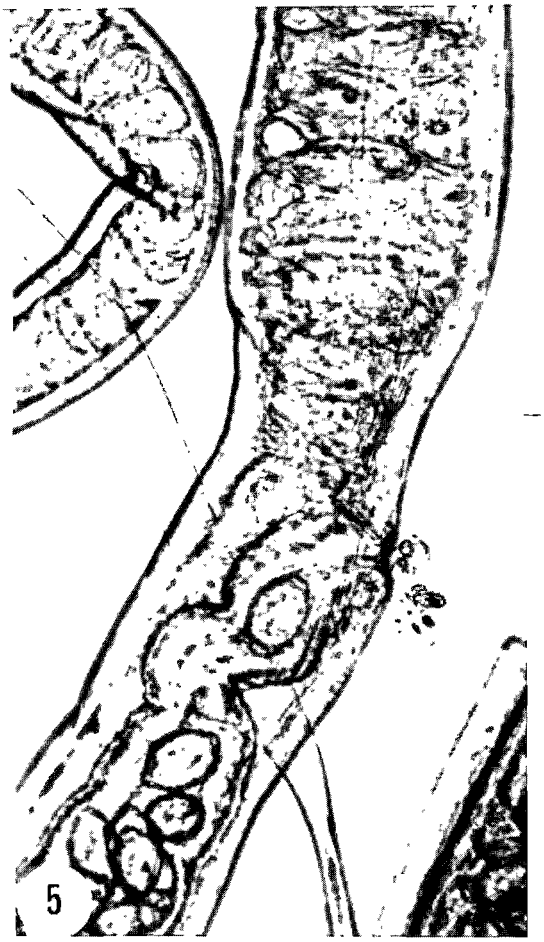



**Fig. 1.** Transverse sections of a worm in the epidermal layer of the skin of the footpad HE X128

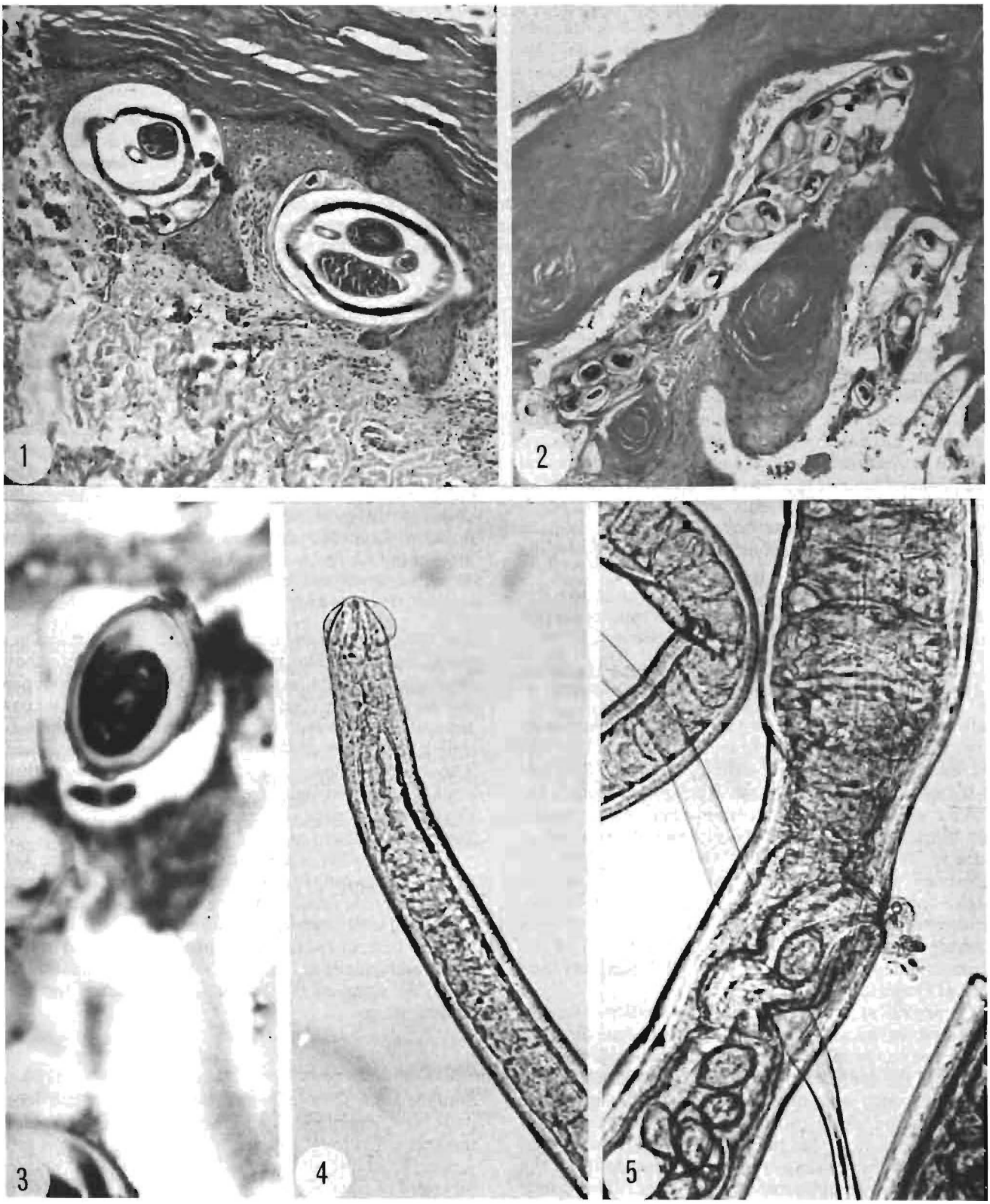
**Fig. 2.** Eggs deposited by migrating female worm. The eggs are within a tubelike structure the material of which comprises a bluish-purple granular substance HE X135

**Fig. 3.** Bioperculate egg with a thick brown capsule and coiled larva inside. HE X534

**Fig. 4.** Cephalic inflation X33

**Fig. 5.** Post-oesophageal swelling anterior to the vulva X32

tion of the epidermis. In these areas the keratin layer had detached from the rest of the skin. In the spaces created by the migrating worms were depositions of eggs surrounded by a bluish-purple granular substance (Fig. 2). The eggs were bioperculate, had thick brown capsules and contained coiled larvae (Fig. 3). The tissue surrounding the worms did not show any inflammatory changes. The dermis just below the migratory

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tion of the epidermis. In these areas the keratin layer had detached from the rest of the skin. In the spaces created by the migrating worms were depositions of eggs surrounded by a bluish-purple granular substance (Fig. 2). The eggs were bioperculate, had thick brown capsules and contained coiled larvae (Fig. 3). The tissue surrounding the worms did not show any inflammatory changes. The dermis just below the migratory

tracts showed oedema and infiltration of a few neutrophils but no worms were encountered here.

The diagnosis of chronic interstitial nephritis was confirmed histologically.

## DESCRIPTION OF THE WORM

The total length of the complete female is 42, 26 mm; the length from the anterior end to the end of the stichosome is 5,81 mm. There is a cephalic inflation, 44–57 μm wide while the width of the worm posterior to the inflation is 35–44 μm (Fig. 4). The nerve ring is situated 60 μm from the anterior end. There are from 130–142 stichocytes. The width at the post-oesophageal swelling is 192–198 μm, at the constriction 132–148 μm and posterior to the constriction 162–176 μm (Fig. 5). The vulva is situated approximately 70–80 μm posterior to the oesophageal constriction; in the complete female this is 5,87 mm from the anterior end. The uterus is filled with bioperculate brown eggs each of which contains a larva and measures 63–72 μm by 35–44 μm.

## DISCUSSION

Although *Anatrichosoma* sp. have not as yet been recorded in South African vervet monkeys and baboons, or in the human population, the most likely surce of infestation of the cat was these primates as its hunting habits covered an area inhabited by these animals. In a survey conducted to determine the incidence of infestation of *Anatrichosoma* in several species of African monkeys kept in research centres in the United States of America, it was found that 29% of 198 monkeys were hosts to the parasite. The five species of monkeys involved were *Erythrocebus patas* (red patas), *Cercopithecus talapoin* (talapoin), *C. aethiops, Cercocebus galeritus* (mangabey) and *Papio* sp. (baboon)[6]. Unfortunately the species of *Anatrichosoma* involved were not described, although they appeared to differ from *A. cutaneum* and *A. cynamolgi*[6]. *A. cynamolgi, A. rhina* and *A. nacepobi* all parasitise the nasal mucosa of the rhesus monkey[2,3] and they too differ from the *Anatrichosoma* sp. found in the footpads of the cat described in this paper. The worms recovered from the cat cannot be assigned to a known species of *Anatrichosoma* as males were not recovered. It does seem, however, that the feline worm is a different species from the 4 known species, as the total length of the intact female worm is 42,26 mm; this is much greater than the total length of the females of the 4 known species viz. *A. cutaneum* 22–24 mm[7]; *A. cynamolgi* 17,8–19,6 mm[2]; *A. rhina* 18,03–27,04 mm[3]; *A. nacepobi* 16,86–19,08 mm[3].

The severity of the lesions in the cat leads one to consider this as an example of cutaneous larval migrans in an abnormal host. The lesions caused by *A. cutaneum* in the skin of rhesus monkeys were characterised by blister formation due to the females being present in the epithelium of the skin. The inflammatory reaction consisted of oedema, neutrophil and eosinophil infiltration and later giant cell and fibroblast reaction[7]. In the nasal cavity of monkeys infested with this nematode the lesion is characterised by firm grey-white plaques on the mucous membranes. The parasites burrow in the epithelium immediately above the subepithelium and cause extensive damage to this layer. There is also a marked hyperplasia and parakeratosis of the epithelium overlying the worms[1].

In the cat there was extensive sloughing of the epidermis of the footpads. It appeared as if all 4 feet were simultaneously infested and that sloughing coincided with the maturation of the worms and their migration in the epidermis.

Capillarid nematodes have also been recorded from man in Vietnam and Japan. In these instances the lesions caused by the parasite was typical of creeping eruption due to aberrant migration of nematodes in the skin[4,5].

## ACKNOWLEDGEMENTS

We wish to thank the technical staff of the Department of Pathology for their assistance, as well as Mr. R. Watermeyer for preparation of the photographs, Prof. R.C. Tustin for reading the manuscript and Mrs. V. Käber for the typing thereof.

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# SODIUM HYALURONATE IN EQUINE TRAUMATIC ARTHRITIS

D.H.G. IRWIN

ABSTRACT: Irwin, D.H.G. **Sodium hyaluronate in equine traumatic arthritis.** *Journal of the South African Veterinary Association* (1980) **50** No 4 231–233 (En) Newmarket Equine Clinic, P.O. Box 4107, 1451 Alrode, Transvaal, Republic of South Africa.

Thirty seven intra-articular injections, each 2 ml sodium hyaluronate were made into 32 arthritic joints in 15 racing Thoroughbreds and one part-bred polo pony. One joint failed to improve at all, 16 became sound with one injection, 4 became sound with 2 injections, and 11 joints showed various degrees of response. Eleven of 14 horses became sound for flat racing. Discussion relates to the technique of using sodium hyaluronate, some clinical and pathological aspects of traumatic arthritis, joint lubrication and a brief review of some properties of sodium hyaluronate which have a bearing on arthritis.

## INTRODUCTION

Arthritis of the fetlock and carpal joints is a serious cause of economic loss by precluding maximum athletic performance, by enforced idleness and by early retirement of racing thoroughbreds in the Transvaal. These two disease entities are second only to the hoof problems (which arise mainly from improper hoof paring and shoeing) as a cause of lameness in race horses here. Therefore, when in 1975 the author learned of work done by Balazs, communication between us led finally to sodium hyaluronate (SH) being made available for trials in South Africa*, which started in 1978.

## MATERIAL AND METHODS

The SH used was Healon R Vet.† The patients were horses presented for treatment in the course of private practice. Radiographs were made to exclude horses with chip fractures of the carpus, but not all fetlocks were radiographed. Table 1 gives a summary of the patients, and Column 6 shows in which horses intra articular anaesthesia was used to make sure the lameness originated in the joint, as described by Asheim & Lindblad (1976). One consideration included in this brief trial was conformation. Column 7 of Table 1 describes the conformation of the affected joint/s, where a simple classification of good, fair and poor was chosen, based on standard zootechnological criteria. Case 16 was a head strong and difficult horse who required draining of a deep abscess, extensive dental care and deworming besides SH injection. All four procedures were done at one session under general anaesthetic. All other SH injections were done standing, with a tranquilizer in jittery subjects.

## RESULTS

The results obtained are listed in Table 1, and are highly acceptable to us. Even Case 1 was more rewarding that the bare statistics reveal, in that this horse won a race in the B division before relapsing to a milder lameness. Case 9 was not subjected to local analgesic intra-articular block because he was to race in a few days, and he won in the top division. It is possible that Case 14 (a polo pony) suffered an undisplaced carpal bone fracture which we failed to demonstrate radiologically. In all it appears that in private clinical practice SH is a useful treatment for traumatic arthritis in racing thoroughbreds. The results show relief of signs and a curative effect. The number of cases is too small to draw conclusions, but inspection of the results shows a tendency for results to be superior in horses with good or fair conformation to results in poorly conformed horses.

In practice the decision to employ SH depends upon the balance of economic forces: price of the drug and its administration, cost of spelling or remaining idle on a training yard and stake earnings potential besides capital outlay and the realistically good chance of recovery. Certainly it is one of the great advances made in equine practice in recent years.

## DISCUSSION

Intra-articular injection into the carpal joints seldom presents technical difficulty, but hypertrophy of the synovial membrane in the fetlock has proved embarrassing, and it is recommended that if slick intra-articular access is not confidently anticipated, a wheal of local analgesic should be placed over the puncture site.

Although evaluation of conformation is highly empirical and subjective it is never-the-less of the greatest importance because the trial of speed, hardness of going (underfoot) and conformation are indivisible in the aetiology of traumatic arthritis, to which of course is added immaturity in two and three year old horses.

The question of injecting local analgaesic into a joint to be sure of the diagnosis of arthritis, and only arthritis, followed by SH if indicated needs amplification. Asheim & Lindblad are not clear as to whether they injected SH into recently (less than 30 min) blocked joints, or if the SH was given a day or more later. We have used both systems, but unfortunately did not record the intervals.

A small percentage (possibly 8–10%) of horses show local inflammation at the site of local analgesic injections when nerves and joints are blocked. One of our patients (not recorded in the results table, but which responded to SH, is accident prone and often goes lame from a variety of causes). He is a big chestnut colt and when local analgesic is injected he sometimes presents an embarrassing reaction. Recently the trainer allowed me to inject one ml of two different local analgaesics at two sites on the side of the horses neck. He showed no reaction to mepivacaine hydrochloride‡ but from Hour 6 to Hour 30 he showed a distinct inflammatory reaction to lignocaine§.

*by courtesy of Intal Ethical Promotions.
†Pharmacia AB Uppsala, Sweden.
‡Carbocaine (2%) – Registered Trade Mark.
§Centaur Labs (Pty) Ltd. (2%).


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Table 1: SUMMARY OF PATIENTS TREATED BY INTRA-ARTICULAR SODIUM HYALURONATE

| *Case* | *Age* | *Colour* | *Sex* | *Joints* | *Blocked out* | *Confor-mation* | *Days Sore Pre Inj.* | *Effect at at 10 days* | *Effect at 90 days* |
|---|---|---|---|---|---|---|---|---|---|
| 1 | 4 | Ch | g | lf. rf. | No | Good | >150 | sound | Relapse[1] |
| 2 | 2 | B | f | lf. rf. | Yes | Fair | >60 | sound | sound |
| 3 | 5 | Dkb | g | lf. lfrc. | Yes | Fair | >150 | impr. | sound |
| 4 | 3 | B | g | rfrc. lrc. | Yes | Poor | >200 | impr. | poor |
| 5 | 2 | B | g | rrc. lfrc. | Yes | Fair | >90 | impr. | sound |
| 6 | 3 | B | g | rfrc. lic. | No | Fair | 60 | impr. | fair[2] |
| 7 | 3 | B | c | ric. | Yes | Poor | 45 | impr. | poor |
| 8 | 3 | B | g | lf. rf. | Yes | Fair | >90 | impr. | sound |
| 9 | 5 | B | g | lf. | No | Good | 3 | sound | sound |
| 10 | 3 | B | c | rf. | No | Fair | 21 | sound | sound |
| 11 | 3 | Ch | c | lrc. lrcic. | Yes | Good | 30 | sound | sound |
| 12 | 4 | B | g | rrcic. | Yes | Fair | >180 | impr. | fair |
| 13 | 2 | B | f | rtt. | No | Good | 7 | impr. | sound |
| 14 | 9 | B | f | lrcic. | Yes | Fair | 10 | Nil | v. poor[3] |
| 15 | 3 | Ch | c | rrcic. | Yes | Good | 21 | sound | sound |
| 16 | 5 | Br | g | lic. | No | Good | >150 | sound | sound |

NOTES: No hind fetlocks treated
l = left (near)
r = right (off)
f = fetlock
rc = radio-carpal
ic = intercarpal
tt = tibio-tarsal
1 = retreated: sound
2 = fetlocks cured, knees relapsed
3 = arthroscopy or arthrotomy indicated

## TRAUMATIC ARTHRITIS

The sequence of events in a horse's fetlock joint exposed to working trauma is briefly as follows. Clinically the horse becomes lame (if one fetlock is affected) or shortens his stride ("goes pottery") if both fetlocks are affected. The fetlock is warmer than normal, the joint capsule is distended, forced flexion causes pain and the metacarpal pulse is increased. Aspiration of synovia reveals a thin watery fluid which is often blood-stained in acute episodes, and which is rich in lymphocytes. These cells are harmful in that they tend to over-react to the insults visited upon the working joint: when they change into their various definitive cell types they liberate lytic enzymes, thin the synovia and lead to restriction of joint mobility (Nilsson & Persson 1973).

The pathological changes set in motion have a self-perpetuating tendency even without further work load. More work at this time speeds up the disease process. Pain causing lameness is closely correlated to synovial tissue changes such as hyperaemia, oedema and thickening of the fibrous capsule (Nilsson 1973) but, in contrast, joint cartilage lesions such as pannus and marginal erosions show little correlation to pain and lameness.

The lymphocytes present in the abnormal synovial fluid have several options including a change to fibroblasts which are then free to migrate to nearby solid tissue, and once on terra firma, are able to multiply and, as is their nature, to lay down fibrous strands. The resulting proliferation of the synovial membrane and the deep folds created are the cause of difficulty in placing a needle into the joint space and are most easily visualized by those veterinary surgeons with experience of open joint surgery. The joint suffers reduced mobility from the normal healthy state where forced flexion gives 270° MC III–P1. Other lymphocyte descendents, through migration and performance of their "defensive roles", cause the hyaluronate concentration in the synovial fluid to decrease (Nilsson & Persson 1973).

These changes are also seen in men suffering from arthritis of the great bearing joints – the knee and hip (Balazs 1974), and again it is considered that a central, if not the essential ingredient of the pathology is a reduction in the viscosity of the synovial fluid which accompanies reduction in the molecular weight of the biopolymer, hyaluronate.

## JOINT LUBRICATION AND HYALURONATE

In 1743 Hunter described the lubrication aspects of synovial fluid and in 1934 Meyer & Palmer isolated a muco polysaccharide from the eyes of cattle, which is now called hyaluronate, and is responsible for the high viscosity of synovia (Meyer et al 1939). Hyaluronate is widely distributed in the animal kingdom and is the same basic molecular configuration in all species from bacteria to man. The molecule is a linear biopolymer of a recurring disaccharide unit, containing one molecule of N-acetylglucosamide and one molecule of sodium glucuronate linked by a glycosidic bond, yielding a final molecular weight of $1 - 4 \times 10^6$ the larger of which are of the magnitude of a bacterium.

In vitro studies have shown that hyaluronate exerts inhibition of cell mobility, leucocyte chemotaxis, leucocyte phagocytosis, specific lymphocyte stimulation and virus infectivity. The material exerts a stimulating effect upon hyaluronate synthesis and a sealing effect upon the joint permeability barrier and, finally, it binds specifically to cartilage cells (Balazs et al 1967b).

Hyaluronate possesses a viscoelastic property of "shock absorbtion" by being able to allow half the delivered mechanical energy to be dissipated as heat whilst the other half is stored as elastic energy which is rapidly uncoupled by intra-molecular gyrations (Balazs 1974a). However, Radin (1972) argues that this effect is small because the film is thin.

Hyaluronate is not the major cartilage-on-cartilage lubricant (Wilkens 1968, and Radin et al 1970), but

possibly protein complexes of glyco-proteins are. However, the soft tissue of joints undergo great frictional stress and it is specifically hyaluronate which lubricates the ligaments and synovial membranes. (Radin et al 1971).

Intra-articular injection of cortcoids has long been used as an anti-inflammatory for arthritis in horses, (Rooney 1972) but untoward effects are serious (O'Conner 1968). A combination of corticoids and SH showed greater benefits than corticoids alone (Rydell *et al* 1970).

The next step was to use SH alone for traumatic arthritis in horses: the results were very good in that symptomatic relief occurred and a strong curative effect was observed (Asheim & Lindblad 1976), and may be attributed, in summary, to the following actions:

1. Reduction of inflow and inhibition of aggressive leucocytes.
2. Improved soft tissue lubrication.
3. Specific binding of SH to cartilage allowing cartilage rehydration (Wigren et al 1975) and preservation of its surface.
4. A biochemical effect of synovial cell stimulation to synthesize hyaluronate (Balazs & Darzynkiewicz 1973), which could be the key to explaining the curative effects.
5. Immunological effects have not been reported and vigorous attempts to induce them have failed (Richter 1974).

## ACKNOWLEDGEMENTS

Thanks are due to Dr. E.A. Balazs, Dr. J. Helfet, S.L. Miller and the late J. Bloom, for help and encouragement also Dr. D.W. Howell and Dr. D.R. Hoey who treated some of the cases. Mrs. B.J. Tillett and Miss T. Maloon kindly did the typing.

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# EQUINE PYELONEPHRITIS AND UNILATERAL NEPHRECTOMY

D.H.G. IRWIN and D.W. HOWELL

ABSTRACT: D.H.G. Irwin; D.W. Howell; **Equine pyelonephritis and unilateral nephrectomy.** *Journal of the South African Veterinary Association* (1980) **51** No. 4 235–236 (En) Box 4107, 1451 Alrode, Rep. of South Africa.

The clinical and laboratory findings of illness in a 2-year-old Thoroughbred filly are described. The treatment employed, including unilateral nephrectomy, and the macro- and microscopic findings in the diseased kidney are presented and this rare case is discussed briefly.

## INTRODUCTION

Disease of the kidney in adult horses is rare and is, in practical terms, mainly limited to acute renal shut down in severe cases of paralytic myoglobinurea amongst Thoroughbreds in racing stables.

## CASE HISTORY

On 26 November 1979 a 2-year-old bay Thoroughbred filly in training performed well on the track and the trainer was about to nominate her for a race. In the evening she was off-colour and clinical examination revealed a pyrexia of 40,4°C, pulse rate 56/min., harsh respiratory sounds with a respiratory rate of 30/min. On rectal examination an enlarged spleen and mucous covered faecal pellets were found. Laboratory findings were: packed cell volume (PCV) 0,49; red cell count (RCC) 12,4 × $10^{12}/\ell$, white cell count (WCC) 5,7 × $10^{9}/\ell$; blood slides did not reveal babesia parasites. Treatment administered was: 1,5 $\ell$ technical oil by tube; 20m$\ell$ of a 4% trimethoprim with 20% sulfadoxine combination* i.v.; 20m$\ell$ of a pyrazoline/pyrazolidine combination¶ i.v.

Next day her habitus was improved, body temperature 38°C, pulse and respiratory rates were slower. Late in the morning a serosanguinous nasal discharge started, first from one nostril, later from both nostrils and worsened in the afternoon. Laboratory findings were: PCV 0,41, RCC 10,6, WCC rose to 7,8. Blood slides showed the presence of *Babesia equi* (parasitaemia less than 5%). Treatment: trimethoprim/sulphadoxine combination 20ml i.v., 2g oxytetracycline† i.m.

On 28 November the lung sounds, temperature, pulse and respiration were normal. The filly was anorexic and restless. Droppings were small and firm, but rectal examination showed no other abnormalities. Laboratory findings were: PCV 0,38, RCC 9,0 and WCC 11,1. Treatments: trimethoprim/sulphadoxine combination 20 ml i.v., 2g oxytetracycline i.m., 2g ascorbic acid‡ i.m.; magnesium oxide 50g, magnesium sulphate 30g, sodium bicarbonate 30g all by tube.

On 29 November the filly had a body temperature of 38,5°C. Her appetite was minimal and she moved stiffly so as to keep head, neck and body in one straight line. Blood slides were negative for Babesia. Treatment: oxytetracycline 2g.

On 30 November she had a normal habitus but was not feeding, her body temperature was 39,5°C, lungs clear, pulse rising again to 48/min. Laboratory: slides were negative for Babesia although her PCV had fallen to 0,32. Treatment administered was oxytetracycline 2g i.m., 2g ampicillin§ i.m. and 2g ascorbic acid i.m. She was tubed with laxative salts, a vitamin tonic 16cc**, appetite stimulant 40g†† and a strychnine tonic (13,5mg strychnine hydrochloride).

From 1 December to 3 December the clinical picture remained static, but fever reduced to normal and ampicillin was continued daily. Appropriate supportive therapy (vitamins and aperients) was supplied.

On 4 December the filly became listless. The body temperature and pulse rate rose. Rectal examination revealed a tender, non-fluctuating mass in the left sublumbar region, estimated to be 20 × 15 × 10cm. Laboratory findings: the WCC was 17,8. RCC = 8,8.

On 5 December the filly was still dull. She had eaten practically nothing for 8 days but there was no sign of abdominal pain. Droppings were scanty. A pyrexia of 38,7°C. The abdominal mass was estimated at 30 × 20 × 10cm. The WCC had risen to 18,4 and the RCC had fallen to 7,6. Treatments: ampicillin 2g was again injected and 100g sterculia BP 62% and frangula 8%‡‡ was given by tube.

On 6 December the general condition of the filly was deteriorating. The filly was admitted to the clinic for exploratory laparotomy with a presumptive diagnosis of sublumbar abscess.

The filly was prepared for a standing laparotomy. A 15cm incision allowed direct palpation of the mass. Manipulation and gentle finger tip dissection revealed barely recognisable kidney formation. In spite of the grave prognosis, general anaesthesia was induced after taking the necessary steps to retain aseptic technique. 50g of glycerol guiacolate and 1600mg of sodium pentobarbitone, dissolved in 1 litre of water, were given to effect. This was followed by endotracheal intubation for the administration of halothane§§. The incision was enlarged to 28cm. Loops of bowel, partly filled with gas, offered to protrude from the wound. These were deflated by 20g needle puncture and a suction pump. The dissection revealed a greatly enlarged kidney which was removed after tying an appropriate pedicle. The wound was closed by standard surgical technique. The filly rose uneventfully and started eating immediately upon return to her stable. A supporting bandage was applied. The wound healed per primum and the filly has made uneventful recovery. At the time of writing (6 April 1980) she is in hard training again.

*Trivetrin®, Wellcome.
¶Isopyrin & Phebuzine as Tomanol®, Byk Gulden.
†Liquamycin 100®, Pfizer.
‡Ascorbic acid 20% m.v. Saphar.

§Penbritin® Vetlab Chembro.
**BVMO® Burns-Biotec
††Penstim, Vetlab.
‡‡Normacol, Norgine
§§Fluothane


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## PATHOLOGY

The kidney was enlarged with a mass of 1267g. The notch of the renal hilus was less distinct than in a normal kidney but no further macroscopic findings were noted. The histopathologist's report read: "Examination of several sections show a process of severe acute pyelonephritis. The distal and proximal tubules show distention by a mass of acute inflammatory cells. Some chronic changes can be seen in periglomerular thickening of the cortical glomeruli and also slight vascular changes. There is no evidence of neoplasia and no specific aetiological factor can be identified. Diagnosis: acute-on-chronic pyelonephritis."

## DISCUSSION

The findings related above suggest that the primary pathogen was a virus affecting the respiratory tract. The lowered resistance allowed a premunity against *B. equi* to be unbalanced causing biliary fever. (In Transvaal it is not uncommon to find biliary fever in horses as a complication of other stressful conditions.) The anaemia must be attributed, in part, to the biliary fever and partly to the toxic state resulting from the lung condition, the constipation and the pyelonephritis. The WCC rise was presumed to be due to the pyelonephritis. The constipation was attributed to the pyrexia and poor appetite. It appears that the ampicillin contained the kidney infection for 36–48 hours as shown by a reduction in the fever, whereupon the fever again rose on 4 December. Kidney tissue was not submitted for culture because the original aetiological organism would have been masked by the wide variety of antibacterials. The glaring omission is the lack of a urine examination which, had it been done on the 3 or 4 December, may well have lead to the opinion of a hopeless prognosis and euthanasia – because a unilateral pyelonephritis would probably not have been seriously considered. The "average" weight of the left kidney is 700g (Sisson), the enlargement representing an increase by a factor of about 1,7. We have weighed the left kidney from a filly of equivalent mass and found it to weigh 477g.

## ACKNOWLEDGEMENTS

Dr P van Drimmelin kindly conducted the histopathological examination and the trainer Mr C Otto is thanked for his confidence and co-operation.

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## BOOK REVIEW — BOEKRESENSIE

### GENETICS FOR DOG BREEDERS

W.H. FREEMAN, FREDERICK B. HUTT and Company Limited, 20 Beaumont Street, Oxford OX1 2NQ ISBN 0 7167 1069 – 2 board; 245 pages, 53 illustra., Price £7,80 board

Veterinary students have sometimes difficulties in learning genetic principles and they usually do not like fruit flies or chickens for the understanding of the basics of breeding and selections.

Frederick B. Hutt provided the solution to these study problems by writing the exciting book "Genetics for Dog Breeders" which should and will be of interest to every veterinary student and qualified veterinarian.

Many dog breeders also have a burning desire to know more about genetics than the 3:1 ratios they had learned in high school. Again, this book is filling an existing gap and will be of great help to the specialist breeder or also to the dog lover who wants to know more about his animals. Frederick B. Hutt did it again: After his successes with the books "Genetic Resistance to Disease in Domestic Animals" and "Animal Genetics" the present book will certainly be a best seller.

The book is divided into three parts – Principles of Genetics, Genetic Variations in Dogs and Breeds and Breeding. In the first part the principles of Mendelian inheritance, the genetic background of chromosomes and genes and the inheritance of quantitative characters is outlined. Part II concentrates on the genetic variations with special reference to abnormalities and defects of the skeleton and joints, coats and colours, nerves and muscles, blood and cardiovascular abnormalities, eyes and ears and other hereditary abnormalities. Part III gives a bit of breeding philosophy and some suggestions for dog breeders. The most interesting chapters are those on "Breeds and Behaviour" and "Selection and Breeding".

In a glossary the definitions of the most important genetic terms are presented.

In all, a very useful book to veterinarians, not only veterinary students but to all students in the biological field and to all dog breeders and lovers.

D.R. Osterhoff

# ETHOLOGICAL STUDIES WITHIN THE MAN-HORSE RELATIONSHIP*

H.P. VAN NIEKERK

ABSTRACT: H.P. Van Niekerk **Ethological studies within the man-horse relationship.** *Journal of the South African Veterinary Association* (1980) **51** No. 4 237–238 (En) Dept. of Zootechnology, Fac. Vet. Science, Univ. Pretoria, Box 12580, 0110 Onderstepoort, Rep. of South Africa.

