CORRIGENDA

1. Book Review : "CIH Keys to the Nematode Parasites of Vertebrates" Vol 47(1) p 42 : "A.G. Chits" should read "A.G. Chabaud".
2. "The control of ticks, fleas and lice on dogs by means of a Sendran*-impregnated collar" Vol 47(1) p 17 — I.G. Horak: The trade mark of Sendran* is incorrectly attributed to Thuron Industries Inc. It is in fact held by Bayer A.G. Leverkusen.

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EDITORIAL

BLUE INTRAMAMMARIES, ANTIMICROBIAL RESIDUES IN MILK, AND THE VETERINARY PROFESSION

The presence of antimicrobial residues in milk constitutes a potential public health hazard and more often results in starter failures in cultured dairy products such as cheese and yoghurt. Repeated requests from dairy scientists and the manufacturers of dairy products have now resulted in action in terms of Act 36 of 1947. As from 1st January 1977 all "stock remedies" registered for intramammary therapy of bovine mastitis will contain one of the blue-green food dyes (Food Blue No 2 and No 3, Food Green No 4*) as a marker for the presence of the antimicrobial substances present in the remedy. Such remedies are already on the market, and all undyed mastitis stock remedies will have to be off the shelf before the end of this year. ("Stock remedies" are available without prescription).

The primary purpose of including a marker dye in mastitis remedies is to enable the milk producer and his farm workers to recognise that milk from a treated cow contains residues of the remedy during and for some time after treatment. It is further intended as an aid to the producer who wants to sell only residue-free milk. By synchronising excretion of the marker dye and the antimicrobial substance(s) the pharmaceutical industry has provided the milk producer with a visible indicator of the presence of such residues in milk. Dye marked intramammary remedies are therefore of considerable benefit to the milk producer, industry and the consumer. This has been proved in Australia (State of Victoria). Dye marked mastitis remedies are also in use in France and Japan.

Dye concentration methods will enable health officials and milk reception personnel to detect subvisible concentrations of dye in bulked market milk, but this method cannot replace conventional microbiological methods of monitoring the presence of antimicrobial residues. The reason is the fact that intramammary therapy is not the only source of milk contamination. There is always some transfer from treated to untreated quarters, and parenteral administration, including intra-uterine therapy, also results in residues in milk. It is also not unusual for antimicrobials, intended for intramuscular or intravenous use, to be administered to the quarter via the teat canal and then, of course, there are the prescription intramammary formulations which are not subject to registration in terms of Act 36/1947 and need not therefore be dye marked at this stage. Such medicines are channeled through the veterinarian for use, sale or prescription for treatment of his patients. Dye concentration tests on market milk would thus be

VAN DIE REDAKSIE

GEKLEURDE MASTITISMIDDELS, ANTIMIKROBIESE RESTE EN DIE VEEARTS

Die aanwesigheid van antimikrobiese reste in melk is 'n potensieel-gesondheidskadelike faktor maar is meer dikwels verantwoordelik vir produksieprobleme t.o.v. gekweekte suiwelprodukte soos kaas en joghurt. As gevolg van herhaalde versoeke van suiweltegnoloë en vervaardigers van suiwelprodukte word nou kragtens Wet 36 van 1947 opgetree. Vanaf 1 Januarie 1977 moet alle "veemiddels" wat vir binnespeense behandeling van mastitis geregistreer is, een van die blou-groen voedselkleurstowwe (Voedselblou N^{r}2 en N^{r}3, Voedselgrown N^{r}4*) bevat as merker vir die aanwesigheid van die antimikrobiese substanse in die middel. Sulke middels word alreeds bemark. Alle ongemerkte "veemiddels" vir mastitis moet voor 31 Desember 1976 van die handelaar se rak af wees ("Veemiddels" is sonder voorskrif beskikbaar).

Die primêre doel van insluiting van 'n merkerkleurstof is om aan die melkprodusent en die melkers te toon dat melk van 'n behandelde kwart tydens en vir enkele dae na behandeling nog reste van die middel bevat. Dit is verder 'n hulpmiddel vir die produsent wat daarin belang stel om slegs reste-vrye melk te bemark. Deur die uitskeiding van die antimikrobiese stof en die kleurstof te sinkroniseer het die farmaseutiese bedryf die produsent voorsien van sigbare tekens van die aanwesigheid van sodanige reste in die melk. Kleurstofgemerkte binnespeense middels strek derhalwe tot aansienlike voordeel vir die melkprodusent, die bedryf en die verbruiker. Dit is reeds bewys in die Australiese staat van Victoria en gekleurde mastitismiddels word ook in Japan en Frankryk gebruik.

Terwyl konsentrasiemetodes beskikbaar is waarmee gesondheidsbeamptes en melkontvangspersoneel subsigbare konsentrasies van kleurstof in grootmaatplaasmelk kan vasstel, kan hierdie metodes nie die konvensionele mikrobiologiese metodes vir die monitor van antimikrobiese reste in melk vervang nie. Die rede hiervoor is dat binnespeense behandeling nie die enigste bron van sodanige besoedeling is nie. Daar is altyd 'n mate van oordrag, via die bloedstroom, van antibiotika vanaf die behandelde na ander kwarte. Verder lei parenterale toediening, insluitend intra-uterine toediening, ook na reste in die melk. Dit is ook bekend dat antimikrobiese stowwe wat vir binnespierse en binne-aarse gebruik verpak word, soms binnespeens toegedien word. Dan is daar nog die voorskrif mastitismiddels wat nie aan registrasie kragtens Wet 36/1947 onderhewig is nie en derhalwe totnogtoe nie met kleurstof gemerk moet word nie. Laasgenoemde word deur die veearts gebruik, verkoop of voorgeskryf vir die behandeling van diere onder sy

* Colour Index Nos 42090, 42045 and 44090 respectively.

of no use in detecting residues resulting from the use of these undyed antimicrobial formulations.

Despite their obvious advantages, there will always be some milk producers who, for reasons best known to themselves, will try to avoid the use of dye marked intramammaries. Recent pressure selling of unmarked registered mastitis remedies to get them off the shelf before the end of the year may well add to this. It must therefore be anticipated that practicing and other veterinarians will be requested to prescribe or supply unmarked therapeutic formulations. The profession has a clear obligation to emphasize the purpose and value of dye-marked "stock remedies". It should under no circumstances permit itself to become party to any attempts to evade the use of dye marked remedies without good and just cause.

But the veterinarian's obligation, both moral and legal, goes a long way further. Firstly, no substances which are scheduled in terms of Act 101/1965 may be used, sold or prescribed except by a veterinarian and then only for the treatment of **animals (patients) under his care and control.** This means, *inter alia* that the veterinarians' receptionist or lay assistant may not sell such medicines, even to *bona fide* clients.

Where the veterinarian does use, sell or prescribe antimicrobial substances in the course of his professional activities, he has a very clear obligation to ensure that the owner of the animal(s) is fully and properly informed about the method of use of the medicine, **including the fact that milk from treated cows and quarters should not be used during and after treatment** for a specified period of time. Should a milk producer be prosecuted for the presence of antimicrobial residues in his milk supply, or should the producer or the dairy product manufacturer suffer economic loss because of such residues, there is little doubt that the veterinarian can be held responsible, if it can be shown that he did not properly inform the milk producer.

To obviate any misunderstanding which may result from verbally communicating the necessary information it is suggested that the issue of printed instructions would be advisable. The manufacturers and distributors of prescription intramammaries might well supply veterinarians with suitably worded leaflets, or otherwise the veterinarian could produce his own.

It cannot be overstressed that the veterinarian at present enjoys the **privilege of unrestricted use** of therapeutic substances scheduled in terms of the Medicines Control Act 101/1972. The recent restriction of the range of stock remedies registered in terms of Act 36/1947 has emphasized this privilege. Let us always bear in mind that this is a privilege, not a right; that it can be withdrawn; and that we should guard it with the full sense of responsibility which we as a profession possess — a responsibility to others as well as ourselves.

sorg. Kleurstofkonsentrasietoetse op marksmelk sou dus van geen nut wees as aanduiding van reste afkomstig van sulke bronne nie.

Ten spyte van die duidelike voordele daaraan verbonde moet verwag word dat sekere produsente, om redes slegs aan hulle bekend, sal poog om die gebruik van kleurgemerkte mastitismiddels te vermy. (Die huidige grootskaalse verkoopspogings om ongekleurde "veemiddels" vir mastitis voor die einde van die jaar van die rak af te kry mag daartoe bydra). Dit moet derhalwe verwag word dat praktiserende en ander veeartse onder druk sal verkeer om ongekleurde voorskrif mastitismiddels te voorsien. Die professie het 'n baie duidelike plig om die doel en voordele van die gebruik van gekleurde "veemiddels" vir mastitis te beklemtoon. Onder geen omstandighede kan lede van die professie toelaat dat hulle sonder baie goeie rede mededadig word aan pogings om die gebruik van gekleurde mastitismiddels te vermy nie.

Die wetlike en morele plig van die veearts strek egter veel verder. Eerstens mag geen substanse wat kragtens Wet 101/1965 geskeduleer is, verkoop of voorgeskryf word behalwe deur 'n veearts en dan slegs vir behandeling van *diere (pasiënte) onder sy sorg en beheer.* Dit beteken o.a. dat die veearts se hulppersoneel nie sodanige voorskrifmiddels mag verkoop selfs aan sy bona fide kliënte nie.

Waar die veearts wel antimikrobiese middels in die loop van sy professionele bedrywighede gebruik, verkoop of voorskryf, lê daar by hom 'n duidelike plig om te verseker dat die eienaar van die diere ten volle ingelig is betreffende die metode van gebruik daarvan, *insluitend die feit dat melk van behandelde koeie en kwarte vir 'n gespesifiseerde tydperk na behandeling nie bemark moet word nie.* Indien 'n melkprodusent vervolg sou word omrede daar antimikrobiese reste in sy melkvoorraad gevind is, of sou 'n produsent of die vervaardiger van suiwelprodukte geldelike verliese a.g.v. sodanige reste sou ly, so die veearts ongetwyfeld daarvoor aanspreeklik wees indien bewys kan word dat hy nie die produsent behoorlik ingelig het nie.

Ten einde enige misverstand a.g.v. mondelingse oordrag van die nodige inligting te vermy, word voorgestel dat uitreiking van skriftelike instruksies wenslik sou wees. Vervaardigers en verspreiders van voorskrifmiddels vir mastitis kan moontlik veeartse van toepaslik bewoorde strokies voorsien. Anders moet die veearts hulle self opstel.

Daar moet nogmaals benadruk word dat die veearts huidiglik die *voorreg van onbeperkte gebruik* van terapeutiese stowwe kragtens die Medisynebeheerwet 101 van 1972 geskeduleer. Die onlangse beperking van "veemiddels" geregistreer kragtens Wet 36/1947 het hierdie voorreg beklemtoon. Dit bly 'n voorreg, nie 'n reg nie, en dit kan onttrek of beperk word. Elke lid van die professie dra die verantwoordelikheid om die voorreg te beskerm deur streng korrekte optrede in belang van ander sowel as homself.

Jl. S. Afr. vet. Ass. 47(3) 159-160 1976

ADDRESS VOORDRAG

MANKIND'S MOST BASIC NEEDS — HOW CAN THE VETERINARY PROFESSION MORE MEANINGFULLY RESPOND?*

Calvin W. Schwabe, D.V.M., Sc.D.**

It should be obvious to all of us here on this first World Veterinarians' Day that the most critical world needs today are for enough food, adequate health, reasonable environmental quality and societies in which humane values prevail. Meeting these needs is far from a simple matter. It is not a certainty. If we are to be successful, it will require every creative input of which mankind is capable. There are few professions which speak to these several most basic needs of man in such a great variety of ways **individual** veterinarians do. Our extremely varied programme here this week testifies well to that. Yet, despite this wide range of our individual efforts as veterinarians, many of our **collective** responses as a profession fall short of their potential or, at best, seem to lack direction. And even when we in the veterinary profession do respond with a reasonably well-organized approach, our efforts frequently are poorly articulated and understood. And, most unfortunately, this lack of understanding of our purpose is almost as evident within our profession as without.

Where do our professional problems lie in this regard? For example, why is it that most official and popular statements of world food policy have reflected so little creative input from veterinarians and other animal scientists? Or why is it that our profession's full importance to man's health still remains so poorly understood by the public and by officials in government in most countries? And so on. In my opinion, these are merely representative of the types of questions to which the veterinary profession *en toto* must begin to elevate its sights — **now**.

One overriding problem is that, by definition, our field is an extremely broad one, embracing as it does the study, and identification of the relationships to man, of an enormous variety of problems of health and disease in the almost one million species of non-human animals with which we share this globe. As pointed out by Professor Beveridge, the paradox is simply that the world's total of only some 200 000 veterinarians is spread almost impossibly thinly over this immense field for potential veterinary contributions. As a consequence, in only very few areas of its overall societal responsibilities do veterinary medicine's efforts possess anything approaching enough depth. Even in those two countries which together account for over a quarter of the world's total veterinarians, our profession's overall social role has not been fully expressed, much less adequately fulfilled. A result is that almost all veterinarians everywhere are so fully involved day to day with the **particular** so as to almost completely neglect the **general**. Most of us are individually narrow, focusing our efforts far too much upon our many "trees" at the expense of much needed collective expressions of a professionally **holistic** view of our several important "forests".

A first priority for remedying this situation is to begin to much better **document** specifically just what the world's 200 000 individual veterinarians actually **do**. Meetings like this aid in this process, and invariably, whenever we have attempted this anywhere even we ourselves have been surprised at what we now are doing and where our social impact is being felt. As but one example with respect to our health science role, a detailed census three years ago of the 111 faculties of human medicine in the United States showed quite unexpectedly, that 346 veterinarians then held teaching, research or service positions on these faculties. An even more suprising finding was that some 54 percent of this total number of 346 veterinarians were not laboratory animal specialists, the most obvious area for a veterinary input. Instead we have a situation now where 219 veterinarians are serving in American medical schools alone as professors, associate professors and assistant professors in 35 different disciplinary areas ranging from the more familiar areas of microbiology and pathology into less expected areas such as obstetrics, anesthesiology and tropical medicine.

Second to this necessity to document the present variety and extent of our efforts — to compile our record — is the necessity to evaluate this information realistically, to articulate it whenever necessary, and to act accordingly by using it to plan our future efforts in veterinary education and service. For instance, any realistic appraisal of our past and present **research** efforts which are directly or indirectly applicable to human health results in an inescapable conclusion that, despite our small numbers, we veterinarians, from this research point of view, are the second most important human health profession. How many basic medical discoveries have been made by dentists, nurses or pharmacists? Very few. Yet how many of us in or out of our profession appear to be aware of our importance as a human health profession and act accordingly.

Third, there is the clearly evident need everywhere in the world, but particularly in the so-called economically developed countries, for us to subject our governmental instruments for veterinary service and our veterinary schools to intensive, continuing and completely open-minded scrutiny. All human institutions as they age are subject to what I call "progressive ankylosing encephalo-non-creatitis" and many of our veterinary institutions display classic symptoms of this malady. Governmental veterinary services in many countries are organized — and are doing things — almost exactly as they have been for

* Opening address for first World Veterinarians' Day, XXth World Veterinary Congress, Thessaloniki, Greece, July 9, 1975.

** School of Veterinary Medicine, University of California, Davis, California, 95616.

the past seventy or eighty years! A highly creative group of veterinarians around the turn of this century first demonstrated an unusually effective group of investigative and control techniques for diseases *en masse* and identified a group of economically disruptive diseases and animal diseases of major public health importance to which they were applicable. Ever since, we have been applying these techniques to these problems successively, and almost routinely, with but little modification and little innovation or real creativity on our part. In large measure our institutional infrastructures in government have ossified as a result and many individual efforts have become stereotyped, narrow and routine — even to the point of boredom. Traditions are useful when they contribute to the development of a sense of creative and confident citizenship in our profession. When they don't, they need to be toppled and replaced.

A consequence of past veterinary successes in control of many economically disruptive livestock diseases has been new patterns of intensified and integrated animal husbandry to which Professor Várnagy will speak and which is increasingly evident in many countries. But the fact is that, even where these efforts have been most successful, many very costly, less well-defined multi-casual diseases (e.g. neonatal losses, metabolic diseases and reproductive inefficiencies) remain which collectively add greatly to the **unit cost** of producing animal protein and other valuable products. When are our governmental services and veterinary schools going to stop concentrating so much on our old battles against infections — important as they are, and begin to engage wholeheartedly in the epidemiological methodological revolution necessary to begin facing such very large food production and management problems more effectively?

Many politicians and economists are being led to partially erroneous conclusions about the cost and value of animal protein. Why? Because food planning bodies seldom consider adequately such things as the complementary and interdependent natures of animal and plant agriculture, particularly in the Third World; or that much of the sun's energy is stored in plant forms not directly consumable by man, but convertible by animals to high quality human food; or that only 60% of the world's grazing lands at most are suitable for cultivation. (Even in the United States over 54% of feed consumed by **all** classes of livestock together still is forage.) Nor do they consider that large populations of readily improvable livestock already exist in many Third World countries, and finally there are the almost completely neglected practical difficulties of altering man's generally strong cultural preferences for animal protein (even to his neglect of other less costly food commodities and other needs).

Lastly, let us consider the role our schools of veterinary medicine have defined for themselves with respect to these most basic needs of man. Here, in my opinion, lies the crux of our entire professional dilemma world wide. And I say this as an educator who must share fully in the responsibility for what our schools are and what they are not. In my view they are not articulating our social goals and not leading our profession in exploring and opening up new vistas of service and in providing the specialized training required for individual veterinarians to meet effectively this great variety of challenges to man. I know of no veterinary school in the world whose professional and post-graduate programmes at all approach the full breadth of our field or are truly focused upon these basic needs of man to which I referred at the onset and to which individual veterinarians can and do speak. Time will not permit more than the briefest consideration of but one example. The drift along the path of least resistance of veterinary schools in my own country is in the allotment of more and more of each school's resources and efforts to the practice of a high quality of curative medicine on individual pets and companion animals. Elsewhere I have coupled my grave concerns about this drift with my equally strong belief that veterinarians in their communities now play — and in the future can play a far greater, role as very much needed demonstrators, especially to the world's youth, of such basically cherished but short-supplied social values everywhere as compassion, tenderness, love, empathy and a respect for life. In other words, we veterinarians can and do help meet the critical need today throughout the world for a more meaningfully interactive humanism. This, **and this alone,** is the social justification for small animal medicine. Yet how often is this great human need and the veterinarian's response to it made the focus in any veterinary school for instruction in small animal medicine? My friend and neighbour, Harry Roswell, will speak further to this theme.

Fortunately, there are bright spots in our schools, as there are elsewhere throughout our profession, but not enough of them. One exciting innovative possibility has been the new "core and track" teaching program for early specialization — or career orientation if you will — first tried by Dr. Baharsefat's faculty in Iran. We in California have copied their system recently.

Now to briefly summarize the points I would make —

We veterinarians **are** doing a lot, but — we don't know what we're doing.

Our efforts tend to lack effective overall direction, focus and long-range goals.

Many of our service institutions at all levels are so archaic as to be completely inadequate vehicles for the full expression of our profession's purposes and social responsibilities.