Certain aspects of ethology and the horse's senses are discussed to bring about a better understanding between man and horse. Furthermore the behaviour of horses with respect to housing, feeding, breeding, veterinary treatment and work are considered.

## INTRODUCTION

Ethology is an abstract science. In this subject we can only take certain given elements and work hypotheses from them. With good observation, logical insight and a sound knowledge of ethology, we can understand how the horse's mind works, especially within the context of the close man-horse relationship. The delicate emotional stability of the horse determines its usefulness to man. Equine ethology is the study of the horse's reactions to given stimuli.

### Senses

The only difference between the senses of man and those of the horse lies in their function. Appreciation of this is essential if one is to understand the horse's reactions.

#### *Vision*

It is generally accepted that horses are colour-blind; for them colour exists only as a mixture of various intensities of black, grey and white. They are unable to distinguish detail at a distance; also, because of the anatomical position of the eyes, horses see two separate images which may or may not overlap in the centre. Peripheral vision is good. The size of the blind spot in front of the nose and the blind area behind him is determined by the level at which the head is carried. The greatest peripheral vision, and smallest blind area to the rear is obtained with the head in grazing position. This is to the advantage of an animal constantly on the alert for predators[1,5].

Movement, readily appreciated, together with smell, helps the horse to decide whether or not an object poses a threat or not.

The factors which make the horse's vision ideal for its natural state are not always suitable for man. A horse may shy at a piece of paper because of the movement, not because the object itself is threatening. Any strange object is a potential source of danger to an animal whose main form of defence is flight.

Night vision is better than man's, but horses' eyes adapt more slowly to changes in light intensity. This explains why horses are unwilling to walk into a dark stable or horsebox. To do so is tantamount to stepping into a dark hole in the air. The same applies to walking a horse over a dark surface. The problem regarding horseboxes is usually overcome by opening the front door.

The horse's poor (by human standards) vision is compensated for by well developed senses of hearing and smell. Horses familiarize themselves with strange objects by smelling them.

Strangers greet each other by touching muzzles, and so avoid a possible kick or bite. If they are suspicious of each other, they circle, with only the muzzles touching. Should they accept each other, smelling of other parts of the body is allowed and this may end in mutual grooming, and indication that a bond of friendship has formed. A stallion can scent a mare in oestrus over a large distance. The stallion will greet such a mare with a very specific vocalization. The well-known homing instinct is also apparently dependant on smell[1].

#### *Smell and Taste*

Smell and taste go hand in hand. Man often has difficulty in determining what is acceptable to the horse because of marked individual differences. It can be frustrating when the horse objects to something perfectly innocuous, for example an apparently tasteless anthelmintic mixed with the feed. Some horses may reject some feed types on the smell alone, others may dislike the taste.

#### *Audition*

The sense of hearing is better developed than that of man. A horse is able to pinpoint the source of a sound quite accurately by moving his ears. Ears and nose are used together to find out about something that has excited curiosity.

#### *Touch*

The sense of touch is well developed. Very sensitive areas include the withers, the mouth, the flank and elbow regions. Some horses dislike their ears, eyes and bulbs of the heels being touched. One can win a horse's friendship by stroking the withers and back. Most horses enjoy being groomed but some are very ticklish on the very sensitive areas. The tactile hairs around the mouth, lips or muzzle act as fingertips. Together with lips they serve to gather information, to calm foals and to form friendships. Humans put the sensitive mouth to good use as a means of control, but misuse it in the form of hard hands and sharp bit that can lead to a great deal of discomfort. The sense of touch is also used in training – horses tend to move away from pain. For instance, pressure on the ribs will make a horse move forward. The same principle is responsible for undesirable reactions. An ill fitting bit may be responsible for a horse running away or jumping (a natural behaviour pattern). Any frightened horse will run away, or try to fight.

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Often, an apparently stupid action is quite natural, and the only course open to the horse at that time. On the other hand, a trained horse does many things that it would not do in the natural course of events.

### Behaviour

Most behaviour patterns are fixed after birth. Behaviour is a study of cause and effect, cause being what happens, and the effect being the reaction. Many new things happen to the foal after his birth. His immediate source of information is his dam; then in the natural state the other members of the herd and then there is his general environment which may include strange creatures such as man, with his demands.

The newborn foal's immediate requirement is food, obtained by smell and a process of trying. Other behaviour patterns are obtained by imitation – allelomimetic behaviour. These include grazing habits, recognition of danger and imitating sexual behaviour in play.

All behaviour studies are of a basic stimulus and reaction. There are two categories of reflexes, conditioned and unconditioned. Training involves conditioned reflexes, while ethology, the study of animals under natural conditions, is concerned with the unconditioned reflex. Each behaviour pattern is a series of acquired responses. These are divided into three stages viz. stimulus, reaction and reinforcement. The reinforcement is that which modifies the reaction to enable the animal to adapt to certain circumstances. Negative reinforcement occurs when the desired reaction is associated with the removal of pain or discomfort. Positive reinforcement may be a pleasant sensation of a continuation of the present condition.

Horses are naturally curious. Strange objects are watched, then cautiously approached and smelled. The object may or may not then be added to a list of harmless objects.

Horses enjoy routine and once settled, do not change easily. They do, however, adapt easily to new circumstances. Changes in a normally busy routine may lead to boredom. Habits are very difficult to break. Behaviour is modified with each new experience. The greatest single modifying factor is man.

### Housing

The first thing that man does to the horse is to confine him, which immediately creates problems for an animal used to moving around at will. The restrictions may lead to the development of bad habits such as aerophagia, anorexia or dysphagia[4]. Horses may be placed together with, or next to horses that they dislike. This may lead to ethostasis[4].

### Feeding

This presents an area of further restriction especially where the human habit of eating three meals a day has been applied to an animal used to grazing for most of the day. Single or a few large meals, especially of concentrates are eaten quickly, and leave the greater part of the day free in which to do nothing. Boredom sets in. The animal seldom has the sensation of a full stomach. Bad habits, such as dysphagia, of which lignophagia and coprophagia and good examples, begin[4]. Horses adapt quickly to a specific feeding routine. This may easily be used as a form of reward during training[2,3].

### Breeding

Natural breeding is non-selective. Man, however, dictates how, when, and with whom which is totally unnatural. During the natural mating process the animals have more time for the species specific ritual, which may or may not end in mating. With man's interference, it is hardly surprising that stud animals often become difficult to handle.

### Veterinary treatment

Man does try to help the animals in his care, but the horse may not understand why a tube is being pushed down his nose, or some terrible tasting stuff is squirted into his mouth, especially if he is feeling well at the time. Pain inflicted during treatment is also not understood, especially if the animal is already in pain. A horse may be difficult to handle during treatment, or recovery from general anaesthesia may be stormy, irrespective of the amount of pain suffered. This is because the very strong survival instinct makes him want to fight anything or anyone threatening him.

### Work

Horses are used for man's business and pleasure, a situation which places physical stress on the animal and suppresses his natural instincts. He is required to gallop at top speed with a weight on his back, to stop suddenly and to turn, to jump great heights or to pull heavy loads, situations unheard of in the natural state. His ability to adapt to these drastically altered circumstances is incredible.

All in all, the horse is a creature that we could understand much better, if we go to the trouble of studying his ethological make-up, and thus contribute to the welfare of the noble animal.

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# "METAPHYLAXIS" IN POST-PARTUM CONDITIONS IN DAIRY COWS WITH BUTAPHOSPHONE*: A TRIAL UNDER SOUTH AFRICAN CONDITIONS

C.R. PALMER

ABSTRACT: Palmer C.R., 1979 **"Metaphylaxis" in post-partum conditions in dairy cows with butaphosphone: a trial under South African conditions** *Journal of the South African Veterinary Association* **51** No. 4 239–242 (En) Bayer South Africa (Pty) Ltd., Box 1366, 2000 Johannesburg, Rep. of South Africa.

Current literature on the prevention of the "parturition syndrome" in dairy cows using butaphosphone is summarised. A South African trial is recorded in which 2 pre-partum injections of the drug caused a significant reduction in the number of cases of parturition syndrome, as well as a marked reduction in intercalving period in the treated portion of the herd. No difference in lactation yield or culling rate was seen. The financial implication of the reduction in intercalving period is discussed.

## INTRODUCTION

Yield increases in dairy herds are often made at the expense of even greater increases in the incidence of so-called production diseases, for example in the Federal Republic of Germany between 1950 and 1973 the national milk yield increased by 22%, but the national culling rate for infertility increased by 67% and culling for mastitis increased by 117%[7].

Losses due to generalised infectious diseases and localised infections are relatively insignificant (13% in South Africa) while losses from and cullings due to metabolic diseases, reproductive disorders and poor production accounts for 84,2% of cullings and/or deaths[3]. The total losses in South Africa resulting from this group of diseases include the losses from mastitis which are currently estimated at R185,78 × $10^6/a^2$, as well as the loss attributable to poor production which is estimated at R84 × $10^6/a^5$, i.e. a total annual loss of approximately R269 million.

Sommer[7] postulated a central cause for all the conditions grouped under the parturition syndrome, namely abortion, retained afterbirth, metritis and subsequent infertility, mastitis and milk fever; the basic aetiology being liver failure and subsequent decreased gluconeogenesis. He showed that ruminal activity slows down by 33% between the 8th week ante partum and the 5th week post partum. This, plus minor feeding errors causes a pre-calving energy defecit. Over several calvings cumulative liver damage can occur due to acetonaemia and fatty infiltration. The energy defecit will, depending on the animals' particular susceptability and the circumstances prevailing on the farm, result in one or other of the perinatal diseases (Fig. 1). Sommer[7] as well as Zepgi et al.[8] were able to identify animals at high risk due to the above sequence of events as early as 8 weeks before calving by their abnormal SGOT, total cholesterol and serum glucose levels (Fig. 2). They found that by administering butaphosphone antepartum they could reverse these changes and considerably reduce the incidence of parturition syndrome at the subsequent calving in these "high risk" cases. This non-specific "treatment" with a roborant or "metabolic stimulant", which is half treatment and half prophylaxis, they termed "metaphylaxis". The exact dose of butaphosphone and the exact time of its administration is not stated by either worker.

**Fig. 1.** Parturition syndrome caused by precalving stress (after Sommer[7]).

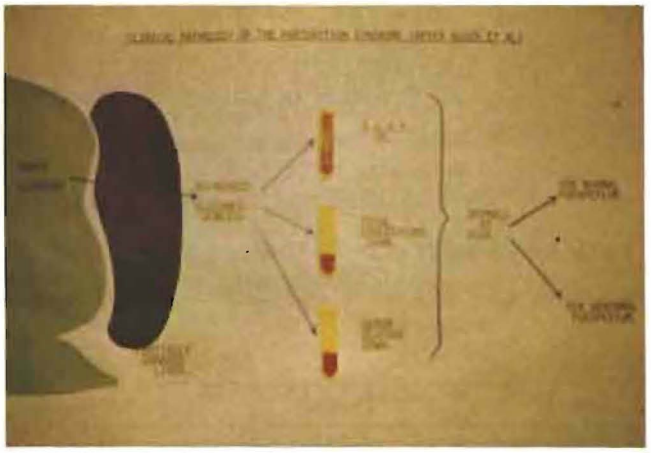

**Fig. 2.** Clinical pathology of the parturition syndrome (after Zepgi et al.[8])

**Fig. 3.** The herd used in the trial

*Catosal®, Bayer Leverkusen



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Table 1: SUMMARY OF THE RESULTS OF SOMMER[7] IN ONE TRIAL

485 Cows at risk

| Control | Treated* | % Reduction |
|---|---|---|
| 73% showed parturition syndrome | 27% showed parturition syndrome | 63% |
| 12% culled | 2% culled | 83% |

*Treatment = Antepartum Metaphylaxis with Butaphosphone

Table 2: SUMMARY OF THE RESULTS OF SOMMER[7] IN ANOTHER TRIAL

54 Cows at risk

| Control | Treated* | % Reduction |
|---|---|---|
| 77% showed parturition syndrome | 16% showed parturition syndrome | 79% |
| 1,61 Treatments per animal required | 0,17 Treatments per animal required | 89% |
| 16% died | 0% died | 100% |

*Treatment Ante-partum = Metaphylaxis with Butaphosphone

The results of the 2 trials of Sommer are summarised in Tables 1 and 2. Zepgi et al.[8] tested 60 cows. They found 30 to be "not at risk". All these showed normal puerperium. The results of the 30 cows identified as being "at risk" are tabulated in Table 3. They administered vitamins A, D and E in addition to butaphosphone (Table 3).

Table 3: SUMMARY OF THE RESULTS OBTAINED BY ZEPGI et al.[8]

30 Cows at risk*

| Control | Treated† | % Reduction |
|---|---|---|
| 50% showed parturition syndrome | 0% showed parturition syndrome | 100% |

*In this herd of 60, the remaining 30 cows were considered "not at risk". All showed normal puerperium.
†Treatment = Butaphosphone + Vit. A, D & E.

## THE SOUTH AFRICAN TRIAL

The requirements for this trial differed from those of Sommer[7] and Zepgi et al.[8] because:

1. It was felt that the determination of the 3 blood parameters in every cow in a commercial herd was impractical, and that the economic feasability of a blanket treatment should be investigated;
2. The real criteria for determining the success of the treatment are the intercalving period and milk yield; and
3. The trial should be conducted over a period of a full year in order to eliminate seasonal differences.

## METHOD

The trial involved 75 Friesland milking cows over a period of exactly one year (29.11.76–23.11.77). According to their expected calving dates every second cow received butaphosphone at a dosage rate of 30mℓ intramuscularly 35 days before calving and again 28 days before calving. The remaining cows acted as untreated conrols. Parameters measured were: Number of cows which developed the parturition syndrome, number of days open, number of inseminations to conception, lactation yield and culling rate.

## HISTORY OF THE HERD

At the commencement of this trial, this herd had been under the personal supervision of Prof. C. Maree (Department of Animal Production, Faculty of Agriculture, University of Pretoria) for 5 years, and due to a regular programme of monthly veterinary examinations the intercalving period had dropped from an unknown high down to 573 days in 1975–1976. The following year (in which this trial was conducted) it dropped further to 401 days. The year after that (1977–1978) a number of cows had to be held back in order to build up the herd yield during the winter to fit in with the milk quota system, and the butaphosphone treatment was discontinued. The intercalving period fell back to 421 days (all figures quoted are from the annual report of the Milk Recording Scheme[6] whose year runs from 1 September to 31 August and will therefore not agree exactly with the average figures in Tables 4 and 5).

Table 4: NUMBER OF COWS SHOWING THE PARTURITION SYNDROME AND NUMBER OF INSEMINATIONS REQUIRED TO INDUCE CONCEPTION

South Africa: 75 random cows

| Controls (36 cows) | | Treated (39 cows) |
|---|---|---|
| Total No. showing parturition syndrome | 9 (retained afterbirth) | 1 (retained afterbirth and metritis) |
| Average days open | 179,4 | 82,8 |
| Total No. of inseminations | 108 | 53 |

Table 5: RESULTS OBTAINED EXPRESSED AS A PERCENTAGE

South Africa: 75 random cows

| Control | Treated | % Reduction |
|---|---|---|
| 25% showed parturition syndrome | 2,5% showed parturition syndrome | 90% |
| 179,4 days open | 82,8 days open | 53,8% |
| 3,0 inseminations per conception | 1,36 inseminations per conception | 46,6% |

The fate of all cows in the trial was followed until January 1979 in order to record their lactation yields and compare culling rates in the 2 groups. Ten of the treated and 21 of the control group were sold (these were neither the best nor culls). Lactation yields of a further 8 treated and 7 control cows were not recorded. These were the deaths and culls recorded in Table 7, plus one animal which was sold out of hand and 2 which had not yet completed their lactations at the time this article was written.

### Nutrition

The heifers receive silage, dairymeal and a proprietary protein/mineral lick. The dry cows receive silage, dairymeal and limited grazing while the cows in milk receive pelleted lucerne, highly-fertilised barley hay (cut green), dairy meal, and vlei grazing during the summer months.

## RESULTS

The results as tabulated in Tables 4 and 5 show the highly significant improvement in intercalving period and number of inseminations to conception. The standardised lactation yield showed no significant difference between the 2 groups (Table 6).

Table 6: STANDARDISED LACTATION YIELD (AVERAGE)

| Controls (11 animals) | Treated (22 animals) | Difference |
|---|---|---|
| 4 776,0 kg | 4 768,7 kg | 0,15% |

*Expressed in kg milk per 305 days

Table 7: CULLING RATE IN THE 14 MONTHS FOLLOWING THE TRIAL PERIOD

| | Controls | Treated |
|---|---|---|
| Died of unknown causes | 1 | 3 |
| Died of bloat | 0 | 1 |
| Fate unknown | 1 | 1 |
| Culled for old age | 1 | 1 |
| Culled for bad legs | 1 | 0 |
| Culled for general debility | 1 | 0 |
| Culled for chronic mastitis | 0 | 1 |
| | 5 | 7 |

### Economics

There was also no difference between the culling rate in the 2 groups in the following 14 months (Table 7). The financial loss for each day that a cow remains open over the accepted ideal of 100 days has been calculated by Perchman[5] as being R1,70 per day in South Africa in 1979. This figure includes losses due to reduced milk production, reduced calf production, feed costs, labour costs, dips, drugs, etc. and capital loss on investment. As the control group had an average of 179,4 days open, this represents an additional cost over the ideal of 79,4 days which, at R1,70 per day, equals R134,98 per cow per annum. The treated group actually came below the ideal number of days open which gives extra savings in feedcosts, etc., but which would be offset to some extent by decreased production in the subsequent lactation.

This figure of a loss of R134,98 per cow per annum does not include the cost of the additional 1,44 treatments per animal which were required or the cost of the additional 1,4 inseminations which were required in the conrol group.

The cost of the butaphosphone treatment at current retail prices is:

| | |
|---|---|
| Drug: 2 × 30mℓ injections | R5,35 |
| Two disposable syringes | R0,62 |
| Total | R5,97 |

In this trial the "investment" in the treatment was thus recouped 22 times in the savings made in the treated group. The 197,4 days open in the control group (i.e. an intercalving period of 459,4 days) is probably close to the national average intercalving period. The average intercalving period for herds participating in the Milk Recording Scheme in 1976 was 425 days[1], i.e. 145 days open equals 45 additional days at R1,70 per day which means a saving of R76,50 per cow per annum or a recoupment on the investment of treatment of 12,8 times.

## DISCUSSION

The results of the previous workers mentioned, indicated that it would be economically feasable to apply blanket treatment to entire dairy herds. This has been adequately proven by the current trial. The physiological mechanisms of beneficial action of butaphosphone on the pre-partum dairy cow have not yet been adequately studied. In horses a similar roborant effect is seen which is manifested as a general improved response to training[6]. This is thought to be mainly due to the increased availability of oxygen to the tissues as a result of the administration of an organic phosphate[3]. It is likely that in cattle an additional direct effect on the smooth musculature of the rumen assists in reversal of the process shown in Fig. 1.

## ACKNOWLEDGEMENTS

Grateful thanks are extended to Mr. John Bradford, manager of the herd for his assistance and meticulous record keeping, and to Prof. C. Maree for his encouragement.

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## BOOK REVIEW

## BOEKRESENSIE

### PHYSICIAN AND NURSE MIGRATION: ANALYSIS AND POLICY IMPLICATIONS. REPORT OF 'A WHO STUDY' by A. MEJIA, H. PIZURKI, E. ROYSTON.

Geneva, World Health Organisation, 1979 (ISBN 92 4 156059 2). 476 pages

The World Health Assembly some years ago adopted a resolution calling for a study of the phenomenon of migration of health manpower as a basis for action.

This book documents the view that the migration of physicians and nurses is not due to a random set of events but is the result of a hierarchy of identifiable factors in both donor and recipient countries.

The study was based partly on the results of a statistical inquiry made by means of questionaires and partly on an examination of the extensive literature related to health manpower migration. The work is presented in three parts – Part One contains a global description and analysis of the migration phenomenon; Part Two consists of individual studies of 13 countries both developing and industrialised. Part Three consists of summaries and conclusions.

Chapters I and II deal respectively with the dimensions and directions of migration, are most enlightening about the factors influencing migration.

Chapter IV discusses the pros and cons of the migration of health personnel and describes the resulting economic losses and gains and the impact on health care. Chapter V deals with the influence of national policies on migration.

The situation in each of 13 countries is analysed under the following headings: Direction and dimensions of migration; Characteristics of migrants; Factors influencing migration; Consequences; and Policy. Where appropriate, a section deals with the export and import of medical education as distinct from medical manpower.

Very extensive documentation as evidenced by numerous tables has been undertaken. The bibliography includes 224 references.

Country-specific studies were carried out on 13 countries which represented various stages of development from the East, Mid-East and the West. As the findings naturally varied a great deal in different countries a special look at the report on Australia which is the nearest akin to South Africa of the countries studied is indicated.

Regarding physicians Australia is both a donor and recipient with the latter somewhat in greater number. In the early 1970's about 12,5% physicians were abroad nearly half in the United Kingdom and about one sixth in the USA. There was also a steady flow to Canada and New Zealand. In the 1966 census 26,5% of Australian physicians were foreign born. This percentage decreased to about 23% in 1972 with more Australians qualifying as physicians.

In 1963, 21% of immigrant physicians were from South Africa but in 1971 this percentage had dwindled to only 3%.

The investigation relative to push and pull factors acting on migration of physicians are summarised in 25 findings.

These can again be telescoped into the observations that political upheavals account for a certain proportion of migration but the most important immediate push factor in a majority of donor countries, particularly the developing ones appears to be overproduction of physicians relative to effective economic demand for medical services for each country. For each country where this is the case the obvious directive therefore would be to produce as many physicians as the country can afford or to take the necessary steps to increase the local demand for the amount and type of medical services implicit in the quantity and quality of physicians produced.

In a brief review it is impossible to do justice to a book as intensive and extensive as the present one. From what has been stated it can be deduced that there is considerable food for thought for the veterinarian.

C.F.B. Hofmeyr.

# *PARAFILARIA BOVICOLA* IN CATTLE AND ITS CONTROL*

A.C. WELLINGTON

ABSTRACT: Wellington A.C., ***Parafilaria bovicola* in cattle and its control.** *Journal of the South African Veterinary Association* (1980) **51** No. 4 243–244 (En) Animal Health Research Station, Maybaker (S.A.)(Pty) Ltd, P.O. Box 1130, 6000 Port Elizabeth Rep. of South Africa. The areas of infestation, life cycle, lesions, method of diagnosis and the control of *Parafilaria bovicola* in cattle in South Africa are discussed.

## DISTRIBUTION, LIFE CYCLE AND LESIONS

*Parafilaria bovicola* is an internal parasite of cattle, which manifests itself by causing bleeding points in the live animal and extensive areas of "bruising" of the carcass on slaughter.

Carmichael & Koster[1] found that the areas of South Africa infested with Parafilariosis are the northern Cape Province, western O.F.S., south-west Transvaal, Transvaal bushveld, the Lowveld, northern Natal, eastern O.F.S., eastern Province and South-West Africa. They further reported that the optimum conditions for the occurrence of *Parafilaria* are:

(i) an annual rainfall of 400 to 700 mm
(ii) a frost period of less than 120 days
(iii) a mean annual temperature of 17,5 to 22,5°C
(iv) an altitude ranging between 800 and 1200 metres above sea level.

Viljoen[4-8] and Nevill[2 3] have described, and are in the process of working out in finer detail, the life cycle of the parasite in the intermediate and definitive hosts and the lesions caused by the parasite in cattle.

The life cycle is as follows: The adult female worm bores her way through the skin from the subcutis to the outer surface, where she lays her eggs in the blood that exudes from the lesion. The intermediate host, the fly (1 of 3 of the *Musca* spp. known to act as the intermediate host), feeds on this blood and in the process ingests eggs containing microfilaria. The eggs hatch in the fly and the larvae reach the third stage within 10 to 14 days and are then ready to leave the fly when next she lands on the host[3]. The infective larvae leave the fly and enter the animal. The parasite now takes approximately 7, 67 and 195 days post-infestation respectively to reach the fourth larval, fifth and adult stage. At approximately 250 days[8] post-infestation, the adult egg-bearing female starts to bore her way through to the surface of the skin.

Subcutaneous lesions (and their colouration) noted on the carcass are due to the migration and presence of the worms. The lesions in the first 30 days post-infestation are prominent and very acute in nature; during the period Day +30 to Day +120 the lesions seem to disappear, possibly being subacute, and from about 120 days onwards post-infestation the chronic form of lesion is noted[8].

## DIAGNOSIS

The only clinical signs seen in the live animal are the presence of bleeding points. Congealed blood may be removed along with the hair and examined microscopically for the presence of microfilaria-bearing eggs. After slaughter, the carcass can be examined for the presence of worms or for the typically coloured oedematous lesions, which are positive for eosinophiles on microscopic examination of a smear of the lesion.

## CONTROL

Viljoen and Boomker[4] tested the efficacy of several compounds against *P. bovicola.* One of the compounds tested, nitroxynil†, was found to be effective when injected subcutaneously twice at 96 hour interval at the dose rate of 20 mg/kg. They found that this treatment brought about a decrease of 90% in the mean number of lesions and a decrease of 93% in the mean eosinophile lesion area when compared to unmedicated control animals. Wellington[9] later found that nitroxynil injected twice at 72 hour interval at a dose rate of 20 mg/kg was equally effective in older animals.

Slaughter of the animals in both these trials took place at plus 9 weeks after treatment of the animals. The reason for this is that Viljoen[5] found that the subcutaneous lesions caused by the worm took approximately 9 weeks after the death of the worm to resorb and return to normal.

Viljoen[6] also found that cattle treated with nitroxynil showed a 99% and 97% decrease in the total number of bleeding points, 5 and 8 months after treatment respectively, when compared with unmedicated controls.

The treatment of cattle with two injections of nitroxynil approximately 10 weeks prior to slaughter has been shown to be very effective for the following reasons:

(i) Wellington[9] showed that unmedicated control animals had a mean mass of 2,5 kg meat trimmed, whereas only a mean of 0,1 kg was trimmed in the treated animals. Thus the owner saved R6,40 per animal after deducting the cost of the drug.
(ii) Viljoen[6 7] estimated that carcass downgrading by one grade caused a financial loss of between R4 and R13 and by two grades, a loss of between R11 and R20.
(iii) Carmichael & Koster[1] found that the financial loss due to the aesthetic element in regard to the meat buyer (not including losses due to downgrading, trimming or condemnation) showed a mean price difference between affected and non-affected carcasses of:

| | |
|---|---|
| for Super Grade | 89 cents/100 kg |
| Prime Grade | 72 cents/100 kg |
| Grade 1 | 93 cents/100 kg |
| Grade 2 | R1,20/100 kg |

Each of the above conditions, either individually or in combination, would partially or completely pay for the treatment of each animal.

*Paper delivered at the SAVA National and International Congress, 1979 held in Johannesburg.

†'Trodax': Maybaker (S.A.)(Pty) Ltd.


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(iv) Heavy infestations of the parasite may have an effect on the growth potential of the animal. This possibility still has to be studied.

Viljoen[7 8] has tried to eliminate the disease from a specific farm, but this seems to be impossible due to the life cycle of the fly and the fact that fences do not stop flies. It is thus felt that if one wants to attempt to eradicate the disease, a massive campaign will have to be undertaken in the infested areas and that each and every animal on every farm will have to be treated.

In conclusion, although nitroxynil has been found to be effective against *P. bovicola* when used correctly, one must keep in mind that the product can be toxic. The use of a scale in ascertaining the live mass of the animal is of importance. I have found that the use of the weighband is not accurate enough to determine the mass and has led to overdosage, causing clinical signs of toxicity in animals in poor condition. Furthermore, animals should not be driven for long distances after treatment and should at all times be handled quietly.

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## BOOK REVIEW — BOEKRESENSIE

### BRITISH MEDICAL BULLETIN – REPRODUCTION VOL 35 NUMBER 2 MAY 1979

Edited by R.V. SHORT

This volume deals with the very fascinating subject of reproduction beginning with the process of meiosis in mammalian oocytes, the processes involved and the factors responsible for the complete development and full maturation of the oocyte. This followed by a review of in-vitro fertilization, embryo transfer and storage, control of early embryonal development and sex determination and differentiation. The embryonal development is concluded with a review on embryonic signals which are responsible for the establishment and maintenance of pregnancy.

Foetal growth and development, diagnostic aids and causes of intra-uterine foetal growth retardation are then discussed in detail followed by a concise review on the mechanisms responsible for the initiation of parturition. The effects of lactation on fertility in the human are also discussed.

Reviews on the neuroendocrine control of gonadotropin secretion gonadotrophic control of ovarian and testicular functions and the role played by prostaglandins in reproductive functions of man and domestic animals and then presented.

The last four reviews are devoted to chromosomal causes of human infertility, the endocrinology of male and female infertility in humans and the recent trends and developments in contraceptive research.

This volume will be of benefit to all those concerned with reproductive endocrinology in the medical and veterinary fields because of its concise but very clear coverage of this subject.

H.M. TERBLANCHE

# MORTALITEIT IN LAMMERS AS GEVOLG VAN VERSTOPPING VAN DIE ABOMASUM DEUR *ORNITHOPUS SATIVUS* Brot. (SEREDELLA) HAARBALLE

D.J. SCHNEIDER* and L. HUGO†

ABSTRACT: Schneider D.J.; Hugo L. **Mortality in lambs due to impaction of the abomasum by *Ornithopus sativus* Brot. (seredella) phytobezoars.** *Journal of the South African Veterinary Association* (1980) **51** No. 4 245–247 (Afr) Veterinary Diagnostic Laboratory, Private Bag X5020, Stellenbosch 7600, Republic of South Africa.

An outbreak of mortality in a flock of Merino x Mutton Merino crossbred sheep where 62 out of 300 4 month old lambs died over a period of 3 weeks, is described. Some of the lambs showed symptoms of illthrift, but most died within a day or two as a result of impaction of the abomasum with obstruction of the pylorus. The obstruction was caused by more-or-less angular, tough phytobezoars varying in size from 15–40 mm in diameter and consisting almost exclusively of the hairs which occur on the leaves and stems of *Ornithopus sativus* Brot.

## INLEIDING

Plantaardige haarballe in die abomasum van herkouers is 'n onbekende verskynsel in die Winterreënstreek van die Republiek van Suid-Afrika. Bath[1,2] egter, het soortgelyke balle beskryf wat oor 'n wye gebied in die Karoo onder skape en bokke voorkom en veeverliese veroorsaak. Hy het die pappushare van die blomme van sekere Karoobossies verdink en ook morfologies identiese balle veroorsaak deur die voer van die ryp blomme van enkele bossies aan lammers en bokkies[1]. Aangesien die huidige probleem voorgekom het op 'n plaas aan die grens van die Ceres-Karoo, was die pappushare van die plaaslike bossies eerste onder verdenking.

Geen ooreenkoms kon egter hier met die hare vanaf die enkele bossies en droë gras, teenwoordig op die land, gevind word nie. By verdere ondersoek het dit geblyk dat die hare in die balle identies is met die hare wat op die blare en stingels van die weidingsgewas *Ornithopus sativus* voorkom. Hierdie is dus die eerste getuienis dat die hare van *O. sativus* balle in die abomasum van skape kan veroorsaak.