With rare exceptions, our schools still accept the narrowest of functions and reflect the narrowest of goals. They are not the creative germinal centres of veterinary medical activity society and the profession have the right to expect them to be. They now follow the profession worldwide rather than lead it. This is intolerable.

Ironically, what all of this means is that **veterinary medicine today is much less than the sum of its parts.** This also is intolerable. For we do possess a much greater capacity to speak to man's condition and his most basic needs than we have been able to articulate collectively.

So, on this first World Veterinarians' Day, let us all rededicate ourselves and our efforts to our beloved profession's great and ancient callings. Mankind's tomorrow is **now**. To face it, we veterinarians of the world must unite our efforts to more adequately meet man's needs — his cravings — for enough food, for health, for an adequate environment **and for a more humane world.** To paraphrase Lenin a bit further, Mr. Chairman, we have nothing to lose but our insularity — society has much to gain.

Thank you, ladies and gentlemen, for the honour of addressing you.

Jl. S. Afr. vet. Ass. 47(3) 161-169 1976

STUDIES ON *PARAFILARIA BOVICOLA* (TUBANGUI 1934)

1. CLINICAL OBSERVATIONS AND CHEMOTHERAPY

J.H. VILJOEN*

ABSTRACT: Viljoen, J.H. **Studies on** *Parafilaria bovicola* **(Tubangui 1934).** *Journal of the South African Veterinary Association* (1976) **147** No. 3, 161-169 (En) Section Entomology, Veterinary Research Institute, Onderstepoort 0110, Rep. of South Africa.

A detailed examination on a group of oxen naturally infested with *Parafilaria bovicola* suggested a prepatent period varying from 238 to 250 days. In these animals 54% of all lesions bled only once, 22% a second time and 24% more than twice. Of all active lesions 42% occurred in the shoulder region and decreased from this area both cranially and caudally. The same tendency was noticeable on carcasses after slaughter. During the observation period May 1974 to February 1975 the number of positive animals increased, reaching peak values during September - October 1974, after which a decline was noticed.

The filaricidal effects of nine compounds were tested. These were suramin (used in combination with diethylcarbamazine citrate), thiacetarsamide sodium, fenchlorphos, phosmet, mebendazole, fenbendazole, levamisole hydrochloride and trichlorphon.

By comparison with carcass lesions on untreated control animals only levamisole hydrochloride and fenbendazole could be classified as reasonably effective. Lesions were reduced by 90% by the former remedy and by 80% by the latter. It would appear that fenbendazole may suppress the egg laying ability of female worms.

Eosinophile infiltrations were found in the majority of smears taken from visible lesions and can be regarded as a constant diagnostic feature thus excluding lesions as a result of bruising.

INTRODUCTION

The lifecycle of *Parafilaria bovicola* in the final host is still unknown. In the closely related *Parafilaria multipapillosa* of horses it has been shown that the prepatent period could vary from 281 to 387 days in horses artificially infested[25].

Clinical observations on animals infested with *P. bovicola* have been made by various authors. Lesions caused by this parasite were usually observed in the Northern hemisphere between December and the following July after which they disappear[6 27]. In the Southern hemisphere this would coincide with the midwinter to midsummer period from June to January. The clinical appearance of these lesions as well as their general distribution in the live animal have also been described[6 22 24 27]. Van den Heever *et al*[39] calculated the percentage distribution of 129 parafilarial lesions on beef carcasses, both in a horizontal and vertical plane. In the horizontal plane (cranio-caudally) they found the neck, withers, thorax, loins and hind quarters, in this sequence, to be the most important areas affected.

With the exception of India, the chemotherapy of *P. bovicola* in cattle has not been undertaken on a very extensive scale. This parasite is common in Indian cattle and various antimonials have been used in the past to combat the disease[12 18 21 30 35]. Active ingredients used were tartar emetic, sodium antimony biscatechol disulphonate, sodium antimony tartrate, lithium antimony thiomalate and other related drugs. Whereas it was possible for these workers to produce clinical recovery a few months after treatment, it is not clear whether these drugs could also kill the parasite.

In addition to the antimonials, suramin and the organo phosphorus compounds trichlorphon have also been used on infested cattle but with very poor results clinically[30].

In the treatment of filariasis in other animals and man, a wide variety of active ingredients have been used. Thiabendazole as a representative of the benzamidazole group has been used in the treatment of *Onchocerca volvulus* in a number of human patients[7]. The results were poor although a slight reduction in the microfilarial counts occurred.

The organo phosphorus compounds, fenthion and dichlorphos have been tested against *Dirofilaria immitis* in dogs but only the former could reduce the adult parasite population significantly over a period of 6 months of treatment[9 10]. The arsenical preparation thiacetarsamide sodium combined with fenthion can eliminate the microfilariae of *D. immitis* from the blood of dogs[19]. While trichlorphon was shown to have a partial microfilaricidal action in human beings affected with *O. volvulus*[8], a degenerative effect on the reproductive system of female worms could also be demonstrated[28 29].

Levamisole hydrochloride is lethal for adult *D. immitis* worms in dogs[37 38] and also reduces the microfilarial counts in the blood of dogs affected with dirofilariasis and *Dipetalonema* spp[1].

The trypanocidal drug suramin in combination with a piperazine derivative has proved to be effective against both adult worms and microfilariae in various filarial diseases[2 3 15 16 20 32].

The original work on the piperazine compounds demonstrated that hetrazan or 1 - diethyl carbamyl - 4 - methyl piperazine hydrochloride was the most promising of all derivatives with an immediate and sustained reduction of microfilariae in cotton rats infested with *Litomosoides carinii* and dogs with *D. immitis*[14]. More recent work has confirmed the microfilaricidal action of hetrazan in heartworm infested dogs[9 36]. In human medicine this drug is used to reduce or eliminate microfilariae in patients suffering from *O. volvulus*[2 15]. Filariasis in horses caused by *P. multipapillosa* has been treated unsuccessfully with hetrazan[11 31].

Arsenical preparations proved to be effective

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against the adult *Dirofilaria* parasite in dogs and were therefore used either alone or in combination with dithiazanine iodide or fenthion to eliminate microfilariae from the blood[1 4 13 34]. The most common arsenicals used were thiacetarsamide sodium, melarsonyl oxide, melarsonyl potassium, arsenamide and dichlorophenarsine hydrochloride. The results against adult worms particularly with thiacetarsamide sodium were good to excellent[5 17 23].

The microscopic appearance of parafilaria lesions have been described in the Indian buffalo[26] and in cattle[27]. From the detailed descriptions especially in the latter host it must be concluded that infiltrations of roundcells, ie. lymphocytes, macrophages and particularly polymorphonuclear cells (eosinophiles) are quite typical of parafilarial lesions.

In the present paper the results of the following investigations are given:

(i) A detailed clinical examination of a group of infested oxen in the vicinity of Onderstepoort.
(ii) Chemotherapeutic trials with at least one or more of the most promising drugs in each of the different classes of antifilarial compounds.
(iii) Frequency distribution of carcass lesions in all animals slaughtered in order to ascertain the most important carcass areas affected.

MATERIALS AND METHODS

Experimental animals

Crossbred Afrikaner × Simmentaler calves from the government experimental farm at Zoutpan in the northern Transvaal approximately 45 km north of Pretoria, were used for the experimental work. These calves were born between October and December 1973, bull calves were castrated at an age of 2 to 3 months, and all calves weaned 8 months after birth.

Preliminary Observations

From June 1974 the experimental farm was visited weekly in order to identify clinically positive animals and to subdivide these animals into a control and eight different treatment groups. On each visit bilateral cutaneous examinations were done on each calf and the position of all new lesions marked on a chart. For further reference the locality of each lesion was indicated on this chart by a horizontal measurement taken from the root of the tail to a point on the topline immediately above the lesion and a vertical measurement from this point to the lesion itself. In these growing animals it was necessary to calibrate both measurements weekly.

The period between birth and the appearance of the first bleeding spots on the different animals in the herd, was used to ascertain the suggested prepatent period of *P. bovicola*. Data on the number and locality of bleeding points in the herd was used to ascertain the frequency distribution of lesions in a horizontal plane in the live animal, while the corresponding information in the control group indicated the peak period in the occurrence of these lesions during 1974 - 1975. Originally the information on the locality of lesions was used to classify these according to the muscle or muscle groups on which they occurred[33]. Subsequently the following muscles were grouped in 5 different body regions as follows:

1. *Head, neck and dewlap:* Facial, mandibular and hyoid muscles. Mm. sternocephalicus, sternohyoidius, brachiocephalicus (anterior two-thirds of both parts) and trapezius (anterior third).

2. *Withers, shoulders, upperforelimbs and pectoral region:* Mm. trapezius (middle third covering the scapula), deltoid (both parts), triceps brachii (long and lateral heads) and pectoralis profundus (posterior deep pectoral). Muscles of the extensor division of the forelegs and ulnaris lateralis.

3. *Back and loins:* Mm. trapezius (posterior third), serratus dorsalis (posterior part), obliquus abdominis internus (lumbodorsal origin) and gluteus medius (lumbar part).

4. *Ribs and flanks:* Mm. latissimus dorsi, serratus ventralis (rib attachment of thoracic part) and obliquus abdominis externus.

5. *Hindquarters and upper legs:* Mm. gluteus medius (excluding lumbar part), biceps femoris, semitendinosus, semimembranosus and tensor faciae latae. Muscles of the tail and lateral muscles of leg.

A haemorrhaging nodule or swelling was considered as a positive infestation and an animal was regarded as suitable for chemotherapeutic studies if it had a history of two or more active episodes of bleeding from each lesion or immediate vicinity during the preceding 6 weeks. In the text, nodules which bleed once or repeatedly are referred to as bleeding points.

During September 1974 sixty young oxen and six heifers were selected from the positive animals and randomly subdivided into the following groups which were marked with eartags of different colours:

(1) One group of ten control animals. These animals were transferred from Zoutpan to Onderstepoort to facilitate eventual slaughter.
(2) Seven treatment groups with at least seven animals per group.
(3) One treatment group comprising the six heifers.

First treatment

The eight different compounds which were included in the present trial are given in Table 1 (first treatment). Non-availability of antimonial preparations excluded them from the present trial. Seven of these treatments took place during the period 16-20 September 1974, while the heifer group was treated during the first week of October 1974. One of these treatments represents a combination of two compounds given simultaneously but by two different routes. Dosage rates and intervals between treatments were obtained from the specifications as supplied by the various companies.

Observations after first treatment

For a period of 6 weeks after the initial treatment all animals were again examined on a weekly basis in order to ascertain the possible effect of each compound. At this stage a successfully treated group was considered as one with a 50% or greater reduction in the number of bleeding lesions. Clinical observations on these groups were continued until slaughter.

Second treatment

All unsuccessfully treated groups were again treated at the end of October 1974 with the most promising remedies but at different dosage levels or intervals than initially. In Table 1 a list of these se-

Table 1: PROGRAMME OF TREATMENT.

Group No.	Animals treated	FIRST TREATMENT					
		Active Ingredient	Tradename or code number*	Date treated	Dosage rate		Route
					Daily	Days treated	
1	7	SURAMIN and DIETHYLCARBAMAZINE CITRATE	NAGANOL (BAYER 205) and CODE 46508-13	18 Sept. 18 Sept. -19 Oct.	12 mg/kg 4,2 g	1 10 (Weekly intervals)	Intra-venously Per Os
2	7	THIACETARSAMIDE SODIUM 1% M/V	CAPARSOLATE	16 - 17 Sept.	2,2 mg/kg twice	2	Intra-venously
3	7	FENCHLORPHOS 60% M/V	RONNEL	16 - 20 Sept.	20 mg/kg	5	Per Os
4	7	PHOSMET 10% M/V	PROLATE	16 - 20 Sept.	20 mg/kg	5	Pour on
5	7	MEBENDAZOLE 5% M/V	MULTISPEC	16 - 20 Sept.	20 mg/kg	5	Per Os
6	7	FENBENDAZOLE 10% M/V	HOE 881 V	16 - 20 Sept.	20 mg/kg	5	Per Os
7	7	LEVAMISOLE HYDROCHLORIDE 7,5% M/V	RIPERCOL-1	16 - 20 Sept.	10 mg/kg	5	Intra-muscular
8	7	TRICHLORPHON 50% M/V	DYLOX INJECT	2 & 9 Oct.	15 mg/kg	2 (Weekly interval)	Intra-muscular

Group No.	Animals treated	SECOND TREATMENT					
		Active Ingredient	Tradename or code number*	Date treated	Dosage rate		Route
					Daily	Days treated	
1	7	LEVAMISOLE-HYDROCHLORIDE 7,5% M/V	RIPERCOL-1	29 and 30 Oct.	15 mg/kg	2	Intra-muscular
2	7	FENBENDAZOLE 10% M/V	HOE 881 V	30 Oct.	100 mg/kg	1	Per Os
3	7	FENBENDAZOLE 10% M/V	HOE 881 V	30 Oct.	75 mg/kg	1	Per Os
4	7	FENBENDAZOLE 10% M/V	HOE 881 V	30 Oct.	50 mg/kg	1	Per Os
5	7	FENBENDAZOLE 10% M/V	HOE 881 V	30 Oct.	30 mg/kg	1	Per Os
6	7						
7	7						
8	7						

* Naganol : 8 — (3 - benzamido — 4 — mathyl — benzamido) naphtalene — 1,3,5, — trisulphonic acid — Bayer Agro-Chem.
Coda 46508-13:1 — diethyl carbamyl — 4 — mathyl piparazina hydrochlorida — S.A. Cyanamid.
Caparsolate : sodium (P — carbamyl phanylarsylane — dithio) diacetata. — Abbott.
Ronnel : 0,0 — dimethyl 0 — (2, 4, 5 — trichlorophenyl) phosphorothioate — Dow.
Prolate : 0,0 — dimethyl S — phthalimidomathyl phosphorodithioate — Datons.
Multispec : methyl 5 — banzoylbanzimidazole — 2 — carbamate — Ethnor.
Hoe 881 V : methyl 5 — (phanylthio) — 2 banzimidazole carbamata — Hoechst.
Ripercol-1 : 1 — 2, 3, 5, 6 — tetrahydro — 6 phenylimidazo (2, 1 — b) thiazolehydrochloride — Ethnor.
Dylox inject : 2, 3 dimethyl (2, 2, 2 — trichloro — 1 — hydroxyethyl) phosphonate — Bayar Agro-Chem.

cond treatments are given under the heading "second treatment".

Observations after second treatment

As during the first post treatment period, animals were again examined weekly in order to assess the efficacy of each treatment. Because of the natural decline in bleeding points in the control animals at this stage a 90% or greater reduction in the active lesions was taken as an indication of a successfully treated group.

Toxicological observations

One week before the first series of treatments one animal for each treatment group was given twice the prescribed daily dose (see Table 1 under first treatment), in order to observe the toxicological effects of the different active ingredients. Trichlorphon and levamisole, however, were administered at 15 mg/kg live mass. In the groups treated with organophosphates blood samples were taken before and after each treatment and the acetylcholinesterase levels determined so that depletion of this enzyme could be counteracted and consequent losses of experimental animals prevented.

Slaughter trials

1. *Controls:* Untreated control animals were slaughtered during the period 23 July to 14 January 1975. The carcass and skin of each animal was thoroughly examined for the presence of live and dead parasites. Encapsulated worms were included as dead worms. The association of these parasites with the previously recorded lesions was also noted. The number as well as the locality of all affected areas of the subcutus were recorded for each carcass.

Lesions were again classified according to the muscle or muscle group on which they occurred, and subsequently under the five headings as previously mentioned.

In order to exclude bruised areas, smears were taken from all affected areas, stained by the Giemsa method and examined for the presence of eosinophiles.

2. *Successfully treated groups:* Two of the groups treated originally showed promising results and were subsequently slaughtered during the period October 1974 to February 1975. From the groups treated a second time, a further two groups were chosen for slaughter although four different groups could be classified as successfully treated. These animals were slaughtered from 5 February to 25 February 1975. Carcass examinations and smears taken were similar to those of the untreated controls.

RESULTS

The results of the clinical observations are summarized in Tables 2 and 3 and illustrated in Figure 1.

In Table 2 the suggested mean and range of the prepatent period of *P. bovicola* are given for the animals in each group as well as for the animals in the herd as such. An overall mean of 250 days or 36 weeks was recorded. Animals were born over a period of two and a half months and the appearance of their first bleeding points covered the same period approximately 36 weeks later.

Table 3 illustrates the frequency distribution of bleeding lesions on each side of the body as well as in the different regions for all animals during the experimental period. On a percentage basis 56% of all

Table 2: APPROXIMATE PREPATENT PERIOD — *P. bovicola.*

GROUP DESCRIPTION OR NUMBER	ANIMALS IN GROUP	DATES OF BIRTH — RANGE —	DATES FIRST BLEEDING POINTS OBSERVED — RANGE —	PREPATENT PERIODS			
				DAYS		WEEKS	
				AVERAGE	RANGE	AVERAGE	RANGE
CONTROLS	10	3.10.73 — 8.11.73	6.6.74 — 15.8.74	265	230 — 303	38	33 — 43
1	8	11.10.73 — 2.12.73	6.6.74 — 22.8.74	249	199 — 328	36	32 — 47
2	7	19.10.73 — 3.12.73	30.5.74 — 1.8.74	239	209 — 268	34	29 — 38
3	7	4.10.73 — 3.11.73	6.6.74 — 8.8.74	261	215 — 302	37	31 — 43
4	7	5.10.73 — 13.11.73	6.6.74 — 18.7.74	250	220 — 286	36	31 — 41
5	7	11.10.73 — 16.12.73	30.5.74 — 15.8.74	239	216 — 273	34	31 — 39
6	7	5.10.73 8.12.73	13.6.74 — 8.8.74	246	224 — 272	35	32 — 39
7	7	13.10.73 21.11.73	6.6.74 — 8.8.74	248	214 — 279	36	31 — 40
TOTAL RANGE		3.10.73 — 16.12.73	30.5.74 — 22.8.74		199 — 328		29 — 47
AVERAGE PER ANIMAL IN HERD				250		36	

Table 3: FREQUENCY DISTRIBUTION OF BLEEDING POINTS ON LIVE OXEN.

GROUP DESCRIPTION OR NUMBER	ANIMALS IN GROUP	BLEEDING POINTS OBSERVED DURING EXPERIMENTAL PERIOD			DISTRIBUTION OF BLEEDING POINTS														
					HEAD NECK AND DEWLAP			WITHERS SHOULDERS AND UPPER FORE-LEGS			BACK AND LOINS			RIBCAGE AND FLANKS			HIND-QUARTERS AND UPPER HIND-LEGS		
		L*	R**	TOT	L	R	TOT	L	R	TOT	L	R	TOT	L	R	TOT	L	R	TOT
UNTREATED CONTROLS	10	101	85	186	27	15	42	44	32	76	11	15	26	10	18	28	9	5	14
1	8	64	42	106	9	6	15	32	16	48	15	11	26	4	2	6	4	7	11
2	7	57	46	103	10	5	15	28	28	56	9	9	18	2	2	4	8	2	10
3	7	43	32	75	9	4	13	23	11	34	5	14	19	3	0	3	3	3	6
4	7	65	45	110	14	10	24	26	17	43	15	9	24	7	5	12	3	4	7
5	7	53	31	84	15	1	16	20	13	33	7	7	14	3	7	10	8	3	11
6	7	31	34	65	5	4	9	11	12	23	9	10	19	3	2	5	3	6	9
7	7	36	34	70	6	9	15	14	11	25	7	7	14	2	4	6	7	3	10
TOTAL		450	349	799	95	54	149	198	140	338	78	82	160	34	40	74	45	33	78
PERCENTAGE		56	44	100			18,6			42,3			20,0			9,3			9,8

*L = Left **R = Right

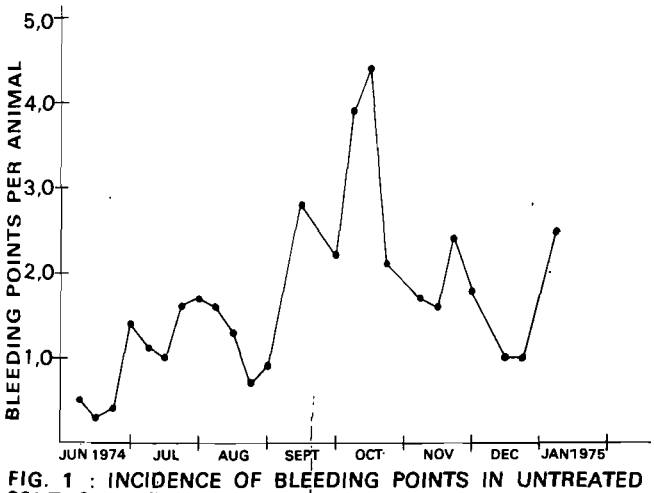

FIG. 1 : INCIDENCE OF BLEEDING POINTS IN UNTREATED CONTROL ANIMALS

lesions was noticed on the left and 44% on the right side of the body. Further analysis showed that 42% of all lesions were concentrated on the withers, shoulders and forelegs, 20% on the back and loins, 18,6% on the head, neck and dewlap and only 9,3% and 9,8% on the ribcage (including flanks) and hindquarters (including the upper parts of hind legs) respectively.

The incidence of bleeding points recorded weekly for the control group only is illustrated in Figure 1. It is evident from this figure that lesions increase from a very low level at the beginning of June to a peak value during mid October after which a decrease was noticed until the end of the experimental period. Because bleeding points as well as repetitions of bleeding points showed the same graphical tendencies, they were all included under the general heading "bleeding points".

The effect of the first and second treatments on the frequency of bleeding spots is summarized in Table 4 and illustrated in Figures 2 - 4.

In Table 4 the total number of bleeding lesions in each group during the 6 week period before and after each treatment for both first and second treatments is compared with the corresponding data in the untreated controls. Figures 2 - 4 illustrate the same tendencies although on a 2 weekly basis. Only levamisole (10 mg/kg x 5) and fenbendazole (20 mg/kg x 5) reduced the frequency of bleeding points by more than 50% during the 6 week period after the first treatment (Table 4) and caused a dramatic reduction in bleeding points after the middle of September 1974. Other groups either showed an increase or a less than 50% decrease in bleeding spots and were therefore considered as less successfully treated. The trichlorphon group was treated only once and at a later date (2 - 9 Oct., 1974) than the others and the 26% decrease in active lesions coincides with the natural decrease in lesions in the control group and is therefore more significant than the mebendazole group with a similar decrease but which was treated earlier (Table 4 and Figure 4). After the initial treatments only the first mentioned two groups were kept for slaughter purposes and the other five groups retreated at the end of October with various levels of these apparently successful drugs. The trichlorphon group of heifers were not available for slaughter purposes and were therefore excluded from further trials.

Results of second treatments with either levamisole (15 mg/kg x 2) and fenbendazole (30-100 mg/kg x 1) are more difficult to interpret because active lesions in the control animals showed the same decreasing tendency at this stage as those of the treatment groups. However, in the four fenbendazole groups haemorrhaging lesions decreased by more than 90% in comparison with the 48% of the untreated controls and exhibited a more dramatic weekly reduction (Table 4 and Figures 2 - 4). Because only two groups were available for slaughter purposes the fenbendazole (100 mg/kg x 1) and fenbendazole (30 mg/kg x 1) were chosen.

In Table 5 the effect of treatment on the number of worms collected, the number of carcasses affected and the number of lesions observed are summarized.

Table 4: EFFECT OF TREATMENT ON FREQUENCY OF BLEEDING POINTS OBSERVED.

FIRST TREATMENT (16 — 20th OCTOBER)				SECOND TREATMENT (LATE OCTOBER)			
GROUP DESCRIPTION OR TREATMENT	TOTAL BLEEDING POINTS 6 WEEKS PRE	TOTAL BLEEDING POINTS 6 WEEKS POST	% INCREASE OR DECREASE	GROUP DESCRIPTION OR TREATMENT	TOTAL BLEEDING POINTS 6 WEEKS PRE	TOTAL BLEEDING POINTS 6 WEEKS POST	% INCREASE OR DECREASE
UNTREATED CONTROLS	79	148	+ 89	UNTREATED CONTROLS	148	76	–48
SURAMIN AND DIETHYL-CARBAMAZINE CITRATE	66	113	+ 71	LEVAMISOLE HYDRO-CHLORIDE 15 mg/kg × 2	113	26	–77
THIACETARSAMIDE SODIUM	66	105	+ 59	FENBENDAZOLE 100 mg/kg × 1	105	2	–98
FENCHLORPHOS	53	90	+ 70	FENBENDAZOLE 75 mg/kg × 1	90	6	–93
PHOSMET	63	129	+104	FENBENDAZOLE 50 mg/kg × 1	129	8	–94
MEBENDAZOLE	61	45	– 26	FENBENDAZOLE 30 mg/kg × 1	45	2	–96
FENBENDAZOLE 20 mg/kg × 5	47	7	– 85				
LEVAMISOLE HYDROCHLORIDE 10 mg/kg × 5	54	15	– 72				
TRICHLORPHON	50	37	– 26				

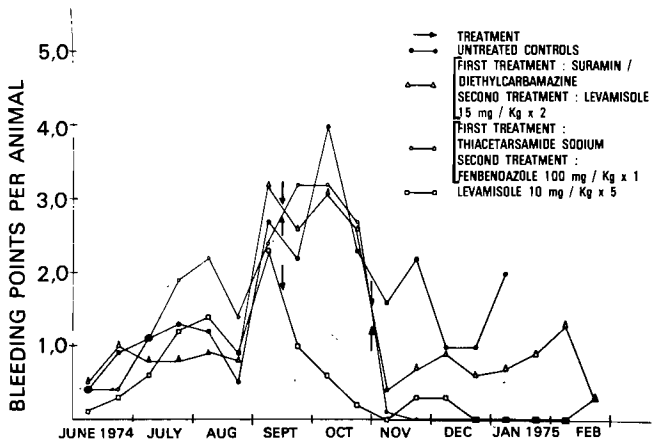

FIG. 2 : EFFECT OF TREATMENT ON BLEEDING POINTS OBSERVED

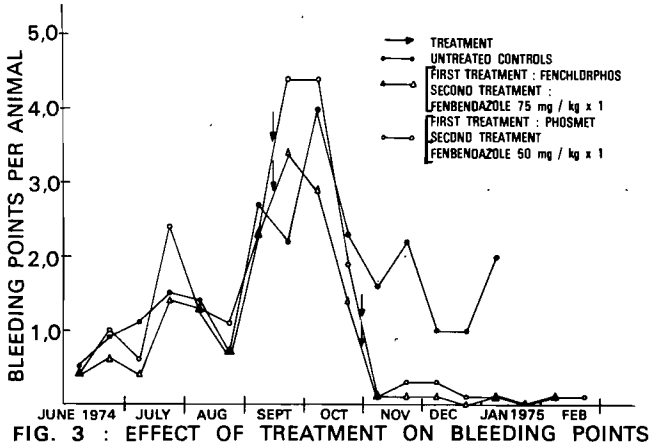

FIG. 3 : EFFECT OF TREATMENT ON BLEEDING POINTS OBSERVED

In comparison with the untreated controls, live worms per treatment group and per carcass in each group were dramatically reduced. Dead encapsulated worms were encountered particularly in the levamisole group and are grouped under "dead worms". All carcasses were affected in the control

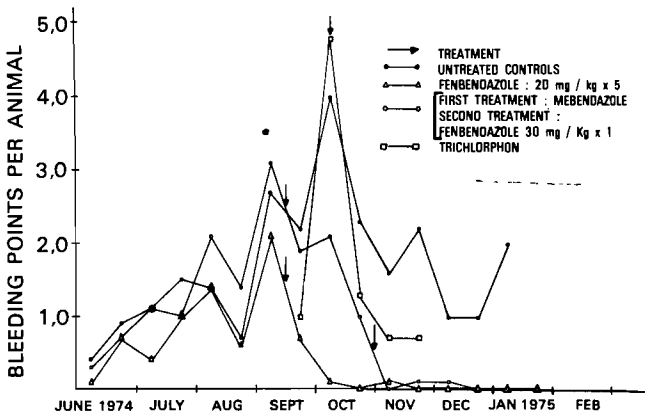

FIG. 4 : EFFECT OF TREATMENT ON BLEEDING POINTS OBSERVED

group, and more than half in each treatment group, but the total number of lesions per treatment group and per carcass in these groups were also drastically reduced. In comparison with the average of 10 lesions per carcass in the untreated controls only one, two and three per carcass were noticed in the levamisole, the two high level fenbendazole and the low level fenbendazole groups respectively.

The appearance of the carcass of an untreated control animal is compared in Figure 5 with that of a treated animal in the levamisole group (Figure 6).

In Table 6 the frequency distribution of carcass lesions in all animals slaughtered is summarized. As in the live animal, lesions seem to decrease caudally and cranially from the withers, shoulders and upper parts of the hind legs. The distribution of bleeding points and carcass lesions were comparable, although a higher percentage of the latter lesions was present on the back, loins, ribcage and flanks and fewer on the withers, shoulders and upper forelegs.

Toxicologically only those animals treated with levamisole at 15 mg/kg live mass showed nervous

symptoms (restlessness, salivation, shivering) but symptoms subsided after 30 - 45 minutes and no losses were recorded.

As illustrated in Figure 7 the acetylcholenesterase levels were reduced by 60% after two treatments with trichlorphon at 15 mg/kg live mass. Other organophosphorus compounds (fenchlorphos and phosmet) depressed this enzyme-system to a minor extent (28 - 35%) after five treatments.

Smears taken from lesions for microscopical examination showed that 89,5% of all affected areas were positive for eosinophiles.

Osipov[25] found similar extended periods in two artificially infested horses.

From the exact position and subsequent history of bleeding spots it was calculated that 54% of all lesions only bled once, 22% a second time and 24% more than twice (3 - 9 x). This suggests that mature females may migrate to adjoining areas and repeat their egg laying cycle.

Table 5: EFFECT OF TREATMENT ON *P. bovicola*, CARCASSES AFFECTED AND LESIONS OBSERVED

GROUP DESCRIPTION OR TREATMENT	ANIMALS SLAUGHTERED	PERIOD OF SLAUGHTER	*P. bovicola* WORMS OBSERVED				CARCASSES AFFECTED	LESIONS OBSERVED				
			ALIVE	DEAD	TOT	* CARCASS AVERAGE (LIVE WORMS)		TOT	* CARCASS AVERAGE	CARCASS FREQUENCY DISTRIBUTION OF LESIONS		
										0	1 — 5	5+
UNTREATED CONTROLS	9	23.7.74 — 14.1.75	56	6	62	6	9	89	10	0	0	9
FENBENDAZOLE 100 mg/kg x 1	5	19.2.75 — 25.2.75	4	0	4	1	3	11	2	2	2	1
FENBENDAZOLE 30 mg/kg x 1	7	5.2.75 — 19.2.75	10	2	12	1	7	21	3	0	5	2
FENBENDAZOLE 20 mg/kg x 5	7	29.10.74 — 19.12.74	2	6	8	0	5	15	2	2	4	1
LEVAMISOLE HYDROCHLORIDE 10 mg/kg x 5	7	29.10.74 — 5.2.75	2	26	28	0	4	7	1	3	4	0

* NEAREST DECIMAL.

DISCUSSION

All clinical observations were made on the group of sixty young oxen. These animals were born over a period of 75 days (3.10.73 - 16.12.73) and exhibited lesions for the first time over a comparable period of 85 days (30.5.74 - 22.8.74). The similarity of these two periods suggests a prepatent period similar to the intervening period of 238 days or the average of 250 days as calculated from all available data (Table 2). The slow maturity of *P. bovicola* can therefore be compared with that of *P. multipapillosa* in horses where

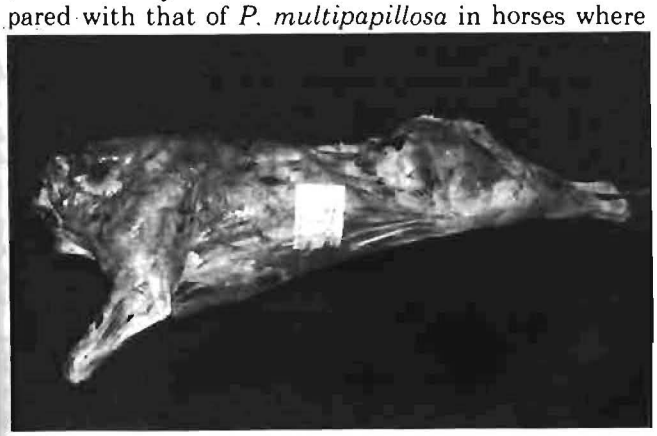

FIG 5

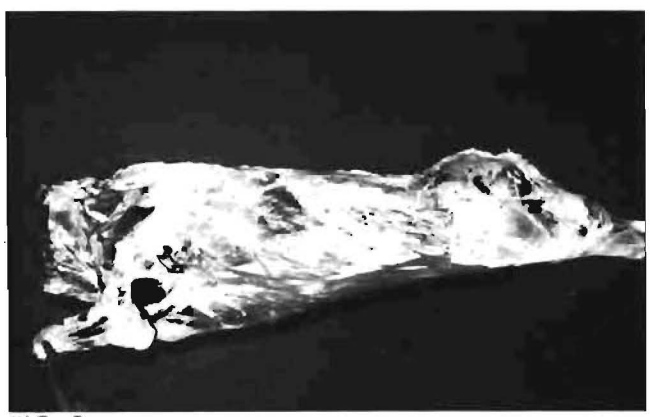

FIG. 6.

In the live animal active lesions usually occurred in the shoulder region (including withers and upper forelegs) and diminished from this region both cranially and caudally. The reason for the abundance of lesions in this region is not clear but might be associated with the route of infection or the predilection of certain areas by the parafilarial worm.

The seasonal incidence of bleeding spots in the control animals showed a rapid increase from June 1974 to peak values during September - October 1974, after which a decline was noticed. Provided vectors were present, the September/October period must have been very favourable for the transmission of *P. bovicola*. At Zoutpan calves are usually born during October/to mid December of each year and infection is therefore probable at a very early and susceptible age.

Table 6: FREQUENCY DISTRIBUTION OF CARCASS LESIONS.

GROUP DESCRIPTION OR TREATMENT	ANIMALS SLAUGHTERED	PERIOD OF SLAUGHTER	CARCASS LESIONS OBSERVED	DISTRIBUTION														
				HEAD NECK AND DEWLAP			WITHERS SHOULDERS UPPER FORE-LEGS			BACK AND LOINS			RIBCAGE AND FLANKS			HIND-QUARTERS AND UPPER HIND LEGS		
				L*	R**	TOT	L	R	TOT	L	R	TOT	L	R	TOT	L	R	TOT
UNTREATED CONTROLS	9	23.7.74— 14.1.75	89	10	6	16	16	15	31	11	7	18	8	7	15	4	5	9
FENBENDA-ZOLE 100 mg/kg x 1	5	19.2.75— 25.2.75	11	0	0	0	3	2	5	2	3	5	0	0	0	0	1	1
FENBENDA-ZOLE 30 mg/kg x 1	7	5.2.75— 19.2.75	21	2	5	7	0	3	3	3	3	6	1	1	2	2	1	3
FENBENDA-ZOLE 20 mg/kg x 5	7	29.10.74— 19.12.74	15	1	2	3	2	3	5	3	1	4	2	1	3	0	0	0
LEVAMISOLE HYDRO-CHLORIDE 10 mg/kg x 5	7	29.10.74— 5. 2.75	7	0	0	0	0	3	3	1	1	2	0	0	0	1	1	2
	TOTAL		143	13	13	26	21	26	47	20	15	35	11	9	20	7	8	15
	PERCENTAGE		100			18,2			32,8			24,5			14,0			10,5

* (L — Left) ** (R — Right)

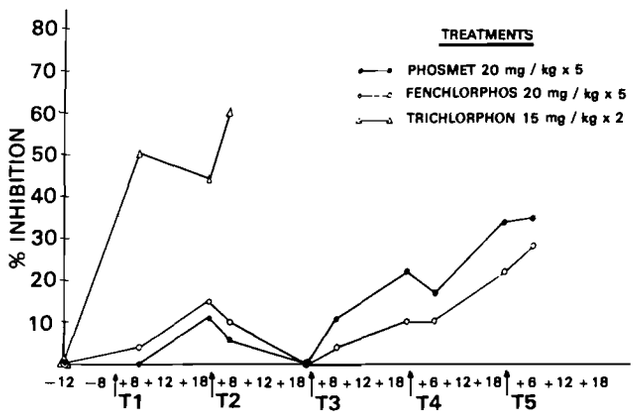

FIG. 7 : PERCENTAGE INHIBITION OF ACETYLCHOLINESTERASE

Drugs in the benzamidazole and tetramisole groups have previously been used in human patients affected with *O. volvulus* and dogs with dirofilariasis[37 38] respectively. Organophosphates were used for the same purposes. No published data of their effect on *P. bovicola* exists and their inclusion in the present trial was therefore considered a necessity. The high dosage rates, as suggested by the different companies, were employed only in the initial treatments, but for all second treatments these dosages were reduced for financial and practical reasons.

Based on a 50% or greater reduction of bleeding lesions in the groups treated originally only two of the eight drugs proved to be reasonably effective. Fenbendazole (20 mg/kg x 5) with a 85% reduction of active lesions appeared to be more promising at this stage than levamisole (10 mg/kg x 5) with a 72% reduction. Both groups were kept for slaughter purposes. The effect of trichlorphon (15 mg/kg x 2) in the heifer group is probably masked and difficult to interpret because these animals were treated just before active lesions showed their seasonal decreasing tendency in the control animals. This group was also not available for slaughter purposes and observations were only carried through until the end of November. Further treatment with trichlorphon appears to be warranted.

During retreatment of the unsuccessfully treated groups levamisole at 15 mg/kg gave poorer results clinically than the four fenbendazole groups at 30 - 100 mg/kg x 1 and were therefore not included in the slaughter trials. Two of the last mentioned groups with a 90% and more reduction of active bleeding spots were regarded as successfully treated and eventually slaughtered.

The fallacy, however, of choosing groups for autopsy purposes on diminishing bleeding spots after treatment is clearly illustrated in Table 5. Carcasses affected as well as lesions per carcass were lower in the levamisole than in all fenbendazole groups.