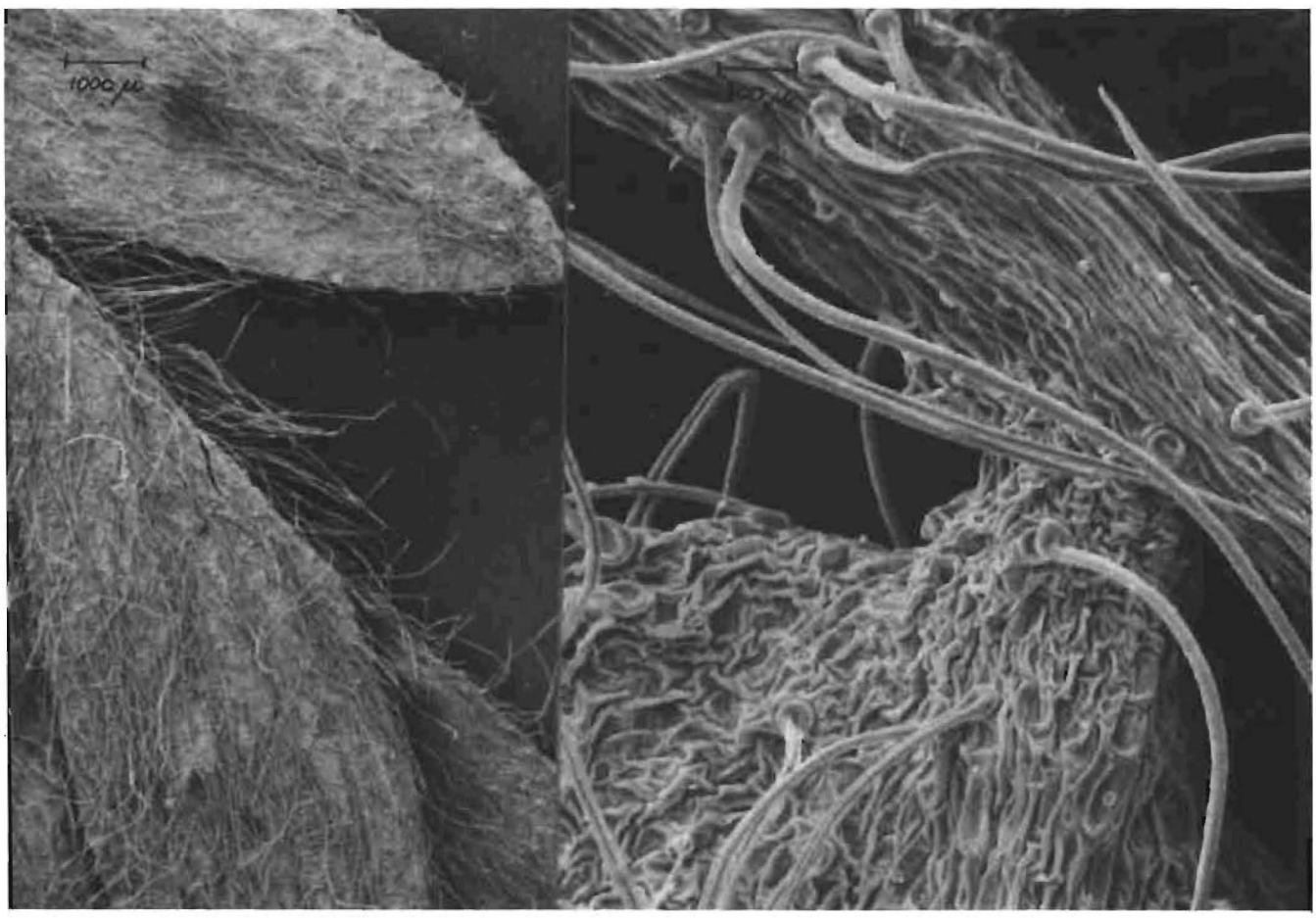


**Fig. 1.** Hare op blare en stam van *Ornithopus sativus*

*Veterinêre Streekslaboratorium, Privaatsak X5020, Stellenbosch 7600.
†Navorsingseenheid vir Plantkunde, Stellenbosch.


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## GESKIEDENIS

Gedurende Februarie 1979 is 'n vrekte onder Merino x S.A. Vleismerino kruisras lammers ondersoek op 'n plaas in die Ceres-Karoo. Toe die lammers tussen 2 en 6 weke oud was, is die ooie met hul lammers vanaf aangeplante weidings naby die opstal, geskuif na 'n kamp sowat 50 ha groot. In hierdie kamp was 'n land sowat 25 ha groot met 'n mooi stand *O. sativus* daarop. Die oorblywende gedeelte het bestaan uit natuurlike veld met hoofsaaklik *Elytropappus rhinocerotis* Less. (renosterbos) daarop. Die skape het onafgebroke in hierdie kamp gebly en uitstekend gegroei, alhoewel daar uiteindelik baie min *O. sativus* oor was. Na sowat 2½ maande in hierdie kamp het die eienaar opgemerk dat 'n aantal van die mooi groot lammers gevrek het en dat enkele van hulle dun en lusteloos voorkom. Toe die mortaliteit toeneem en 25 lammers reeds gevrek het het die eienaar die skape uit die kamp gehaal, die lammers van die ooie geskei en hul op groen lusern weiding naby die woonhuis, in 'n kamp geplaas. Omdat die weiding vinnig min geword het en intussen nog sowat 15 lammers gevrek het, is die lammers na 6 dae uitgehaal en in 'n voerkraal geplaas. Hier het hul skaapafrondingspille en droë gebaalde lusern ad lib. gekry. Alhoewel hier net sowat 6 lammers gevrek het, was daar na 3 dae sowat 40 lammers wat effe siek vertoon het en het die eienaar hul uit die voerkraal geneem en in kampe met aangeplante weiding (grasklawer en lusern) geplaas.

Twee van hierdie dooie lammers is ingebring vir nadoodse ondersoek. Die mortaliteit het geleidelik afgeneem en al die oorblywende lammers (240) is vanaf hierdie groenweiding bemark gedurende die volgende 2 maande. Uit 'n totaal van 302 het 62 lammers gevrek. Nie een van die ooie uit hierdie trop skape het op soortgelyke wyse gevrek nie. Volgens die eienaar is dit hoofsaaklik die grootste lammers wat gevrek het en het sowat een derde gevrek binne 24 uur nadat siektetekens waargeneem is en twee derdes nadat hul vir enkele dae simptome getoon het.

## SIMPTOME

Party lammers is net deur die eienaar dood gevind sonder dat enige tekens van siekte opgemerk was. Meesal, egter, was daar simptome van lusteloosheid, anoreksie, plat abdomen, diarree en vertraagde groei te bespeur. Die aanvang van simptome waargeneem voor dood het gewissel van enkele uur tot 2 of 3 weke.

## NADOODSE ONDERSOEK

Net 4 lammers kon nadoods ondersoek word. Drie hiervan het self gevrek en een is teen die einde van die probleem geslag omdat die lam duidelike simptome van lusteloosheid en vertraagde groei getoon het. Behalwe 'n ligte edeem en kongestie van die longe in meeste skaap gevind, was die ooglopende afwyking 'n verstopping van die abomasum deur plantaardige haarballe.

Hierdie balle was meesal hoekig met effe afgeplatte kante en afgeronde rande. Die kleur was donker khakikleurig wanneer dit in die abomasum gevind word, maar het ligter geword namate dit droog geword het. Die oppervlakte was glad, dof en fluweelagtig. In die nat toestand sink hierdie balle in water, maar wanneer droog dryf dit maklik. Die getal aanwesig per skaap het gewissel vanaf 4 tot ± 30, en die grootte tussen 15–40 mm in deursnee. Die balle was effe veerkragtig, maar besonder taai. Die inhoud bestaan uit een soliede massa baie fyn haartjies met die voorkoms van fluweel. Indien deurgesny was konsentriese lae sigbaar met 'n kern wat nie verskillend vertoon as ander dele van die bal nie. In die geval van elk van die 3 lammers wat self gevrek het, is een van die balle stewig, soos 'n balklep, in die tregtervormige pylorus gedeelte van die abomasum gevind. Die ander balle het dan daaragter die abomasum geheel of gedeeltelik volgepak. Behalwe vir hierdie balle was die abomasum gewoonlik droog en leeg.

Die inhoud van die rumen het effe meer vloeistof as gewoonlik bevat, maar daar was geen balle teenwoordig nie en niks abnormaal kon gevind word nie. Die meeste van die lammers wat gevrek het is deur die eienaar nadoods ondersoek en in elke geval is hierdie balle saamgepak in die abomasum gevind.

Die haartjies vanuit die haarballe is toe mikroskopies vergelyk met alle harige gedeeltes van die enkele bossies en droë gras wat op die land gevind kon word, maar geen ooreenkoms kon gevind word nie. Aangesien geen *O. sativus* plante op daardie stadium op die land gevind kon word nie (daar was wel heelwat saad verspreid op die grond) is hierdie haartjies vanuit die balle toe vergelyk met die haartjies vanaf verskillende *O. sativus* eksemplare vanuit die Staatsherbarium, Stellenbosch. Dit was dadelik duidelik dat hierdie haartjies identies was. Daar is toe bevind dat die abomasum balle feitlik uitsluitlik uit *O. sativus* hare bestaan met enkele ander plantaardige stekels en debris. Die gemiddelde lengte van die hare van *O. sativus* en die uit die haarballe is 1 mm.

**Fig. 2.** Abomasum van lam ongeopen

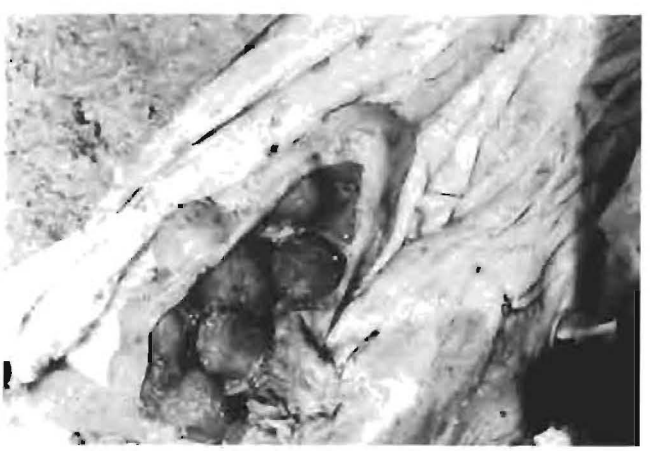

**Fig. 3.** Abomosum van lam geopen

**Fig. 4.** Plantaardige haarballe vanuit lam abomasum

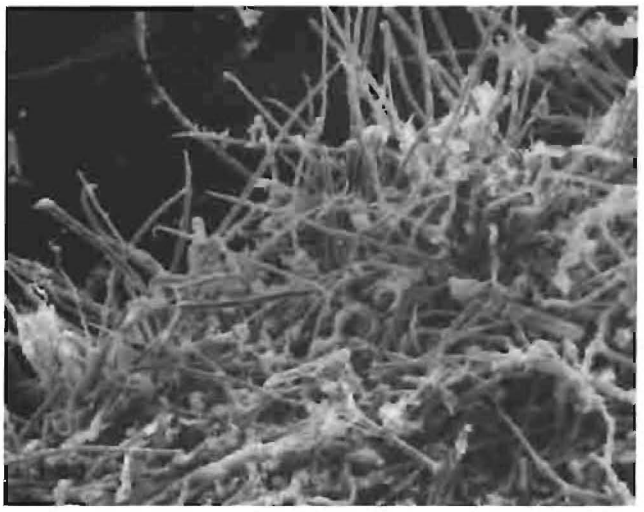

**Fig. 5.** Plantaardige hare vanuit haarbal uit abomasum

Volgens die eienaar het hy 2 van hierdie groep lammers weens swak liggaamlike kondisie moes terughou op die plaas toe die ander bemark is. Toe hul na sowat 2 maande herstel het, is hul op die plaas geslag en sowat 15 balle per skaap in onveranderde toestand in die abomasum gevind.

## BESPREKING

Geen onweerlegbare verklaring kan gegee word waarom soortgelyke haarballe nie al vantevore opgemerk is nie, aangesien *Ornithopus* spesies vir jare al in sekere dele van die Winterreënstreek as weidings- en hooigewas gesaai word.

Die lammers was vir 'n onafgebroke periode van sowat 2½ maande op amper suiwer seredella weiding. Volens die eienaar was die stand *O. sativus* baie mooi en suiwer met baie min gras en onkruid. Aangesien die beskikbare renosterbosveld baie onsmaaklik is sou 'n mens verwag dat die lammers vir die hele periode hoofsaaklik *O. sativus* gevreet het. Hierdie suiwer dieet van *O. sativus* oor 'n lang periode mag in die toekoms blyk 'n belangrike veroorsakende faktor van haarballe in die abomasum van lammers te wees. 'n Mens sou verwag dat die ooie meer geneig sou wees om van die onsmaaklike bossies te vreet om in hul vesel behoeftes te voorsien.

Ander moontlike verklarings is dat die melkstolsels in die abomasum van die lam 'n kern voorsien het waarom haar begin aansamel om uiteindelik 'n haarbal te vorm. Die meer doeltreffende rumen flora van volwasse skape mag seredella hare moontlik afbreek sodat by hulle geen haarballe vorm nie. Volgens beskikbare informasie bly hierdie haarballe vir baie maande in die abomasum van lammers wat aan die lewe bly.

## BEDANKINGS

Ons is dankbaar teenoor Drs Thomson en de Kock, Ceres vir verwysing van die geval en hulp verleen, teenoor Mnre. A.T. Carsens en P. van der Merwe en Mev. Ina Boshoff, N.I.V.V. Stellenbosch vir die fotos en Mnr. S.W. Pienaar, Uitkomst, Ceres vir vriendelik samewerking.

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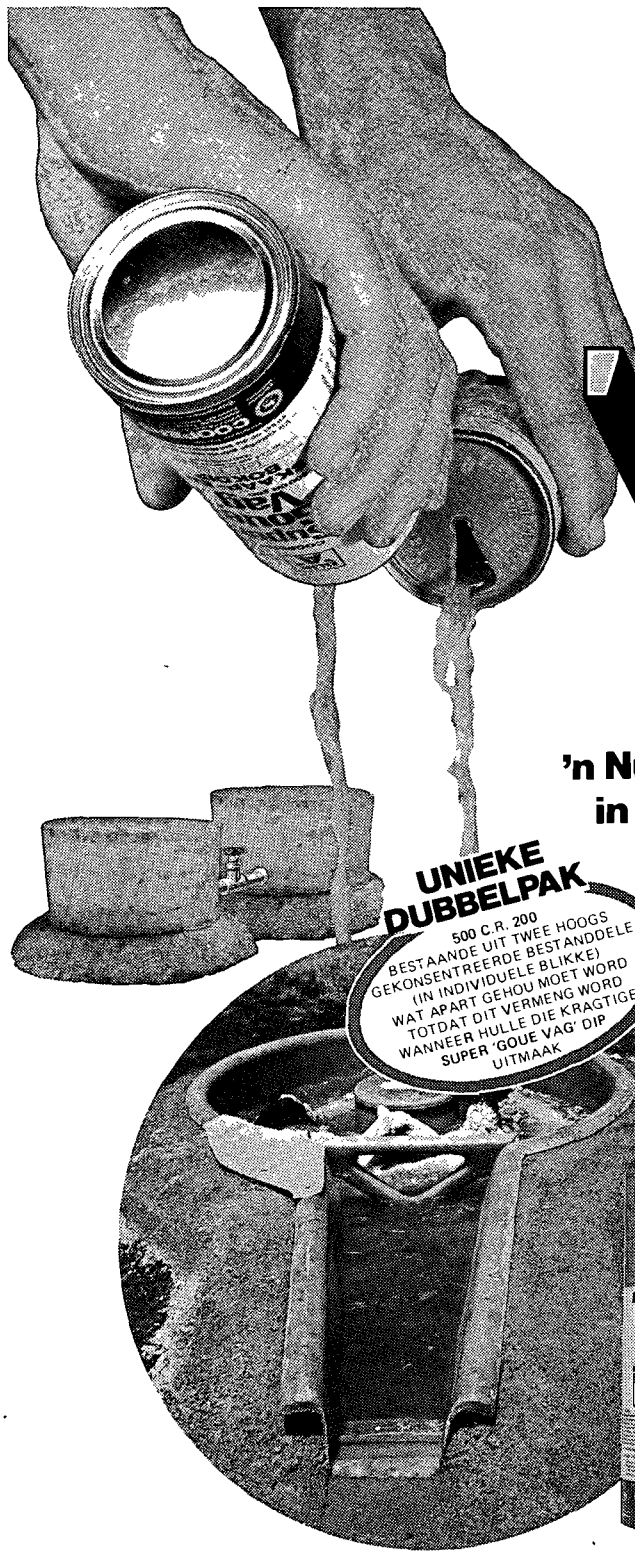


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# THE TREATMENT OF *STILESIA HEPATICA* INFESTATION

ANNA VERSTER and G. MARINCOWITZ*

ABSTRACT: Verster, Anna; Marincowitz G. **The treatment of *Stilesia hepatica* infestation.** *Journal of the South African Veterinary Association* (1980) **51** No. 4 249–250 (En) Veterinary Research Institute, 0110 Onderstepoort, Rep. of South Africa.

Sheep and goats naturally infested with *Stilesia hepatica* were treated with either praziquantel (Droncit, Bayer) or oxfendazole (Systamex, Coopers). Praziquantel at 15 mg/kg was 100% effective in 20 sheep; at 7,5 mg/kg 2 out of 20 sheep were still positive. Oxfendazole at 3,5mg/kg was ineffective. In spite of its efficacy the cost of praziquantel prohibits its routine use as an anthelmintic.

## INTRODUCTION

The liver tapeworm, *Stilesia hepatica,* which occurs in the bile ducts of sheep and other ruminants, occurs widespread in the Republic of South Africa. It causes considerable economic loss as infested livers are condemned at abbatoirs for aesthetic reasons.

Bankov reported that praziquantel (Droncit, Bayer) at 15 mg/kg live mass was 100% effective against *Stilesia globipunctata,* a parasite of the small intestine of sheep and other ruminants. Early work in Kenya seemed to indicate that oxfendazole (Systamex, Coopers) at 5 mg/kg live mass had some effect against *S. hepatica.* (W.J. Jones-Davies, Cooper (S.A.)(Pty) Ltd., P O Box 677, Kempton Park, Transvaal, Personal communication, 1978 to J.A. van Wyk). It was decided to test these two drugs against *S. hepatica* because there was no known effective treatment against this cestode.

Because infestation cannot be diagnosed in the living animal, animals for an anthelmintic trial must be selected from flocks that are known to be heavily infested. This condition was met in a flock of Dorper sheep on natural grazing at Roodeplaat Research Station near Pretoria where we examined 14 sheep and all of them were infested.

Two trials were performed. In the pilot trial animals were treated either with praziquantel or oxfendazole and in the second trial praziquantel was administered at 2 dosage levels.

## PILOT TRIAL

### MATERIALS AND METHODS

Eight sheep and 21 goats from Roodeplaat Research Station were moved to the Veterinary Research Institute where they were housed for the duration of the trial. They were divided into 3 groups and were treated as follows:

*Group 1* Two sheep and 7 goats were treated *per os* with praziquantel at 15mg/kg live mass.
*Group 2* Two sheep and 7 goats were treated *per os* with oxfendazole at 3,5 mg/kg live mass.
*Group 3* Two sheep and 7 goats acted as untreated controls.

Two weeks after treatment the animals were slaughtered and the livers examined for cestodes. The ductus hepaticus, ductus cysticus and ductus choledochus were opened and any cestodes present removed. The liver was subsequently cut in slices 5 mm thick. The slices were firmly pressed between the fingers to extrude any remaining tapeworms.

*Transvaal Region, Agrivaal Building, Edmund St., Pretoria.

### RESULTS

The numbers of animals that were infested with *S. hepatica* are summarized in Table 1.

Table 1: THE NUMBER OF ANIMALS INFESTED WITH *S. HEPATICA* AFTER TREATMENT WITH PRAZIQUANTEL AND OXFENDAZOLE

| | Number of animals infested | |
|---|---|---|
| Treatment | Sheep | Goats |
| Praziquantel | 0/2 | 0/7 |
| Oxfendazole | 2/2 | 4/7 |
| Controls | 4/4 | 1/7 |

### COMMENT

The 4 untreated control sheep and 1 of 7 untreated goats were infested but none of the animals treated with praziquantel (15 mg/kg live mass) harboured *S. hepatica.* Oxfendazole was ineffective as both sheep and 4 of 7 goats were infested. The dose of oxfendazole administered was calculated to treat the animals at 5 mg/kg live mass but shortly before the animals were slaughtered we were informed that the preparation (an experimental batch) was 30% under strength; the actual dose administered was thus 3,5 mg/kg. Subsequently J. Berger (Coopers (S.A.)(Pty) Ltd., P O Box 5034, Greenfields, East London Personal Communication 1980 to J.A. van Wyk) treated sheep at 5 mg and at 7,5 mg/kg but both these dose rates were ineffective against *S. hepatica.*

It appears that the incidence of infestation of the goats on the Research Station is considerably less than that of the sheep as only 1 of 7 untreated goats harboured *S. hepatica.* The goats were therefore considered unsuitable for continued anthelmintic tests in this aspect.

## SECOND TRIAL

This trial was conducted in 62 Dorper sheep in a feedlot at Roodeplaat Research Station.

### MATERIAL AND METHOD

Forty-seven wethers and 15 ewes, approximately 100 days old were moved from the natural pasture to the feedlot and divided into Groups A, B and C for the purpose of a feedlot trial. The animals in each of these groups were subdivided into 3 subgroups, viz. 4, 5 and 6, with the aid of tables of random numbers.


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The animals were treated as follows:

*Group 4.* Twenty sheep (15 whethers, 5 ewes), with praziquantel at 15 mg/kg live mass *per os.*
*Group5.* Twenty sheep (16 wethers, 4 ewes) with praziquantel at 7,5 mg/kg live mass *per os.*
*Group 6.* Twenty sheep (16 wethers, 6 ewes) acted as untreated controls.

The animals were all treated on the same day and slaughtered when their live mass was 40 kg, i.e. 15–61 days after treatment. While the animals were in the feedlot they were fed concentrates only and it is unlikely that reinfestation could have occurred.

In an attempt to quantify the numbers of cestodes, chloroform was injected into the bile ducts shortly after removal of the liver from the carcase (P.C. van Schalkwyk & L. Geyser, Smith Kline, P O Bos 38, Isando, 1979). After about 10 minutes the liver was examined as in the first trial.

## RESULTS

The numbers of animals that were infested are summarized in Table 2.

Table 2: NUMBERS OF SHEEP INFESTED WITH *S. HEPATICA* AFTER TREATMENT WITH PRAZIQUANTEL

| | | Number of sheep | | |
|---|---|---|---|---|
| | Dose of Praziquantel | No. in | Positive | |
| Group | (mg/kg) | Group | Number | % |
| 4 | 15 | 20 | 0 | 0 |
| 5 | 7,5 | 20 | 2 | 10,0 |
| 6 | Untreated | 22 | 20 | 90,9 |

## DISCUSSION:

The results in Group 4 confirm the results of the pilot trial. Praziquantel at 15 mg/kg is 100% effective against *S. hepatica.* When the dose is halved efficacy was still high as only 2 out of 20 sheep were still infested in the group treated with praziquantel at 7,5 mg/kg. According to the classification of the efficacy of anthelmintics used in this country this would warrant a classification of Class II or 100% effective in more than 60% of the animals. To attain Class I, i.e. 100% effective in more than 80% of the sheep, only 1 out of 20 animals may be positive after treatment[2].

An attempt was made to determine the lowest effective dose so that the cost of treatment would be reduced. This was, however, not successful as at the lower dose it is not 100% effective. Moreover at 7,5 mg/kg it would cost R2,00 to treat an animal of 30 kg live mass. Since sheep's liver retails for approximately R1,50 and *S. hepatica* is not pathogenic to sheep[3] it would not be economical to treat infested animals.

The attempt to anaesthetise the cestodes and thus remove them intact in order to determine their numbers was not successful. The scolices are firmly attached to the walls of extremely small bile ducts and despite the use of chloroform, did not all become detached. It was therefore impossible to determine accurately the extent of the infestation. From a meat inspection point of view, the number of cestodes present is unimportant as infested livers in this country are condemned irrespective of the number of worms present.

## ACKNOWLEDGEMENTS

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# NEOSTIGMINE-RESPONSIVE WEAKNESS AND GLOMERULONEPHRITIS ASSOCIATED WITH HEARTWORM *DIROFILARIA IMMITIS* INFESTATION IN A DOG

J. VAN HEERDEN*, ANNA VERSTER† AND D.J. GOUWS‡

ABSTRACT: Van Heerden J.; Verster, Anna; Gouws D.J. **Neostigmine-responsive weakness and glomerulonephritis associated with heartworm *Dirofilaria immitis* infestation in a dog.** *Journal of the South African Veterinary Association* (1980) **51** No. 4 251–253 (En) Dept. of Medicine, Faculty of Veterinary Science, University of Pretoria, Box 12580, 0110 Onderstepoort, Rep. of South Africa.

Microfilariae of *Dirofilaria immitis* were found in blood smears of a Dalmation which showed clinical signs of muscle weakness and membranous glomerulonephritis. The muscle weakness responded to treatment with neostigmine and the microfilariae disappeared after treatment with levamisole.

## INTRODUCTION

Infestation of the dog (which is the natural host) with the filarial nematode, *Dirofilaria immitis* causes heartworm disease which is associated with pathological changes in the heart, lungs, kidney and liver and also occasionally in other organs[1,3,7,8,9,13,14].

The adult heartworms lodge in the right atrium, right ventricle, right ventricular outflow tract, pulmonary arteries and venae cavae. Circulating microfilariae are detectable in peripheral blood about 6 months after the animal is bitten by an infested mosquito[3,7,9,13].

The symptomatology, epidemiology and pathology of dirofilariasis in the dog have been described by Ettinger[3], Rawlings et al.[13] and Levine[9].

The parasite occurs in many parts of the world but is rare in Africa[9]. It has been found in dogs on Pate Island off the coast of Kenya[5] and is common in dogs in Northern Kenya[12]. It was also recorded in Malawi (Nyasaland) by Fitzsimmons[4], but has not yet been found in dogs in the Republic of South Africa.

More than 60 different species of mosquito, some of which belong to the genera *Aedes*, *Culex*, *Anopheles* and *Psorophora* act as vectors of the parasite. The principal vector(s) varies in the different geographical regions[9].

This report deals with the finding of microfilariae of *D. immitis* in a dog with a neostigmine-responsive weakness and glomerulonephritis. Since the non-pathogenic parasite, *Dipetalonema reconditum* is fairly common in dogs in the Republic of South Africa (Verster, unpublished data), it is necessary to differentiate between the microfilariae of these 2 worms.

## CASE REPORT

A 4-year old Dalmation bitch with a live mass of 22 kg, was referred to the Department of Medicine, Faculty of Veterinary Science, University of Pretoria. The owner reported intermittent weakness and lameness mainly of the hindquarters during exercise. The dog was imported from Kenya when it was 2 years of age. Prior to this, a veterinarian had treated the dog for heartworm with a drug, the nature of which is not known to us.

On clinical examination the dog was found to be apyrexic and with a good habitus. Walking and/or trotting, however, precipitated weakness which progressed to lameness of the hindquarters. At a slow pace there was a definite shortening of the stride which were followed by attempts to hop. These ended in the dog assuming a "dog-sitting" position. On palpation of the muscles of the hind legs there was an impression of stiffness but no pain could be elicited even on deep palpation. Slight muscle tremors were observed over the hindquarters.

With forced exercise the periods of activity became shorter while the periods of rest became longer. At all times there was an obvious kyphosis during movement.

On one occasion when paralysis had developed after exercise, the dog was injected subcutaneously with neostigmine methylsulphate** at a dose rate of 0.05 mg/kg. Within 3 min she was able to rise and became fully mobile for approximately 8 h.

Haematological and blood chemistry investigations showed her to be mildly anaemic with an elevated serum protein content. The latter was due to a reversal of the albumin-globulin ratio with a tremendous increase in globulin content. Urinalysis showed a proteinuria with a few hyalin casts and epithelial cells.

At the request of the owner the patient was discharged, a tentative diagnosis of myasthenia gravis having been made. Neostigmine sulphate tablets†† were prescribed at an oral dosage rate of 11,25 mg twice daily. Atropine drops‡‡ were supplied to counteract possible muscarinic side-effects. This treatment was continued for 16 days and at its completion no signs of paresis or paralysis could be precipitated by exercise.

Because of the possible auto-immune nature of myasthenia gravis, blood and serum were examined for auto-immune disease. During examination of LE preparations, which yielded negative results, microfilariae were observed.

The dog was readmitted to hospital and numerous microfilariae of *D. immitis* were found in a fresh droplet of blood as well as in blood samples examined by the modified Knott's technique. The criteria used to identify and differentiate this parasite from *D. reconditum* are listed in Table 1.

Radiographic examination of the chest revealed no abnormalities.

The dog was subsequently treated with levamisole§§ in an attempt to kill the microfilariae. The intended dosing programme was as follows[7]:

First week: 2 mg/kg (daily oral dose)
Second week: 5 mg/kg (daily oral dose)
Third week: 10 mg/kg (daily oral dose)

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†Veterinary Research Institute, Onderstepoort.
‡185 D.F. Malan Drive, Nortchcliff, Johannesburg.

**Neostigmine methylsulphate 2.5 mg/ml, Goldfields Veterinary Medical Supplies, Turffontein, Johannesburg.
††Prostigmin, Roche, Isando, Transvaal.
‡‡Eumydrin, Winthrop Laboratories, Mobeni, Durban.
§§Tramisol – ICI, Braamfontein, Johannesburg.


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Table 1: DIFFERENTIATION OF THE MICROFILARIAE OF *D. IMMITIS* AND *D. RECONDITUM*

| *D. immitis* | *D. reconditum* | *Author* |
|---|---|---|
| Fresh Droplet | | |
| Usually numerous | Always few in number | |
| Undulate in one place | Move across field | 15 |
| In stained smear (Modified Knott) | | |
| Few to numerous | Usually low numbers | 10 |
| Appear straight | Crescent shaped, wavy | |
| Straight tail | Buttonhook tail | |
| Tapered anterior extremity | Parallel anterior extremity | |

Levamisole was given in a liquid form in gelatin capsules. The intended dosing programme was interrupted during the 2nd day of the 3rd week of treatment as the dose rate of 10 mg/kg regularly induced immediate vomition. The dosing schedule was then changed in that 10 mg/kg levamisole was injected intramuscularly once daily for ten consecutive days. Each injection was followed by a period of severe shivering and ataxia during which the dog assumed a recumbent, sternal position. The onset of these clinical signs varied from 4–9 min after injection and lasted for a variable period of time.

Before, during and following treatment with levamisole haematological and blood chemical investigations were conducted. These indicated a non-responsive anaemia, as well as an increased serum protein value which was due to a marked increase in gammaglobulin levels. A rise in serum alkaline phosphotase and SGPT-levels were also observed (Table II).

Table 2: RESULTS OF HAEMATOLOGICAL AND BLOOD CHEMISTRY INVESTIGATIONS BEFORE, DURING AND FOLLOWING TREATMENT WITH LEVAMISOLE

| Date | Before treatment | During treatment | | | | After treatment |
|---|---|---|---|---|---|---|
| | 20.9 | 3.10 | 5.10 | 9.10 | 17.10 | 24.10 |
| RBC × $10^{12}$/litre | 4,20 | 3,62 | 3,45 | 3,66 | | 3,7 |
| Reticulocyte % | | | | 0,008 | | 0,012 |
| Ht | 0,32 | 0,30 | 0,30 | 0,32 | | 0,31 |
| Hb g/litre | 94 | 95 | 83 | 77 | | 91 |
| WBC × $10^{9}$/litre | 10,8 | 7,6 | 7,8 | 8,4 | | 11,0 |
| TSP g/litre | 95 | 97 | | | 102 | 94 |
| Albumin g/litre | 17,3 | 21,5 | | | | 19,2 |
| Globulin g/litre | 82,7 | 78,5 | | | | 80,8 |
| SGPT IU/litre at 25°C | 17 | 21 | | | 15 | 94 |
| SAP IU/litre at 25°C | 36 | 93 | | | 170 | |
| Urea m mol/litre | 7 | 12 | 10 | | 7 | 7 |
| Creatinine u mol/litre | | | 103,1 | | 110 | |

Microfilariae were present in peripheral blood up to the 16th day, or last day of oral treatment with levamisole. On completion of the treatment blood was examined at various times of the day and no microfilariae were found. At a later date blood was again examined for microfilariae but none were found.

When the dog was discharged anaemia and proteinuria were still present but otherwise she was in reasonably good physical health. A diagnosis of glomerulonephritis and a guarded prognosis were given to the owner.

Seven months later the dog was readmitted. There was moderate weight loss, depression and lumbar pain. Investigations for microfilariae were negative. Radiographic examination of the chest was negative, but chemical investigations revealed markedly elevated blood urea and blood creatinine levels, as well as severe proteinuria. Despite relevant treatment for these, her condition worsened and euthanasia was performed.

On post mortem examination both kidneys were swollen and whitish in colour. No *D. immitis* or other parasites were observed. Histopathological examination of the kidneys demonstrated a severe diffuse subacute membranous glomerulonephritis with diffuse focal interstitial plasma cell infiltrations.

## DISCUSSION

As there was no evidence of heart and/or lung involvement in this case, it can only be assumed that the patient was successfully treated with a drug effective against adult *D. immitis* before it was brought into the Republic of South Africa. Microfilariae can circulate in the blood stream for a period of more than 2 years after the adults have been destroyed. It is propable that in this case they were responsible for the inducement of the glomerulonephritis. This has been described in *D. immitis* infestation[1,13].

The association of a *D. immitis* infestation, elevated gamma globulin levels and symptoms of myasthenia gravis or a neostigmine-responsive syndrome is an interesting and intriguing observation. Termination of symptoms of myasthenia gravis with anticholinesterase treatment has been reported in the dog[6]. In one instance microfilariae of *D. reconditum* were associated with a neostigmine-responsive weakness in a dog[11].

In the present instance high levels of gamma globulins might have interfered with post-synaptic membrane depolarization at the myoneural junctions. In most patients with myasthenia gravis, antibodies to acetylcholine receptor are present and they may have a pathogenic role in the disease. In an autoimmune attack these antibodies are directed specifically against the acetylcholine receptors and result in a receptor deficit[2].

Hindquarter paralysis or paresis due to myopathy, vascular changes or neural changes induced by *D. immitis* has been described in the dog[8,14]. These changes are usually irreversible and easily discernable on post mortem investigation.

This report is presented mainly to bring to the attention of local veterinarians and authorities the possibility of introducing dirofilariosis into the Republic of South Africa. All imported dogs should be thoroughly examined for the presence of *D. immitis*. The long latent period before microfilariae appear in circulation should also be borne in mind. Introduction and subsequent establishment of the parasite in the Republic of South Africa might be possible via local mosquitoes as genera that are known vectors of *D. immitis,* also occur in the Republic of South Africa.

## ACKNOWLEDGEMENTS

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## BOOK REVIEW

## BOEKRESENSIE

# CLINICAL EXAMINATION OF CATTLE

GUSTAV ROSENBERGER with the collaboration of G. DIRKSEN, H. GRÜNDER, E. GRUNERT, D. KRAUSE and M. STÖBER

Paul Parey, Berlin and Hamburg 1979 pp XVI 453, Figs. 478, Tabs. 52, Publ. Price R84,35

This first English edition is the translation by Roy Mack of the 2nd Edition (1977) of „Die klinische Untersuchung des Rindes". The first German edition of this book appeared in 1964. It created so much interest that it was translated into Spanish, Italian and Polish. After going out of print in 1972, work was begun on a completely revised and updated edition which was then published in 1977. Because of the high standard of the text and the lack of equivalent work in the English language, translation was considered worth while.

The systematic, thorough and effective clinical examination remains the corner-stone of successful clinical diagnosis in bovine medicine. This book greatly contributes towards this, and every student and graduate in clinical veterinary medicine will benefit substantially from it.

The contents of the text are very systematically arranged and start with a section on methods of handling, restraint and sedation. The next section deals with case history and general examination. The final section includes the specific system examination. The contents of this section can be summarised as follows:

(i) Aspects of normal organ physiology and anatomy.
(ii) General and special methods of examination, e.g. palpation; percussion; auscultation; centesis; endoscopy; biopsy. In this respect the text very effectively both re-emphasizes and gives one a fresh look at the value of general methods of examination. Methods for special examination are simple and practical and thus brings it within reach of the general practitioner.
(iii) Examples of pathological states and changes from normal.
(iv) Applicable laboratory examinations on blood, urine, faeces and organ fluids.

S.R. van Amstel

# THE ISOLATION OF *BRUCELLA ABORTUS* BIOTYPE I FROM SEROLOGICALLY POSITIVE REACTORS FOLLOWING ON $S_{19}$ ADULT INOCULATION OF DAIRY COWS

S. HERR and D. ROUX

ABSTRACT: Herr S.; Roux D. **The isolation of *Brucella abortus* biotype I from serologically positive reactors following on $S_{19}$ adult inoculation of dairy cows.** *Journal of the South African Veterinary Association* (1980) **51** No. 4 255–257 (En). Veterinary Research Institute, 0110 Onderstepoort, Rep. of South Africa.

The incidence of infection following adult inoculation is such that this method alone cannot be relied on to control the spread of brucellosis. *Brucella* infection is present in a high percentage of serologically positive cases using the interpretation of titres as applied in South African.One premature and one full term calf born to cows 7–9 months after adult vaccination as well as the placentae, discharges and colostrum were all heavily infected with *B. abortus* biotype I.

## INTRODUCTION

In studies correlating serological with cultural findings on abattoir specimens in bovine brucellosis usually only the serologically positive cases were investigated[2 4 7 8]. No such investigation has been conducted in South African since the introduction of the routine use of the Rose Bengal (RBT) and Complement Fixation (CFT) tests. It is important that on the one hand animals condemned by the tests be proved to be harbouring the infection at least in a high percentage of cases. On the other hand it is equaly important to recognize that a low percentage of infected animals fall in the serologically negative class if any control programme is to have a chance of success. Thus the titre at which an animal is adjudged positive is critical and in this study both positive and negative animals were subjected to cultural techniques to see whether the serological criteria as applied in South Africa correllated well with the cultural findings.

Recent work, in the United States of America in particular, has led to a re-evaluation of adult inoculation as part of brucellosis control[1 3 4 5 9]. There is a tendency in South Africa, however, to inoculate an infected herd and not to apply further control measures, despite warnings as to the dangers inherent in this practice[3]. The incidence of brucellosis following adult inoculation is often in the region of 10 percent[4 7]. In this study an opportunity arose in which the isolation of *Brucella abortus* could be attempted from the discharges, colostrum, milk and aborted and full-term calves of cows which had been inoculated as adults and which had subsequently developed significant serological titres when the complement fixation test (CFT) was applied.

## MATERIALS AND METHODS

### Animals

A herd of 22 Friesland-type dairy cows was presented for investigation in July 1979 after one had aborted. *Brucella abortus* biotype I was isolated from the foetal and placental material of this animal. She was immediately isolated. The herd history was of a single abortion due to brucellosis in July 1978. The cow was immediately slaughtered and the whole herd inoculated with a dose of $4\times10^{10}$ live organisms of $S_{19}$ strain *Brucella abortus* vaccine in December 1978.

There had been no prior inoculation of the animals with this vaccine except in the case of one animal, No. 921, which had been inoculated as a calf.

### Serological Tests

The herd was monitored using the RBT, Serum Agglutination test (SAT), CFT and Milk Ring test (MRT) carried out according to procedures of the Bovine Brucellosis Serology Manual of the Veterinary Research Institute, Onderstepoort (December 1979)*. The criteria of the official Brucellosis Eradication Scheme as applied to adult vaccinated animals are set out in Table 1, and were used to evaluate the serological results. The CFT was used as the definitive test and any animal showing a reaction of 60 IU/ml or higher in the CFT was classed as positive.

Table 1: INTERPRETATION OF SERUM AGGLUTINATION TEST AND COMPLEMENT FIXATION TEST TITRES

| Vaccination History | SAT IU/ml | CFT IU/ml | Evaluation |
|---|---|---|---|
| | ≤ 67 | ≤ 24 | Negative |
| Adult Vaccination with | 80–134 | 30–49 | Suspicious |
| with S19 | ≥ 160 | ≥ 60 | Positive |

### Specimens, Isolation and Typing Methods

Five serologically positive cows together with 5 that showed serological reactions but were classified as negative were slaughtered. Specimens were taken from retropharyngeal, lumbar, iliac and supramammary lymph nodes, spleen, udder and uterus or uterine content if pregnant. Both bacteriological and biological isolation were attempted. Serological tests were carried out on guinea-pigs 3 and 6 weeks after inoculation of suspect material. Details of the isolation and typing procedures are more fully described in a second paper by the same authors[6].

## RESULTS

The results of the RBT, SAT and CFT are presented in Tables 2, 3 and 4. Of the original 22 cows 11 were negative to all tests throughout the period of this investigation and are not reflected in the tables. Initially on 3 July 1979 only one cow, (921), showed a positive reaction while Cow 1216 had a high titre in the SAT but was completely negative in the CFT and was therefore considered a vaccine reaction. As Cow 921 was the only animal that had been inocculated previously, her pos-

*Obtainable from the authors on request


0038-2809/80/04/0255–0257

Table 2: ROSE BENGAL TEST

| | RBT (1979) | | | |
|---|---|---|---|---|
| Cow No. | 3 July | 4 September | 3 October | 8 October |
| 1141 | – | – | – | – |
| 1164 | – | P | P | P |
| 1216 | – | – | P | P |
| 1269 | – | – | – | P |
| 2026 | P | P | P | P |
| 2636 | – | – | P | P |
| 2675 | – | – | – | P |
| 7893 | – | P | – | – |
| 9341 | – | P | P | P |
| 9542 | – | – | P | P |
| 921 | P | ND | ND | ND |

P = Positive, – = Negative, ND = Not Done

Table 3: SERUM AGGLUTINATION TEST

| | SAT IU/ml (1979) | | | |
|---|---|---|---|---|
| Cow No. | 3 July | 4 September | 3 October | 8 October |
| 1141 | – | – | – | – |
| 1164 | – | 23 | 20 | 20 |
| 1216 | – | – | 424 | 424 |
| 1269 | – | – | 20 | 20 |
| 2026 | 160 | 134 | 268 | 320 |
| 2636 | – | – | 34 | 20 |
| 2675 | – | – | – | 23 |
| 7893 | – | 34 | – | – |
| 9341 | – | 34 | 424 | 424 |
| 9542 | – | – | 424 | 424 |
| 921 | 424 | ND | ND | ND |

– = Negative, ND = Not Done
*Note:* A five tube test was applied where the maximum titre was 424 IU/ml

Table 4: COMPLEMENT FIXATION TEST

| | CFT IU/ml (1979) | | | |
|---|---|---|---|---|
| Cow No. | 3 July | 4 September | 3 October | 8 October |
| 1141 | – | – | – | – |
| 1164 | – | 36 | – | 21 |
| 1216 | – | – | 784 | 784 |
| 1269 | – | – | – | – |
| 2026 | – | – | 145 | 98 |
| 2636 | – | – | 60 | 98 |
| 2675 | – | – | – | – |
| 7893 | – | 145 | – | – |
| 9341 | – | 98 | 784 | 784 |
| 9542 | – | – | 784 | 784 |
| 921 | 784 | ND | ND | ND |

– = Negative, ND = Not Done
*Note:* The maximum titre in the test was 784 IU/ml

itive titre was difficult to interpret and because she was a high producer she was kept on in the herd but in isolation. She aborted an 8 month premature calf on 27 August 1979. *Brucella abortus* biotype I was isolated from the placenta, discharges, colostrum and various organs of the calf which was sacrificed for this purpose. This cow was removed from the herd but not before she had a chance to break loose and run through the milking area with the heavily infected placenta still attached.

Serological results on 4 September 1979, 8 days after this abortion, showed that Cows 7893 and 9341 had developed positive titres while Cow 1164 fell in the suspicious category. Cow 1216 still had what was interpreted as a vaccine reaction. On 3 October and 8 October 1979 Cow 7341 showed an eightfold rise in CFT titre while Cow 7893 dropped to negative. Cow 1216 had developed a very high CFT titre and was now classed as positive and no longer as a vaccine reaction. In addition Cows 2026, 2636 and 9542 had now developed positive reactions while the suspicious reactor, 1164, had become negative.

Individual cow MRT's done on 18 and 26 September and 9 October 1979 were positive one or more times in the case of Cows 1141, 1164, 9341 and 9542 as seen in Table 5. *Brucella abortus* biotype I was isolated from the milk of Cows 1164, 9341 and 9542 on one or more occasions. Cow 2026 calved normally on the day of slaughter, she delivered a full term, clinically sound, viable calf which was sacrificed. The placenta, calf, discharges and colostrum were all infected.

Table 5: MILK RING TEST

| | MRT (1979) | | |
|---|---|---|---|
| Cow No. | 18 September | 26 September | 9 October |
| 1141 | P | – | – |
| 1164 | P | P | – |
| 1216 | – | ND | ND |
| 1269 | – | ND | ND |
| 2026 | – | ND | ND |
| 2636 | – | ND | ND |
| 2675 | – | ND | ND |
| 7893 | – | ND | ND |
| 9341 | P | P | P |
| 9542 | – | ND | P |
| 921 | ND | ND | ND |

P = Positive, – = Negative, ND = Not Done

A summary of the final serological diagnosis based on the CFT results of 8 October 1979 is presented in Table 6. Five cows, Nos. 1216, 2026, 2636, 9341 and 9542 were positive reactors. These 5 together with 5 serologically negative animals, Cows 1141, 1164, 1269, 2675 and 7893 were slaughtered and isolation attempted from abattoir specimens. Table 7 summarises these isolation results. All isolates were *B. abortus* biotype I and from 4 of the 5 serologicaly positive animals isolation was successful while a guinea-pig gave a serologically positive response 6 weeks after having been inoculated with organ material from the fifth animal. Of the serologically negative cows successful isolation was achieved in only one out of the five.

Table 6: SEROLOGICAL DIAGNOSIS

| Cow No. | SAT IU/ml | CFT IU/ml | Interpretation |
|---|---|---|---|
| 1141 | – | – | Negative |
| 1164 | 20 | 21 | Negative |
| 1216 | 424 | 784 | Positive |
| 1269 | 20 | – | Negative |
| 2026 | 320 | 98 | Positive |
| 2636 | 20 | 98 | Positive on CFT |
| 2675 | 23 | – | Negative |
| 7893 | – | – | Negative |
| 9341 | 424 | 784 | Positive |
| 9542 | 424 | 784 | Positive |
| 921 | 424 | 784 | Positive |

Table 7: CORRELATION OF SEROLOGICAL DIAGNOSIS WITH ISOLATION RESULTS

| Cow No. | Serological Diagnosis | Isolation at Calving/ Abortion | Isolation from milk | Isolation at slaughter | Guinea-pig isolation | Guinea-pig serology |
|---|---|---|---|---|---|---|
| 1141 | – | ND | ND | – | – | – |
| 1164 | – | ND | P | – | – | – |
| 1216 | P | ND | ND | P | P | P |
| 1269 | – | ND | ND | P | P | P |
| 2026 | P | P[1] | ND | P | P | P |
| 2636 | P | ND | ND | – | – | P |
| 2675 | – | ND | ND | – | – | – |
| 7893 | – | ND | ND | – | – | – |
| 9341 | P | ND | P | P | P | P |
| 9542 | P | ND | P | P | P | P |
| 921 | P | P[2] | ND | ND | ND | ND |

– = Negative, ND = Not Done, P = Positive

1 Full term live calf

2 Live Calf ± 8 months

## DISCUSSION

The fact that the serum of only 2 of the 22 cows had reactions to any of the tests 7 months after adult inoculation agrees to some extent with findings of Worthington *et al.*[11], but probably applies only to animals having no prior contact with the infective agent either as vaccine or field infection. Numerous reports from the field indicate more persistant titres in all tests where calfhood vaccination had preceded the full dose of $4x10^{10}$ organisms as was used in vaccinating these cows.

The occurrence of a 10 percent infection rate following adult vaccination is not uncommon[4 7] and the total of 8 animals infected (this includes the original abortion in July 1979 and the isolation from milk in Cow 1164) gives a 36 percent infection rate in the 10 month period following adult vaccination. To what extent this very high rate can be attributed either to cows latently infected at the time of inoculation or to the abortion and spread that occurred in August 1979, with 5 cows developing titres immediately thereafter, must remain a matter for conjecture. What is abundantly clear, however, is that despite adult inoculation infected material from late abortions and full term calvings is the prime source of infection.

Although the numbers included in his study are too few to make any statistical deductions it is nevertheless heartening that all serologically positive animals proved to be infected. The fact that a single serologically negative animal was infected must be expected to occur in a disease with such a prolonged incubation period.

The value of serologically monitoring a herd is illustrated by the fact that both cows found to be infected at abortion or calving had developed positive titres beforehand, although in the case of Cow 2026 this was only a week or two prior to calving. Both these animals were serologically positive on the day of calving or aborting and although it is generally accepted that some cows may abort and only develop a positive titre some 2–3 weeks later[10], the testing of the cow serologically at the time of abortion or calving will help identify a large percentage of infected animals. This will facilitate the early removal of such animals and is in itself a valuable procedure as it is generally accepted that the greatest spread of organisms in vaginal discharges occurs in the first weeks following on abortion or calving. Nevertheless, it remains true that no cow can be regarded as clear of infection unless she has proved to be serologically negative 2–3 weeks after calving or abortion[10].

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# A CRITICAL EFFICACY TEST OF CAMBENDAZOLE IN EQUIDS: THE USE OF THE GEOMETRIC MEANS TO ASSESS EFFICACY*

J.P. LOUW, SANTA MEYER and J. SCHRÖDER

ABSTRACT: Louw J.P.; Meyer Santa; Schröder J. **A critical efficacy test of cambendazole in equids: the use of the geometric means to assess efficacy.** *Journal of the South African Veterinary Association* (1980) **51** No. 4 259–261 (En) MSD Research Centre, Private Bag 3, 1685 Halfway House, Republic of South Africa.

The anthelmintic efficacy of a 44,5% paste formulation of cambendazole was evaluated in a critical trial performed on 5 horses and a donkey. A dosage of 20 mg/kg showed reduction of mean parasite burdens as follows: Strongylinae >99%; Cyathostominae 94%; immature and adult *Oxyuris equi* 89 and >99%; *Probstmayria vivipara* >99% and *Habronema muscae* 97%. *Parascaris equorum* and *Anoplocephala perfoliata* were present in small numbers in individual animals only, and while all were removed by treatment, the small numbers did not justify calculation of a percentage efficacy for these 2 parasites.

## INTRODUCTION

The efficacy of cambendazole, (isopropyl 2-(4-thiazolyl) -5-benzimidazole carbamate) against nematodes and cestodes was first described in sheep and cattle[5,6,7]. A number of controlled[1,2] and critical[4] anthelmintic trials evaluating the efficacy of cambendazole suspension, paste and pellets in horses have been reported.

This paper reports the results of a critical anthelmintic trial done on 5 naturally infested horses and one donkey, treated with cambendazole 44,5% paste† at 20 mg/kg.

## MATERIALS AND METHODS

### Animals

On 2 different occasions 3 horses, and 2 horses and one donkey selected on faecal worm egg count, were purchased and transported to our laboratory. Worm egg counts were monitored for a number of days prior to day of treatment (Day 0) and continued until slaughtered on the fifth day. On Day 0 all animals were treated with cambendazole 44,5% paste at 20 mg/kg live mass.

### Processing of excreta

Commencing on day of treatment, animals were stabled in separate pens and the total daily faecal output was collected for each animal and processed separately. The first operation was to remove all large parasites, e.g. adult *P. equorum, O. equi,* Strongylinae and *A. perfoliata.* This was done by carefully flaking the faeces with the fingers, and then removing only the large parasites for later counting and identification.

The next step was to collect, on a dry mass basis, the necessary aliquots for macroscopic and microscopic examination. The mass of the total faecal output per animal was determined daily. Of this, two one-tenth aliquots were then collected by mass, washed separately on a sieve with 152 $\mu$m aperture and preserved with 10% formalin for later examination. A further two 1/100 aliquots were collected, washed on a sieve with 38 $\mu$m aperture and preserved with 10% formalin for later examination.

During the whole period between treatment and slaughter the feed intake of the first 3 horses was restricted in an attempt to reduce the amount of faeces expelled. This practice was abandoned in animals treated subsequently.

### Post mortem procedure

At slaughter the different parts of the gastro-intestinal tract were separated from each other for processing. The stomach and small intestine were cut open in separate containers and the mucosa washed thoroughly. The ingesta of these 2 parts were washed *in toto* on a sieve with 150 $\mu$m aperture and preserved in separate jars for later examination. The caecum and colon were

Table 1: DAILY FAECAL WORM EGG COUNTS (EGGS PER GRAM) BEFORE AND AFTER TREATMENT ON DAY 0

| | | DAY | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Animal No. | | −7 | −6 | −5 | −4 | −3 | −2 | −1 | 0 | +1 | +2 | +3 | +4 | +5 |
| Horses | White | 1500 | 1600 | 900 | 850 | 1050 | 350 | 400 | 850 | 400 | 525 | 250 | 0 | 0 |
| | Foal | 1200 | 450 | 350 | 850 | 600 | 650 | 350 | 500 | 750 | 600 | 150 | 0 | 0 |
| | Brown | 400 | 650 | 1700 | 1850 | 1300 | 1900 | 1050 | 3250 | 4100 | 1700 | 150 | 0 | 0 |
| | * | 0 | 950 | 500 | 450 | 450 | 7500 | 350 | 450 | 400 | 350 | 350 | 500 | 200 |
| | 99 | NS | 1150 | 1200 | NS | NS | 1800 | 1300 | 1200 | 1300 | 0 | NS | NS | 0 |
| | 355 | NS | 650 | NS | NS | NS | 1300 | 1300 | 1650 | 550 | 0 | NS | NS | 0 |
| Donkey | 401 | NS | NS | 1600 | NS | NS | 950 | NS | NS | 700 | 0 | NS | NS | 0 |

* – Brown horse parascaris egg count
NS – No sample

*Presented at the 8th Annual General Meeting, Parasitological Society of Southern Africa, Durban, July 17–18, 1979.

†Equiben: MSD


0038-2809/80/04/0259–0261

opened together in a large container and the mucosa washed thoroughly. The ingesta was then made up to a suitable volume by adding water. Two pressure pumps were used to agitate the material by bubbling air through it. During this operation two one-tenth and two 1/100 aliquots were collected and the rest of the material discarded. The one-tenth aliquots were washed on a 150 $\mu$m aperture sieve and the 1/100 aliquots on a 38 $\mu$m aperture sieve. All aliquots were preserved for later examination.

### Worm counting

Total microscopic counts were done on both stomach and small intestinal ingesta. One of the two one-tenth aliquots collected from the large intestine was examined macroscopically and the parasites counted and removed for later identification. One of the 1/100 aliquots collected from this organ was examined microscopically and the worms counted and identified.

The burdens of large parasites (*P. equorum, O. equi* and Strongylinae) were computed by using the macroscopic counts while the burden of Cyathostominae and *P. vivipara* were calculated from the microscopic counts.

For identification the key as compiled by Lichtenfels[8] was used.

### Assessment of anthelmintic efficacy.

The geometric means of the total numbers of each parasite recovered after treatment (i.e. ante plus post mortem worm burdens) were used to calculate the efficacy of treatment. Where an animal was not infested with a particular parasite, that animal was ignored in calculating the geometric mean for that parasite.

## RESULTS AND DISCUSSION

Daily faecal worm egg counts prior to and following treatment are treated in Table 1. These counts (excluding *P. equorum*) became negative 2–4 days after treatment.

Table 2 summarises the worm counts between treatment and slaughter (ante mortem worm counts) and at the time of slaughter. The majority of Cyathostominae still present at slaughter appeared dead and decayed. In a critical test each animal serves as its own control, obviating the need for a uniform parasite burden as is the case in a controlled trial. It is impractical to carry out a statistical evaluation on a trial of this type, and efficacy has to be judged with care where small numbers of a parasite are present.

Strongylinae numbers were reduced by >99%. This result is in agreement with the consistently good results obtained by various authors[1 2 4] although they only included parasites of the genus *Strongylus* in this class. The system suggested by Lichtenfels[8] appears to be a more sensible way of classing morphologically similar genera together under the same sub-family to include the four genera, *Strongylus, Oesophagodontus, Triodontophorus* and *Craterostomum.* In the past many of the so called "small strongyles" were often larger than the so called "large strongyles" and equally dissimilar to the other genera of the small strongyle group of parasites.

Cyathostominae burdens were reduced by 94% even though large numbers were still present in some horses. This reduction agrees with that reported by other workers[1 2 4]. A possible explanation for the large number of these worms still present in some horses could be the fact that these horses (Brown, Foal and White) were slaughtered only 5 days after treatment. In addition their feed intake was restricted, possibly delaying the movement of digested material throught the alimentary tract. Drudge[3] found the action of benzimidazoles in

Table 2: RESULTS OF ANTE AND POST MORTEM WORM COUNTS

| Animal No. | | Strongylinae | Cyathostominae | *P. equorum* | *O. equi* L4 | *O. equi* ad | *P. vivipara* | *H. muscae* | *A. perfoliata* |
|---|---|---|---|---|---|---|---|---|---|
| Ante mortem worm counts | | | | | | | | | |
| Horses | White | 742 | 112 400 | 0 | 400 | 0 | 0 | 0 | 0 |
| | Foal | 174 | 20 730 | 0 | 400 | 0 | 0 | 200 | 0 |
| | Brown | 848 | 122 600 | 3 | 3 800 | 0 | 1 295 000 | 600 | 0 |
| | No. 99 | 1 454 | 12 100 | 0 | 200 | 114 | 1 000 | 700 | 4 |
| | 355 | 0 | 18 000 | 0 | 100 | 0 | 703 000 | 200 | 0 |
| Donkey | 401 | 657 | 28 300 | 0 | 2 100 | 30 | 3 237 100 | 300 | 0 |
| Post mortem worm counts | | | | | | | | | |
| Horses | White | 0 | 4 600 | 0 | 100 | 0 | 0 | 0 | 0 |
| | Foal | 0 | 1 500 | 0 | 300 | 0 | 0 | 0 | 0 |
| | Brown | 65 | 69 050 | 0 | 2 800 | 0 | 100 | 12 | 0 |
| | No. 99 | 0 | 1 200 | 0 | 100 | 0 | 0 | 59 | 0 |
| | 355 | 10 | 100 | 0 | 0 | 0 | 0 | 79 | 0 |
| Donkey | 401 | 0 | 4 400 | 0 | 100 | 0 | 1 500 | 10 | 0 |
| Geom. Mean | | 1,2 | 2 512 | | 77 | 0 | 9,8 | 10,7 | |
| Geom. Mean total (Ante and post mortem) | | 322 | 39 715 | | 731 | 58 | 233 026 | 381 | |
| % Reduction | | >99 | 94 | >99 | 89 | >99 | >99 | 97 | >99 |
| Range % (critical test) | | 0–100 | 63,9–99,4 | 100 | 57,6–100 | 100 | 99,9–100 | 71,7–100 | 100 |

the horse particularly slow. Considering these possibilities and the fact that the majority of the worms still present in these horses at slaughter were dead and decayed, this trial might not reflect the true efficacy of the drug against the Cyathostominae.

*P. equorum* was present in only one of the horses. Here the drug was completely effective. A number of workers have reported exceptional efficacy of cambendazole against *P. equorum* adults and immatures[1,4].

*O. equi* immatures and adults were reduced by 89% and >99% respectively. Only 2 horses harboured adult parasites, which have been said to occur mainly in the younger animal although immatures are common in older horses[3].

*P. vivipara* was present in 4 of the 6 animals but occurred in large numbers. Treatment reduced burdens of this parasite by >99%.

*Habronema muscae* was present in 5 of the 6 animals. Efficacy against this parasite is difficult to assess in a critical trial due to the fact that parasites living in the anterior part of the alimentary tract are usually digested *en route* when killed[4]. This results in a rather unfair picture of the efficacy of an anthelmintic. In this trial an efficacy of 97% was nevertheless recorded.

The *A. perfoliata* which were present in one animal only, were removed completely.

## ACKNOWLEDGEMENTS

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# BOTHRIOCEPHALOSIS IN THE COMMON CARP IN THE EASTERN TRANSVAAL

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ABSTRACT: Boomker J.; Huchzermeyer F.W.; Naudé T.W., **Bothriocephalosis in the common carp in the Eastern Transvaal.** *Journal of the South African Veterinary Association* (1980) **51** No. 4 263–264 (En) Dep. Parasitology, Faculty of Veterinary Science, University of Pretoria, Box 12580, 0110 Onderstepoort, Rep. of South Africa.

The first outbreak of bothriocephalosis in common carp in South Africa is recorded. This condition was caused by *Bothriocephalus acheilognathi,* a pseudophyllid tapeworm not previously identified in this country. Methods to limit its spread are suggested.

## INTRODUCTION

Although more than 70 species of the pseudophyllid tapeworm *Bothriocephalus* occur in freshwater fish all over the world, few species of the genus are pathogenic. Probably the most pathogenic is *Bothriocephalus acheilognathi* (syn: *B. phoxini, B. gowkonensis*)[4], and this species has been responsible for high mortalities in carp.

This tapeworm originally was a parasite of the grass carp *(Ctenopharyngodon idella)* and the silver carp *(Hypothalmichthys molitrix)* in the southern parts of China. With the brisk trade in these 2 carp species it rapidly spread to other countries by means of infested fish, and has adapted itself successfully to the common carp *(Cyprinus carpio)*. It is presently found in most countries in Europe and Asia, in the United States of America and New Zealand[4], and has now also been found in South Africa.

Silver carp were first introduced into South Africa in March 1975, and grass carp in September 1975[5]. Both species of fish are kept at the Lowveld Fisheries Research Station, Marble Hall, Transvaal, to determine their feeding requirements, breeding potential and effect on indigenous fish species. After completion of these investigations their release into impoundments and rivers may be considered. The Research Station at Marble Hall is also a major supplier of fry of the common carp to commercial carp farmers.

This paper reports mortalities due to infestation with *B. acheilognathi* in a commercial carp production unit within the Komatipoort area of the Eastern Transvaal. It is believed to be the first record in South Africa of mortalities in young carp as a result of infestation with *B. acheilognathi.*

## MATERIALS AND METHODS

In April, 1980, a commercial carp producer in the Komatipoort area, Eastern Transvaal, experienced heavy mortalities in young, 150–250 mm long carp. He noted that the abdomens of many of the dead fish were distended, and suspected infectious dropsy of carp to be the cause. To confirm his diagnosis, 24 live carp from the affected pond were presented to the Department of Infectious Diseases, Faculty of Veterinary Science, at Onderstepoort for examination.

Necropsies were performed on all the live carp, and various tissues were collected for bacteriological, virological and toxicological examinations. The water in which the fish was received was also routinely subjected to water quality tests, as it had been collected from the affected pond.