In comparison with the untreated controls, treatment in all groups slaughtered seemed to be reasonably effective with the emphasis on the levamisole group. The percentage of lesions per carcass compared with untreated controls were as follows — levamisole 10 mg/kg x 5 (10%), fenbendazole 100 mg/kg x 1 or 20 mg/kg x 5 (20%) and fenbendazole 30 mg/kg x 1 (33%).

Distributions of carcass lesions at slaughter is similar to that of the actual bleeding spots in the live animal. Differences in results must be attributed to the migration of live worms from one area to another and the consequent overlapping of lesions in adjacent regions. The results of frequency distribution obtained by Van der Heever *et al*[39] illustrate the same tendency as the present data, especially if lesions on withers and thorax are grouped together.

After five daily treatments with the organophosphorus compounds phosmet as a pour on, and fenchlorphos per os, (both at 20 mg/kg live mass) the acetylcholinesterase levels in the blood fell by only 28 - 35%. These two organophosphorus compounds,

although ineffective against *P. bovicola*, must therefore be regarded as very safe at the dosage rates employed. Trichlorphon on the other hand with a 60% inhibition after two treatments at 15 mg/kg live mass should be used with caution and preferably with acetylcholinesterase determinations as a background. In the present trial this high inhibition as well as the factors previously mentioned terminated the use of this organophosphorus compound.

Almost 90% of all affected areas were positive for eosinophiles. This polymorphonuclear cell must therefore be regarded as quite typical of all parafilarial lesions.

CONCLUSION

The results of the present trials show that at the dosage rates employed it is possible to reduce lesions to a greater or lesser extent but that this would not be an economical proposition except in cases where cost is not a decisive factor. Further work on the chemotherapy of *P. bovicola* is, however, still in progress and appears to be more promising.

ACKNOWLEDGEMENTS

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CESTODOSIS IN BATTERY-HOUSED LAYING HENS

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ABSTRACT: Abrams, L. **Cestodosis in battery-housed laying hens.** *Journal of the South African Veterinary Association* (1976). **47** No. 3 171-173 (En) Faculty of Veterinary Science, University of Pretoria, Box 12580, Onderstepoort, 0110. Republic of South Africa.

Cestodosis in battery-housed laying hens severely reduced egg production particularly at the time of peak production. Hens were able to consume large numbers of *Musca domestica*, the intermediate host of *Choanotaenia infundibulum* following the use of an aerosol insecticide to control flies. A considerable discharge of cestodes followed the use of "Lintex" in the feed resulting in a marked improvement in egg production. The use of an insect growth regulator in the feed showed promise in controlling the breeding of flies.

INTRODUCTION

Cestodosis in poultry is most commonly associated with birds maintained on the floors of intensive and semi-intensive houses or on the ground of open range systems where they have easy access to the intermediate hosts of the various cestodes of fowls. Slugs of the genera *Limax, Arion, Cepaeca* and *Agriolimax* are the intermediate hosts of *Davainea proglottina*[2]; ants of the genera *Tetramorium* and *Pheidole* of the genus *Raillietina*[2]; earthworms of *Amoebotaenia spenoides*[2]; the house fly; *Musca domestica* and beetles, *Aphodaius* spp., *Geotrupes sylvaticus, Calathus* spp. and *Triboleum* of *Choanotaenia infundibulum*.[2]

In this report an unusual outbreak of cestodosis involving a flock of approximately 90 000 battery-housed hens is recorded. It was possible to establish the erosive and economic effects of *C. infundibulum* infestation in hens, since the owner maintained accurate records of production, whereas similar records are not usually maintained for birds kept in semi-intensive or open range systems.

HISTORY

The owner of a large egg-producing unit complained that egg production was approximately 10% below the anticipated level and considerably lower than in previous years, while the young hens did not attain the normal peak production of 85 - 90%. The birds on this farm were purchased as point-of-lay pullets at 18 weeks of age, and were immediately housed in cages; at no stage were they kept on the floor. Pyramid-type batteries with four hens to a cage were housed in 15 sheds. The passages between the cages were cemented, but in 10 small sheds the manure fell directly on to uncemented areas, whereas in five larger sheds the floors were cemented throughout.

The food troughs were of an open type and placed immediately in front of the hens while the water was delivered in sealed plastic pipes with "Hart" cups. At one stage considerable water spillage took place due to incorrect installation of the water pipes, resulting in areas of wet manure in which large numbers of fly larvae were observed.

The manure was regularly removed from the houses and dumped in heaps outside and adjacent to the fowl houses pending removal to the vegetable lands. The frequency of vegetable plantings determined the length of time manure heaps remained near the hen houses, sometimes for as long as 6 weeks. The summer of 1974/1975 was characterised by unusually heavy rains followed by considerable heat and humidity which together with the accumulated manure provided ideal conditions for fly breeding.

Adult *M. domestica* in large numbers flew into the fowl houses and settled on the ceilings, walls and cage frames. In an attempt to reduce the number of flies in the houses, the walls and ceilings were regularly sprayed, sometimes three or four times a week, with a pyrethrin insecticide using a pump which dispersed it in a fine mist. Dead and dying flies fell into the troughs and were readily consumed by the hens with no apparent harmful effects caused by the ingestion of insecticide.

CLINICAL EXAMINATION

On inspection of the birds no obvious symptoms were observed, and feed consumption was normal. All birds on this farm were regularly immunised against Newcastle disease, and also vaccinated against infectious bronchitis and fowl pox. The daily mortality was minimal and the total monthly mortality was less than 1%.

Six birds were taken at random for necropsy from each of the two houses where egg production was at its lowest. In the majority of birds the jejenums were heavily infested with tape worms, and in some specimens the intestines were all but totally occluded. In some birds ovulation had ceased, whereas others were still actively in lay. The only pathological change seen was a catarrhal enteritis; this was confirmed histologically. Most birds from the other houses subsequently sacrificed and necropsied were, with the exception of a group of birds only recently acquired, found to be cestode infested. The worms were identified as *C. infundibulum* (Dr Anna Verster personal communication, Veterinary Research Institute, Onderstepoort) who was also able to determine the presence of cysticercoids in adult flies. The owner was advised to stop insecticidal spraying immediately in the houses and all hens were treated with a cestocide.

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Choanotaenia infundibulum (Bloch) occurs in the jejenum of the fowl, is up to 25 cm long and the segments are markedly wider posteriorly.[2] The scoleces bear 16 to 20 slender hooks and attach to the intestinal wall where they produce local irritation. The eggs voided in the faeces of the hen are ingested by fly larvae present in the manure and subsequently develop into cysticercoids in adult flies which, when ingested with the flies by hens, develop into tape worms. The minimum time required from feeding the cysticercoid stage to production of gravid proglottids in chickens 12 days old, is 13 days, so the life cycle is completed in approximately 3 - 4 weeks. It is estimated that an adult fly can contain up to 40 - 50 cysticercoids (Dr Anna Verster personal communication, Veterinary Research Institute, Onderstepoort). Should a hen have consumed only two or three heavily infected flies (quite likely from the number of flies present) it then becomes obvious that the worm load in some hens could be expected to be extremely high.

TREATMENT

The hens were treated with Lintex* (Bayer Agrochem) at the rate of 50 mg per kg live mass; each hen weighed approximately 2 kg and consumed on average 110 g of feed per day. Ten tonnes of feed were medicated and given to the birds once the troughs were emptied of unmedicated feed. Medicated feed was consumed within 16 hours and 24 hours later the manure was covered by a thick layer of excreted worms. Production increased gradually 7 - 10 days later.

Two or 3 weeks after this treatment it was observed that tape worm segments were being voided in the droppings and autopsies revealed a moderate infestation of tape worms in some hens. A second treatment of Lintex at the same dosage level was given, but in view of the persistent enteritis, feed medicated with furazolidone (100 g of activity per tonne) was provided for 10 days. A further discharge of worms took place and was followed within 2 weeks by a steady increase in egg production by as much as 12% in young hens.

Three weeks after the second administration of Lintex close inspection of the droppings again revealed tape worm segments. The dose of Lintex was increased to 100 mg per kg live mass and this feed was fed for 2 days. Once again there was an elimination of worms, but considerably fewer than followed the first and second treatments.

At the higher dosage level of Lintex no signs of toxicity or interference with egg production were encountered. Treatment proved to be successful, as during the subsequent period of 3 months the incidence of tape worm infestation was reduced to a level where the hens were free from parasites for all practical purposes.

Limited use of Terenol† (Hoechst) indicated that it might be used as an alternative drug in the treatment of cestodosis of poultry.

Once the birds were free from worms, successive intakes of young hens resumed peak production at 85 - 90%, and the others continued to maintain a high level of production. During the Spring of 1975, however, the number of flies again became alarmingly high when the owner decided to spray the houses with insecticide as before and this was followed within several weeks by a decline in egg production. Tape worm segments could again be seen in the droppings, and on autopsy at least 60% of the birds carried a heavy load of young tape worms.

The birds were again treated with Lintex at 100 mg per kg live mass for 2 days with the same spectacular results.

DISCUSSION

The heavy infestation of the hens took place by inadvertently making the infected intermediate hosts readily available in a somewhat unusual manner.

The young hens arrived on the farm free from cestodes at approximately 18 weeks of age and were immediately exposed to the cestode infestation when fly spraying was commenced. They normally reach peak production at approximately 28 weeks of age, when the worm load could have been expected to be high, and when the hens' nutritional requirements were optimal. This would explain their inability to reach peak production.

The erosive effect of cestodosis on the economy of the laying unit can be assessed by the decrease in total egg production of approximately 10% as well as a reduction in egg size, which resulted in a daily loss of approximately R250,00.

If diagnosis and treatment had been delayed the intestinal lesions could have become aggravated and led to debilitation. The severity of the effect on egg production and the degree of response to medication in instances of delayed treatment are matters for speculation.

The obvious aim of cestocidal treatment is to totally dislodge the tape worm so that no scoleces, from which new growth can occur remain attached. It is apparent that a single treatment of Lintex at 50 mg per kg live mass was insufficient to achieve this requirement, as the hens were still heavily infested three weeks after the first treatment. No further massive ingestion of cysticercoids took place as fly spraying ceased at the time the diagnosis was made. Even after a second treatment worms, although markedly reduced in numbers, were still present. Only after dosage at 100 mg per kg live mass for two days (a total of 400 mg of Lintex per bird) were the hens for all intents free of worms. It is therefore suggested that a minimum of 100 mg Lintex per kg live mass for two consecutive days, followed by a second treatment at the same dosage level two or three weeks later, be used for the elimination of *C. infundibulum* infestation in laying hens.

The control of flies on most laying farms presents considerable problems as flies are a public nuisance, particularly in peri-urban areas where many large poultry farms are situated. Although extensive use is made of a number of insecticides to control flies, they are not particularly effective since the only successful method of fly control is to attack their breeding sites i.e. the manure. Even if the manure is removed from hen houses at daily or weekly intervals, its disposal remains a problem. A hen produces 30 kg of faeces during her productive life. On a moderately small unit of 10 000 hens some 300 tonnes of manure is produced annually, and if not properly disposed of presents an excellent site for fly breeding.

Several disposal techniques may be used, e.g. the

* Niclosamide N - (2' Chloro 4 - nitrophenyl) Chlorosalicylamide.

† Resorantel 2 - 6-Dihydroxy-Benzoic acid - 4 - Bromanilide.

installation of manure drying machines. The dried poultry waste can be recycled into feeds for layers and pullets at an inclusion rate of no more than about 10%; can be fed to ruminants as an excellent source of protein; or used as fertilizer.

A promising approach in controlling fly breeding is the addition of an Insect Growth Regulator to feed. It is voided unmetabolised in the faeces and interferes with the life cycle of the fly by preventing the pupae from developing into adults. Small scale trials with one of these compounds has produced encouraging results, and its application is now being evaluated in the field.

The length of time that *C. infundibulum* eggs remain viable in manure, particularly when spread out thinly on lands, remains unanswered. It would appear that the eggs remain viable for several months — at least during the winter when sunlight is considerably reduced. This hypothesis is based on the evidence that the worm load became large enough to depress egg production within three weeks after flies were sprayed in the houses, during the spring of 1975, indicating that numerous flies were carrying cysticercoids which may have survived as eggs from the previous summer's infestation.

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THE RELATIONSHIP BETWEEN TOTAL AND IONIZED CALCIUM IN THE BLOOD OF SHEEP WHEN CALCIUM LEVELS ARE ALTERED

P.C. BELONJE*

ABSTRACT: Belonje, P.C. **The Relationship between Total and Ionized Calcium in the blood of sheep when calcium levels are altered.** *Journal South African Veterinary Association* (1976) **47** No. 3 175-181 (En) Dept. Human and Animal Physiology, University of Stellenbosch, Stellenbosch 7600, Republic of South Africa.

In a series of eight experiments attempts were made to change the levels of calcium in the blood of sheep by intravenous infusions of calcium, EDTA and thyrocalcitonin or changing the acid-base balance by various means. When total calcium levels changed these changes were mirrored closely by changes in the physiologically important ionized calcium fraction ($r = 0{,}839$). It is concluded that the total calcium determinations remain valuable indicators of changes in the physiological level of calcium in the blood.

INTRODUCTION

Previous studies[3] [4] have shown that in normal sheep the correlation between total and ionized calcium is not of a sufficiently high order to be able to predict the physiologically active ionized calcium levels from total calcium values. If, however, the ratio between the two is not reasonably constant in both normal and abnormal animals, as predicted on the basis of the law of mass action governing the dissociation of a weak electrolyte of calcium proteinate[12] [13] [14] [15], then the validity of the results of total calcium analyses performed on abnormal animals must be in doubt. In fact if ionized calcium levels change in a manner which is not related to total calcium levels, the results of a great deal of research which has been done on calcium during abnormal conditions must be regarded as worthless.

With this in mind a series of experiments was designed to determine the ratio between total and ionized calcium when blood calcium was raised by calcium infusions or lowered by calcium chelation. Furthermore it has been shown *in vitro* that an increase in pH decreases the diffusible calcium in fowl blood[26] and both diffusible[7] and ionized calcium[17] [18] in human sera by increasing the binding of calcium ion to serum proteins[11] [21]. For this reason the ratio between total and ionized calcium was also determined during changes in acid-base balance induced by the administration of base or acid or the reduction of blood carbon dioxide by hyperventilation. Finally, the effect of the calcium lowering hormone, thyrocalcitonin, on the relationship between total and ionized calcium was determined.

MATERIALS AND METHODS

Animals: A total of 18 healthy year-old South African Mutton Merino wethers and maiden ewes were used in the experimental series. The sex and mass of each animal will be mentioned in each experiment.

Collection of blood specimens: The animals were handled carefully to avoid excitement and were bled in the standing position. If only a few samples were taken then the jugular vein was occluded only momentarily to locate it for piercing and then free-flowing blood was collected. If a number of samples were taken an indwelling cannula was inserted into the jugular prior to the experiment. When intravenuous infusions were done blood was always withdrawn from the opposite jugular vein. In all cases the free-flowing blood was withdrawn anaerobically through the same needle or catheter into three plastic syringes (Plastipak : Becton, Dickinson & Co.). After each syringe was filled the point was sealed with a plastic cap and the syringe placed in crushed ice. The first syringe (10 ml capacity containing 100 IU dry heparin) was centrifuged point downwards after removing the plunger. The plasma obtained was then kept at –20°C for total calcium and protein analyses. The second syringe (1 ml capacity containing 2,5 IU dry heparin) was used for the collection of blood for the determination of ionized calcium. It has been shown that this level of heparin does not interfere with ionized calcium determinations while still inhibiting blood coagulation[22] [6]. This capped syringe, filled with blood, was then centrifuged point upwards after cutting off the protruding section of the shaft of the plunger. The plasma could then be withdrawn anaerobically through the tip of the syringe into the syringe used in the calcium ion-exchange electrode system. The third syringe (1 ml capacity containing 10 IU dry heparin) was filled and a small metal agitator was introduced quickly through the tip before capping. These specimens were used for acid-base determinations. The blood was well mixed and the probe of the micro-electrode was introduced through the tip of the syringe to withdraw a sample for pH determination. The syringe itself was then used to place blood into the equilibration chambers for the determination of the other acid-base parameters.

Analytical methods

(i) Ionized calcium determinations: Ionized calcium was determined by means of an Orion Model 99-20 Serum Calcium Flow-thru System coupled to a Model 801 Digital pH/mV meter. The same precautions and method of processing the plasma samples and bracketing of each deter-

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mination with a standard were used as described previously[3, 4]. The time during which the blood specimens were being centrifuged was sufficient to allow them to reach ambient temperature similar to that of the standards.

(ii) Total plasma calcium determinations: Plasma was diluted 1 in 20 with 0,1 per cent lanthanum solution by means of an automatic pipette (Micromedic Systems Inc.) and compared with commercial standards (Hopkin & Williams Ltd.) also diluted with 0,1 per cent lanthanum on a Varian Techtron Model 1200 atomic absorption spectrophotometer.

(iii) Total plasma protein determinations: Samples were analysed by the biuret method of Weichselbaum[29] and read on a Beckman Model B spectrophotometer.

(iv) Blood pH, PCO_2 and standard bicarbonate determinations: These parameters were determined by means of the Astrup technique using a Radiometer Blood Micro System BMS 2. It was not the object of the study to determine accurately the acid-base parameters in sheep but merely to determine relative changes in pH, PCO_2 and standard bicarbonate (bicarbonate at pH = 40 mm Hg). Venous blood was freely available and was therefore used. The limitation of using venous blood for critical acid-base studies are realised. However, Tasker (1971)[25] has pointed out that there is great difficulty in obtaining arterial or "arterialized" blood in animals and therefore most clinical studies of acid-base status in spontaneous disease as well as studies on normal values have been based on venous blood specimens. As venous blood was used a closer approximation of true PCO_2 values was obtained by first correcting the buffer line by means of the formula:—

$$\frac{100 - \text{actual } 0_2 \text{ saturation}}{100} \times \text{Haemoglobin \%} \times 0{,}03 =$$

the correction to be added to the Base excess and Buffer base curves on the Siggaard Andersen nomogram[23]. As relative results were of more importance than absolute results, a standard correction was applied in each case assuming that all samples contained 15 g haemoglobin with an oxygen saturation of 50 per cent.

Statistical Methods: Correlation coefficients and the slope of the regression line were calculated as described by Snedecor & Cochran[24].

THE EXPERIMENTAL SERIES

Experiment 1: The relationship between total and ionized calcium when blood calcium is raised by calcium infusions.

PROCEDURE

Two ewes, Sheep A (33 kg) and Sheep B (40 kg) were used. Both animals were bled for zero time samples after which 4 g $CaCl_2$ in 250 ml water was infused intravenously over a period of 5 min. Blood was then taken 95; 150; 190 and 270 min after infusion. Blood taken before 95 min after the infusion was so high in calcium that it was impossible to determine the ionized calcium levels. Plasma was analysed for total and ionized calcium and total protein.

RESULTS

The results of the analyses are presented in Table 1.