Tapeworms were collected and fixed in 70% ethyl alcohol or boiling glycerine alcohol. Whole mounts of the tapeworms were prepared after staining with Mayr's acid haemalum, differentiating in acid 70% ethyl alcohol and dehydrating in graded ethyl alcohol. After clearing in oil of cloves, the worms were mounted on glass slides in Canada balsam.

## RESULTS

Bacteriological, virological and toxicological tests proved negative, and the mortalities could, therefore, not be related to infectious diseases or toxic substances.

Tapeworms were present in the intestines of half of the fish examined and burdens varied from 2–12 worms. They were from 42,5–195 mm long.

The tapeworms were tentatively identified as *Bothriocephalus acheilognathi* and forwarded to the Tierärtzliche Hochschule, Hannover, West Germany, where the identification was confirmed.

Macroscopic examination of the intestines revealed petechial haemorrhages in the mucosa of the small intestine. In the heavier infestations, the intestine was markedly distended, although no distension of the abdomens of the fish examined was noted. All infested fish, however, appeared stunted in comparison to non-infested fish from the same pond. Blockage of the intestines occurred only in 2 fish, each of which harboured 12 tapeworms.

## DISCUSSION

There is little doubt that *B. acheilognathi* was imported into this country with infested grass and silver carp. This has been found to be the case in all countries in which the parasite has been identified[2].

This cestode has a life cycle that lasts approximately 30–34 days from ingestion of the egg by a small crustacean intermediate host (e.g. *Cyclops*) to recovery of the adult worms[3]. As a number of crustaceans can act as intermediate hosts[3], and as carp ponds are usually fertilized to ensure an abundance of zooplankton on which carp fry feed, very high levels of infestation can be reached in a relatively short time.

During the early stages of the development of the tapeworm in the fish, few symptoms are seen. Should such infested fish be transported to other production units, mortalities may occur at the new venue at a later date. It is then often difficult to determine the origin of the infested fish, especially if the mortalities are not excessive. Fish farmers accept a certain percentage of deaths, and necropsies are not performed on all the fish

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that die. The fish in this case were obtained as fry, partially from the Marble Hall Research Station and partially from another source. It is reasonable to assume that the fry obtained from Marble Hall had become infested at the Research Station, where both grass and silver carp are kept. Some of these common carp fry died a considerable time after they had been received at the production unit in the Komatipoort area.

The parasite seems to be more pathogenic to young carp. Scott & Grizzle[6] found local haemorrhages of the intestinal mucosa, necrosis of the muscularis mucosa and, in severe infestations, blockage and distension of the intestine to be the most common lesions. The live fish received for examination showed some of the lesions described by Scott & Grizzle[6], and probably represented that part of the population that harboured either smaller cestodes or were less severely infested. Those that were severely affected had died on the farm, and were unfortunately not presented for necropsy.

Brandt, Schoonbee & Can As[1] recently found a *Bothriocephalus* spp. in common carp from the Lowveld Fisheries Research Station, Marble Hall. Although they do not provide a specific identification of this worm, it can be assumed to be *B. acheilognathi.* The facts that the farmer from the Komatipoort area obtained carp fry from the Research Station, and that the parasites from his fish were positively identified as *B. acheilognathi,* support this assumption.

The parasite is not difficult to control. Körting[2] advocates the use of niclosamide (Lintex, Bayer) at 500–600 g per 100 kg of dry food, fed at the rate of 1,5 kg per 100 kg of live fish mass, 2–3 times at intervals of 2 weeks. Brandt et al.[1] found that niclosamide mixed into pelleted food and fed at 5% of the total fish mass for 5 consecutive days, resulted in the elimination of the parasite. To control reinfestation, treatment should be repeated after 3–4 weeks[1]. Under semi-intensive and intensive conditions simultaneous control of the intermediate host by the use of trichlorphon in the water might also be considered.

It is concluded that regular examination of fish for the presence of *B. acheilognathi,* together with strict quarantine and regular treatment of all imports, as well as treatment of fish and ponds on affected farms, will do much to contain the parasite and prevent its spread to other parts of the country.

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ABSTRACT: A.S. de Graaf, P.D. Shaughnessy, R.M. McCully & Anna Verster, 1980. **Occurrence of Taenia solium in a Cape fur seal (Arctocephalus pusillus).** *Onderstepoort Journal of Veternary Research,* 47, 119–120 (1980).

The larval stage of *Taenia solium* was revovered from the brain, skeletal muscles, heart, lungs and liver of a Cape fur seal collected near Cape Town. This is apparently the second record of the larval stage of this cestode in a marine mammal.

ABSTRACT: A. Lucia Lange, 1980. **Tissue culture studies on a suspected lysosomal storage disease in Abyssinian cats.** *Onderstepoort Journal of Veterinary Research,* 47, 121–127 (1980).

Cell cultures prepared from the spleen and mesenteric lymph nodes of affected kittens were subjected to histochemical and ultrastructural studies. Macrophages in the cultures contained lipid in the cytoplasm and the ultrastructural studies revealed lysosomes containing lamellae similar in appearance to phospholipids.

ABSTRACT: J.D. Bezuidenhout & C.J. Stutterheim, 1980. **A critical evaluation of the role played by the red-billed oxpecker Buphagus erythrorhynchus in the biological control of ticks.** *Onderstepoort Journal of Veterinary Research,* 47, 51–75 (1980).

*Buphagus erythrorhynchus* uses 4 feeding methods–scissoring, plucking, pecking and insect catching. During the day the birds spend 68% of their time feeding, with peaks of activity during the early morning and late afternoon. A total of 21 641 ixodid ticks were found in 53 stomachs examined, with a range of between 16 and 1 665 per stomach. *Boophilus* and *Rhipicephalus* were the most important genera eaten. Thirty Diptera, also found in the stomachs, accounted for 0,4% of the diet by mass. The food of the nestlings consisted of 45,6% ticks, 19,4% Diptera and 35,0% hair and tissues.

When kept in captivity, *Buphagus* was able to account for an appreciable reduction in the numbers of *Boophilus* on cattle, reaching a figure of 95,7% reduction for adult ticks. In controlled experiments *Buphagus* showed the highest preference for *Boophilus decoloratus, Rhipicephalus appendiculatus* and *Hyalomma truncatum.* The daily food intake of a captive bird was 14,7g, which is equivalent to 7 195 engorged *Amblyomma hebraeum* larvae. Three acaricides, namely, amitraz, chloromethiuron and DDT, did not cause any clinically detectable toxicity in captive birds during a 5-day period.

# EXERTIONAL MYOGLOBINAEMIA IN BLACK WILDEBEEST, AND THE INFLUENCE OF GRADUATED EXERCISE*

A.M. HARTHOORN

ABSTRACT: Harthoorn A.M. **Exertional myoglobinaemia in black wildebeest and the influence of graduated exercise** *Journal of the South African Veterinary Association* (1980) **51** No. 4 265–270 (En) Transvaal Division of Nature Conservation, Private Bag X209, 0001 Pretoria, Rep. of South Africa.

## INTRODUCTION

Myoglobinaemia (and its concommitant myoglobinuria) may be regarded as one of the cardinal signs of the capture myopathy syndrome. It is readily seen in antelopes that have been subjected to unusual stress either in the form of running or after isometric exertion while roped or in a net. The serum level of myoglobin appears to be a reliable indicator of the amount of stress to which the animal has been subjected in relation to its capability to withstand such stress, but also to its survival potential. In relation to the latter it should be said that there is every indication that the animal may be fairly severely stressed with substantial levels of myoglobin in the bloodstream, but survive if released into natural or near-natural surroundings. Such an animal is likely to succumb to myoglobinaemic nephrosis if kept under conditions of close confinement with continuing adrenergic discharge, raised lactic acid levels, and possible dehydration due to unwillingness to drink out of water containers (black wildebeest on farmland during drought condition have been known to die of thirst rather than drink out of drums) and the loss of condition usually associated with holding in pens. Unlike other indicators of exertional stress such as creatine phosphokinase, the myoglobin appears to reach its highest level at the time of capture, which may be the only time that a blood sample may be drawn. For the purposes of these tests, the discolouration of the blood serum has been termed myoglobin. In fact, previous work has indicated that some haemoglobin is also present, but this is being ignored as largely irrelevant for the purpose of this experiment.

An important aspect of the capture myopathy syndrome is myoglobinuria. This is symptomatic of breakdown in muscle fibres resulting on exceptional exertion as occcurs during the capture of wild or para-wild animals. Experimentally the manual restraint of captured Sable antelope will induce a rapid rise in plasma myoglobin, and a severe hyperkalaemia rising in approximately 1 h to lethal levels. Non-acute deaths in wild animals after capture are mostly associated with myoglobinaemic nephrosis. Sublethal myoglobinaemia is a typical occurence in animals such as black wildebeest captured mechanically for experimental purposes whether capture was effected by driving into a plastic funnel, into nets or a combination of the two. When animals were subjected to regular and incremental exercise or training, however, the incidence of myoglobinaemia dropped to undetectable levels or to zero. The regime entailing semi-weekly drives at moderate speed through an open ended capture funnel also induces a reduction in the rise of potassium and of creatine phosphokinase and oxaloacetic-transaminase which under the previous capture conditions had risen to substantially high levels. It is concluded that myoglobinaemia, and its concomitants, myoglobinuria and myoglobinaemic nephrosis, may be prevented by suitable training procedures which are readily carried out in para-wild animals after restriction in suitably sized enclosures and may even be applicable to wild animals after attraction into similar or modified enclosures prior to close confinement and handling.

*Paper read at the South African Veterinary Association Congress, Rand Afrikaans University, Johannesburg, 5 September 1979

## CAPTURE MYOPATHY

### 1. Definition of capture myopathy

Capture myopathy is the term favoured to describe a condition occuring in wild animals on or after capture[2]. Clinical symptoms are signs of pain, stiffness and disability of certain muscles and muscle groups, leading to paresis, torticollis, prostration and paralysis. Death may occur immediately after capture in hyper-acute cases, or up to 12 h later in the acute cases. The principal manifestations are dyspnoea, tachycardia, areflexia, and a disinclination to move. Discolouration of the urine is frequently seen. Characteristic signs are dullness, anorexia, and progressive lack of response to stimuli.

### 2. Outline of the Pathological Picture

The post-mortem picture typically shows pale areas in the muscles that are asymetrically bilateral. The lesions of exertional myopathy are apparently identical with those of nutritional myopathy due to selenium and vitamin E deficiency, and show hyaline degeneration with loss of cross striation in the muscle fibres. Ruptures in the skeletal and cardiac muscle fibres lead to haemhorrages. Where death is not acute there is necrosis and mineralisation, with sarcolemmal proliferation and fibrosis of the muscle tissue. Necrotic lesions are seen in adrenals, liver, spleen, lung and lymph nodes. The kidney usually shows signs of inflammation, necrosis and tubulorhexis.

### 3. Contributing factors

Contributing factors to the condition are: maximal speed of chase, prolonged exertion without rest, fear, handling, manual restraint by bonds or in a net, prolonged stress as in crating or transport, holding in crates or in pens, subjection to fear stimuli over periods of time, such as noise, renewed and even minor stresses, such as repeated moving and transport, nutritional deficiency and hyperthermia.

### 4. The situation on provincial reserves and on game farms

Changes in the methods and the patterns of capture in


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the Transvaal provincial reserves has greatly reduced the capture mortality over the last half dozen years. In 1973 the death rate over the 1–2 thousand animals captured and relocated a year was just under 15%, in 1974 it was under 5%. Last year (1978) total deaths from all causes during capture holding and relocation was down to 1,1%. Contributing factors to the former mortality rate was undoubtedly: the general use of nets for capture with the accompanying long periods of manual restraint, the capture of individuals rather than family groups, individual crating and transport, prolonged holding in slatted pens, further handling and transport after a resting period of a week or so[1] and a precipitative change of fodder. Favourable modifications during the latter years have undoubtedly been: the reduction of the speed of the chase, periodic rests during drives over long distances, almost total absence of handling, the capture of family or herd groups, the transport to the destination almost immediately after capture, with absence of holding or quarantine, the use of plastic sheeting or hessian funnels and corrals, and reduction of fear to the minimum in the subject animals through the elimination of shouting and other noise, and minimal contact between the animals and the capture personnel.

## 5. Exercise as a remedial factor

Notwithstanding this satisfactory reduction in mortality, research has continued in an attempt to formulate a system where stress is reduced to a minimum. There are two principal reasons for this objective. Firstly, mortality is not a satisfactory criterion of the success of a capture method and many animals may survive albeit significantly stressed. Such animals may die if, during a similar exercise, environmental factors are not so favourable, or if other stressing factors are added such as longer holding or quarantine. Secondly, the method of driving using a helicopter and plastic sheeting requires highly trained and knowledgeable teamwork. Consequently it has proved very much less successful in the hands of game farmers and others who have attempted to use it, so that a method more suitable for the relatively inexperienced or less well equipped is necessary. Other and relatively minor aspects are the escalating expense of a helicopter, and the pressure of time whenever such is used. Also the problems of handling and holding newly captured stock and so on, including the difficulties associated with the sudden change in food when animals are caught off the veld and then held in stockades.

Examination of newly caught animals indicates that during capture, wild animals are subjected to a degree of exercise stress that is both unusual and unnatural, and that the ability to contend with such stress has no survival value under natural conditions. Symptoms exhibited by animals after an intense chase at (their) maximal speed or pace were clearly analagous to that of azoturia in untrained or rested horses. Animals captured on game farms or on provincial reserves are even less well adapted to sudden and intense exercise than those captured in the more natural areas or in National Parks of Africa, and which also exibit capture myopathy. The hypothesis that wild and para-wild animals are basically unadapted to prolonged running and consequently suffer from stress and myopathy when stampeded over substantial distances by car or helicopter was substantiated by running eland on an exercise track. These animals exhibited spectacular drops in levels of stress indicators such as creatine phosphokinase (CPK) following exercise as compared to the first run (see Fig. 1). On the basis of these results, which were substantiated in other species of ungulates, it was felt that: on the one hand any prolonged and fast run should be avoided, and on the other the animals would be adapted to eventual capture. This was achieved by a process of mock capture.

## 6. Mock capture as an element in training and taming

The basis of training and taming is that of mock capture, in which the animals are subjected to runs through a double-ended capture funnel. This procedure can be carried out with a minimum of coersion while the end of the corridor or funnel remains open and the animal are induced to pass through without driving. This exercise permits familiarisation with the plastic sheeting and capture module, and also trains the animal physically through a series of incremental runs. It appears that the passage by the animals through the funnel and corridor module on a number of occasions, without actually being subjected to capture or stress, has a taming effect in that they are familiarised with the process and possibly impressed with its seemingly inocuous nature. In any case the animals subjected to this process became tamer and less inclined to panic, and are able to tolerate subsequent close confinement better than animals not so conditioned.

## 7. The mechanics of mock capture

### *(a) Enticement into enclosures*

The basic module for the taming and training procedure has an area of 10 ha and measures 400 × 250 m. Where the extent of the enclosure in which the animals are to be caught or are kept is considerably larger, or in the case of animals in parks, modules of approximately this size are built and the animals are attracted into these in a number of ways. The enticement procedure is most easily carried out by means of water if other watering points in the area can be rendered unavailable. Otherwise the animals may be attracted by means of salt, lucern or other hay, or by standing crops such as sunflower. Under ideal circumstances the animals are permitted to enter the enclosure freely and remain undisturbed for weeks or even for months, or possibly on a permanent basis throughout the year until capture is required. When the correct group or composition of animals has entered and capture is desired, the gate(s) is closed.

### *(b) Dimensions and construction of capture corrals*

Depending on the species of animals to be captured, the sides of the corral is constructed of game fencing with or without wire mesh. The fence may or may not have to be lined with plastic material. The type of construction depends not only on the species of animal, but its familiarity or otherwise with fencing. It is difficult to lay down hard and fast rules. Recently newly captured impalas were released into a 40 ha enclosure

FIG.1

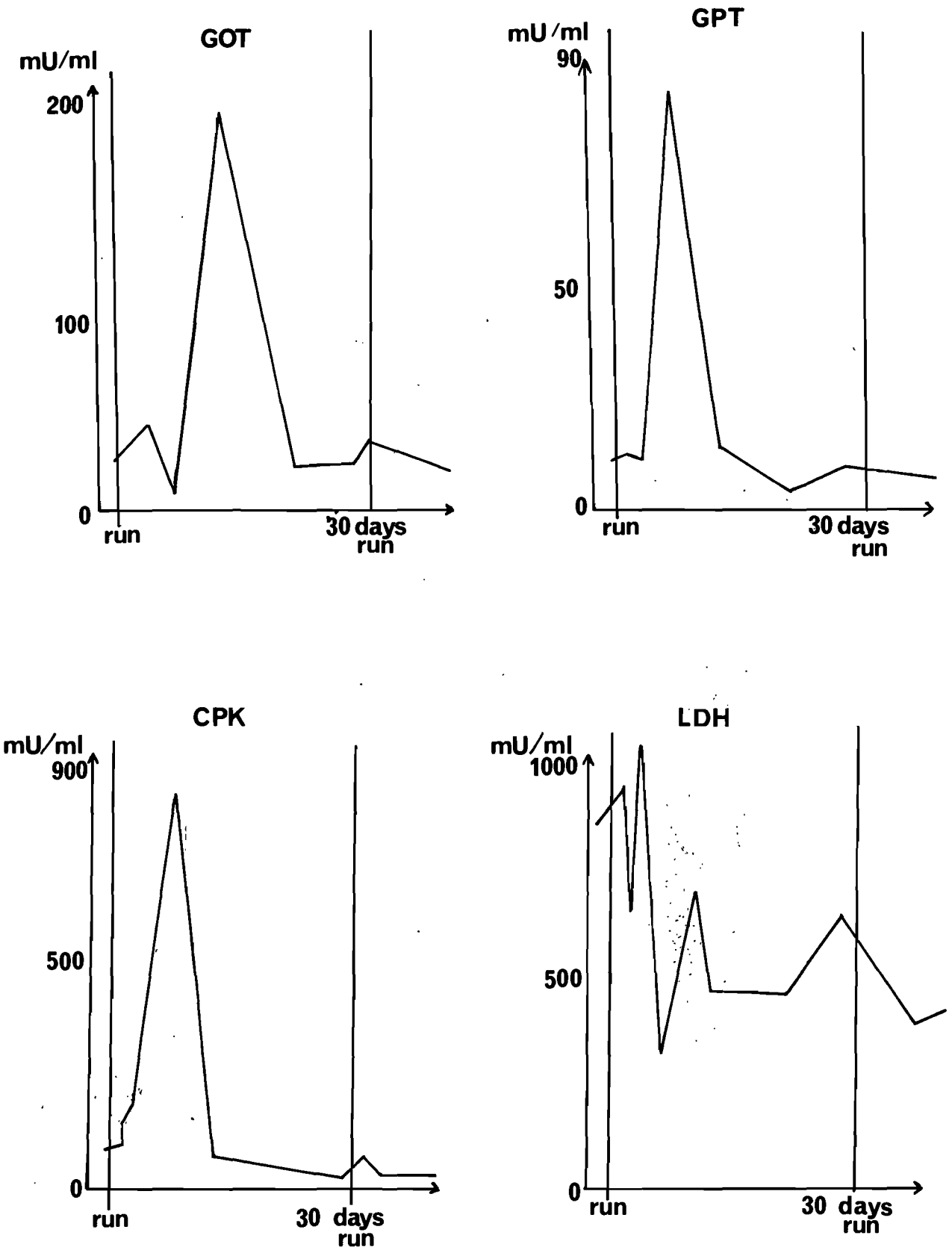


**Fig. 1.** Levels of GOT, GPT, CPK, and LDH before and after exercise training

composed of wire mesh, and surprisingly none came to harm or tried to jump through the fence, while blesbok, generally regarded as a much more tractable animal, broke through fencing of a capture corral. It may be mentioned that whereas the cost of such a structure as described may be substantial, it is a capital asset, and should be useable for a decade or more without additional expense apart from minor maintenance. The ideal structure is composed of 4 paddocks or corrals each of 10 ha, but two such modules placed end to end will suffice. With two modules the animals are enclosed in

FIG.2

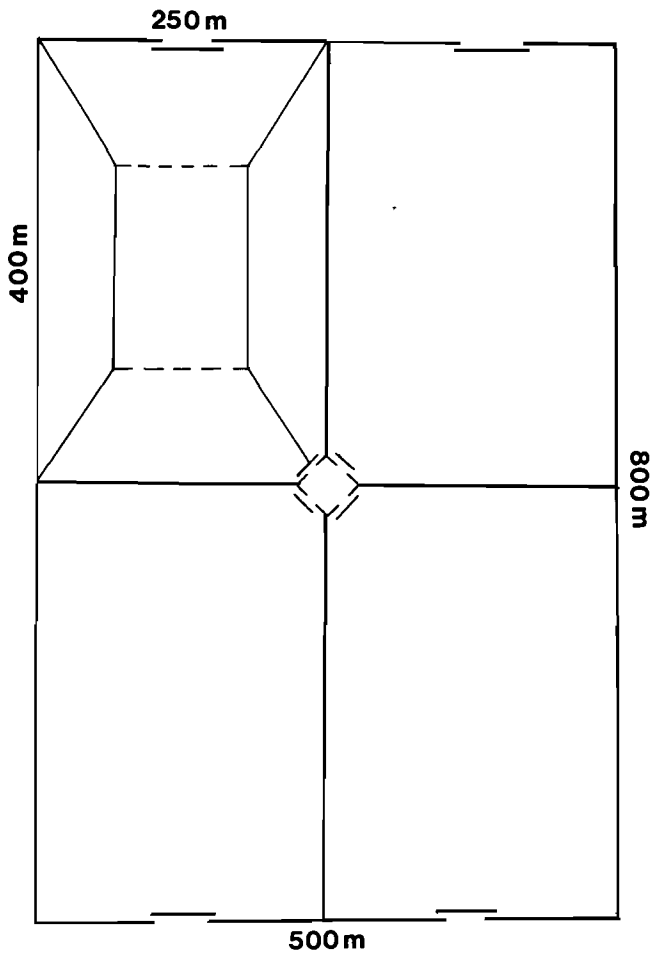


**Fig. 2.** The corral system used for the capture and subsequent taming and training of antelope.

one and the structure for taming and training is built in the other. Where four modules are built as in Fig. 2, the central system of gates gives scope for segregation of groups of animals, or continuation of the capture exercise without disturbance to animals already enclosed.

*(c) Plastic sheeting training structure*

The training structure is usually composed of polypropylene woven fibre plastic sheeting of a neutral colour. This may be 1 or 2 m high depending on the species to be driven through, for instance 1 m for black wildebeest *(Connochaetes gnu)* and 2 m for Sable antelopes *(Hippotragus equinus)*. The plastic sheeting is fixed by short lengths of wire to horizontal strands of fencing wire connecting poles placed about 10 m apart. The double funnel and corridor system is depicted in Fig. 2. Each of the two funnels should connect with the long sides of the corral fence so that the animals are forced to travel through the funnel and corridor each time they are disturbed. In such a structure it is very easy to induce the animals to move or run through the funnels and corridor with the minimum of coersion. The period of training during both experimental and practical tests has been 1 month or slightly longer, with runs being effected twice a week.

*(d) Capture techniques*

The connecting corridor is equiped at each end with a plastic curtain on runners (top and bottom) that may be rapidly closed. When actual capture is required, the animals are induced to run through as usual, and one curtain is closed just before their arrival at the one end. As they turn the other curtain is also closed. A truck may then be backed to one end of the corridor for loading.

By the end of the month, the animals are thoroughly adapted to most of the procedure. A normal group of animals will have been caught which have had further opportunity to react to each other on a herd basis. The strangeness of the plastic material or hessian will now have minimal impact, the animals will be in a good nutritive state, and used to the type of fodder that is being fed in addition to the natural grazing. The truck may be dug down to facilitate loading, and the ramp and floor covered with earth, litter, and preferably also the animals' dung. The tempo of the final chase need now be no more than that of any practice run, so that the animals should suffer from no exertional stress.

## 8. Results

The effect of one monthly capture and a period of bi-weekly exercise on black wildebeest was determined on the basis of the presence of intravascular myoglobin, to obviate the need of repeated capture and bleeding which is necessary when determining the rise in indicators such as CPK. The results are depicted in Fig. 3. When capture was carried out monthly there was little taming effect, and the serum myoglobin levels fell only gradually. When the animals were subjected to bi-weekly runs through the plastic funnel module, the myoglobin fell rapidly to zero or to undectable levels.

It is clear from the levels of myoglobin, that the effect of the taming and training programme is not permanent, and when a month was subsequently allowed to elapse without the bi-weekly runs the myoglobin levels rose again as the animals ran faster and were less easy to handle, for sampling.

## 9. Advantages

The obvious advantages of this capture method is the reduction in stress as indicated by levels of CPK, etc. but more particularly by the reduced levels of myoglobin in the blood serum. There are, however, a number of concurrent advantages which may be especially valuable to the game farmer, or to the staffs of animal parks which have a smaller routine in animal capture than do the provincial reserves. These are the following:

(a) Most or even all the capture work may be carried out by the personnel immediately concerned with the day to day care of the animals.

FIG. 3

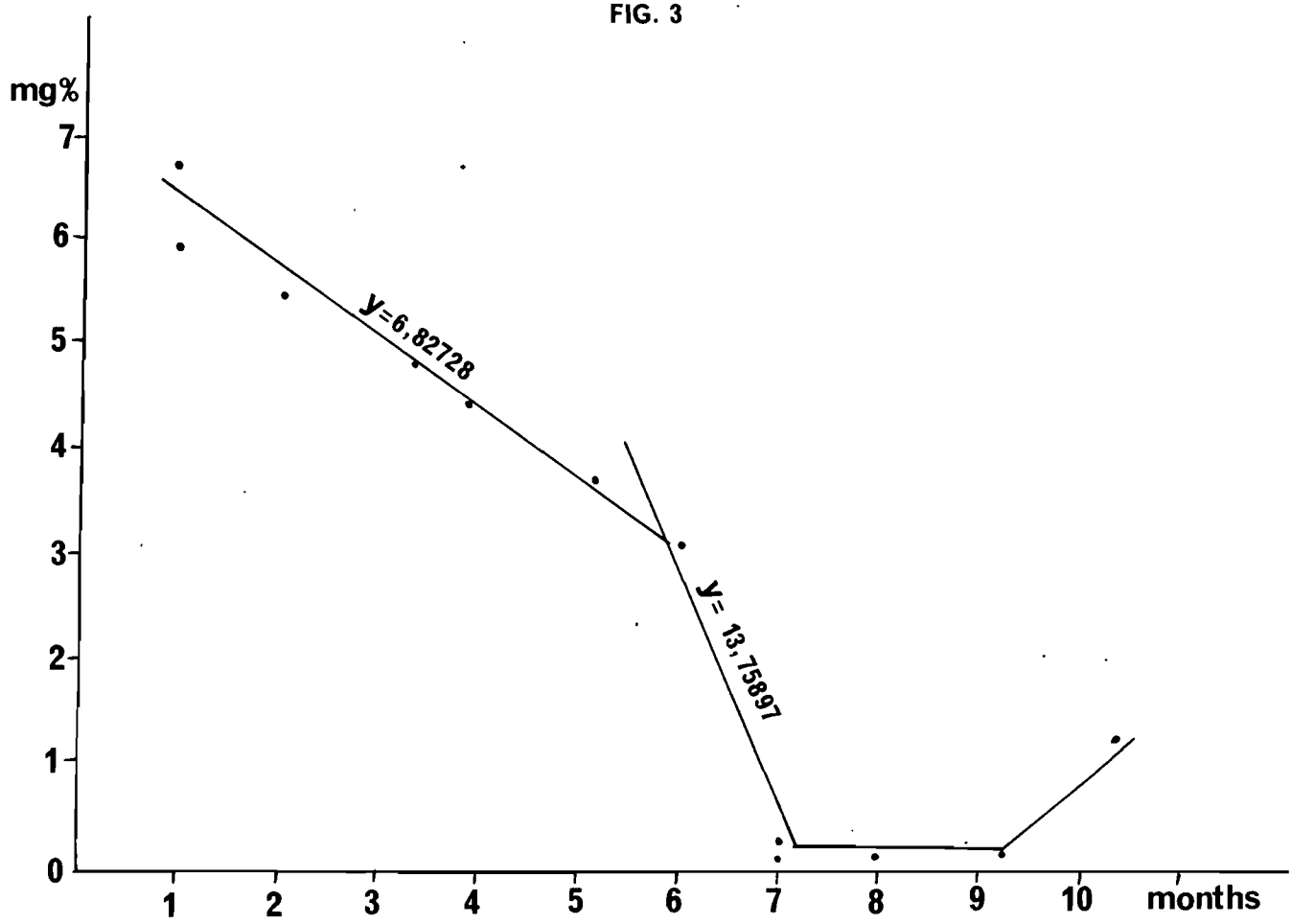


**Fig. 3.** Levels of free myoglobin in black wildebeest firstly during monthly capture and secondly after semi-weekly training runs.

(b) The enclosure of the animals can be carried out both without specialised teams but also with the minimum use of vehicles or the use of a helicopter.

(c) The timing or urgency of the capture operation becomes far less than is the case if a helicopter and a specialised team is hired, and also the animals can now be enclosed well before a critical day such as an auction, as the holding for relatively long periods becomes not only less of a problem, but even an advantage.

(d) The animals do not deteriorate on holding as is the case where they are driven and then placed in pens, in fact the holding in 10 ha enclosures tends to improve the nutritional status of the animals. Also the animals are accustomed over a period of a month or so to other types of food, lucerne hay or concentrates thus obviating a sudden change of diet. As in this system there is usually no period during which animals refuse food and/or water as is frequently the case of animals which are driven and held in pens.

(e) The capture preparation may now be spread over a much longer part of the year, as hyperthermia is less likely to occur. It thus no longer becomes necessary to drive animals during the latter part of winter when they are in poor condition, a factor responsible for a proportion of the mortalities in the past. Animals may in fact be induced to use the enclosures all the year round and be enclosed virtually at any time.

(f) The cost factor should be reduced over several years of capture operations, and become very much more predictable as the recurrent expenses are very small.

(g) The losses in stock (which have been well over 50%) and have been a heavy burden for game farmers, should now be much reduced whether the animals are captured for sale or relocation, or for the meat market. This method of capture has already proved a valuable asset for cropping both in wild areas and on game farms, and the enclosed animals are easily killed at an optimum time during the hours of darkness using a light and a light calibre rifle.

(h) The facilitation of capture should be an encouragement to putative game farmers and encourage the acquisition of game animals on farms.

## DISCUSSION

Myoglobinaemic nephrosis was recorded as a constant symptom in almost all animals that died in pens from 1–6 weeks after capture. The wildebeest in this experiment did not show symptoms of nephrosis suggesting that certain levels of myoglobin in the bloodstream may be tolerated if the animals are released in natural or near-natural surroundings, and may be associated with the degree of sympathetic tonus, lactate levels in the blood, normal exercise and so forth. The apparent tolerance to myoglobin of animals that are not confined suggests that a much lower mortality can be expected when animals are released soon after capture instead of being confined to stalls for periods of time before being moved to new surroundings, and this tallies with the

more recent routine of the capture unit whereby animals are moved within 24 h whenever possible and the sharp reduction in the death rate that has been achieved.

The increasing popularity of game farming or the stocking of sections of farmland with game animals such as blesbok *(Damaliscus dorcas)* brings such animals more closely within the province of the veterinarian. Both from the game farmers' as well as the veterinarians' viewpoints, it appears advantageous to evolve a system of capture that can be described in detail, is fairly simple to operate, is of a clearly defined cost structure, and which can be operated without specialist expertise. Such a system also becomes necessary for the veterinary treatment of such animals, and this is likely to become increasingly necessary as these animals are kept in confined surroundings and on farm land.