Table 1: THE EFFECT OF INTRAVENOUS INFUSIONS OF 4g $CaCl_2$ INTO SHEEP ON PLASMA TOTAL CALCIUM, IONIZED CALCIUM AND TOTAL PROTEIN

Sheep	Sampling periods	Time in minutes	Total calcium mg/100ml	Ionized calcium mg/100ml	Total protein g/100ml
A	Preinfusion	0	10,1	4,80	6,52
	Postinfusion	95	12,9	7,70	6,52
	Postinfusion	150	10,8	6,30	6,21
	Postinfusion	190	10,4	5,90	6,23
	Postinfusion	270	10,1	5,10	6,43
B	Preinfusion	0	9,8	4,90	6,82
	Postinfusion	95	12,4	7,80	6,99
	Postinfusion	150	11,0	6,20	6,36
	Postinfusion	190	11,2	6,10	6,39
	Postinfusion	270	10,7	5,30	6,54

DISCUSSION

From the results it is apparent that in both animals as total calcium rose and then gradually fell back to normal, ionized calcium followed a very similar course ($P<0,01$; $r = 0,952$). Total protein levels showed no particular change.

Experiment 2: The relationship between total and ionized calcium when blood calcium is lowered by means of EDTA.

PROCEDURE

Two wethers, Sheep C (46 kg) and Sheep D (39 kg) and two ewes Sheep E (40 kg) and Sheep F (28 kg) were used. The EDTA (disodium ethylenediaminetetra-acetate) was made up as a 4,7 per cent (m/v) solution[19] and the pH was adjusted to 6,3 to prevent alkalizing the blood. The EDTA was introduced into the jugular vein through an indwelling cannula (Portex, o.d. 0,75 mm) by means of a perfusing apparatus (Unita (II), Braun).

Sheep C : A zero time blood specimen was taken after which 40 ml EDTA was infused over 40 min. Blood specimens were then taken 3, 27 and 63 min. after infusion stopped.

Sheep D : A zero time blood specimen was taken and a total of 90 ml EDTA was infused over 85 min when a second blood specimen was taken. Another specimen was taken at 110 min (140 ml EDTA) and another at 120 min (160 ml EDTA) when infusion stopped. Further specimens were taken 40 and 80 min after infusion stopped.

Sheep E : A zero time blood specimen was taken and a total of 70 ml EDTA was infused over 35 min when a second specimen was taken. Another was taken at 57 min (114 ml EDTA) when infusion stopped. Further specimens were taken 38 and 83 min after infusion stopped.

Sheep F : A zero time blood specimen was taken and a total of 90 ml EDTA was infused over 45 min when a second specimen was taken. Another specimen was taken at 63 min, (126 ml EDTA) when infusion stopped. Further specimens were taken 22, 62 and 132 min after infusion stopped.

The plasma was analysed for total non-chelated calcium, ionized calcium and total protein. Total non-chelated calcium was determined by chelometric titration using EDTA[28] instead of determining total calcium by atomic absorption. In the ionized calcium

determinations, the lowest of the commercial standards (Orion A standard; 2 mg/100 ml) was higher than the lowest ionized calcium values found. Two lower standards were therefore prepared. The first was made by adding 0,6 ml 0,002 M EDTA solution to 5 ml of the A standard to give a final calcium ion concentration of 0,93 mg/100 ml. The second by adding 0,9 ml 0,002 M EDTA solution to 5 ml of the A standard to give a final calcium ion concentration of 0,47 mg/100 ml.

RESULTS

The results of the analyses are presented in Table 2.

Table 2: THE EFFECT OF INTRAVENOUS EDTA INFUSIONS IN SHEEP ON PLASMA NON-CHELATED CALCIUM, IONIZED CALCIUM AND TOTAL PROTEIN.

Sheep	Sampling periods	Time in minutes	Total non-chelated calcium mg/100ml	Ionized calcium mg/100ml	Total protein g/100ml
C	Preinfusion	0	10,2	5,20	6,47
	Postinfusion	3	8,2	3,90	6,04
	Postinfusion	27	9,6	4,20	6,16
	Postinfusion	62	10,1	4,50	6,08
D	Preinfusion	0	10,6	4,70	6,51
	Infusion	85	7,5	2,60	6,31
	Infusion	110	4,5	1,28	6,08
	Infusion	120	3,9	0,83	6,01
	Postinfusion	40	6,0	2,30	6,18
	Postinfusion	80	8,1	3,00	6,33
E	Preinfusion	0	11,4	4,80	5,98
	Infusion	35	5,6	2,40	6,31
	Infusion	57	3,9	1,36	6,04
	Postinfusion	38	6,3	2,90	6,26
	Postinfusion	83	7,5	3,60	6,36
F	Preinfusion	0	11,6	4,60	6,59
	Infusion	45	5,8	1,76	6,53
	Infusion	63	3,8	0,81	6,60
	Postinfusion	22	5,6	2,10	6,80
	Postinfusion	62	6,4	2,60	6,77
	Postinfusion	132	7,1	3,30	6,94

DISCUSSION

Once again the total and ionized calcium values followed one another closely ($P<0,01$; $r = 0.962$). Total protein levels showed no particular change.

Experiment 3 : The relationship between total and ionized calcium after intravenous injections of thyrocalcitonin Barlet[2] has shown that a single intravenous injection of thyrocalcitonin does not influence total calcium levels in the sheep while a continuous infusion over a number of hours causes a profound drop in total calcium. This experiment was designed to determine whether single injections of thyrocalcitonin affected ionized calcium levels while not affecting total calcium.

PROCEDURE

Two wethers, Sheep G (40 kg) and Sheep H (46 kg) were used. Zero time blood specimens were taken and then each animal was given an intravenous injection of 1250 Medical Research Council (MRC) mU thyrocalcitonin (Thyrocalcitonin; Calbiochem) in 0,2M acetate buffer pH 4,62 (Merck) containing 0,1 per cent bovine albumin (Merck). The animals were bled 30 min later after which each received a further intravenous injection of 1250 MRC mU thyrocalcitonin prepared as above. The animals were then bled 40 and 115 min after the second injection. The plasma was analysed for total and ionized calcium and total protein.

RESULTS

The results of the analyses are presented in Table 3.

Table 3: THE EFFECT OF INTRAVENOUS INJECTIONS OF THYROCALCITONIN IN SHEEP ON PLASMA TOTAL CALCIUM, IONIZED CALCIUM AND TOTAL PROTEIN

Sheep		Time in minutes	Total calcium mg/100ml	Ionized calcium mg/100ml	Total Protein g/100ml
G	Zero sample: then 1st injection	0	9,9	4,65	5,99
	2nd sample: then 2nd injection	30	9,7	4,65	6,04
	3rd sample	40	10,0	4,70	6,13
	4th sample	115	9,6	4,75	6,13
H	Zero sample: then 1st injection	0	9,6	4,65	6,69
	2nd sample: then 2nd injection	30	9,5	4,20	6,86
	3rd sample	40	9,5	4,50	6,41
	4th sample	115	9,6	4,60	6,41

DISCUSSION

In both animals there was very little change in the total calcium as was expected. Furthermore the thyrocalcitonin did not significantly affect the ionized calcium levels either. Total protein levels showed no particular change.

Experiment 4 : The relationship between total and ionized calcium during a change in the acid-base balance induced by intravenous bicarbonate administrations.

PROCEDURE

Two wethers, Sheep I (35 kg) and Sheep J (38 kg), were used. A 6 per cent $NaHCO_3$ solution was introduced into the jugular veins using the same cannulae and perfusing apparatus as in Experiment 2.

Sheep I : A zero time blood specimen was taken and then 3,75 ml of the $NaHCO_3$ solution was infused per min. After 60 min (225 ml $NaHCO_3$) a second specimen was taken and another at 120 min (450 ml $NaHCO_3$) when the infusion was stopped. Further specimens were taken 33, 78; 158 and 228 min after the infusion stopped.

Sheep J : A zero time blood specimen was taken and then 5 ml of the $NaHCO_3$ solution was infused per min. After 60 min (300 ml $NaHCO_3$) a second specimen was taken and another at 90 min (450 ml $NaHCO_3$) when the infusion was stopped. Further

specimens were taken 33, 78; 158 and 228 min after the infusion stopped.

Sheep J : A zero time blood specimen was taken and then 5 ml of the $NaHCO_3$ solution was infused per min. After 60 min (300 ml $NaHCO_3$) a second specimen was taken and another at 90 min (450 ml $NaHCO_3$) when the infusion was stopped. Further specimens were taken 53 and 123 min after the infusion stopped.

Plasma was analysed for total and ionized calcium and total protein. Blood was analysed for pH, PCO_2 and standard bicarbonate.

RESULTS

The results of the analyses are presented in Table 4.

DISCUSSION

The bicarbonate infusion caused a rise in standard bicarbonate levels and, although there was a measure of compensatory increase in PCO_2, there was a rise in blood pH. This resulted in a small but definite fall in the level of total calcium which rose back to normal in Sheep J. The changes in total calcium were again mirrored by changes in ionized calcium although the values were not significantly correlated.

There were small changes in total protein and these are thought to be the result of the diluting effect of the intravenous infusion.

Experiment 5 : The relationship between total and ionized calcium during a change in the acid-base balance induced by intraruminal sodium hydroxide administration.

As it appeared that the intravenous infusions had not had a large effect on the calcium in the blood and since intravenous infusions disturb the intravascular fluid dynamics if large quantities are used, it was decided to administer a strong base in a relatively small quantity of fluid into the rumen.

PROCEDURE

Two wethers, Sheep K (30 kg) and Sheep L (32 kg) were used.

Sheep K : A zero time blood specimen was taken and then the animal received 1 litre of water containing 40 g NaOH by stomach tube. Blood specimens were taken 105, 195, 315 and 435 min after administration.

Sheep L : A zero time blood specimen was taken and then the animal received 1 litre of water containing 80 g NaOH by stomach tube. Blood specimens were taken 75, 250, 330 and 380 min after administration. The final specimen at 380 min was taken 5 min after the death of the animal.

The same analyses were performed as in Experiment 4.

RESULTS

The results of the analyses are presented in Table 5.

Table 4: THE EFFECT OF INTRAVENOUS SODIUM BICARBONATE INFUSIONS IN SHEEP ON PLASMA TOTAL CALCIUM, IONIZED CALCIUM, TOTAL PROTEIN, BLOOD pH, PCO_2 AND STANDARD BICARBONATE.

Sheep	Sampling periods	Time in minutes	Total calcium mg/100ml	Ionized calcium mg/100ml	Total protein g/100ml	pH	PCO_2 mm Hg	Bicarbonate mEq/l
I	Preinfusion	0	10,2	4,30	6,65	7,427	41,0	23,7
	Infusion	60	9,7	3,60	6,04	7,550	45,0	38,0
	Infusion	120	9,6	3,30	6,10	7,587	48,0	43,0
	Postinfusion	33	9,8	3,50	6,45	7,565	44,0	39,0
	Postinfusion	78	9,7	3,60	6,65	7,512	50,0	37,5
	Postinfusion	158	9,7	3,76	6,66	7,530	42,0	34,5
	Postinfusion	228	9,7	3,65	7,00	7,521	43,0	34,8
J	Preinfusion	0	9,7	4,70	6,20	7,399	44,0	25,2
	Infusion	60	8,8	3,80	5,35	7,544	51,0	41,0
	Infusion	90	8,3	3,60	5,13	7,620	48,0	47,5
	Postinfusion	53	10,1	3,88	5,78	7,540	45,0	38,0
	Postinfusion	123	9,9	3,92	5,91	7,461	53,0	35,5

Table 5: THE EFFECT OF INTRARUMINAL ADMINISTRATIONS OF SODIUM HYDROXIDE IN SHEEP ON PLASMA TOTAL CALCIUM, IONIZED CALCIUM, TOTAL PROTEIN, BLOOD pH, PCO_2 AND STANDARD BICARBONATE.

Sheep	Sampling periods	Time in minutes	Total calcium mg/100ml	Ionized calcium mg/100ml	Total protein g/100ml	pH	PCO_2 mm Hg	Bicarbonate mEq/l
K	Predosing	0	9,8	3,50	5,38	7,428	38,0	24,0
	Predosing	105	9,6	3,25	5,60	7,435	47,0	28,8
	Postdosing	195	9,3	3,00	5,34	7,467	46,0	30,8
	Postdosing	315	8,8	2,85	5,23	7,457	51,0	32,4
	Postdosing	435	8,7	2,70	5,27	7,492	48,5	33,5
L	Predosing	0	10,7	4,40	5,21	7,345	45,0	21,7
	Postdosing	75	9,3	3,60	4,93	7,380	50,0	26,0
	Postdosing	150	9,0	3,30	4,95	7,459	49,0	32,0
	Postdosing	225	8,1	3,00	4,95	7,385	56,0	29,5
	Postdosing	330	7,6	2,40	5,07	7,420	45,0	26,2
	Postdosing	380	7,6	2,20	5,12	7,117	78,0	17,5

DISCUSSION

Although the increases in pH were not as large as those found when bicarbonate was infused intravenously the resultant drop in both total and ionized calcium was far more profound. The reason for this is unclear at present but the important point is that in both animals the decline in total calcium was mirrored closely by a decline in ionized calcium ($P<0,01$; $r = 0,913$). It is also of interest to note that whereas there was a dramatic fall in blood pH in Sheep L 5 min after death this did not affect either the total calcium level or the relative relationship between total and ionized calcium.

Total protein levels showed no particular change.

Experiment 6 : The relationship between total and ionized calcium during a change in the acid-base balance induced by intraruminal lactic acid administration.

As alkalosis caused a decrease in both total and ionized calcium it was considered necessary to determine the effect of acidosis on these parameters. For this purpose lactic acid was administered intraruminally by stomach tube at levels somewhat higher than those which would give symptoms of lactic acidosis[27].

PROCEDURE

Two wethers, Sheep M (27 kg) and Sheep N (33 kg), were used.

Sheep M : A zero time blood specimen was taken and then the animal received 1 litre of water containing 140 g pure lactic acid by stomach tube. Blood specimens were taken 105, 195, 315 and 435 min after administration.

Sheep N : A zero time blood specimen was taken and then the animal received 1 litre of water containing 280 g pure lactic acid by stomach tube. Blood specimens were taken 75, 150, 225, 330 and 450 min after administration.

The same analyses were performed as in Experiment 4.

RESULTS

The results of the analyses are presented in Table 6.

DISCUSSION

The administration of the lactic acid caused a decline in the blood pH of both sheep, but by 195 min in Sheep M and 225 min in Sheep N the pH had risen to above the predosing level. In Sheep M there was a small decline in total calcium followed by a rise to the pretreatment level. On the other hand, in Sheep N, which received the higher dose of lactic acid, the opposite occurred and calcium rose at first and later returned to normal. The important point however is that the changes in total calcium were again closely mirrored by changes in ionized calcium ($P<0,01$); $r= 0,932$). Changes in total protein were small although it appeared to increase slightly in Sheep N.

Table 6: THE EFFECT OF INTRARUMINAL ADMINISTRATIONS OF LACTIC ACID IN SHEEP ON PLASMA TOTAL CALCIUM, IONIZED CALCIUM, TOTAL PROTEIN, BLOOD pH, PCO_2 AND STANDARD BICARBONATE.

Sheep	Sampling periods	Time in minutes	Total calcium mg/100ml	Ionized calcium mg/100ml	Total protein g/100ml	pH	PCO_2 mm Hg	Bicarbonate mEq/ℓ
M	Predosing	0	10,2	3,50	5,76	7,361	45,0	23,5
	Postdosing	105	10,3	3,50	5,42	7,275	42,5	17,7
	Postdosing	195	9,8	3,30	5,53	7,359	41,0	21,5
	Postdosing	315	9,8	3,30	5,81	7,395	38,5	22,0
	Postdosing	435	10,4	3,45	6,03	7,393	37,0	21,0
N	Predosing	0	10,8	4,00	5,77	7,227	34,5	13,5
	Postdosing	75	11,4	4,50	6,14	7,179	41,0	14,4
	Postdosing	150	11,7	4,50	6,20	7,177	46,0	14,6
	Postdosing	225	12,0	4,60	6,20	7,247	34,0	14,2
	Postdosing	330	11,9	4,10	6,57	7,230	37,0	14,4
	Postdosing	450	10,7	3,90	6,30	7,305	34,0	16,5

Table 7: THE EFFECT OF INTRARUMINAL ADMINISTRATIONS OF DISTILLED WATER IN SHEEP ON PLASMA TOTAL CALCIUM, IONIZED CALCIUM, TOTAL PROTEIN, BLOOD pH, PCO_2 AND STANDARD BIRCARBONATE.

Sheep	Sampling periods	Time minutes	Total calcium mg/100ml	Ionized calcium mg/100ml	Total protein g/100ml	pH	PCO_2 mm Hg	Bicarbonate mEq/ℓ
O	Predosing	0	10,1	3,70	5,91	7,444	40,0	26,2
	Postdosing	105	10,0	3,50	5,66	7,435	39,5	25,0
	Postdosing	195	10,4	3,70	5,93	7,345	43,0	21,4
	Postdosing	315	10,1	3,50	5,75	7,389	38,5	21,5
	Postdosing	435	10,5	3,50	5,71	7,412	37,0	22,3
P	Predosing	0	10,8	4,30	5,17	7,408	40,0	23,5
	Postdosing	75	10,5	4,40	4,81	7,373	43,0	23,4
	Postdosing	150	10,9	4,40	4,87	7,372	46,0	24,0
	Postdosing	225	10,8	4,30	4,86	7,385	43,0	23,5
	Postdosing	330	10,9	4,30	4,99	7,412	38,5	23,0
	Postdosing	450	10,8	4,40	5,02	7,388	38,5	21,5

Experiment 7 : The relationship between total and ionized calcium in normal sheep during the course of a day.

This experiment served as a control for the two previous experiments and also gave some indication of the variation in calcium levels during an eight hour period.

PROCEDURE

Two wethers, Sheep O (33 kg) and Sheep P (37 kg), were used. Zero time blood specimens were taken and then the animals received 1 litre of distilled water by stomach tube. Sheep O was bled at 105. 195. 315 and 435 min and Sheep P at 75. 150. 225. 330 and 450 min after the administration.

The same analyses were performed as in Experiment 4.

RESULTS

The results of the analyses are presented in Table 7.

DISCUSSION

It is apparent that the administration of the water had little or no effect and that the levels of the various blood components remained remarkably constant during the course of the day.

Experiment 8 : The relationship between total and ionized calcium during a change in the acid-base balance induced by respiratory alkalosis.

PROCEDURE

Two wethers, Sheep Q (45 kg) and Sheep R (42 kg) were used. A zero time blood specimen was taken from each animal and then they were placed in a crate covered with tarpaulin and surrounded by heating elements and fans. The heat caused panting (160 — 220/min.) and the fans ensured a continuous circulation of fresh air. The animals were bled 110 and 420 min after being placed in the crate.

The same analyses were performed as in Experiment 4.

RESULTS

The results of the analyses are presented in Table 8.

DISCUSSION

In Sheep Q, PCO_2 decreased from 44 to 32,5 mm Hg and there was a small increase in standard bicarbonate so that the pH rose by 0,090 units. On the other hand, there was a small but definite fall in both total and ionized calcium similar to the decrease found when $NaHCO_3$ was infused intravenously or NaOH administered by stomach tube.

In Sheep R, PCO_2 decreased markedly from 50 to 25,5 mm Hg but there was a compensating decrease in standard bicarbonate from 25 to 18,7 m Eq/L and the pH rise was limited to only 0,037 units. Although total calcium remained constant there was a very small decrease in ionized calcium of a magnitude similar to that in Sheep Q.

Although these changes are small and well within the normal range it does appear from this and the foregoing experiments that it is pH as such and not PCO_2 or bicarbonate levels that affect calcium levels.

Changes in total protein were small and the slight rise seen in Sheep R may be the result of slight dehydration in this animal.

GENERAL DISCUSSION ON THE EXPERIMENTAL SERIES

The object of this series of experiments was to determine whether any significant change in total calcium was reflected by a similar change in ionized calcium. Previous studies[3, 4] have shown that in normal sheep total and ionized calcium levels are not well correlated (r = 0,42). However in Experiments 1; 2; 4; 5; 6 where total calcium levels were changed there was a high degree of correlation between the two (r = 0,-839; r required for significance at the 1 per cent level = 0,318) (Fig. 1). It would appear then that for all practical purposes total calcium determinations are a fairly reliable indication of the physiological level of calcium in the blood.

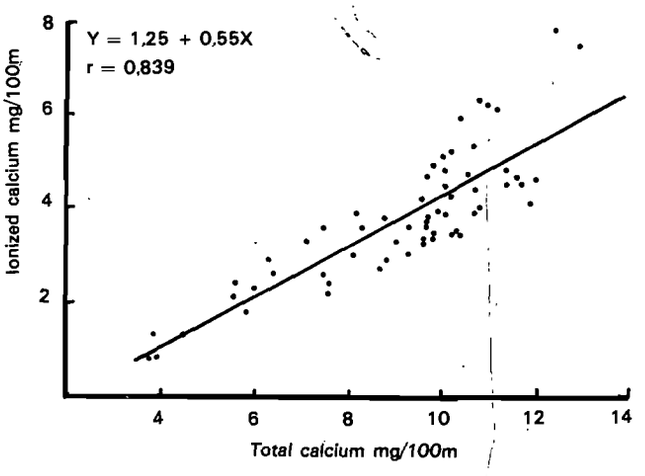

Fig. 1: Relationship between plasma total and ionized calcium when calcium levels were changed by experimental means.

Table 8: THE EFFECT OF TEMPERATURE INDUCED RESPIRATORY ALKALOSIS OVER 420 MIN IN SHEEP ON PLASMA TOTAL CALCIUM, IONIZED CALCIUM, TOTAL PROTEIN, BLOOD pH, PCO_2 AND STANDARD BICARBONATE.

Sheep	Sampling periods	Time in minutes	Total calcium mg/100ml	Ionized calcium mg/100ml	Total protein g/100ml	pH	PCO_2 mm Hg	Bicarbonate mEq/l
Q	Zero time sample	0	10,4	4,70	6,89	7,368	44,0	22,7
	2nd Sample	110	10,1	4,75	6,84	7,400	38,5	22,5
	3rd Sample	420	9,6	4,55	6,87	7,458	32,5	23,2
R	Zero time sample	0	9,9	4,75	6,57	7,393	50,0	25,0
	2nd Sample	110	9,9	4,65	6,89	7,351	38,5	19,6
	3rd Sample	420	9,9	4,60	7,10	7,430	25,5	18,7

While this study was in progress publications have appeared which substantiate these findings. So. for instance, it has been found in human patients with various disturbances of calcium metabolism that the correlation coefficient between total and ionized calcium was 0,94[9]. Similarly in human patients with hyperparathyroidism the correlation coefficient was found to be 0,85[10]. It has also been shown in rats that parathyroid hormone increase both total and ionized calcium while thyrocalcitonin decreases both levels[5].

In sheep again, it has been shown that, similar to findings in Experiment 4, intravenous infusions of bicarbonate decrease both total and ionized calcium and that this again increases the secretion of parathyroid hormone[8]. An investigation into the hypocalcaemia associated with normal parturition or with milk fever in cows has shown that total and ionized calcium are closely correlated under these conditions ($r = 0,985$)[6]. These latter workers suggest that little advantage is gained in testing for plasma ionized calcium instead of total calcium in the routine laboratory test for milk fever.

On the other hand, it has been reported[1] that in cows, 24h after oestrus or after oestrogen administration, there was a sharp transient drop in ultrafiltrable calcium while total calcium generally remained normal. However, later work in which an ion-selective electrode was used has not substantiated the above findings as a drop in ionized calcium was not found under similar circumstances[16].

In conclusion, then ionized calcium determinations may be necessary in the critical evaluation of certain conditions affecting calcium metabolism[17, 20, 9] but total calcium determinations remain valuable indicators of changes in the physiological level of calcium in the blood.

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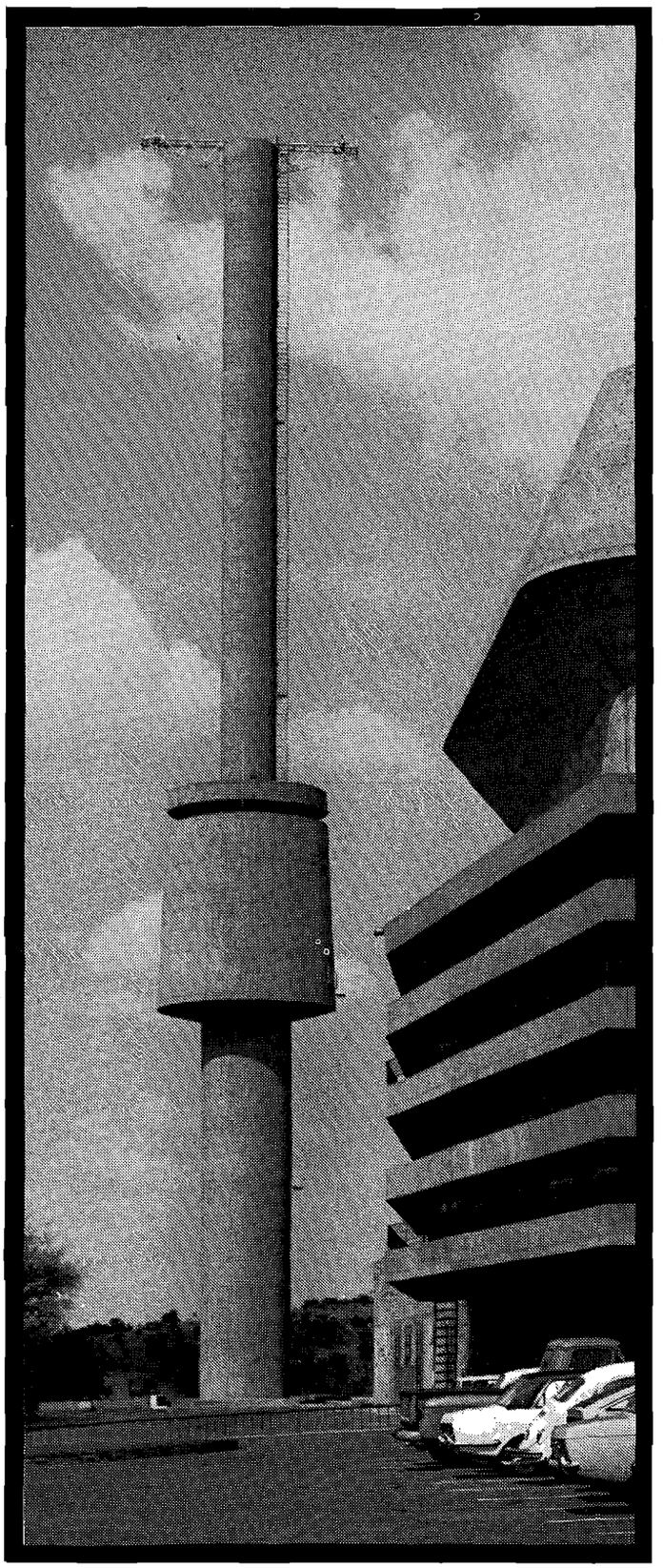

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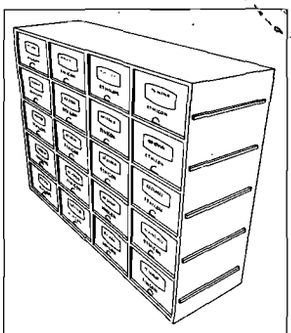

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Jl. S. Afr. vet. Ass. 47(3) 183-186 1976

THE INFLUENCE OF SEASONAL CHANGES IN THE DETERMINATION OF SELENIUM IN LIVER OF VARIOUS ANIMALS BY NEUTRON ACTIVATION ANALYSIS AND HIGH-RESOLUTION GAMMA SPECTROMETRY

A.M. HARTHOORN[1] AND J. TURKSTRA[2]

ABSTRACT: Harthoorn, A.M.[1]; Turkstra, J[2]. **The influence of seasonal changes in the determination of selenium in liver of various animals by neutron activation analysis and high-resolution gamma spectrometry** (1976). *Journal South African Veterinary Association* (1976) **47** No. 3, 183-186 (En). [1]Nature Conservation Division, P. Bag X209, 0001 Pretoria. [2]Atomic Energy Board Pelindaba. Republic of South Africa.

Selenium levels in the liver of animals living in the Umfolozi Game Reserve in Natal and in the Sabi Sand Nature Reserve in the Eastern Transvaal were studied by instrumental neutron activation analysis. The distribution of the selenium content was followed for about 16 months and attempts have been made to explain seasonal fluctuations of the selenium level.

INTRODUCTION

Nutritional Myopathy due to selenium deficiency has been recognised as an important factor in domestic livestock[3]. The possibility of selenium deficiency has also been investigated in zoo animals[11]. Overt myopathies are likely to occur when animals from selenium deficient areas are subjected to stress. Paradoxically the danger of this type of myopathy developing during transit has increased since improved methods of chemical capture techniques and tranquillisation have been developed. These methods have eliminated the necessity for keeping animals in holding pens on artificial feed for an interim period as was the procedure when using mechanical capture methods.

The literature on myopathies in captured wild animals has been reviewed by Harthoorn and Young[5]. Selenium deficient areas have been well documented in the United States, less well known in other countries, and virtually undocumented in Africa.

Selenium levels in normal biological tissue range from 0,005 to 0,5 ppm[2] with the result that only ultramicromethods can be used for selenium analysis. Colorimetry[4] and fluorimetry[6] have been utilized for the quantitative analysis of selenium in biological materials. A more modern analytical technique, neutron activation followed by high resolution gamma-ray spectrometry, has also been used extensively for the analysis of trace elements in biological tissues because of its inherent high sensitivity for many elements[10].

Five stable isotopes of selenium exist in nature. Table 1 gives the relevant nuclear data for radionuclides which are produced from selenium by neutron activation[7]. Only three of these radionuclides, namely ^{75}Se, ^{77M}Se and ^{81}Se, can be obtained with high specific activity. Neethling, Brown and De Wet[8] employed the shortlived radionuclide ^{77M}Se for the instrumental radioactivation analysis of selenium in biological material. Because of possible interferences associated with this fast technique, satisfactory results cannot generally be obtained at very low concentrations, unless a thin NaI(T1) scintillation crystal or a Ge(Li) detector is applied[1]. The virtually pure beta emitter ^{81}Se has been used to a limited extent[2]. The radionuclide most commonly used for the determination of selenium in biological material is ^{75}Se[9]. A limitation of the use of this radionuclide is the length of irradiation time required to achieve a high activity, but the long half-life ($t_{\frac{1}{2}} \doteq 120$ days) allows sufficient time for careful radiochemical separation and activity measurements. It was therefore decided to investigate the use of high-resolution gamma spectrometry for the direct determination of selenium in liver samples of wild animals at regular intervals over a period of 16 months.

MATERIALS AND METHODS

Preparation of the liver samples:

About 30 liver samples weighing approximately 30 g were collected from animals culled for various purposes from two areas in South Africa namely the Umfolozi Game Reserve in Natal and the Sabi Sand Nature Reserve in the Eastern Transvaal. The animals investigated were impala, white rhinoceros, blue wildebeest and warthog. The samples were immediately placed in standard bottles containing formalin solution. Subsequently pieces of liver tissue weighing approximately 2 g were taken from the centre of the sample, finely chopped, heated to a constant weight at 108°C and then ground in a agate mortar to obtain a more or less homogeneous powder. Approximately 500 mg of liver powder was accurately weighed into polyethylene containers and sealed.

Preparation of the standards:

A stock solution analytical grade selenium dioxide was prepared and diluted to contain 2 µg Se per ml of solution. Reference standards used were approximately 0,5 ml of the solution in polyethylene containers. The weights of the standard solutions were recorded and, after careful evaporation to dryness, each container was sealed.

NBS Bovine Liver Standard Reference Material (SRM 1577) containing 1,1 ± 0,1 µg selenium per gram of sample was also used as a reference standard. Accurately weighed samples (~ 250 mg) of this standard were sealed in polyethylene containers.

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Paper delivered at the South African Veterinary Association Congress held at Durban 8 - 12th September, 1975.

TABLE 1 : NUCLEAR DATA FOR RADIONUCLIDES PRODUCED FROM SELENIUM BY IRRADIATION IN A THERMAL REACTOR[7]

Target isotope	Abundance (%)	Activation cross-section (barn)	Product isotope	Half-life	Gamma-ray energy (keV)	Activity of Se after activation for one half-life $0 = 1 \times 10^{12}$ n cm $^{-2}$ sec $^{-1}$ (mC/g)
^{74}Se	0,87	30	^{75}Se	120 *d*	97 121 136 199 265 280 305 401	25•
^{76}Se	9,02	22	^{77m}Se	17,5 s	162	97•
^{78}Se	23,52	0,38 0,05	^{79m}Se ^{79}Se	3,9 *m* $6,5 \times 10^{4}$ y	96 nil	2,9 —
^{80}Se	49,82	0,08 0,53	^{81m}Se ^{81}Se	57 m 18,6 *m*	103 1 % gamma 272 280 550 560 830	1,5 25•
^{82}Se	9,19	0,05	^{83m}Se	60 s	350 650 1 010 2 020	0,46
		0,004	^{83}Se	25 m	225 358 520 710 833 1 060 1 310 1 880 2 290	0,04

Irradiation:

For convenience of counting in the determination of selenium, the samples and standards were irradiated separately and in fixed sequence for precisely 90 seconds. The relative value of the integrated neutron fluxes were determined for some samples and standards by using gold monitor samples. Irradiations were done in the pneumatic facility of SAFARI-1, an ORR-type reactor, in a thermal neutron flux of $2,89 \times 10^{13}$ n cm $^{-2}$ sec $^{-1}$. Westcott's epithermal index, r. for this irradiation, position is 0,0087.

Measurement of gamma activity:

Gamma spectrometry of the irradiated samples and standards was done exactly 20 seconds after each irradiation by placing them in a fixed position from the Ge(Li) detector. The detector used was a 50 cm^3 coaxial Ge(Li) diode (Princeton Gamma Tech.) connected to an uncooled TC 135 M Tennelec preamplifier. The output pulses were amplified by a TC 200 Tennelec amplifier. Spectrum analysis was performed on an Intertechnique 4 000-channel analyzer (Model SA 44). The resolution of this counting system is 3 keV (full width at half maximum) for the 1 333 keV photopeak of ^{60}Co. Data for peak analysis were recorded on magnetic tapes which were processed by computer. Yule's[12] smoothed first derivative method was applied to obtain the true peak counts under the photopeaks of interest.

RESULTS

A gamma spectrum of a liver sample after 90 seconds of irradiation and 20 seconds of decay time is shown in Fig. 1. The 162 keV photopeak of ^{77}Se is well separated from other gamma photopeaks. No interference due to the 198 keV photopeak of ^{190} can be observed.

It was observed by radioactivation that the Bovine Liver Standard used contains 1,14 µg of selenium per gram of sample compared to the prepared selenium reference standard. Fig. 2 shows the seasonal variation in selenium content in the liver of the animals.

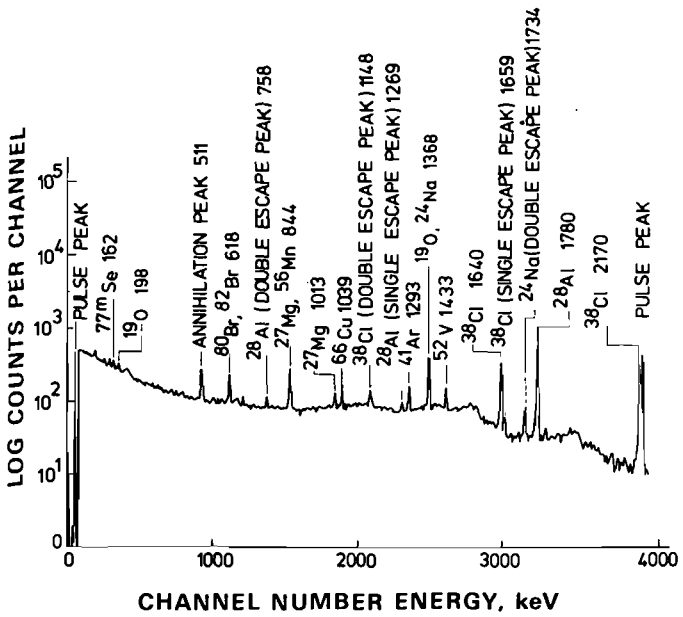

FIG. 1

The results obtained for the selenium concentration in the liver samples are given in Table 2.

Table 2: SELENIUM CONTENT IN ANIMAL LIVER DETERMINED BY NEUTRON ACTIVATION ANALYSIS.

Date animal killed	Neutron Activation Analysis Se (ppm)
73-09-02	0,78
73-12-21	0,76
73-12-21	0,64
74-01-07	0,71
74-01-07	0,74
74-02-05	0,54
74-02-05	0,88
74-03-04	1,01
74-03-08	0,99
74-03-26	1,14
74-04-20	1,14
74-04-23	0,68
74-05-10	0,94
74-05-23	1,35
74-06-12	1,28
74-06-18	0,72
74-07-02	0,82
74-07-28	1,1
74-08-08	0,59
74-08-21	0,68
74-09-07	0,5
74-09-23	1,11
74-10-07	1.22
74-10-25	0,99
74-11-02	1,04
74-11-23	0,86
74-12-07	0,69
74-12-19	0,45
75-01-09	0,83
75-01-21	0,97

DISCUSSION

The results of the analyses show remarkable conformity considering that the samples were derived from a number of different species and several geographical locations. The animals include grazers such as warthog (25), white rhinoceros (1) and blue wildebeest (1), and the impala (3) which is a facultative browser. All the animals follow a definite curve; even the impala which are shown separately, show the same trend. The work on wild animals is inevitably done on smaller numbers than on cattle where large numbers of samples are obtained from slaughter houses.

A few of the results do not ift into a general curve. Exceptionally low values were found in lactating and, in one case, pregnant warthog. The unnumbered dots represent impala which, possibly as facultative browsers, appeared to deviate somewhat from the others and were therefore excluded from the averages. These impala samples were derived from animals in predominantly overstocked land at Sabi Sand Game Reserve.