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## BOOK REVIEW — BOEKRESENSIE

### PARASITOLOGY FOR VETERINARIANS

J.R. GEORGI with a chapter on antiparasitic drugs by V.J. THEODORIDES
W.B. Saunders Co, Philadelphia, USA 1980 pp VI + 460, Fig. 270, Tab. 5, Publ. price £18=50

There are 3 parts:

Part 1 Fundamentals of Parasitology (Chapters 1–9)

The arthropods are introduced in Chapter 1 and the arthropods and helminths Chapters 2–8 are well-described. The sure hand of Ron Fayer is present in Chapter 9 on the Class Sporazoa but the other protozoa are superficially dealt with.

Part 2 Diagnosis of Parasitism (Chapters 10–15)

I might well give the author a few tips on an examination post mortem (Chapter 14) but the rest of this part is excellent, particularly Chapter 15 which contains the host parasite list which the author has illustrated.

Part 3 Clinical Parasitism of Domestic Animals (Chapters 16–19) In Chapter 16 I merely mention in passing that Tom Miller (1966a, b, 1971) spent many years of his life working on *Ancylostoma caninum* and *A. braziliense* and developed a vaccine which was commercially available in the USA. Anna Verster (1965) published a monograph on *Echinococcus granulosus* and in Africa at least, hydatid cysts in cattle are fertile not sterile. I doubt whether the treatment of cats for cestodes with arecoline hydrobomide or nitroscanate is ever used with safety nor are any chlorinated hydrocarbons (e.g. lindane) or organic phosphate insecticides used in cats.

The reviewer does not agree with many of the statements and hypotheses propounded by Prof. Georgi in Chapter 17, particularly those concerning immunity and hypobiosis in nematodes.

In Chapter 18 p 386 it should be noted that thiabendazole paste (1 g thiabendazole 5 g petroleum jelly) kills *Habronema* larvae and Ivamectin 0,2 mg/kg or Nitroxynil 20mg/kg are effective against *Parafilaria bovicola.* Taffs (1959, 1964a, b, c) who has done classic studies on *Ascaris suum* should have been mentioned in Chapter 19.

In Chapter 20 by V.J. Theodorides there is a comprehensive list of antiparasitic drugs particularly anthelmintics. Like all out colleagues in the USA he has my deepest sympathy. The Food and Drug Administration (FDA) and the Environmental Protection Agency have done their best to smother any initiative our colleagues may show even forcing Theodorides (no doubt for his own protection) to state on p 399:

> "If there is any disparity between the information presented in this chapter and that printed on the label or package insert, *always* follow the manufacturers recommendations"

Many of the excellent compounds in this chapter are synthesized in the USA and the benzimidazoles particularly have very limited use or are not approved by the FDA. Oddly enough arsenic which you cannot destroy and the most toxic of all anthelmintics, nicotine sulphate are registered as cestocides but the best drug for cestodes praziquantel is not approved. For *Fasciola* hexachlorathane is acceptable, but less toxic compounds such as nitroxynil, rafoxanide and oxyclozanide are not registered.

I think the author might warn his readers that albendazole at 25mg/kg 3 times a day for 5 days for horses can be toxic.

In my opinion this book is well worth buying and a useful addition to any veterinarian's library.

R.K. Reinecke

# STUDIES OF FELINE BABESIOSIS 3. HAEMATOLOGICAL FINDINGS

G.J. FUTTER*, P.C. BELONJE† AND A. VAN DEN BERG†

ABSTRACT: Futter G.J., Belonje P.C., van den Berg A., **Studies on feline babesiosis 3. Haematological findings.** *Journal of the South African Veterinary Association* (1980) **51** No. 4.271–280 (En) 22 Blue Route Centre, Tokai Road, 7945 Retreat, Rep. of South Africa.

Haematological changes were monitored in 20 experimentally infected and 70 clinical cases of feline babesiosis. There was a rapid drop in the haematocrit, haemoglobin and erythrocyte count. Experimental animals were most anaemic about 3 weeks after infection. Variations were encountered. There were no definite trends in the erythrocyte indices although the erythrocytes were often macrocytic and hypochromic. No significant changes were found in total leukocyte counts.

## INRODUCTION

As Futter & Belonje pointed out in a review article on feline babesiosis[3], little has been published on the haematology of domestic cats with babesiosis. For this reason we conducted an in depth study on the haematological changes in 20 experimental artificially infected cats and 70 clinical naturally infected cases.

## MATERIALS AND METHODS

### Animals

The 20 experimental artificially infected (Table 1) and 70 clinical naturally infected cats were the same as detailed in a previous article[4].

### Data collected from experimental animals

Blood samples: Free flowing blood samples were collected from mainly the jugular and occasionally the cephalic vein using a lightly heparinised syringe. The syringes had been flushed with a solution of 200 IU heparin/ml normal saline. Samples were collected twice weekly, but not necessarily on the same days. In 5 of the cats, samples were collected 2 weeks prior to splenectomy.

Haematocrits (Ht) were determined twice weekly using the standard micro-haematocrit method.

Haemoglobin (Hb) concentrations were determined photometrically twice weekly by the standard cyanmethaemoglobin method using Drabkin's solution.

Blood erythrocyte counts were determined once weekly by means of an improved Neubauer counting chamber using Hayem's Solution for diluting the samples.

Blood leukocyte counts were determined once weekly by means of an improved Neubauer counting chamber using Tuerk's Solution as a diluent.

### Data collected from clinical cases

Blood samples were taken as described above.

Ht was determined using standard heparinised Wintrobe tubes and centrifuged in an MSE Minor centrifuge at 3 000 rpm for 35 min.

Hb concentrations were determined by the standard

Table 1: DESCRIPTION OF CATS, SOURCE OF INFECTION AND SUBSEQUENT LIFESPAN

| Cat | Sex | Age | Origin of infection | Number of days from inoculation until death | Remarks |
|---|---|---|---|---|---|
| 1 | Female | Adult | Clinical case from Cape Point | 114 | Splenectomised – Main source of infection for experiment |
| 2 | Female | Adult | | 118 | |
| A | Male | Adult | Cat 1 | 12 | Splenectomised trial run |
| B | Female | 6 weeks | Cat 2 | 32 | Splenectomised trial run |
| C | Male | Adult | Cat 1 | 73 | Splenectomised experimental |
| D | Female | Adult | Cat 2 | 35 | Splenectomised experimental |
| E | Female | Adult | Cat 1 | 18 | Splenectomised experimental |
| F | Male | Adult | Cat 2 | 14 | Splenectomised experimental |
| G | Female | Adult | Cat 1 | 27 | Splenectomised experimental |
| H | Female | Adult | Cat 2 | 21 | Splenectomised experimental |
| I | Female | Adult | Cat 1 | 21 | Splenectomised experimental |
| J | Female | Adult | Cat 2 | 14 | Splenectomised experimental |
| K | Female | Adult | Cat C | 45 | Splenectomised experimental |
| M | Pregnant Female | 6 months | Cat D | 20 | Splenectomised experimental |
| N | Female | 4 months | Cat D | 53 | Non-splenectomised experimental |
| O | Female | 4 months | Cat D | 53 | Non-splenectomised experimental |
| P | Female | 8 months | Cat D | 49 | Non-splenectomised experimental |
| Q | Male | 8 months | Cat D | 39 | Non-splenectomised experimental |
| R | Female | 10 months | Cat D | 42 | Non-splenectomised experimental |
| S | Pregnant Female | Adult | Cat C | 38 | Non-splenectomised experimental |

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0038-2809/80/04/0271–0280

cyanmethaemoglobin method and read on an Ames blood analyser.

For the purpose of this study the clinical cases were grouped into 3 categories:

Group 1: Cats with Ht greater than 0,16
Group 2: Cats with Ht 0,13 to 0,16
Group 3: Cats with Ht less than 0,13

### Statistical methods

These were performed as outlined by Snedecor & Cochran[12].

## RESULTS

### Experimental Animals

#### Haematocrit

*(a) Splenectomised cats (Table 2).*

A rapid drop in the Ht was recorded after infection in all the cats, with the mean Ht falling from 0,29 on Day 0 to 0,09 on Day 21. By this stage, 6 of the cats had already died. From Day 21 onwards for the remaining cats the mean Ht rose slightly and fluctuated between 0,10 and 0,13. On Day 35 Cat D, with a Ht of 0,06, was euthanatised – this was the lowest recorded Ht at this stage of the experiment. As shown in Table 2 the lowest Ht measured for each cat following infection ranged from 0,06 to 0,10. On Day 45, 3 Cats (C 1 and 2) were still alive and the mean Ht was 0,13. From Day 45 onwards the Ht of these 3 animals fluctuated from 0,11 to 0,20 with the terminal Ht being 0,05, 0,06 and 0,07 on Days 73, 114 and 118 respectively.

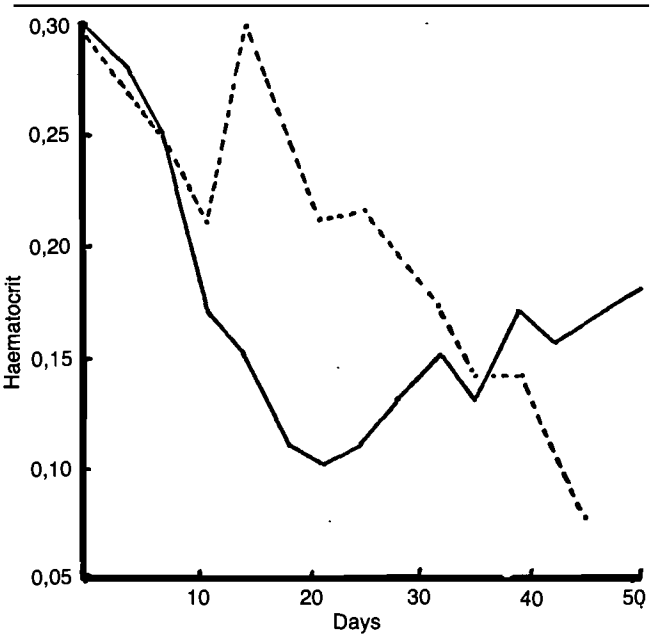


**Fig. 1.** Mean haematocrit in non-splenectomised and splenectomised cats inoculated with *B. felis* infected blood.

*(b) Non-splenectomised cats (Table 3)*

A drop in the Ht was also recorded in the non-splenectomised cats. There was, however, a large variation in the Ht between individual cats. Cats Q and R did not conform to the general pattern and in fact their Ht rose at the time when those of the other cats were falling rapidly. In Cat R the Ht dropped from 0,21 on Day 39 to 0,05 on Day 42. The mean Ht prior to inoculation was 0,31 and the lowest mean Ht, which was recorded on Day 21, was 0,14. The lowest Ht measured for each cat following infection ranged from 0,05 to 0,09.

#### Haemoglobin concentration (Hb)

*(a) Splenectomised cats (Table 4)*

The Hb levels closely followed the drop in Ht. The lowest individual Hb values varied from 1,7 to 3 g/d*l*. The lowest mean Hb value of 2,5 g/d*l* was recorded on Day 28. From Day 45 onwards the Hb of Cats C 1 and 2 fluctuated between 2,7 and 6,6 g/d*l* with the terminal Hb being 1,0; 1,8 and 2 g/d*l* on Days 73, 114 and 118 respectively. It is significant to point out that the Hb of Cat C dropped 3,4 g/d*l* during the final 3 days and that of Cat 1 dropped 4,5 g/d*l* during the final 7 days.

*(b) Non-splenectomised cats (Table 5)*

The Hb levels again closely followed the pattern found in the Ht. The lowest recorded individual Hb values varied from 1,9 to 2,5 g/d*l*. The mean Hb prior to inoculation was 9,7 g/d*l*. The lowest mean Hb of 4,1 g/d*l* was recorded on Day 35. It is interesting to note the sudden decrease in Hb in Cat R which fell from 5,1 to 1,9 g/d*l* in the final 3 days.

#### Blood erythrocyte count (RBC)

(a) Splenectomised cats (Table 6)
The drop recorded in the erythrocyte counts was similar to that of the drop in Ht and Hb values. The lowest recorded erythrocyte count for the individual cats varied from 0,9 to 1,9 × $10^{12}/l$. Erythrocyte counts were not performed on Cats 1 and 2.

(b) Non-splenectomised cats (Table 7)
The pattern for erythrocyte counts once again followed the trends in Ht and Hb. The lowest erythrocyte counts in individual cats ranged from 0,7 to 2,2 × $10^{12}/l$.

#### Erythrocyte indices

1. Mean Corpuscular Haemoglobin Concentration (MCHC)

*(a) Splenectomised cats (Table 8)*

The MCHC fluctuated throughout the experiment, but did tend to fall when the animal was near terminal stage and/or in the initial haemolytic crisis

*(b) Non-splenectomised cats (Table 9)*

A similar pattern was recorded for the non-splenectomised cats with the lowest mean MCHC being 28 g/d*l*. In general the MCHC was higher with less fluctuations.

2. Mean Corpuscular Haemoglobin (MCH)

*(a) Splenectomised cats (Table 10)*

An increase in the MCH was noted in the splenectomised cats. This increase occurred irregularly and no obvious pattern could be determined. The mean of the highest values was 19,7 ± 3,8 pg. Cats C and D, however, demonstrated the rise on Days 21 and 28 which occurred at the same time as the initial haemolytic crisis. Cat H (13,9 – 25,0 pg) and Cat 1 (13,9 – 23,0 pg) showed a significant rise in the MCH during the course of the disease.

*(b) Non-splenectomised cats (Table 11)*

In these cats a similar fluctuation was observed. The mean of the highest values was 21 ± 4,1 pg. Cat N showed a MCH of 22,9 pg at Day 24 and on Day 49 a figure of 12,3 pg was recorded. The highest figure recorded in this group was 27,1 pg in Cat R, on Day 42, the day the animal died.

3. Mean Corpuscular Volume (MCV)

*(a) Splenectomised cats (Table 12)*

In these cats significant increases in the MCV were noted. In Cats C and D the rise was parallel to the increase in MCH on Days 21 and 28 with a return to normal figures on Day 35. The greatest increase was recorded in Cat M with MCV rising from 54 f*l* on Day 0 to 102 on Day 20.

*(b) Non-splenectomised cats (Table 13)*

In comparison to the splenectomised cats no distinct difference was seen in the MCV of non-splenectomised cats. Cats O and S showed no increase in their MCV for the entire experiment. The values for the other cats fluctuated but never rose to the same degree as the splenectomised cats. The highest valued recorded in this group (79 f*l*) was on Day 24 in Cat N.

Blood leukocyte count (WBC)

*(a) Splenectomised cats (Table 14)*

In these cats the total leukocyte counts fluctuated considerably. Extreme counts ranged from 1,7 to 128,0 × $10^9/l$, but in general, although slightly raised, the majority of counts was within the normal range.

*(b) Non-splenectomised cats (Table 15)*

The total leukocyte counts in these cats showed similar fluctuations as to those in the splenectomised group. Extreme counts ranged from 3,5 to 37,2 × $10^9/1$.

Effect of splenectomy in experimental cats

Table 16 shows the comparison of the values prior to splenectomy and then again 14 d later. As can be seen there was a significant change ($p<0,01$) in the mean Ht, Hb and RBC. A moderate change ($p<0,05$) in the MCHC, MCH and WBC was recorded.

**Clinical cases**

Haematology at initial diagnosis (Tables 17, 18 and 19)

*(a) Haematocrit*

The vast majority of the 70 cats had an Ht of less than 0,25 with a variation from 0,09 to 0,40.

*(b) Haemoglobin Concentration*

The levels closely followed the Ht levels in the 3 groups. The Hb level in the majority of cases was below 7,5 g/d*l* with a variation from 2 to 12,5 g/d*l*.

*(c) Mean Corpuscular Haemoglobin Concentration*

The mean MCHC of the 3 groups was similar (29, 30 and 30 g/d*l*). Individual variations were also noted within the groups, the greatest individual variation occurring in Group 3 (22–35 g/d*l*).

Haemotology during recovery (Tables 17, 18 and 19)

It was possible to take blood from 34 treated cats in the recovery stage of the disease. The time lapse between the initial sampling and the second was not the same in all cases.

*(a) Haematocrit and Haemoglobin Concentration*

A rapid recovery to higher Ht and Hb levels was recorded with an average daily increase in the Ht and Hb of 0,018 and 0,5 g/d*l* respectively.

*(b) Mean Corpuscular Haemoglobin Concentration*

The mean MCHC calculated during the recovery stages in the 3 groups were similar to each other (Group 1: 29, Group 2:28, Group 3:30) and to the initial values shown above.

## DISCUSSION

Blood Erythrocyte Values

The Ht of all the splenectomised and 4 of the non-splenectomised cats declined rapidly from Day 0 to Day 21 after which it fluctuated according to the individual cat's ability to cope with the infection. The non-splenectomised cats were apparently better able to cope as seen in Cats N, O and P with Ht levels of 0,26, 0,19 and 0,21 a few days after the lowest values had been recorded. However, it is interesting to point out that splenectomised Cats C 1 and 2 survived the longest (73, 114 and 118 days).

Non-splenectomised Cat S was pregnant at the commencement of the experiment and the low initial Ht of 0,21 is to be expected[1]. On Day 17 her Ht had fallen to its lowest level (0,08) after which it fluctuated only slightly. She delivered 3 weak kittens on Day 25 (Ht 0,11) and died on Day 35 (Ht 0,08).

Non-splenectomised Cats Q and R did not conform to the pattern and, as can be seen on Fig. 1, the mean Ht of these 2 animals fell to 0,21 on Day 11 and then rose to 0,305 on Day 14. From this Day Q's Ht steadily declined until Day 39 when the cat died (Ht 0,07). After Day 14 R's Ht fluctuated between 0,17 and 0,28

being 0,21 on Day 39. This was followed by a precipitious drop to 0,05 within 3 days when the animal died.

The rapid drop in Ht and subsequent decline in the animal's health that occurred in many of the cats is of practical clinical importance. Many cats can cope with the infection and adapt to the anaemia without the owner being aware of the situation. This can probably occur over a fair length of time and then, due to some unknown reason, there is a sudden decline in the Ht and the condition of the animal.

A fall in the Hb concentration and a reduction in the blood erythrocyte counts followed the pattern of the Ht. Cats Q and R were again the exception.

Erythrocyte indices: No definite tendency could be established when examining the mean MCV, MCHC and MCH values. When the figures of the individual cats were examined the erythrocytes were often macrocytic, i.e. MCV greater than 55 fl and hypochromic, i.e. MCHC less than 30 g/dl[9]. As is to be expected this tendency occurred when there was an influx of polychromatophilic macrocytes which do not have a full complement of haemoglobin related to cell size. At the same time there was usually an increase in MCH (greater than 17,5 pg)[8]. Greater changes in the erythrocyte indices were seen in the splenectomised cats and in fact splenectomy itself had a moderate influence on the MCHC and MCH.

In canine babesiosis the anaemia is essentially normocytic and normochromic[6]; this is to be expected in an acute haemolytic anaemia. In feline haemobartonellosis, however, the erythrocytes are often macroytic and hypochromic[5] which is more in line with a chronic haemolytic anaemia, as has been found in feline babesiosis in this study.

During the recovery stage the Ht, Hb and RBC returned rapidly to normal levels. An average daily recovery rate of 0,018 in Ht was recorded. A lower mean daily recovery rate of 0,0075 to 0,0083 in Ht was found in phlebotomy induced anaemias in cats[11], but it has been shown that the regenerative response in haemolytic anaemia is greater than in blood loss[8].

In general the leukocyte counts remained within the normal range ($5,5 - 19,5 \times 10^9/l$[8], $4 - 28 \times 10^9/l$[7]). An occasional mild leukocytosis was recorded but this is a common occurrence in cats[10]. The extremely high leukocyte count recorded in Cat M ($128 \times 10^9/l$) on terminal Day 20 can be accounted for by the presence of inspissated foetuses that were found on *post mortem* examination[13].

### The effects of splenectomy

Initially cats were splenectomised to facilitate transmission of the parasite[14]. However, it was soon found that this was unnecessary as transmission was as successful in the non-splenectomised cat.

Splenectomy *per se* caused a significant ($p<0,01$) decrease in Ht, Hb and RBC, a moderate ($p<0,05$) decrease in MCHC and MCH and a moderate ($p<0,05$) increase in WBC. Nevertheless, the general course and effects of the disease were similar in the splenectomised and non-splenectomised animals although the latter were not as severely affected.

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Table 3: CHANGES IN HAEMATOCRIT IN NON-SPLENECTOMISED CATS FROM DAY OF INOCULATION (DAY 0) WITH *B. FELIS* INFECTED BLOOD

| Cats | Days after infection | | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 0 | 3 | 4 | 7 | 10 | 11 | 14 | 17 | 18 | 20 | 21 | 24 | 25 | 28 | 31 | 32 | 35 | 38 | 39 | 42 | 45 | 46 | 49 |
| N | 0,32 | — | 0,31 | 0,27 | — | 0,10 | 0,11 | — | 0,07 | — | 0,11 | 0,11 | — | 0,14 | — | 0,19 | 0,20 | — | 0,26 | 0,23 | — | 0,23 | 0,21 |
| O | 0,31 | — | 0,25 | 0,23 | — | 0,13 | 0,11 | — | 0,12 | — | 0,09 | 0,10 | — | 0,19 | — | 0,14 | 0,14 | — | 0,17 | 0,19 | — | 0,17 | 0,13 |
| P | 0,35 | 0,36 | — | 0,20 | 0,18 | — | 0,16 | 0,15 | — | 0,10 | — | 0,12 | — | 0,21 | 0,14 | — | 0,18 | 0,21 | — | 0,15 | 0,16 | — | 0,07 |
| Q | 0,39 | — | 0,26 | 0,24 | — | 0,21 | 0,36 | 0,32 | — | — | 0,21 | — | 0,15 | 0,18 | — | 0,12 | 0,11 | — | 0,07 | — | — | — | — |
| R | 0,30 | — | 0,28 | 0,26 | — | 0,21 | 0,25 | 0,21 | — | — | 0,21 | — | 0,28 | 0,19 | — | 0,21 | 0,17 | — | 0,21 | 0,05 | — | — | — |
| S | 0,21 | — | 0,23 | 0,18 | — | 0,12 | 0,11 | 0,08 | — | — | 0,12 | — | 0,11 | 0,08 | — | 0,09 | 0,08 | — | — | — | — | — | — |
| Mean | 0,31 | | 0,28 | 0,23 | | 0,16 | 0,18 | | 0,16 | | 0,14 | | 0,15 | 0,17 | | 0,15 | 0,15 | | 0,18 | 0,16 | | 0,19 | 0,14 |
| SD | 0,06 | | 0,05 | 0,03 | | 0,05 | 0,10 | | 0,09 | | 0,06 | | 0,07 | 0,05 | | 0,04 | 0,05 | | 0,07 | 0,08 | | 0,04 | 0,07 |

Table 5: CHANGES IN HAEMOGLOBIN (g/d$\ell$) IN NON-SPLENECTOMISED CATS FROM DAY OF INOCULATION (DAY 0) WITH *B. FELIS* INFECTED BLOOD

| Cats | Days after infection | | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 0 | 3 | 4 | 7 | 10 | 11 | 14 | 17 | 18 | 20 | 21 | 24 | 25 | 28 | 31 | 32 | 35 | 38 | 39 | 42 | 45 | 46 | 49 |
| N | 9,4 | — | 8,2 | 8,8 | — | 3,2 | 3,3 | — | 2,1 | — | 3,5 | 3,2 | — | 3,9 | — | 5,3 | 5,7 | — | 7,9 | 7,2 | — | 7,1 | 5,8 |
| O | 9,7 | — | 7,2 | 8,2 | — | 4,3 | 4,5 | — | 3,1 | — | 2,2 | 3,1 | — | 5,3 | — | 4,2 | 3,9 | — | 5,4 | 5,2 | — | 4,9 | 4,2 |
| P | 10,4 | 11,0 | — | 6,4 | 5,9 | — | 4,7 | 3,4 | — | 3,2 | — | 3,4 | — | 5,6 | 4,4 | — | 4,8 | 6,8 | — | 4,3 | 3,9 | — | 2,0 |
| Q | 12,0 | — | 7,6 | 7,0 | — | 6,7 | 9,2 | 9,0 | — | — | 7,4 | — | 4,8 | 4,4 | — | 3,6 | 3,5 | — | 2,5 | — | — | — | — |
| R | 9,5 | — | 8,7 | 8,6 | — | 6,7 | 7,4 | 7,2 | — | — | 7,4 | — | 8,3 | 6,8 | — | 6,3 | 4,3 | — | 5,1 | 1,9 | — | — | — |
| S | 7,1 | — | 7,3 | 6,6 | — | 3,9 | 3,2 | 2,8 | — | — | 3,5 | — | 3,5 | 2,6 | — | 2,9 | 2,3 | — | — | — | — | — | — |
| Mean | 9,7 | | 8,3 | 7,6 | | 5,1 | 5,4 | | 4,6 | | 4,5 | | 4,4 | 4,8 | | 4,5 | 4,1 | | 5,5 | 4,7 | | 5,3 | 4,0 |
| SD | 1,6 | | 1,4 | 1,1 | | 1,5 | 2,4 | | 2,8 | | 2,3 | | 2,0 | 1,6 | | 1,2 | 1,2 | | 2,0 | 2,2 | | 1,6 | 1,9 |

Table 9: CHANGES IN MEAN CORPUSCULAR HAEMOGLOBIN CONCENTRATION (g/d$\ell$) IN NON-SPLENECTOMISED CATS FROM DAY OF INOCULATION (DAY 0) WITH *B. FELIS* INFECTED BLOOD

| Cats | Days after infection | | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 0 | 3 | 4 | 7 | 10 | 11 | 14 | 17 | 18 | 20 | 21 | 24 | 25 | 28 | 31 | 32 | 35 | 38 | 39 | 42 | 45 | 46 | 49 |
| N | 29 | — | 26 | 33 | — | 32 | 30 | — | 30 | — | 32 | 29 | — | 28 | — | 28 | 29 | — | 30 | 31 | — | 31 | 28 |
| O | 31 | — | 29 | 37 | — | 33 | 41 | — | 26 | — | 24 | 31 | — | 28 | — | 30 | 28 | — | 32 | 27 | — | 29 | 32 |
| P | 30 | 31 | — | 32 | 33 | — | 29 | 23 | — | 32 | — | 28 | — | 27 | 31 | — | 27 | 32 | — | 29 | 24 | — | 27 |
| Q | 31 | — | 29 | 29 | — | 32 | 26 | 28 | — | — | 35 | — | 32 | 24 | — | 30 | 32 | — | 36 | — | — | — | — |
| R | 32 | — | 31 | 33 | — | 32 | 30 | 34 | — | — | 35 | — | 30 | 36 | — | 30 | 25 | — | 24 | 38 | — | — | — |
| S | 34 | — | 32 | 37 | — | 33 | 29 | 35 | — | — | 29 | — | 32 | 33 | — | 32 | 29 | — | — | — | — | — | — |
| Mean | 31 | | 30 | 34 | | 33 | 31 | | 29 | | 31 | | 30 | 29 | | 30 | 28 | | 31 | 31 | | 28 | 29 |
| SD | 2 | | 2 | 3 | | 1 | 5 | | 5 | | 4 | | 2 | 4 | | 1 | 2 | | 4 | 5 | | 4 | 3 |

Table 2: THE CHANGES IN HAEMATOCRIT IN SPLENECTOMISED CATS FROM DAY OF INOCULATION (DAY 0) WITH *B. FELIS* INFECTED BLOOD

| Cats | Days after infection | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 0 | 3 | 7 | 10 | 14 | 17 | 20 | 21 | 24 | 27 | 28 | 31 | 35 | 38 | 42 |
| C | 0,30 | — | 0,28 | 0,25 | 0,25 | 0,16 | — | 0,09 | 0,09 | — | 0,10 | 0,15 | 0,11 | 0,13 | 0,14 |
| D | 0,28 | — | 0,26 | 0,24 | 0,15 | 0,12 | — | 0,09 | 0,09 | — | 0,10 | 0,09 | 0,06 | — | — |
| E | 0,31 | 0,31 | 0,29 | 0,26 | 0,17 | 0,10 | — | — | — | — | — | — | — | — | — |
| F | 0,24 | 0,24 | 0,16 | 0,11 | 0,10 | — | — | — | — | — | — | — | — | — | — |
| G | 0,28 | 0,27 | 0,32 | 0,23 | 0,21 | 0,16 | — | 0,10 | 0,11 | 0,11 | — | — | — | — | — |
| H | 0,25 | 0,26 | 0,18 | 0,12 | 0,14 | 0,09 | — | — | — | — | — | — | — | — | — |
| I | 0,32 | 0,30 | 0,29 | 0,23 | 0,11 | 0,08 | — | 0,07 | — | — | — | — | — | — | — |
| J | 0,23 | 0,27 | 0,22 | 0,18 | — | — | — | — | — | — | — | — | — | — | — |
| K | 0,27 | 0,31 | 0,27 | 0,17 | 0,09 | 0,12 | — | 0,10 | 0,11 | — | 0,10 | 0,09 | 0,12 | — | 0,11 |
| M | 0,24 | 0,25 | 0,15 | 0,07 | 0,11 | 0,12 | 0,13 | — | — | — | — | — | — | — | — |
| 1 | 0,40 | — | 0,36 | — | 0,16 | — | — | 0,10 | — | — | — | 0,12 | — | 0,11 | — |
| 2 | 0,33 | — | 0,31 | — | 0,26 | — | — | 0,09 | — | — | — | 0,12 | — | 0,14 | — |
| Mean | 0,29 | 0,28 | 0,26 | 0,19 | 0,16 | 0,12 | — | 0,09 | 0,10 | — | 0,10 | 0,11 | 0,10 | 0,13 | 0,13 |
| SD | 0,05 | 0,03 | 0,07 | 0,07 | 0,06 | 0,03 | — | 0,01 | 0,01 | — | — | 0,03 | 0,03 | 0,02 | 0,02 |

Table 4: CHANGES IN HAEMOGLOBIN (g/dℓ) IN SPLENECTOMISED CATS FROM DAY OF INOCULATION (DAY 0) WITH *B. FELIS* INFECTED BLOOD

| Cats | Days after infection | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 0 | 3 | 7 | 10 | 14 | 17 | 20 | 21 | 24 | 27 | 28 | 31 | 35 | 38 | 42 |
| C | 8,8 | — | 7,7 | 7,9 | 6,5 | 4,8 | — | 3,0 | 2,2 | — | 2,5 | 4,1 | 3,3 | 3,2 | 3,5 |
| D | 8,0 | — | 7,8 | 6,4 | 4,0 | 2,6 | — | 2,3 | 2,5 | — | 2,2 | 2,2 | 1,7 | — | — |
| E | 8,6 | 8,6 | 8,5 | 7,6 | 4,8 | 2,1 | — | — | — | — | — | — | — | — | — |
| F | 7,1 | 7,2 | 5,2 | 3,6 | 2,1 | — | — | — | — | — | — | — | — | — | — |
| G | 8,3 | 7,9 | 7,5 | 8,0 | 6,2 | 4,6 | — | 2,6 | 2,7 | 3,0 | — | — | — | — | — |
| H | 6,1 | 7,1 | 4,6 | 3,8 | 3,0 | 2,2 | — | — | — | — | — | — | — | — | — |
| I | 8,6 | 8,4 | 8,0 | 6,9 | 4,4 | 2,4 | — | 2,1 | — | — | — | — | — | — | — |
| J | 6,0 | 8,8 | 6,2 | 5,4 | — | — | — | — | — | — | — | — | — | — | — |
| K | 7,7 | 9,3 | 8,0 | 6,1 | 3,2 | 3,0 | — | 2,9 | 3,0 | — | 2,8 | 2,6 | 3,1 | 3,4 | 2,7 |
| M | 6,8 | 8,2 | 4,6 | 2,1 | 2,6 | 3,9 | 2,9 | — | — | — | — | — | — | — | — |
| 1 | 12,5 | — | 11,0 | — | 4,5 | — | — | 2,8 | — | — | — | 3,5 | — | 3,2 | — |
| 2 | 10,0 | — | 9,5 | — | 5,5 | — | — | 3,0 | — | — | — | 3,5 | — | 2,7 | — |
| Mean | 8,2 | 8,2 | 7,4 | 5,8 | 4,3 | 3,2 | — | 2,7 | 2,6 | — | 2,5 | 3,2 | 2,7 | 3,1 | 3,1 |
| SD | 1,8 | 0,8 | 1,9 | 2,0 | 1,5 | 1,1 | — | 0,4 | 0,3 | — | 0,3 | 0,8 | 0,9 | 0,3 | 0,6 |