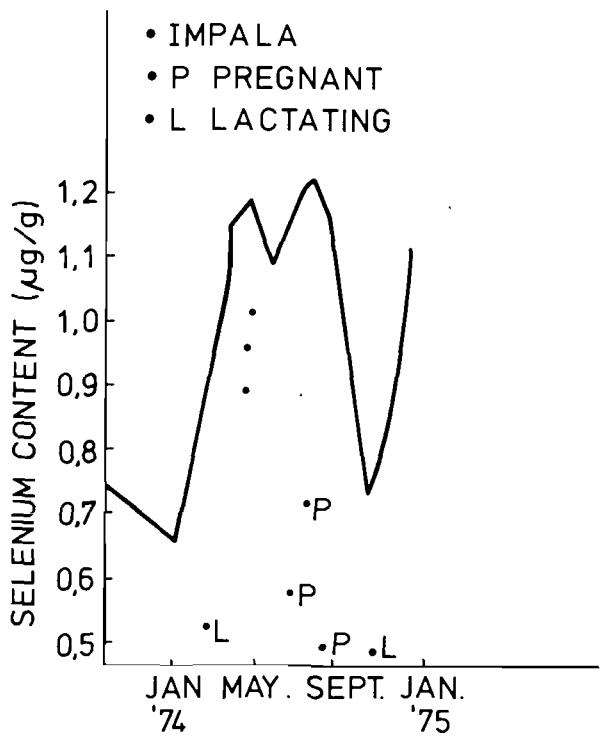

FIG. 2

The first low levels occur in September, dropping gradually until December (Fig. 2), after which the values commence to rise steeply, reaching peak values in April and May. The levels do not follow the same pattern as the rains. The first substantial rains fell in September, but the rise in liver selenium content occurred in December and January. This suggests a low selenium content of the first flush of herbage.

There appears to be a dip at the apex of the curve. This may be an artifact caused by scatter of results at this season. Other samples collected at this time which have still to be processed, will doubtlessly give substance to this section of the curve so that the trend can be more exactly determined.

The low values of 1975 are higher than those in 1974, and the curve rises earlier and more steeply. This phenomenon may be due to unseasonable rain which occurred during the winter in May. As the rainfall in 1975 was unusual both in quantity and distribution, it is likely that the 1974 section of the curve represents the more usual pattern. The difference between the two lower legs of the curve suggests that the selenium values in years of low rainfall may be lower.

As increased susceptibility to capture myopathy in wild animals during the latter part of the dry season is

generally accepted, although factors such as low forage protein value are undoubtedly a major cause, the low levels of selenium during this time of year are likely to be a contributory factor.

ACKNOWLEDGEMENTS

Our thanks are due to the Atomic Energy Board for permission to publish this paper, and to Messrs Darryl Mason, Norman Deane, Ian Crabtree and Pat Donaldson, and Dick Garstang for help in the collection of liver samples.

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BOOK REVIEW

BOEKRESENSIE

VETERINARY TOXICOLOGY

E.G.G. CLARKE and MYRA L. CLARKE

Baillière Tindall, London pp. 438, Figs. 0, Tables 5, Publ. Price R19,55

The first edition of Veterinary Toxicology by R.F. Garner became available in 1957 and soon became a standard text-book. In 1967 the third edition was published, after revision by Clarke and Clarke, with the title "Garner's Veterinary Toxicology". Eight years later, in 1975, there is once again a first edition titled "Veterinary Toxicology" by Clarke and Clarke. New sections have been added but the basic outlay of the previous edition is maintained.

In the Introduction the authors deal with the important factors of absorption, distribution, accumulation, detoxification and the elimination of toxic substances. Other basic facts concerning toxicology are also discussed in this chapter.

Part Two deals with mineral and inorganic substances, and is discussed in the format previously used; viz. : Forms and occurrence of substance, the absorption and excretion; the symptoms and lesions; the confirmation of the diagnosis and then the treatment and control.

Part Three, a chapter dealing with toxic gases and vapours, is a new addition. This is a very short chapter, only eight pages, but covers the essentials.

The following part is one of three that covers organic compounds; this chapter more specifically the drugs. This is a very wide field and most often discussed in pharmacological text-books, but the most important veterinary drugs are included in this chapter.

Part Five deals with the pesticides. The importance of the toxicology of the pesticides in veterinary medicine can never be overemphasised These compounds are becoming more and more important in agriculture and animal husbandry. Useful information could be found on all the important pesticides in use.

Organic compounds — Miscellaneous, is the heading of the following part. Substances not classified in the two previous chapters are described here, ranging from acetic acid to toxic fat disease in chickens.

The section in toxic plants forms the major portion of the book, 120 pages. Many names and description of South African plants appeared here, not always a complete description as one would prefer, but it stands to reason that this is not possible in a publication of this kind. The authors must be complimented on the manner in which they have presented Part Seven. Though the information is sometimes brief, the references given enable the reader to continue his study.

Part Eight covers the mycotoxins. This is a fairly new field and we hope that the authors will elaborate on this chapter in future editions, especially for the benefit of practitioners who often do not have specialized text-books available.

The penultimate section is a short one on venomous bites, stings and doping. This is followed by the section dealing with radio-active materials. This is a field of which every veterinarian should have some basic knowledge.

This book is once again an outstanding text-book for students, research workers and the practitioner. The way the information is presented and arranged as well as the long lists of references makes this a must on every book-shelf.

A.I.

Jl. S. Afr. vet. Ass. 47(3) 187-189 1976

PULMONARY HYPERTENSION IN RELATIONSHIP TO ACIDAEMIA AFTER MAXIMUM FORCED EXERCISE

A FURTHER PRELIMINARY REPORT

A.M. HARTHOORN* AND E. YOUNG†

ABSTRACT: Harthoorn, A.M.; Young E. **Pulmonary hypertension in relationship to acidaemia after maximum forced exercise.** *Journal South African Veterinary Association* (1976) **47** No. 3, 187-189 (En). Nature Conservation Division P. Bag X209, 0001 Pretoria. Republic of South Africa.

Pulmonary hypertension was shown to occur in wild young zebra and wildebeest subjected to forced exercise over 2 to 5 km distance. Measurements of pulmonary arterial pressure were made on a Devices recorder connected to a catheter passed via the jugular vein. Systemic pressure was recorded after puncture of the dorsal aorta. The electrocardiogram, PO_2, PCO_2 and pH were determined and enzyme levels such as CPK were estimated. Reduction of the pulmonary hypertension occurred after bicarbonate infusion.

INTRODUCTION

A preliminary report on an increase in the pulmonary artery pressure in animals subjected to intensive exercise was made by Harthoorn, Young, Burger & Whyte[9]. This change of pulmonary artery pressure was accompanied by a fall in systemic pressure and by clinical deterioration. Further work has substantiated these results and given indication of the operative mechanisms involved and the means whereby these changes in vascular pressure may be rectified.

MATERIALS AND METHODS

Free living zebra *Equus burchelli* aged approximately 2 years, and blue wildebeest *Connochaetes taurinus* aged approximately 6 to 9 months, were separated from their herds in the Mtomene and Mananga areas in the Kruger National Park and subjected to forced exercise under simulated capture conditions. The animals were chased by motor vehicles over distances ranging from 2 to 5 kilometres and captured by seizing ears or tail. A total of six zebra and six wildebeest were captured. Of these, three animals died soon after capture. Two of these were the only mature animals — a bull and a stallion. Another died from misadventure following a sudden movement during aortic puncture.

Immediately on capture, measurements were made of body temperature, heart rate and respiratory frequency. Two 10 ml peripheral blood samples were drawn from the recurrent tarsal, using *Terumo Venoject* tubes. The measurements and blood sample collections were repeated at 20 minute intervals.

Central samples of venous blood were obtained by means of a *PE 200 Intramedic* catheter passed through a 12 gauge needle inserted into the jugular vein, and into the right ventricle. Arterial samples were drawn, in the majority of cases, from the aorta by means of a *PE 100 Intramedic* catheter passed through a 28 cm long, 15 gauge needle inserted into the dorsal aorta through the 13th intercostal space in zebra and the 9th intercostal space in blue wildebeest. The skin area to be punctured was clipped, disinfected and anaesthetised with procaine hydrochloride before making a small stab wound with a *Bard-Parker rib-back* no. 11 pointed scalpel blade. On puncture of the aorta, the catheter was inserted for a distance of approximately 50 cm, after which the needle was withdrawn from the tissues and taped together with catheter on the skin alongside the spine, so as to immobilise the catheter and eliminate drag from the weight of the needle. Where this procedure was not followed, arterial samples were obtained by puncture of a superficial artery. Central venous samples and arterial catheter samples were drawn into 5 cc heparinised *Plastipak* plastic syringes, and for direct arterial puncture, 2 cc glass heparinised *Interchangeable* syringes fitted with an 18 gauge needle.

More latterly aortic puncture has been effective using a 16 gauge needle and using a *PE 50 Intramedic* catheter. All catheters were fitted with *Clay Adams* metal-plastic catheter adaptors at the proximal end to fit luer syringes or to connect with the transducer. The needle is directed cranially only sufficiently to pass the catheter towards the heart, and not to puncture the aorta more than a short distance forward of the area of skin puncture. Particular care must be taken to restrain the animal at the moment of puncture of the aorta to preclude multiple punctures or transfixion of the vessel.

The electrocardiogram was recorded using a *Devices* electrocardiograph and heat sensitive paper. The atrial, ventricular and pulmonary artery pressures were recorded via the *PE 200 Intramedic* catheter in the jugular vein, and the systemic pressures from the aortic catheter using a *4-327-L221 Consolidated Electrodynamics* transducer.

Blood oxygen and carbon dioxide pressures, as well as blood bicarbonate, base excess and total CO_2 were

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estimated from anaerobic blood samples using an *Astrup Radiometer* as described by Harthoorn & Young[8]. Enzyme levels (GOT, GPT, CPK, LDH) from regular peripheral samples were estimated as described by Harthoorn, Young & York[10].

Treatment consisted of an intravenous infusion made up of 1 000 milliequivalents sodium bicarbonate dissolved in 1 litre of a balanced electrolyte solution* given at a rate of approximately 1 litre per 250 kg body weight.

RESULTS

The exact levels of pulmonary arterial pressure are difficult to ascertain owing to muscular movements of the captured animals as also dyspnoea associated with acidaemia. Peaks up to 100 mm Hg were recorded in several instances.

The mean value for all animals for pulmonary artery pressures, taken about 30 minutes after capture, was 70/19 mm Hg, falling to 62/12 mm Hg 5 minutes after infusion of the bicarbonate solution.

The fall in pulmonary artery pressure after the infusion was, in all cases accompanied by signs of clinical improvement. There was a reduction in dyspnoea and tachycardia, a normalisation of systemic blood pressure, and an increase in alertness.

Pulmonary artery pressure was not in all cases high immediately after capture, but tended to rise during the first 30 to 60 minutes after capture (Table 1). The values soon after capture are sporadic owing to the different times taken for the catheterisation procedures. The rise in pulmonary artery pressure is accompanied by a fall in blood pH in zebra (Table 2), and a rapid deterioration of the clinical condition during the first 60 minute period. In some cases, recording was complicated by bloat in the captured wildebeest.

Table 1: BLOOD PRESSURE CURVES IN ZEBRA AND WILDEBEEST AFTER FORCED EXERCISE (AVERAGES OF 12 ANIMALS)

	On capture	after 30 min.	5 min. after infusion*
venous			
zebra	8/-6	8/-6	12/6
wildebeest	—	—	—
atrial			
zebra	8/0	8/0	20/4
wildebeest	13/2	—	12/-6
ventricular			
zebra	30/-6	70/-6	70/-5
wildebeest	98/15	—	48/3
pulmonary			
zebra	40/15	70/19	62/12
wildebeest	98/40	—	45/28
systemic			
zebra	154/130	130/110	155/112
wildebeest	98/65	—	105/70

* 1 000 milliequivalents of bicarbonate in 1 litre solution of balanced electrolyte solution.

* *Normosol*, Abbott, Johannesburg.

Table 2: pH VALUES IN ZEBRA AND WILDEBEEST AFTER FORCED EXERCISE (TOTAL 12 ANIMALS) — AVERAGES*

Immediately after capture	30 minutes later	after half infusion+	after all infusion+
zebra			
6,77	6,69	7,13	7,29
wildebeest			
6,95	7,18	7,58	7,62
* std. dev. =			
zebra			
0,18	0,13	0,11	0,12
wildebeest			
0,11	0,02	0,03	0,03

\+ 1 000 milliequivalents of bicarbonate in 1 litre solution of balanced electrolyte solution.

PO_2 and PCO_2 values in arterial and venous blood samples are shown in Table 3. Blood CPK and LDH levels rose considerably in zebra but no marked changes occurred in the enzyme levels in wildebeest.

Electrocardiogram recordings showed mainly extreme tachycardia, arrhythmias and extra systoles.

Table 3: PO_2 AND PCO_2 VALUES IN ZEBRA AND WILDEBEEST AFTER FORCED EXERCISE (TOTAL 12 ANIMALS)

Immediately after capture (range of values)		After 30 minutes' restraint (range of values)	
PO_2 mm Hg			
arterial	74 — 84	arterial	80 — 90
venous	15 — 25	venous	25 — 35
PCO_2 mm Hg			
arterial	28 — 35	arterial	35 — 38
venous	40 — 50	venous	42 — 45

Table 4: ENZYME VALUES IN ZEBRA AND WILDEBEEST AFTER FORCED EXERCISE (TOTAL 12 ANIMALS) — AVERAGES

Immediately after capture		after 1 hour's restraint
GOT mU/ml		
zebra	139	112
wildebeest	68	70
GPT mU/ml		
zebra	6	8
wildebeest	14	18
CPK mU/ml		
zebra	28	134
wildebeest	700	740
LDH mU/ml		
zebra	377	630
wildebeest	890	840

DISCUSSION

It may be postulated that the lung is one of the target organs for stress in zebra, and possibly also in antelope such as wildebeest. It has been determined that the basic haemodynamic disturbances caused by stress and hypotension differ among the various species. In the dog, the intestine appears to be the most sensitive organ, becoming engorged with blood as a result of hypotension and shock; in man the most marked changes appear in the kidneys, and in the rabbit it also appears that the lung is the primary target organ[12].

Changes in the lung occur also in dogs which, when deprived of normal oxygen tension during anaesthesia, exhibit an increased pulmonary vascular resistance which is associated with a decrease in cardiac output. The latter fell from 2,45 litres per minute to 1,95 litres per minute, while the pulmonary artery pressure rose from 14 to 22,5 mm Hg[2]. When the dogs were treated with an alpha adrenergic blocker, no rise in pulmonary vascular resistance or in pulmonary artery pressure occurred, suggesting that the increase in vascular resistance was mediated through alpha adrenergic receptors. It was pointed out that the phenomenon was due to adrenergic discharge rather than any direct effect of oxygen lack[2].

After capture, our animals showed normal blood PO_2 and PCO_2 levels[8]. Recent work (unpublished) has, however, indicated that there is a considerable drop in cardiac output under the conditions of stress during which the pulmonary artery pressures were monitored.

Pulmonary hypertension in ponies at high altitudes was ascribed to hypoxia by Bisgard, Orr & Will[3] and in the llama *Lama glama* by Banchero, Grover & Will[1], although the blood pH was not measured. Similar results were obtained in swine by McMurtry, Frith & Will[13].

Ateriospasm occurs in man as a result of massive adrenergic discharge. Normal values for circulating catecholamines are less than 1 µg per litre of blood volume. These levels, which are not increased during minor surgical procedures rise during radical surgery, such as extra corporeal bypass, to values of 10 to 30 µg/l[11 14 6]. Adrenaline infusion at a rate sufficient to cause tachycardia and cardiac arrhythmias had only very minor influence on the pulmonary blood pressure in immobilised sable antelope *Hippotragus niger*[7].

Further evidence may indicate that pulmonary vascular resistance is associated with sympathetic discharge. Increased pulmonary vascular resistance in dogs induced by hypothermia and cooling of the pulmonary circulation, could be largely abolished by alpha blockade[15]. Also pulmonary oedema (a constant major cause of death in our untreated zebra), was abolished by means of alpha blockade in man[3]. More recently, an increase in pulmonary vascular resistance has been established as a result of stimulation of the upper thoracic chain in *Papio* species using isolated lung lobes[4]. It was noted that the greater degree of anxiety and excitement of the animals during capture and anaesthesia, the less the responsiveness of the pulmonary vascular bed to nerve stimulation, due apparently to the transmitter concerned which had been partially exhausted by the initial excitement of the animals.

From the results of treatment with bicarbonate infusion and the evidence cited above, it appears probable that the rise in pulmonary artery pressures, reported in this paper is at least partially due to the action of low pH values. The fact, however, that the pulmonary artery pressure failed to return to the generally accepted normal levels after infusion suggests the implication of another factor or factors, possibly sympathetic in origin. Work to corroborate this theory is in progress.