Table 6: THE CHANGES IN BLOOD ERYTHROCYTE COUNT ($\times 10^{12}/\ell$) IN SPLENECTOMISED CATS FROM DAY OF INOCULATION (DAY 0) WITH *B. FELIS* INFECTED BLOOD

| Cats | Days after infection | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | 0 | 7 | 14 | 20 | 21 | 27 | 28 | 35 | 42 |
| C | 5,9 | 5,3 | 5,0 | — | 1,4 | — | 1,1 | 2,0 | 1,9 |
| D | 5,8 | 5,0 | 2,4 | — | 1,2 | — | 1,0 | 1,3 | — |
| E | 5,2 | 5,2 | 3,0 | — | — | — | — | — | — |
| F | 4,8 | 3,0 | 1,9 | — | — | — | — | — | — |
| G | 4,6 | 5,1 | 4,9 | — | 1,5 | 1,9 | — | — | — |
| H | 4,4 | 2,3 | 1,2 | — | — | — | — | — | — |
| I | 6,2 | 6,0 | 2,0 | — | 0,9 | — | — | — | — |
| J | 4,4 | 4,8 | — | — | — | — | — | — | — |
| K | 5,3 | 5,0 | 1,9 | — | 1,9 | — | 2,1 | 2,6 | 2,0 |
| M | 4,2 | 4,7 | 1,4 | 1,2 | — | — | — | — | — |
| Mean | 5,1 | 4,6 | 2,6 | — | 1,4 | — | 1,4 | 2,0 | 2,0 |
| SD | 0,7 | 1,1 | 1,4 | — | 0,4 | — | 0,6 | 0,7 | 0,1 |

Table 7: CHANGES IN BLOOD ERYTHROCYTE COUNT ($\times 10^{12}/\ell$) IN NON-SPLENECTOMISED CATS FROM DAY OF INOCULATION (DAY 0) WITH *B. FELIS* INFECTED BLOOD

| Cats | Days after infection | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 0 | 4 | 7 | 11 | 14 | 17 | 18 | 20 | 24 | 25 | 28 | 32 | 35 | 39 | 42 | 45 | 46 | 49 |
| N | — | 5,6 | — | 3,6 | — | — | 1,0 | — | 1,4 | — | — | 4,0 | — | 4,0 | — | — | 4,0 | 4,7 |
| O | — | 5,7 | — | 3,8 | — | — | 2,1 | — | 2,2 | — | — | 3,2 | — | 3,8 | — | — | 2,9 | 2,3 |
| P | 6,1 | — | 5,6 | — | 3,7 | — | — | 2,2 | — | — | 4,0 | — | 3,7 | — | 2,2 | 2,2 | — | — |
| Q | — | 7,2 | — | 4,3 | — | 4,9 | — | — | — | 3,4 | — | 2,5 | — | 1,1 | — | — | — | — |
| R | — | 6,6 | — | 4,0 | — | 4,1 | — | — | — | 4,6 | — | 4,3 | — | 3,3 | 0,7 | — | — | — |
| S | — | 5,3 | — | 3,6 | — | 2,1 | — | — | — | 2,5 | — | 1,9 | — | — | — | — | — | — |
| Mean | — | 6,1 | — | 3,9 | — | 2,8 | | — | 2,8 | | — | 3,2 | — | 3,1 | 1,5 | 3,0 | | 3,5 |
| SD | — | 0,8 | — | 0,3 | — | 1,6 | | — | 1,2 | | — | 1,0 | — | 1,3 | 1,1 | 0,9 | | 1,7 |

Table 8: CHANGES IN MEAN CORPUSCULAR HAEMOGLOBIN CONCENTRATION (g/d$\ell$) IN SPLENECTOMISED CATS FROM DAY OF INOCULATION (DAY 0) WITH *B. FELIS* INFECTED BLOOD

| Cats | Days after infection | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 0 | 3 | 7 | 10 | 14 | 17 | 20 | 21 | 24 | 27 | 28 | 31 | 35 | 38 | 42 |
| C | 29 | — | 28 | 32 | 26 | 30 | — | 33 | 24 | — | 25 | 27 | 30 | 25 | 25 |
| D | 29 | — | 30 | 27 | 27 | 22 | — | 26 | 28 | — | 22 | 24 | 28 | — | — |
| E | 28 | 28 | 30 | 29 | 29 | 21 | — | — | — | — | — | — | — | — | — |
| F | 30 | 30 | 33 | 33 | 20 | — | — | — | — | — | — | — | — | — | — |
| G | 30 | 29 | 23 | 35 | 30 | 29 | — | 26 | 25 | 26 | — | — | — | — | — |
| H | 24 | 27 | 26 | 32 | 21 | 25 | — | — | — | — | — | — | — | — | — |
| I | 27 | 28 | 30 | 31 | 30 | — | — | 30 | — | — | — | — | — | — | — |
| J | 26 | 33 | 30 | 30 | — | — | — | — | — | — | — | — | — | — | — |
| K | 29 | 30 | 31 | 36 | 36 | 25 | — | 29 | 27 | — | 28 | 29 | 26 | — | 26 |
| M | 28 | 33 | 30 | 30 | 24 | 33 | 22 | — | — | — | — | — | — | — | — |
| 1 | 31 | — | 31 | — | 28 | — | — | 28 | — | — | — | 29 | — | 29 | — |
| 2 | 30 | — | 31 | — | 21 | — | — | 33 | — | — | — | 29 | — | 27 | — |
| Mean | 28 | 30 | 29 | 32 | 27 | 26 | — | 29 | 26 | — | 25 | 28 | 28 | 27 | 26 |
| SD | 2 | 2 | 3 | 3 | 5 | 4 | — | 3 | 2 | — | 3 | 2 | 2 | 2 | 1 |

Table 10: CHANGES IN MEAN CORPUSCULAR HAEMOGLOBIN (pg) IN SPLENECTOMISED CATS FROM DAY OF INOCULATION (DAY 0) WITH *B. FELIS* INFECTED BLOOD

| Cats | Days after infection | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | 0 | 7 | 14 | 20 | 21 | 27 | 28 | 35 | 42 |
| C | 15,0 | 14,5 | 13,1 | — | 21,4 | — | 22,7 | 16,5 | 18,4 |
| D | 13,8 | 15,5 | 16,6 | — | 19,1 | — | 22,0 | 13,1 | — |
| E | 16,5 | 16,3 | 16,0 | — | — | — | — | — | — |
| F | 14,8 | 17,3 | 10,5 | — | — | — | — | — | — |
| G | 18,0 | 14,7 | 12,7 | — | 17,3 | 15,8 | — | — | — |
| H | 13,9 | 20,0 | 25,0 | — | — | — | — | — | — |
| I | 13,9 | 13,3 | 17,0 | — | 23,0 | — | — | — | — |
| J | 13,6 | 13,0 | — | — | — | — | — | — | — |
| K | 14,5 | 16,0 | 16,8 | — | 15,3 | — | 13,3 | 11,9 | 13,5 |
| M | 16,2 | 10,0 | 18,6 | 22,8 | — | — | — | — | — |
| Mean | 15,0 | 15,1 | 16,3 | — | 19,2 | — | 19,3 | 13,8 | 16,0 |
| SD | 1,4 | 2,7 | 4,2 | — | 3,1 | — | 5,2 | 2,4 | 3,5 |

Table 11: CHANGES IN MEAN CORPUSCULAR HAEMOGLOBIN (pg) IN NON-SPLENECTOMISED CATS FROM DAY OF INOCULATION (DAY 0) WITH *B. FELIS* INFECTED BLOOD

| Cats | Days after infection | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 0 | 4 | 7 | 11 | 14 | 17 | 18 | 20 | 24 | 25 | 28 | 32 | 35 | 39 | 42 | 45 | 46 | 49 |
| N | — | 14,6 | — | 8,7 | — | — | 21,0 | — | 22,9 | — | — | 13,3 | — | 19,8 | — | — | 17,8 | 12,3 |
| O | — | 12,6 | — | 11,3 | — | — | 14,8 | — | 14,1 | — | — | 13,0 | — | 14,2 | — | — | 16,9 | 18,3 |
| P | 17,2 | — | 11,4 | — | 12,7 | — | — | 14,5 | — | — | 14,0 | — | 13,0 | — | 19,5 | 18,6 | — | — |
| Q | — | 10,6 | — | 15,6 | — | 18,4 | — | — | — | 14,1 | — | 14,4 | — | 22,7 | — | — | — | — |
| R | — | 13,2 | — | 16,8 | — | 17,6 | — | — | — | 18,0 | — | 14,7 | — | 15,5 | 27,1 | — | — | — |
| S | — | 13,8 | — | 10,8 | — | 13,3 | — | — | — | 14,0 | — | 15,3 | — | — | — | — | — | — |
| Mean | — | 13,0 | — | 12,6 | — | 17,0 | | — | 16,6 | | — | 14,1 | — | 18,1 | 23,3 | 17,8 | | 15,3 |
| SD | — | 1,5 | — | 3,4 | — | 3,0 | | — | 3,9 | | — | 1,0 | — | 3,9 | 5,4 | 0,9 | | 4,2 |

Table 12: CHANGES IN MEAN CORPUSCULAR VOLUME (*fl*) IN SPLENECTOMISED CATS FROM DAY OF INOCULATION (DAY 0) WITH *B. FELIS* INFECTED BLOOD

| Cats | Days after infection | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | 0 | 7 | 14 | 20 | 21 | 27 | 28 | 35 | 42 |
| C | 51 | 53 | 50 | — | 64 | — | 91 | 55 | 74 |
| D | 48 | 52 | 63 | — | 75 | — | 100 | 46 | — |
| E | 60 | 56 | 57 | — | — | — | — | — | — |
| F | 50 | 53 | 53 | — | — | — | — | — | — |
| G | 61 | 63 | 43 | — | 67 | 58 | — | — | — |
| H | 57 | 78 | — | — | — | — | — | — | — |
| I | 52 | 48 | 55 | — | 78 | — | — | — | — |
| J | 52 | 46 | — | — | — | — | — | — | — |
| K | 51 | 54 | 47 | — | 53 | — | 44 | 46 | 55 |
| M | 54 | 32 | 79 | 102 | — | — | — | — | — |
| Mean | 54 | 54 | 56 | — | 67 | — | 78 | 49 | 65 |
| SD | 4 | 12 | 11 | — | 10 | — | 30 | 5 | 13 |

Table 13: CHANGES IN MEAN CORPUSCULAR VOLUME (*fl*) IN NON-SPLENECTOMISED CATS FROM DAY OF INOCULATION (DAY 0) WITH *B. FELIS* INFECTED BLOOD

| Cats | Days after infection | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 0 | 4 | 7 | 11 | 14 | 17 | 18 | 20 | 24 | 25 | 28 | 32 | 35 | 39 | 42 | 45 | 46 | 49 |
| N | — | 55 | — | 28 | — | — | 70 | — | 79 | — | — | 46 | — | 65 | — | — | 58 | 45 |
| O | — | 44 | — | 34 | — | — | 57 | — | 44 | — | — | 44 | — | 45 | — | — | 59 | 57 |
| P | 57 | — | 36 | — | 43 | — | — | 45 | — | — | 53 | — | 49 | — | 68 | 73 | — | — |
| Q | — | 36 | — | 49 | — | 65 | — | — | — | 44 | — | 48 | — | 64 | — | — | — | — |
| R | — | 42 | — | 52 | — | 51 | — | — | — | 61 | — | 49 | — | 64 | 71 | — | — | — |
| S | — | 43 | — | 33 | — | 38 | — | — | — | 44 | — | 47 | — | — | — | — | — | — |
| Mean | — | 44 | — | 39 | — | 56 | | — | 54 | | — | 47 | — | 60 | 70 | 63 | | 51 |
| SD | — | 7 | — | 11 | — | 13 | | — | 16 | | — | 2 | — | 10 | 2 | 8 | | 8 |

Table 14: CHANGES IN TOTAL BLOOD LEUKOCYTE COUNT ($\times 10^9/\ell$) IN SPLENECTOMISED CATS FROM DAY OF INOCULATION (DAY 0) WITH *B. FELIS* INFECTED BLOOD

| Cats | Days after infection | | | | | | |
|---|---|---|---|---|---|---|---|
| | 0 | 7 | 14 | 21 | 28 | 35 | 42 |
| C | 6,4 | 2,4 | 19,9 | 10,0 | 22,2 | 19,1 | 9,2 |
| D | 8,6 | 7,0 | 8,3 | 11,7 | 7,8 | 9,4 | — |
| E | 12,3 | 8,8 | 13,6 | — | — | — | — |
| F | 15,5 | 24,6 | 38,4 | — | — | — | — |
| G | 11,9 | 13,0 | 15,9 | 35,0 | 1,7† | — | — |
| H | 11,8 | 15,2 | 20,2 | — | — | — | — |
| I | 21,9 | 9,8 | 12,7 | 13,4 | — | — | — |
| J | 11,4 | 6,1 | — | — | — | — | — |
| K | 14,6 | 11,4 | 15,6 | 24,1 | 15,8 | 11,4 | 21,6 |
| M | 18,0 | 23,0 | 31,6 | 128,0* | — | — | — |
| Mean | 13,2 | 12,1 | 19,6 | 37,0 | 11,9 | 13,3 | 15,4 |
| SD | 4,5 | 7,1 | 9,6 | 45,5 | 9,0 | 5,1 | 8,9 |

*Day 20
†Day 27

Table 15: CHANGES IN TOTAL BLOOD LEUKOCYTE COUNT ($\times 10^9/\ell$) IN NON-SPLENECTOMISED CATS FROM DAY OF INOCULATION (DAY 0) WITH *B. FELIS* INFECTED BLOOD

| Cats | Days after infection | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 0 | 4 | 7 | 11 | 14 | 17 | 18 | 20 | 24 | 25 | 28 | 32 | 35 | 39 | 42 | 45 | 46 | 49 |
| N | — | 11,4 | — | 9,0 | — | — | 21,0 | — | 24,5 | — | — | 31,4 | — | 28,1 | — | — | 11,6 | 11,4 |
| O | — | 11,0 | — | 7,3 | — | — | 12,1 | — | 16,2 | — | — | 12,2 | — | 11,8 | — | — | 12,0 | 13,0 |
| P | 9,9 | — | 8,5 | — | 11,0 | — | — | 22,0 | — | — | 13,2 | — | 12,5 | — | 14,4 | 15,2 | — | — |
| Q | — | 9,5 | — | 13,4 | — | 12,8 | — | — | — | 10,1 | — | 15,4 | — | 3,5 | — | — | — | — |
| R | — | 6,8 | — | 12,3 | — | 12,1 | — | — | — | 11,8 | — | 12,4 | — | 22,2 | 4,4 | — | — | — |
| S | — | 8,3 | — | 21,5 | — | 26,0 | — | — | — | 37,2 | — | 13,6 | — | — | — | — | — | — |
| Mean | — | 9,4 | — | 12,7 | — | 16,8 | | — | 20,0 | | — | 17,0 | — | 16,4 | 9,4 | 12,9 | | 12,2 |
| SD | — | 1,9 | — | 5,5 | — | 6,4 | | — | 11,1 | | — | 8,1 | — | 10,9 | 7,1 | 2,0 | | 1,1 |

Table 16: HAEMATOLOGY PRE-SPLENECTOMY AND 14 DAYS POST-SPLENECTOMY

| | Pre-Splenectomy | Post-Splenectomy | Significant differences |
|---|---|---|---|
| Ht | 0,35 ± 0,03 | 0,25 ± 0,02 | $p < 0,01$ |
| Hb (g/d$\ell$) | 10,1 ± 0,6 | 7,0 ± 1,0 | $p < 0,01$ |
| RBC ($\times 10^{12}/\ell$) | 6,2 ± 0,4 | 4,6 ± 0,4 | $p < 0,01$ |
| MCHC (g/d$\ell$) | 29,0 ± 1,0 | 27,0 ± 2,4 | $p < 0,05$ |
| MCH (pg) | 16,5 ± 0,7 | 15,2 ± 1,8 | $p < 0,05$ |
| MCV (*f$\ell$*) | 57,0 ± 2,0 | 54,0 ± 5,9 | NS |
| WBC ($\times 10^9/\ell$) | 8,5 ± 2,9 | 13,5 ± 2,8 | $p < 0,05$ |

NS = Not Significant

Table 17: INITIAL (I) AND RECOVERY (R) Ht AND Hb (g/d$\ell$) LEVELS OF FIELD CASES OF *B. FELIS* IN GROUP 1 (Ht > 0,16)

| Ht | | | Hb (g/d$\ell$) | | | |
|---|---|---|---|---|---|---|
| I | R | Average Daily Rise | I | R | Average Daily Rise | Days between I and R |
| 0,29 | 0,31 | 0,005 | 9,0 | 9,5 | 0,1 | 4 |
| 0,25 | 0,29 | 0,013 | 8,0 | 10,0 | 0,7 | 3 |
| 0,24 | 0,28 | 0,010 | 7,5 | 7,5 | 0 | 4 |
| 0,22 | 0,26 | 0,002 | 7,0 | 8,0 | 0,1 | 19 |
| 0,21 | 0,29 | 0,011 | 5,2 | 9,5 | 0,6 | 7 |
| 0,20 | 0,30 | 0,014 | 0,50 | 9,5 | 0,6 | 7 |
| 0,20 | 0,37 | 0,012 | 5,5 | 11,0 | 0,4 | 14 |
| 0,19 | 0,30 | 0,016 | 6,0 | 8,5 | 0,4 | 7 |
| 0,18 | 0,30 | 0,011 | 5,0 | 8,6 | 0,3 | 11 |
| 0,18 | 0,26 | 0,027 | 5,0 | 7,0 | 0,7 | 3 |
| 0,18 | 0,33 | 0,021 | 5,3 | 8,5 | 0,5 | 7 |
| 0,17 | 0,28 | 0,021 | 5,0 | 7,5 | 0,4 | 6 |
| 0,17 | 0,44 | 0,034 | 5,0 | 11,0 | 0,8 | 8 |
| Mean±SD 0,015±0,009 | | | Mean±SD 0,4±0,3 | | | |

Table 18: INITIAL (I) AND RECOVERY (R) Ht AND Hb (g/dℓ) LEVELS OF FIELD CASES OF *B. FELIS* IN GROUP 2 (Ht 0,13–0,16)

| Ht | | | Hb (g/dℓ) | | | |
|---|---|---|---|---|---|---|
| I | R | Average Daily Rise | I | R | Average Daily Rise | Days between I and R |
| 0,15 | 0,25 | 0,010 | 4,0 | 8,0 | 0,4 | 10 |
| 0,15 | 0,28 | 0,019 | 4,0 | 7,0 | 0,4 | 7 |
| 0,14 | 0,30 | 0,013 | 4,5 | 9,0 | 0,4 | 12 |
| 0,14 | 0,20 | 0,009 | 3,3 | 4,5 | 0,2 | 7 |
| 0,14 | 0,33 | 0,017 | 4,5 | 9,6 | 0,7 | 11 |
| 0,14 | 0,28 | 0,020 | 4,5 | 9,0 | 0,6 | 7 |
| 0,14 | 0,30 | 0,020 | 5,0 | 7,5 | 0,3 | 8 |
| 0,14 | 0,25 | 0,036 | 5,0 | 7,0 | 0,7 | 3 |
| 0,13 | 0,36 | 0,026 | 3,4 | 9,5 | 0,8 | 9 |
| Mean±SD 0,019±0,009 | | | Mean±SD 0,5±0,2 | | | |

Table 19: INITIAL (I) AND RECOVERY (R) Ht AND Hb (g/dℓ) LEVELS OF FIELD CASES OF *B. FELIS* IN GROUP 3 (Ht < 0,13)

| Ht | | | Hb (g/dℓ) | | | |
|---|---|---|---|---|---|---|
| I | R | Average Daily Rise | I | R | Average Daily Rise | Days between I and R |
| 0,12 | 0,20 | 0,001 | 3,5 | 7,5 | 0,5 | 8 |
| 0,12 | 0,33 | 0,018 | 3,5 | 10,0 | 0,5 | 12 |
| 0,12 | 0,28 | 0,020 | 3,5 | 9,0 | 0,7 | 8 |
| 0,12 | 0,28 | 0,027 | 4,0 | 6,0 | 0,7 | 6 |
| 0,12 | 0,36 | 0,012 | 4,0 | 11,0 | 0,4 | 20 |
| 0,11 | 0,31 | 0,023 | 3,5 | 9,6 | 0,7 | 9 |
| 0,10 | 0,31 | 0,021 | 3,5 | 10,5 | 0,7 | 10 |
| 0,10 | 0,35 | 0,036 | 2,8 | 10,0 | 1,0 | 7 |
| 0,10 | 0,33 | 0,019 | 3,0 | 7,5 | 0,4 | 12 |
| 0,10 | 0,16 | 0,010 | 2,5 | 5,5 | 0,8 | 4 |
| 0,09 | 0,19 | 0,020 | 3,0 | 5,0 | 0,4 | 9 |
| Mean±SD 0,02±0,008 | | | Mean±SD 0,6±0,2 | | | |

ABSTRACT: De Villiers, Ethel-Michele & D.W. Verwoerd, 1980. **Presence of Herpesvirus ovis DNA sequences in cellular DNA from sheep lungs affected with jaagsiekte (pulmonary adenomatosis).** *Onderstepoort Journal of Veterinary Research*, 47, 109–112 (1980).

To investigate further the possible involvement of *Herpesvirus ovis* in the aetiology of jaagsiekte, the kinetics of reassociation of viral DNA and DNA isolated from tumour tissue as well as from cell cultures derived from it were studied. Although DNA-DNA hybridization could be demonstrated in 2 cases of jaagsiekte, no correlation was found between the presence of *Herpesvirus ovis* genome sequences and the occurrence of the disease.

ABSTRACT: N.T. van der Walt, 1980. **A haemagglutination and a haemagglutination inhibition test for bluetongue virus.** *Onderstepoort Journal of Veterinary Research*, 47, 113–117 (1980).

Haemagglutination of bluetongue virus (BTV) was demonstrated for the first time by making use of a purified preparation of the virus. The reaction was found to be independent of variations in the pH, temperature, buffer system and origin of the erythrocytes used in the test. A haemagglutination inhibition test, subsequently developed, was demonstrated to be serotype specific. the storage of the virus for indefinite periods was facilitated by lyophilization of BTV in the presence of a low concentration of sucrose.

ABSTRACT: B.C. Jansen, 1980 **The aetiology of ram epididymitis.** *Onderstepoort Journal of Veterinary Research*, 47, 101–107 (1980).

A wide variety of organisms from the environment invade the preputial cavity of rams. Various of these organisms can be isolated from the deeper parts of the male genital tract, and especially from the accessory glands.

Some of the bacteria present in the sheath can be stimulated to migrate to the deeper parts of the genital tract by injections into the host animal of luteinizing-hormone-releasing-hormone and injections of pregnant mare serum gonadotrophin.

The increased levels of luteinizing hormone and follicle-stimulating hormone in surgically prepared cryptorchids also stimulate migration of the organisms.

The natural mode of development of genital infections in rams is formulated as follows: The preputial cavity of a ram becomes invaded by various organisms through contact with the environment. When, under the influence of systemic hormonal stimulation, the genitalia undergo development, suitable conditions are created for the migration of some of the bacteria in the sheath to the deeper-lying organs of the genital tract such as the vesiculae seminales, epididymides and testes. In these organs the bacteria can possibly initiate a pathological process.

ABSTRACT: J. Boomker, 1980. **The haemocytology and histology of the haemopoietic organs of South African freshwater fish. II. Erythrocytes and thrombocytes of Clarias gariepinus and Sarotherodon mossambicus.** *Onderstepoort Journal of Veterinary Research*, 47, 95–100 (1980).

This paper describes the light-microscopic appearance of both the erythrocytes and the thrombocytes and their developmental stages of catfish *(Clarias gariepinus)* and Mozambique bream *(Sarotherodon mossambicus)*. Apart from some minor differences in the shape and staining affinities of the polychromatophilic erythrocytes, the erythrocytic series is similar in catfish and bream. The thrombocytes and the reactive stages of thrombocytes of both species are similar to those of birds. A possible mechanism of erythro- and thrombopoiesis is postulated.

**CASE REPORT** **GEVALVERSLAG**

# THE TREATMENT OF *FILAROIDES OSLERI INFESTATION* WITH ALBENDAZOLE

J. VAN HEERDEN and S.W. PETRICK*

ABSTRACT: van Heerden J., Petrick S.W., **The treatment of *Filaroides osleri* infestation with albendazole.** *Journal of the South African Veterinary Association* (1980) **51** No. 4 281 (En) Dep. Medicine, Fac. Vet. Science, Univ. Pretoria, 0110 Onderstepoort.

A dog infested with *Filaroides osleri* was dosed with albendazole at 9,5 mg/kg every day for 55 days. Clinical signs of disease disappeared after 30 days but a few nodules were still present in the left bronchus 373 days after the onset of treatment. The anthelmintic caused an increase in lymphocyte and decrease in neutrophil count.

## CASE REPORT

A mongrel dog 18 months of age with a body mass of 14 kg was presented with a history of laboured breathing, nocturnal orthopnoea and occasional vomition or attempted vomition. Clinical signs were noted for at least 3 months prior to admission. The dog had been treated on 2 occasions with disophenol (Ancylol, Cyanamid), sulphamethazine and vitamin E by the referring veterinarian.

On clinical examination he was in good physical condition, the respiratory rate was elevated and breathing was somewhat laboured. The rectal temperature was normal. A dry cough which often ended in retching was sometimes heard. Radiographic examination of the chest revealed no abnormalities but a bronchoscopic examination under general anaesthesia showed that the lumen of the distal trachea was almost completely occluded by numerous granulomatous lesions in its mucosa. The lumens of the bronchi were not visible.

Both pharyngeal and laryngeal swabs taken under general anaesthesia were rinsed in physiological saline solution. Larvae of *Filaroides osleri* were subsequently demonstrated by microscopic examination of the centrifuged saline solution.

The dog was thereafter treated with albendazole (Valbazen, Smith Kline Animal Health Products) at 9,5 mg/kg live mass every day for 55 days.

The clinical response to treatment and the results of subsequent bronchoscopic examinations are listed in Table 1.

Total and differential white cell counts were monitored before, during and after treatment and are summarized in Table 2. A reversal in neutrophil: lymphocyte ratio which occurred from Day O to Day 86 was noted. The cause of the eosinophilia observed on Days 116 and 373 was not investigated.

## CONCLUSION

Treatment every day for almost 2 months (55 days) with albendazole at 9,5 mg/kg/day caused the clinical cure of a dog infested with *F. osleri* in 3 months (99 days).

Table 1: CHANGES IN CLINICAL SIGNS AND BRONCHOSCOPIC EXAMINATION OF A DOG TREATED EVERY DAY WITH ALBENDAZOLE FOR 55 DAYS AT 9,5 mg/kg

| Days | Clinical signs | Bronchoscopic examination |
|---|---|---|
| 0 | Dyspnoea, orthopnoea, tachypnoea, cough | Severe granulomatous tracheitis. Bronchi not visible |
| 17 | Slight dyspnoea | Granulomas smaller, bronchial openings visible |
| 30 | Occasional cough | Granulomas in trachea and bronchi fewer and smaller |
| 99 | Clinically normal | A few small granulomas present |
| 373 | Clinically normal | A few very small granulomas in left main bronchus only |

Table 2: TOTAL AND DIFFERENTIAL WHITE CELL COUNTS, BEFORE, DURING AND AFTER TREATMENT

| Day | 0 | 44 | 72 | 86 | 116 | 373 |
|---|---|---|---|---|---|---|
| WCC $10^9/\ell$ | 9,3 | 7,6 | 7,3 | 7,9 | 7,4 | 9,3 |
| Neutrophils | 0,67 | 0,53 | 0,42 | 0,38 | 0,50 | 0,57 |
| Lymphocytes | 0,25 | 0,41 | 0,49 | 0,54 | 0,27 | 0,19 |
| Monocytes | 0,02 | 0 | 0,04 | 0,05 | 0,08 | 0,05 |
| Eosinophils | 0,05 | 0,06 | 0,05 | 0,03 | 0,15 | 0,19 |
| Basophils | 0 | 0 | 0 | 0 | 0 | 0 |

## ACKNOWLEDGEMENTS

We are indebted to Miss Annaline van Heerden for performing the laboratory investigations.

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# SERUM ANTIBODY LEVELS BEFORE AND AFTER ADMINISTRATION OF LIVE CANINE DISTEMPER VACCINE TO THE WILD DOG *LYCAON PICTUS*

J, VAN HEERDEN, W.H. SWART* and D.G.A. MELTZER†

ABSTRACT: Van Heerden, J.; Swart W.H.; Meltzer D.G.A. **Serum antibody levels before and after administration of live canine distemper vaccine to the wild dog Lycaon pictus.** *Journal of the South African Veterinary Association* (1980) **51** No. 4 283–284 (En) Dept. Medicine, Fac. Vet. Science, Univ. Pretoria, 0110 Onderstepoort, Rep. of South Africa.

Antibodies against canine distemper virus were determined before and after the administration of a live canine distemper virus vaccine to 16 wild dogs *Lycaon pictus*. Antibody levels were either very low or non-detectable. The results were inconclusive.

## INTRODUCTION

Canine distemper is a highly contagious viral disease of domestic dogs manifested by signs of generalized infection and/or central nervous system disturbance. The disease has been reported in various members of the families Canidae, Hyenidae, Mustelidae, Procyonidae and Viverridae[2]. Although it is often assumed that all members of the family Canidae are susceptible, confirmation of canine distemper virus infection by laboratory examination is often lacking[1,9].

A disease manifesting clinical signs typical of those of canine distemper has been described in a member of the family Procyonidae following the administration of a vaccine containing live attenuated distemper virus[5].

Bere[1], Hofmeyr[4], and Young[9] reported the occurrence of canine distemper in the wild dog *Lycaon pictus*. Hofmeyr[4] based his diagnosis of the disease in 2 zoo animals on clinical signs and macroscopic post mortem findings. A concommitant *Toxoplasma* infection was reported in one individual.

Bere[1] reported as follows: "Colonel Hoier observes that when conditions are too easy, the wild dogs lose something of their natural resistance to disease and become particularly susceptible to distemper ..." but did not give any further information as to how a diagnosis of "distemper" was made.

Young[9] stated that wild dogs are particularly susceptible to canine distemper but did not mention the source of his information.

The wild dog although belonging to the family Canidae is classified under its own and exclusive subfamily Cimocyoninae[6]. It is therefore not a true dog.

The following report deals with a serological investigation for the presence of antibodies against canine distemper virus after the administration of canine distemper vaccine to wild dogs.

## MATERIALS, METHODS AND RESULTS

Sixteen wild dogs were bled before and after the administration of canine distemper vaccine. These dogs were captured as puppies in the wild (as part of a salvage programme) and were held in captivity as Groups A, B and C in 3 different localities. The vaccine used was a commercially available product containing live attenuated canine distemper virus§.

Group A consisted of 3 wild dog puppies which were kept in close proximity to domestic dogs. They were first bled at approximately 9 weeks of age and subsequently vaccinated 3 times at monthly intervals with canine distemper vaccine. Serum antibody titres were finally determined at approximately 6 months of age.

The 4 dogs in Group B were bled at approximately 6 months of age and then vaccinated once. The serum antibody titre determination was based on serum samples collected 2 months later. The dogs were kept in an enclosure on a breeding farm of the National Zoological Gardens in the Pretoria District.

The 9 animals in Group C were bled at approximately 3 months of age prior to vaccination. The serum antibody titre determination was based on a serum sample collected 2 weeks later. The animals were kept in an enclosure in a private game reserve in the Northern Transvaal.

Serum antibody levels were determined by means of

Table 1: RESULTS OF SERUM NEUTRALIZATION TESTS FOLLOWING VACCINATION

| | | 1:4 | 1:8 | 1:16 | 1:32 | SN |
|---|---|---|---|---|---|---|
| Group A | 1. | 0/5* | 3/5 | 3/5 | 5/5 | 1:9 |
| | 2. | 4/5 | 3/5 | 5/5 | 5/5 | 1:4 |
| | 3. | 2/5 | 1/5 | 2/5 | 2/5 | 1:18 |
| Group B | 1. | 0/4 | 1/4 | 2/4 | 2/4 | 1:19 |
| | 2. | 2/4 | 2/4 | 3/4 | 2/4 | 1:9 |
| | 3. | 1/5 | 2/5 | 1/5 | 3/5 | 1:19 |
| | 4. | 1/4 | 1/4 | 2/4 | 3/4 | 1:15 |
| Group C | 1. | 3/4 | 4/4 | 4/4 | 4/4 | 1:4 |
| | 2. | 5/5 | 5/5 | 5/5 | 5/5 | 1:4 |
| | 3. | 4/4 | 4/4 | 4/4 | 4/4 | 1:4 |
| | 4. | 5/5 | 5/5 | 5/5 | 5/5 | 1:4 |
| | 5. | 5/5 | 5/5 | 5/5 | 5/5 | 1:4 |
| | 6. | 5/5 | 5/5 | 5/5 | 5/5 | 1:4 |
| | 7. | 5/5 | 5/5 | 5/5 | 5/5 | 1:4 |
| | 8. | 4/5 | 5/5 | 5/5 | 5/5 | 1:4 |
| | 9. | 5/5 | 5/5 | 5/5 | 5/5 | 1:4 |

*SN challenge level = 316 TCID
SN challenge virus – Onderstepoort CD virus adapted for use on Vero cell line

*Private Practitioner, Nelspruit.
†National Zoological Gardens, Pretoria.

§Enduracel d-h, Smith Kline Animal Health, Box38, 1600 Isando.


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the standard serum neutralization test. The prevaccination antibody level in all animals was lower than 1:4. The post-vaccination antibody levels are given in Table 1.

## DISCUSSION

The wild dog in the Republic of South Africa is an endangered species whose range is largely restricted to the larger national parks[3]. The protection of breeding or non-breeding packs of wild dogs in captivity is thus of the utmost importance. The random administration of vaccine(s) against disease(s) which might possibly affect them is not only an unsatisfactory undertaking but also a potentially dangerous one[5]. Itakura et al.[5] described fatal canine distemper in lesser pandas following the administration of live canine distemper vaccine. One of us (W.H.S.) has observed distemper-like clinical signs in a wild dog pup after the administration of a live canine distemper vaccine. Conjunctivitis, rhinitis as well as posterior paresis were observed.

The serological investigation described here yielded inconclusive results. Serum samples taken before the administration of vaccine did not reveal a protective antibody level. The very low antibody level obtained following vaccination or repeated vaccinations might have been due to a) poor multiplication of the attenuated virus in the tissues of wild dogs, b) resistance on the part of the animals to the virus, or c) in addition, in the case of Group C, the 2 week interval between vaccination and serum antibody determination might have been too short for antibody development to have occurred.

Nevertheless high or low antibody levels need not necessarily indicate whether or not wild dogs are susceptible to canine distemper. Determination of antibody levels also does not evaluate cell-mediated immunity. The only satisfactory method of determining their susceptibility to canine distemper or of evaluating the protective value of serum antibody would be to challenge with a virulent strain of virus.

It is, however, of some significance to note that the 3 pups in Group A were reared under conditions where they had excellent chances of naturally contracting canine distemper virus infection. They did not do so despite the fact that no or very low serum antibody levels were determined and that they were hypoproteinaemic and relatively weak for some time after capture[7].

It is unfortunate that Hofmeyr's diagnosis of canine distemper in the wild dogs was not confirmed by laboratory examination[4]. It is well known that the clinical signs of toxoplasmosis in domestic dogs can resemble those of canine distemper[8].

## ACKNOWLEDGEMENTS

We are indebted to Norden Laboratories, Lincoln, Nebraka, USA, for undertaking the serological determinations. This investigation was made possible through kind permission of the Director of the National Zoological Gardens and the Director, Nature Conservation Division, Transvaal Provincial Administration.

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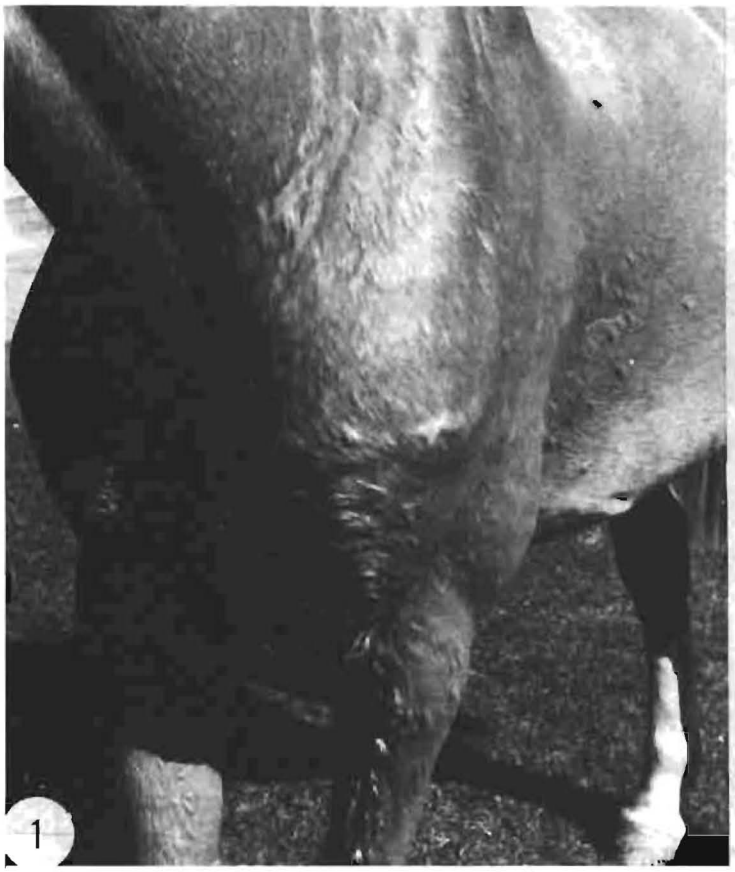
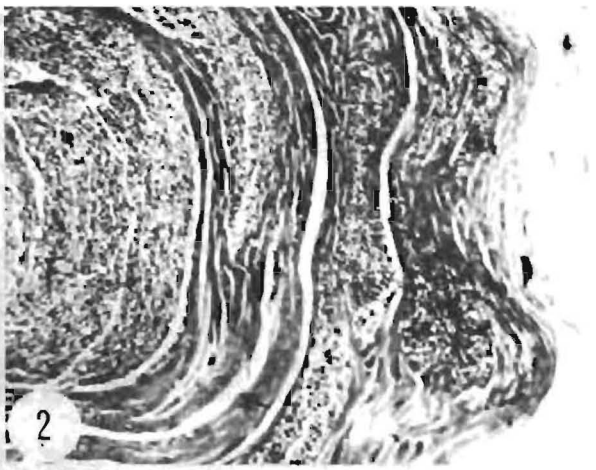
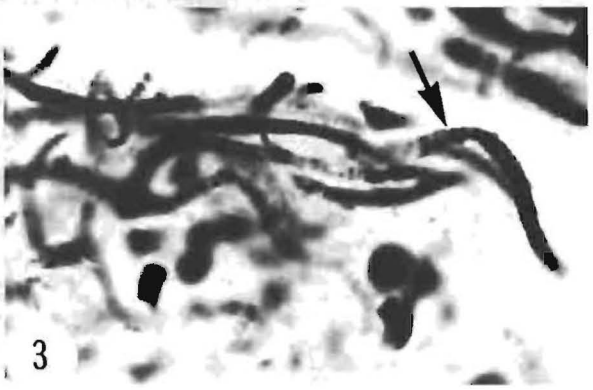
**TREFFERBLAD** **FEATURE PAGE**







## DERMATOFILOSE BY 'N PERD

Dermatofilose is 'n kroniese eksudatiewe korsvormende dermatitis veroorsaak deur *Dermatophilus congolense* by perde. Die toestand is meer bekend in skape en beeste en was in die verlede na verwys as streptotrikose. Fig. 1 toon die verhewe omskrewe letsels op die lyf en bene van 'n 5 jaar oue Palamino hings. Die letsels het oorspronklik soos urtikarie gelyk maar het later 'n duidelike droë roof gevorm. Behandeling het bestaan uit daaglike wassing met povidone-jodium sjampoe* asook binnespierse inspuiting van 15 ml dihidrostreptomisien sulfaat† bid vir 4 d en 20 ml penisilien‡ daagliks vir 10 d. Algehele genesing het gevolg.

Histopatologies (Fig. 2) word daar 'n duidelike laagvorming in die kors gesien. Daar is etlike lae van gekeratiniseerde epiteelselle wat geskei word deur lae van degeneratiewe inflammatoriese selle (hoofsaaklik polimorfonukleêr) Akantose en enkele intra-epidermale mikroabsesse is waarneembaar.

Die *D. congolense* organismes is beste sigbaar as vertakkende filamentagtige hifadrade in die gekeratiniseerde epiteel van die buitenste haarskede. Fragmentasie in die dwarste gee 'n baie kenmerkende vorm aan die organismes (deur die pyl aangedui in Fig. 3). Die organismes is Gram-positief en kleur ook duidelik met Giemsa kleuring.

Ingestuur deur
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## EQUINE DERMATOPHILOSIS

Equine dermatophilosus is a chronic exudative scab-forming dermatitis caused by *Dermatophilus congolense.* The condition is well known in sheep and bovines where it was known as streptotrichosis. Fig. 1 shows the raised circumscribed lesions on the body and legs of a 5 year old Palamino stallion. The lesions, originally resembled urticaria but later a dry scab formed. Treatment consisted of daily washing with povidone-iodine shampoo* as well as intramuscular injections of 15 ml dihydrostreptomycin sulphate† bid. for 4 d and 20 ml penicillin‡ daily for 10 d. Complete recovery followed.

Upon histopathological examination (Fig. 2) the stratification of the scab is clearly visible. There are several layers of cornified epithelium separated from each other by layers of degenerated inflammatory cells (mainly polymorphonuclear). Acanthosis and some intra-epidermal microabscesses are observed.

The *D. cogolense* organisms are best demonstrated in the keratinised epithelium of the external hair sheath. Transverse fragmentation is characteristic for this organism (indicated by an arrow in Fig. 3). The organisms are Gram positive and stained well with Giemsa's stain.

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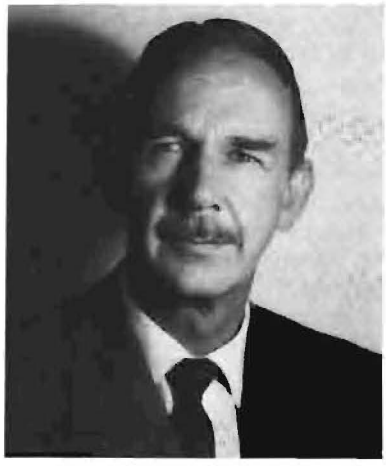
## MARTHINUS CHRISTOFFEL LAMBRECHTS



Die Goue Medalje van die Suid-Afrikaanse Veterinêre Vereniging word vanjaar toegeken aan dr M. C. Lambrechts ter erkenning van sy besondere bydraes tot die ontwikkeling van staatsveterinêre medisyne in Suid-Afrika as 'n wetenskap en die daarstelling van 'n landswye diagnostiese diens.

Marthinus Christoffel Lambrechts is op 20 Maart 1912 in die Wolmaransstad-distrik gebore en verwerf 'n eersteklas matrieksertifikaat aan die Hoërskool Wolmaransstad in 1931. Hy behaal in 1936 die BVSc graad aan die Fakulteit Veeartsenykunde van die Universiteit Pretoria te Onderstepoort.

Dr Lambrechts begin sy loopbaan as veearts by die Allerton Laboratorium naby Pietermaritzburg in 1937, maar word in dieselfde jaar oorgeplaas na Umtata in Transkei. Sy vernaamste pligte aldaar was om die voorgeskrewe beheermaatreëls vir Ooskuskoors en skaapbrandsiekte toe te pas.

Dit is hier waar hy die epidemiologie van Ooskuskoors deur persoonlike ondervinding intiem leer ken het en die grondslag gelê is vir die besondere bydraes wat hy tot die uitroeiing van die siekte gemaak het. Hy het inderdaad 'n sleutelrol gespeel in een van die grootste wetenskaplike prestasies van staatsveterinêre medisyne in die wêreld, naamlik die uitroeiing van Ooskuskoors in Suid-Afrika.

Die uitroeiing van Ooskuskoors was die vrug van die toepassing van basiese wetenskaplike kennis oor die siekte wat op 'n presiese, besonder verantwoordelike wyse deurgevoer is. Dit was egter veel meer as net 'n militaristiese tipe operasie. Dit het oor meer as 50 jaar gestrek en in die proses is 'n wetenskap van sy eie geskep.

Vanweê sy besondere ervaring met die beheer van Ooskuskoors is dr Lambrechts in 1944 na Noord-Natal oorgeplaas waar 'n uiters problematiese situasie in die vorm van een van die laaste broeiplekke van Ooskuskoors geheers het. Doelgerigte optrede was nodig wat hy ten spyte van sterk teenkanting op onversteurde, vasberade, maar nogtans diplomatiese wyse toegepas het.

Op dr Lambrechts se aandrang is 'n uitslagbeleid in die finale fase van die uitroeiingskampanje ingevoer. Dit is gedoen omdat hy met sy fyn waarnemingsvermoë, ten spyte van laboratoriumuitsprake (waarmee tot in die sestigerjare volgehou is) dat daar geen draertoestand by Ooskuskoors is nie, tot die gevolgtrekking gekom het dat herstelde beeste wel as draers van die siekte optree. Die gevolg was dat 'n siekte wat 'n beraamde verlies van 5,5 miljoen beeste tot gevolg gehad het in 1954 totaal uitgeroei is.

Dr Lambrechts is in 1953 as Teringbeampte aangewys met die doel om uiteindelik 'n uitwissingsskema vir tuberkulose daar te stel. Voordat die skema egter in werking gestel kon word was dit nodig om op verskeie aspekte van die siekte navorsing te doen. Hy het veral navorsing op suurvaste organismes wat die sogenaamde velletsels veroorsaak gedoen. Hy het die organismes vir die eerste keer in die land gekweek en 'n proeftuberkulien berei. Sodoende is die diagnose van tuberkulose in Suid-Afrika op 'n beter grondslag geplaas. Na 'n uitgebreide oorsese studiereis het hy in 1958 'n omvattende verslag opgestel wat as grondslag gedien het vir die latere uitroeiingskema wat tans met groot sukses toegepas word.

Een van dr Lambrechts se belangrikste monumente is die diagnostiese diens wat hy ingestel het nadat hy in 1960 beheer van die Afdeling Veeartsenydiens oorgeneem het. Hy het na vore gekom met 'n nuwe benadering en die klem verskuif van 'n militaristiese siektebeheerstelsel na 'n voorkomende gesondheidsdiens.

Ten spyte van baie sterk teenkanting het hy hom met kenmerkende entoesiasme vir die instelling van 'n diagnostiese diens beywer. Hierdie diens is tans besig om 'n immer toenemende invloed op die veebedryf te hê en is reeds bykans onontbeerlik vir die veeboer sowel as privaat praktisyn. Voorts is die Afdeling Veeartsenydiens na aanleiding van 'n memorandum wat hy aan die Rautenbachkomitee voorgelê het struktureel sowel as funksioneel in 1962 omskep tot die meer doeltreffende instelling van vandag. Die sentra waarvandaan die Afdeling Veeartsenydiens tans 'n diagnostiese diens lewer is besig om die veeartsenykundige wetenskap in Suid-

Afrika op 'n voorheen ongekende skaal te bevorder. Die stigting van verdere sentra, soos wat dr Lambrechts dit destyds beoog het, word tans in die vooruitsig gestel.

Dr Lambrechts se ondervinding met die bekamping van bek- en klouseer, veral tydens die groot uitbreek in Springs in 1957, het hom 'n waardevolle lid, en in sommige gevalle voorsitter van verskillende bek- en klouseer kommissies gemaak. Sy bydrae hier het 'n groot invloed op die latere doeltreffende bekamping van die siekte en die instandhouding van ons internasionale handel in landbouprodukte gehad.

Hy het Suid-Afrika internasionaal op hoë veterinêre vlak gedien. Met sy aangename persoonlikheid en breë kennis van sy vakgebied het hy groot agting onder sy vooraanstaande kollegas afgedwing. Hy het byvoorbeeld in 1969 die "Medal for Dedicated Service to Africa" van die "Royal African Society" ontvang.

As Direkteur van Veeartsenydiens het hy gereeld kongresse en vergaderings in die buiteland bygewoon en talle referate, ook ten behoewe van veeartsenykundige navorsing, voorgedra. Daardeur het hy Suid-Afrika se aansien as een van die leiers op die gebied van die veeartsenykundige wetenskap hoog gehou en sy Afdeling se reputasie as een van die doeltreffendstes van sy soort uitgebou.

Dr Lambrechts het sy professie ook op getroue wyse gedien. Hy het vanaf 1956 to 1971 op die Raad van die SAVV gedien en hy was lid van die Veeartsraad vanaf 1961 tot 1970. Voorts was hy vanaf 1961 tot 1971 dosent in staatsveterinêre medisyne aan die Fakulteit Veeartsenykunde te Onderstepoort en het hierdeur hierdie deel van die veeartsenykundige wetenskap bevorder.

Dr Lambrechts is inderdaad 'n waardige wenner van die SAVV se Goue Medalje. Deur hom te vereer, vereer ons ook sy gade wat hom deurgaans getrou bygestaan het, asook die instelling wat hy met soveel toewyding en entoesiasme gedien het, naamlik die Afdeling Veeartsenydiens.

**TOEKENNING** **AWARD**

# JACK BOSWELL-TOEKENNING VIR 1980

## ABRAHAM PAUL SCHUTTE

Die Jack Boswell-Toekenning vir 1980 van die Suid-Afrikaanse Veterinêre Vereniging word aan dr A. P. Schutte toegeken. Die besonder entoesiastiese en onbaatsugtige wyse waarop hy die veterinêre professie en die SAVV gedien het en nog steeds dien is inderdaad by veeartse dwarsdeur die land so goed bekend dat verdere woorde eintlik oorbodig is.

Abraham Paul Schutte is op 14 Januarie 1935 gebore. Hy kwalifiseer in 1959 as veearts by die Fakulteit vir Veeartsenykunde te Onderstepoort, behaal in 1964 die M. Med Vet. (Gyn). graad en in 1969 'n Spec. Doct. (met lof) te Gent, België.

Hy begin sy loopbaan as veearts in privaat praktyk, wend hom dan na veterinêre diagnostiek, doseerwerk en navorsing en was tot onlangs staatsveearts in diens van die Afdeling Veeartsenydiens. Tydens sy termyn as seksiehoof van die Reproduksie-afdeling van die Navorsingsinstituut vir Veeartsenykunde te Onderstepoort word hy in 1974 tot Assistent-Direkteur van Veeartsenykunde in sy persoonlike hoedanigheid op sterkte van sy besondere wetenskaplike prestasies bevorder.

Dr Schutte se intieme betrokkenheid by SAVV aangeleenthede begin reeds in 1961 toe hy lid van die Komitee en later voorsitter van die Produksie- en Reproduksie-groep word. In 1973 word hy as Raadslid van die SAVV verkies en drie jaar later in 1976 tot President van ons Vereniging. Hy dien vir twee jaar met onderskeiding in die hoogste amp van die SAVV waarna hy hom weens sy verplasing na Mosselbaai nie weer verkiesbaar stel nie.

Tydens sy ampstermyn as President en Raadslid bereik hy 'n vlak van produktiwiteit in die diens van sy professie wat min hom sal kan nadoen. Hy dien op 'n breë spektrum van vaste en ander komitees van die SAVV soos die Uitvoerende Komitee, Finansies-komitee, Opleidingskomitee, die Komitee insake Veterinêre Spesialisasie en die Veeartswetkomitee. Hy geniet ook buite sy professie besonder hoë aansien as veterinêre spesialis. Hy is byvoorbeeld sedert 1977 lid van die Minister van Landbou en Visserye se Adviesraad vir Diereproduksie, asook lid van die Minister se Adviseskomitee vir Kunsmatige Inseminasie sedert 1973. Hy is ook lid van 'n Internasionale Komitee vir Kl en Reproduksie.

Hy was betrokke by, of self verantwoordelik vir, die opstel van verskeie omslagtige memoranda wat almal basies dieselfde doel voor oë gehad het, naamlik die bevordering van die veearts en sy professie. Deur middel van hierdie in diepte studies het hy die toekomsbehoeftes ten opsigte van veterinêre mannekrag en opleiding vir ons land bepaal. Ook het hy die saak van die staatsveearts en die landelike praktisyn by elke moontlike geleentheid en op die hoogste vlak herhaaldelik by wyse van memoranda en persoonlike onderhoude bepleit.

Awie Schutte is een van die grootste kampvegters vir 'n tweede veterinêre fakulteit vir die Republiek van Suid-Afrika en het onvermoeid gepoog om die besluitnemers positief te beïnvloed om aan die SAVV se versoeke in hierdie verband gehoor te gee. Hy het meer onderhoude met Ministers gevoer tydens sy termyn as President van die SAVV as enigiemand voor hom, alles met die uiteindelike doel om die saak van die veearts en die gehalte van sy opleiding te bevorder.

Selfs waar hy nou geografies taamlik afgesonderd is het hy met onvermoeide ywer voortgegaan met sy oorredingswerk. Hy het in die afgelope 18 maande meer as 50 voordragte voor onder andere die Parlementêre Landbou-studiegroep, tak-en groepkongresse van die SAVV, boeredae, landbou studiegroepe en landbou-koöperasies gelewer. Dit is dus duidelik dat ook in sy nuwe werksfeer dr Schutte op kenmerkende, onverskrokke wyse en met onblusbare energie voortgegaan het om alle geleenthede ten volle te benut om sy professie se aansien uit te bou.

Tydens die lang jare van lojale, tydrowende diens aan die professie is hy getrou en met eindelose geduld deur sy gade Kinnie bygestaan. Sy het inderdaad 'n onbetwiste aandeel in hierdie toekenning losgeslaan.

Dr. Schutte voldoen aan alle vereistes wat vir die Jack Boswell-toekenning gestel word. Hy is 'n besonder waardige ontvanger van hierdie gesogte toekenning.

## FAKULTEIT VEEARTSENYKUNDE
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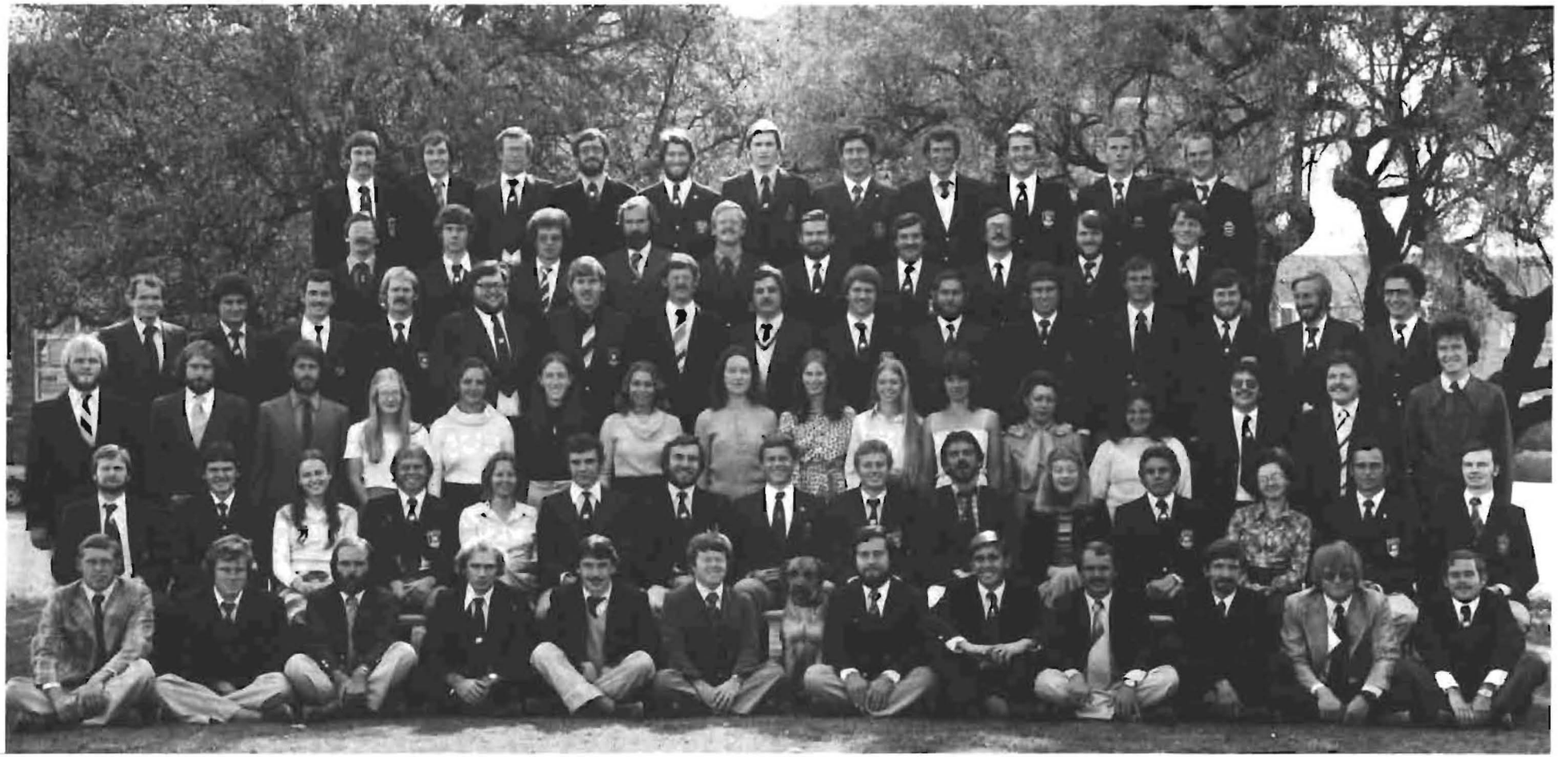

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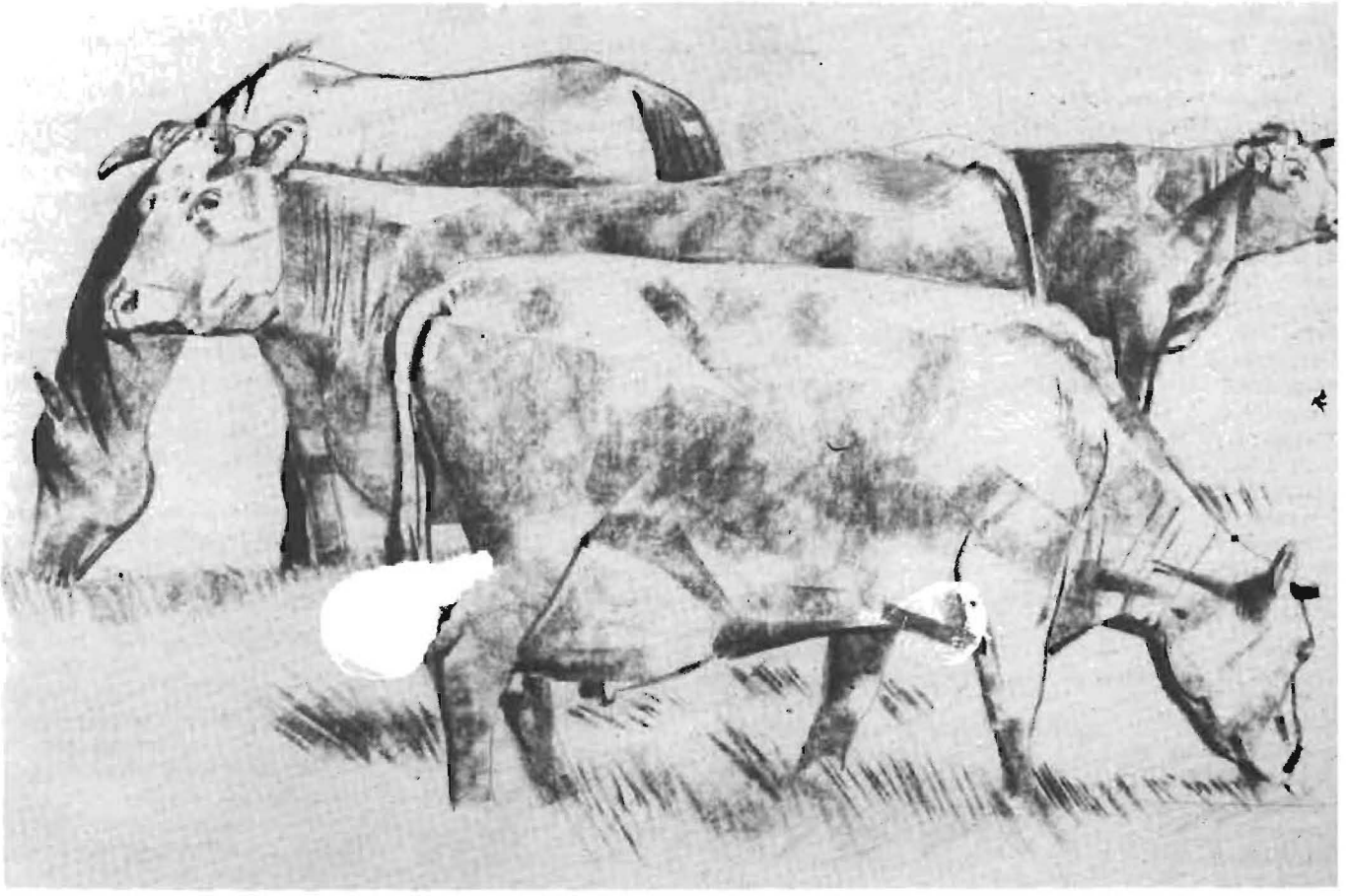

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