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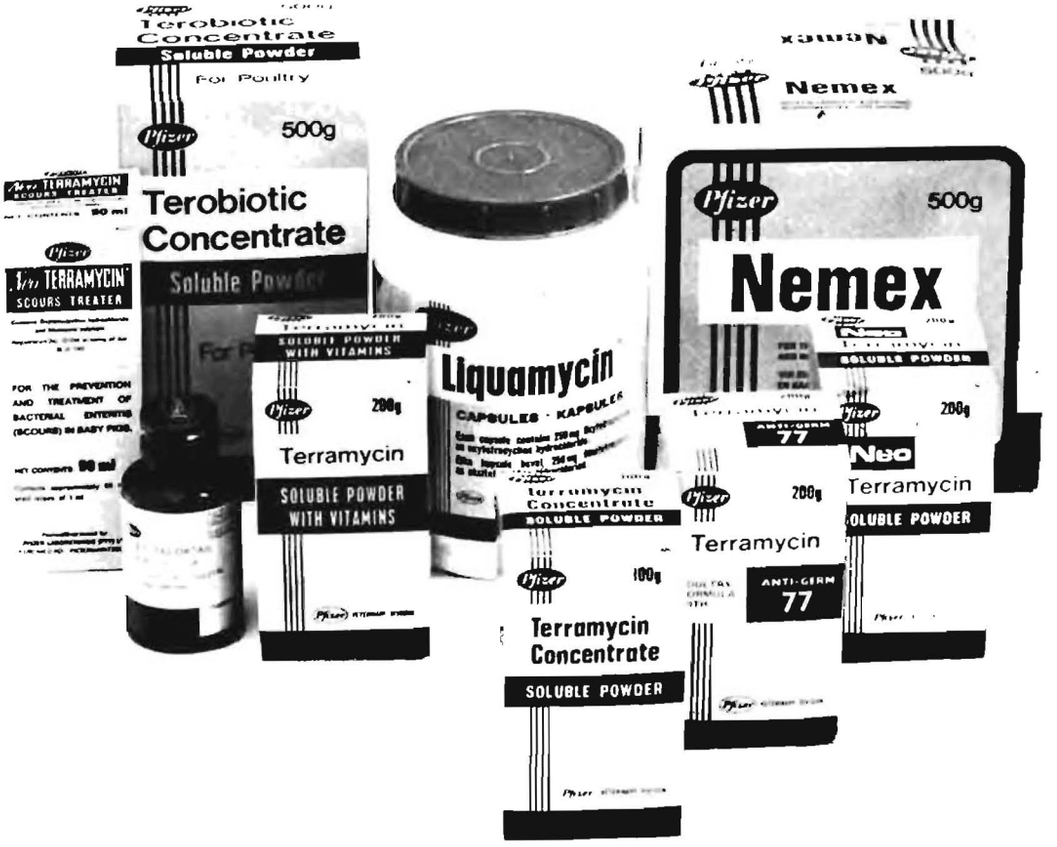

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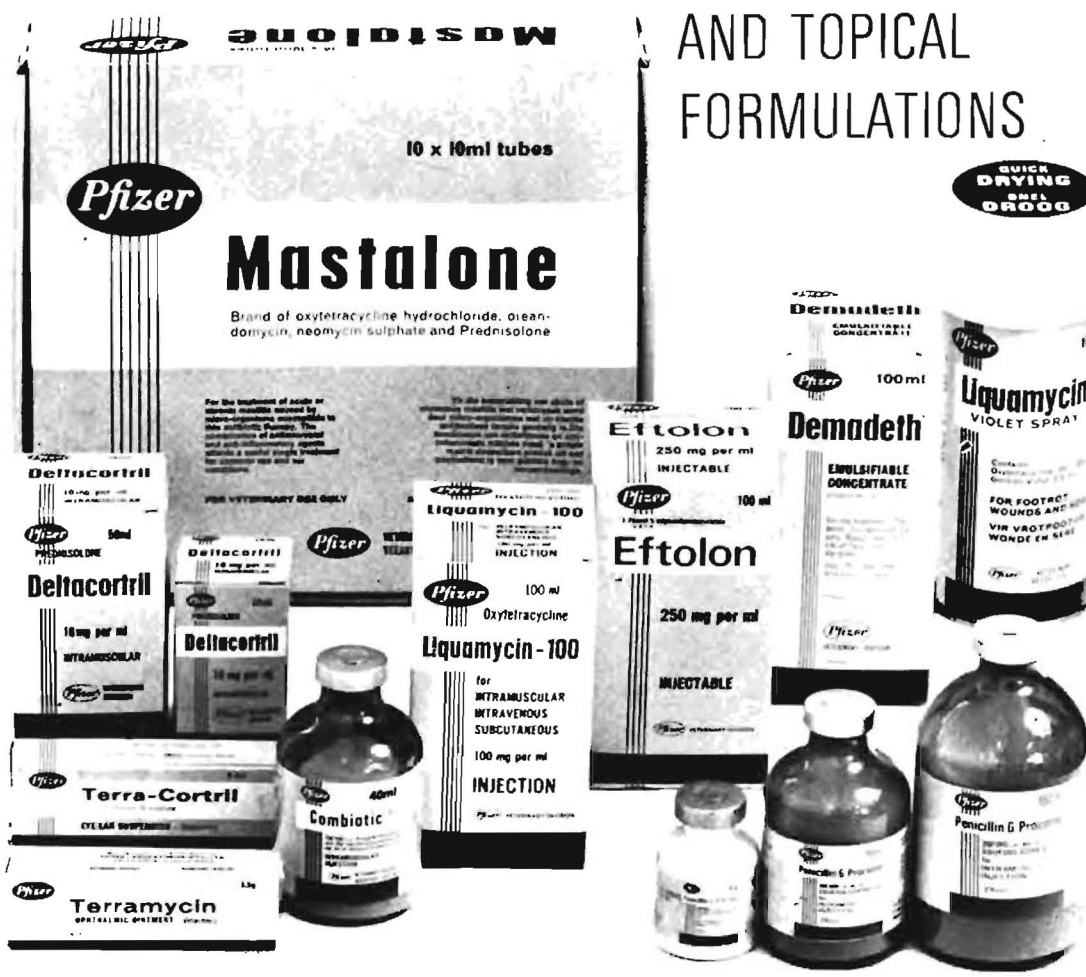

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PATHOGENESIS AND TREATMENT OF *ESCHERICHIA COLI* INFECTIONS IN CALVES†

R.J. BYWATER*

ABSTRACT: Bywater, R.J. **Pathogenesis and treatment of** *Escherichia coli* **infections in calves.** *Journal of the South African Veterinary Association* (1976) **47** No. 3, 193-195 (En) Beecham Pharmaceuticals (Research Division) Walton Oaks, Dorking Road, Tadworth, Surrey, England.

Two clearly defined types of *E. coli* infection are recognised and the factors predisposing and giving rise to pathogenicity are discussed. The mode of action of enterotoxins in the secretary mechanism is thought to be through stimulation of adenyl cyclase activity. Treatment and prevention of the disease is considered in relation to the pathogenesis of the infection.

PATHOGENESIS

Calf diarrhoea is a world wide problem and is the main source of morbidity and mortality in the first weeks of life.

While there has been recent attention to viral aetiology of diarrhoea both in the USA[13] and in the UK[26] nevertheless it seems probable that *E. coli* remains important either as a primary cause of disease or secondary to viral or nutritional factors.

Two clearly defined types of *E. coli* infection are recognised and depend to some extent on the immune status of the calf[12].

1. *Septicaemic colibacillosis*

This is seen where colostrum is taken in insufficient amounts or the time of ingestion is delayed. In either case colostral immunoglobulin is not absorbed in sufficient amounts. If the calf is agammaglobinaemic then it is highly susceptible to invasion of the body by strains of *E. coli* from the gut or pharynx.

The immunoglobulin responsible for protection of the calf against septicaemia is IgM[12]. This fraction is the first of the immunoglobulins to be prevented from leaving the gut after birth[18], which means that early and adequate intake of colostrum is particularly important in preventing this form of the disease.

2. *Enteric colibacillosis*

In this case the colostral intake and absorption may be adequate to prevent septicaemia, but may be less than optimal[14]. In this case the calf is more susceptible to diarrhoea which is not usually complicated by septicaemia.

The most clearly defined form of enteric colibacillosis is that where enteropathogenic strains are involved. To initiate disease, it has been suggested[23] that a strain of *E. coli* must be able to multiply in the upper intestine and must be able to produce enterotoxin.

Multiplication in the upper intestine: The ability to multiply in the upper intestine has been associated with possession of a specific K antigen. This antigen is common to strains of *E. Coli* enteropathogenic for calves and pigs and so was previously known as the "common antigen"[25]. Recently this has been designated the K99 antigen[17].

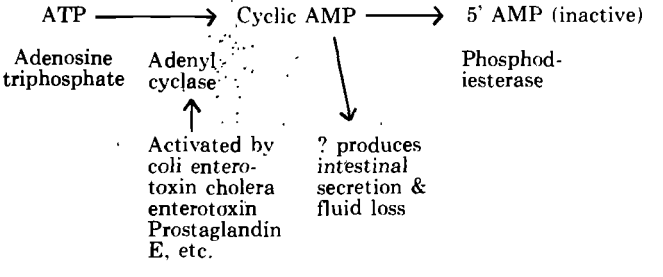
The K99 antigen in calves thus corresponds to the K88 antigen in pigs and is governed by a transmissible plasmid. It acts as a virulence determinant without which virulence is either absent or considerably reduced.

Enterotoxin production: Certain strains of *E. coli* produce enterotoxins which when placed in ligated segments of intestine in appropriate species cause dilation[22 10]. The enterotoxins produced have been classified as either heat labile or heat stable. The main characteristics of the two toxins are shown below:—

Heat Labile (LT)	**Heat Stable (ST)**
Inactivated at 65°C	Stable at 65°C
Antigenic	Non antigenic
Largely intracellular	Largely extracellular
Large molecule	Small molecule

In strains of *E. coli* associated with disease in calves, ST appears to be the predominant factor. The ability to produce enterotoxin is governed by a bacterial plasmid, and so can be transmitted[24].

The mode of action of enterotoxins in the secretory mechanism is thought to be through stimulation of adenyl cyclase activity.



This stimulation of adenyl cyclase by coli enterotoxin has been demonstrated in laboratory animals, but its importance in the natural disease condition has not yet been finally established. However if confirmed, the mechanism does present possibilities for the therapeutic use of agents which lower cyclic AMP, either through inhibiting adenyl cyclase or by stimulating phosphodiesterase activity.

The estimates of the incidence of clearly enteropathogenic strains (i.e. gut dilating strains) in field outbreaks of diarrhoea among calves has varied

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from a Belgian estimate of 12%[21], to 27,5% in Canada[11]. However Fisher and Martinez[8] referred to the above as "enterotoxic colibacillosis" and have suggested that a syndrome of "enteric colibacillosis" exists associated with a variety of strains of *E. coli* which are not gut-dilating and so do not fit the above classification. While this suggestion remains to be proved, it is of interest that Moon[15] has presented the hypothesis that the distinction between enteropathogens and non-enteropathogens is not absolute and is rather one of degree. Thus non-enteropathogens may produce enterotoxins, but in smaller amounts than those produced by the gut-dilatory strains and that these may be not demonstrable by this method.

Some support for this is given by experiments using Thiry-Vella loops in calves to assess enterotoxin activity[5]. In these experiments, strains from healthy calves were compared with those from scouring and septicaemic animals in their capacity to induce fluid secretion in calf Thiry-Vella loops. The results (Table 1) show that while strains from diarrhoeic calves were more potent stimulators of secretion than strains from healthy or septicaemic animals, nevertheless the latter strains both produced some secretion. It is therefore possible that the "enteric colibacillosis" described by Fisher and Martinez[8] may involve a number of weak enterotoxin producers rather than a single potent enterotoxin producer.

Table 1: FLUID EXUDATE RESPONSE TO CULTURE FILTRATES (SEMI-SOLID AGAR MEDIUM) OF STRAINS FROM DIFFERENT GROUPS OF CALVES.

	Healthy (faecal swabs)	Diarrhoeic (upper intestinal swabs)	Septicaemic (Heart blood swabs)
n	6	18	8
Fluid exudate (ml/45 min) ± SEM	11,3 ± 1,6	19,3 ± 1,1	10,6 ± 0,8
Significance (t test)	P<0,01		P<0,001

TREATMENT

The principal aims of treatment should be:—

1. Controlling bacterial infection
2. Restoring blood volume/rehydration
3. Correcting acidosis/electrolyte inbalances

1. *Control of bacterial infection*

Antibiotics and antibacterials remain the principal means of controlling bacterial infection. There is however some dispute concerning their efficacy. Trials have indicated benefit from antibiotic use[6] although later it was pointed out[7] that the findings may have been affected by the immune status of the calves used. Fisher & de la Fuente[7] were unable to show any benefit of treatment with chloramphenicol or nitrofurazone in comparison with untreated controls. Moreover Boyd, Baker & Leyland[1] found disappointing results from antibiotics although in their trial no untreated controls were included.

On the other hand Wray & Thomlinson[28] found antibiotics of at least some value (ampicillin was used on one farm to successfully treat diarrhoea, and decrease the percentage of pathogenic *E. coli* in faeces samples). They cautiously suggested that, if the sensitivity pattern of the pathogenic organism is known then antibiotics may be of value in preventing invasion or multiplication of pathogenic strains of *E. coli*. However Boyd, Baker & Leyland[1] found *in vitro* sensitivity testing a poor indicator of therapeutic response to antibiotics and concluded it was an unreliable indication of therapeutic value.

In view of the difficulty of including untreated control animals in field trials, experimental diarrhoea was used to assess the efficacy of diarrhoea treatment with a new semisynthetic penicillin, amoxycillin. In these experiments, calves bought in the market were dosed with 4g of sodium bicarbonate followed by 10 ml of a 6 hour brain-heart infusion broth culture of *E. coli* strain B44 (0/9 : K30 (?) K99). About 70% became diarrhoeic, usually about 3 - 4 days after challenge, implying that reinforcement of infection from the environment was occurring.

As calves became diarrhoeic they were allocated to treatment with amoxycillin powder 400 mg twice daily or to a placebo (lactose). The calves were assessed daily for diarrhoea.

Table 2: AMOXYCILLIN DISPERSIBLE POWDER, 400 mg TWICE DAILY IN TREATMENT OF EXPERIMENTALLY INFECTED CALVES.

	Amoxycillin	Placebo	Significance
Numbers treated	20	20	
Mortality	1 (5%)	6 (30%)	P<0,05*
Cumulative score in survivors ± SEM	6,32 ± 0,63	9,50 ± 0,53	P<0,01†
Days scour in survivors ± SEM	3,9 ± 0,08	5,7 ± 0,15	P<0,01†

* chi-squared test.
† t test

The results in Table 2 show that in the experimental conditions involved, a distinct benefit was obtained from antibiotic treatment. Moreover the calves received no other form of treatment such as fluid replacement, dietary restrictions etc. which might have increased the recovery rate. This confirmed the widespread impression that antibiotic use in the therapy of scours can, under some circumstances be very valuable. The appropriate antibiotic must be chosen on the basis of a combination of clinical experience on the farm involved and the use of sensitivity testing of faecal swabs. The latter may occasionally give misleading information possibly as a result of rapid development of resistance or as a result of differences between faecal flora and that in the small intestine where the fluid loss is occurring.

With antibiotic use in this context as in any other, it is important that treatment should begin as early as possible in the course of the disease, and should be given in adequate amounts over an adequate period.

2. & 3. *Restoration of blood volume, pH and electrolyte status*

Ideally this should be by slow intravenous infusion

of electrolyte solutions containing appropriate alkalinising agents. However slow intravenous administration under field conditions is often impracticable. More practicable is subcutaneous administration, although here the volume that can be given is limited and circulation to the region may be poor in moribund animals.

Recently attention has been given to oral replacement of fluid by solutions which contain glucose and/or glycine. The rationale is that *E. coli* enterotoxin has no effect on glucose absorption in calves[4]. Thus, since glucose absorption facilitates water absorption, the administration of glucose containing solutions may help to rehydrate animals suffering from enterotoxin induced dehydration. Such an approach has been successful in treatment of human patients with cholera[16].

Some evidence has been presented that use of glucose-electrolyte or glucose-glycine-electrolyte solutions by mouth can be beneficial in treatment of diarrhoea in calves[3,2]. While further evidence is needed, such an approach has the merit of convenience and simplicity, although care should be taken not to overload the animals with glucose and so produce a fermentative diarrhoea.

PREVENTION OF *E. COLI* INFECTIONS IN CALVES

1. *Colostrum*

Careful attention to the early ingestion of adequate amounts of colostrum is extremely important[12]. An adequate amount is at least 3 pints and while absorption is optimal at birth, it continues for 6 hours *post partum*. However, if infection has occurred before ingestion of colostrum, then the benefit is largely lost, even though the colostrum is given during the six hour period[12]. Thus ideally, ingestion must be very soon after birth.

2. *Vaccination*

Vaccines against enteropathogenic *E. coli* have so far been of only limited value, owing to the multiplicity of strains involved. However, recent advances have raised new possibilities. *In utero* vaccination via amniotic fluid has been used experimentally[9] and it has been shown that oral immunisation with heat inactivated *E. coli* and *Salmonella* antigens improves health and growth rate in calves, and reduces the need for antibiotic treatment[19].

Other possibilities for vaccines include protection against virulence determinants such as enterotoxins or the K99 antigen but these have yet to be used in calves.

3. *Hygiene*

It has been shown that the incidence and severity of diarrhoea increases with time of occupation of a calf *house*[20,27]. Wray & Thomlinson[28] found that use of uncontaminated houses was very important in treating the cycle of infection. This should therefore be included where practicable in any policy for control of *E. coli* associated diarrhoea on problem farms.

CONCLUSION

The control of *E. coli* diarrhoea in calves depends on balanced use of therapy and preventive measures. The greater understanding of mechanisms involved in the disease should increase the efficacy of the treatments available.

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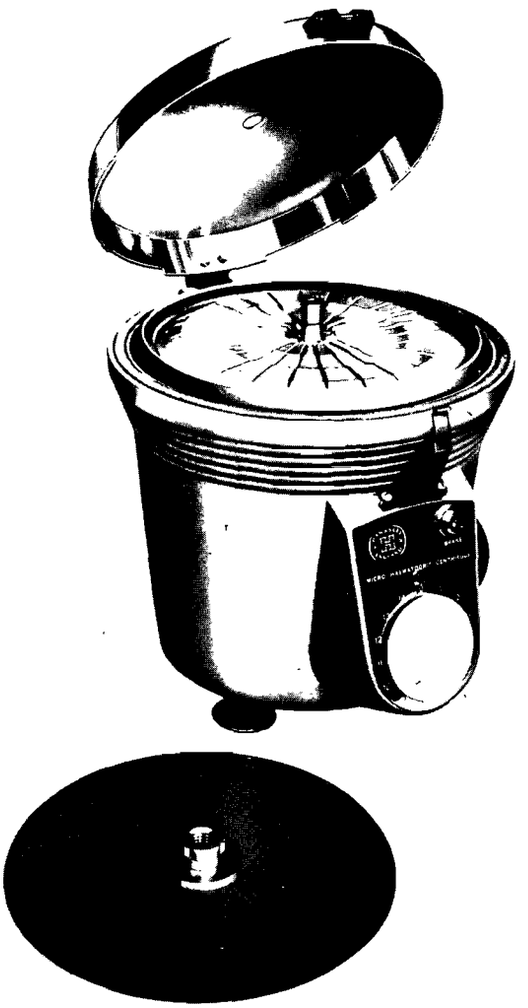

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CASEOUS LYMPHADENITIS IN SHEEP — METHODS OF INFECTION

G. NAGY*

ABSTRACT: Nagy, G. **Caseous Lymphadenitis in Sheep — Methods of Infection.** *Journal of the South African Veterinary Association* (1976) **47** No. 3 197-199 (En) Private Bag X536, 6970 Beaufort West, Republic of South Africa.

Depending on the route of infection, experimental exposure of 30 sheep to *Corynebacterium pseudotuberculosis* resulted in either mucopurulent vaginitis, prosthitis, conjunctivitis, rhinitis, or in subcutaneous abscessation. Caseous lymphadenitis, from which the organism could be recovered in 52 out of 56 instances, occurred in every instance with the exception of animals infected by the preputial route.

INTRODUCTION

The observation[6] that Merino sheep are more prone to caseous lymphadenitis and that the prescapular lymph nodes show a higher frequency of abscessation than do other nodes prompted this investigation into the various portals of entry of *Corynebacterium pseudotuberculosis* into the body. Once these are fully understood more effective methods of preventing the disease can be introduced.

In most cases infection gains entrance into the body through shearing and other skin wounds and less commonly through naval and docking wounds or by inhalation of infected dust or the ingestion of contaminated material[2, 4]. Small injuries in the gastrointestinal tract may also play a role[1]. It has been suggested that infected faeces of sheep suffering from the disease, play an important role in the transmission of the organism by contaminating shearing and other skin wounds of the newborn at birth[5]. Pulmonary "abscesses" have been produced by intravenous injection of cultures of *C. pseudotuberculosis*[3].

Experimental infection has been established by placing a broth culture of *C. pseudotuberculosis* on the freshly shorn skin of sheep or by spraying artificially contaminated sheep dip onto the skin[7].

The presence of *Corynebacterium spp.* has been established in the vagina of sheep by examination of vaginal swabs while[8] mucopurulent prosthitis, similar to that frequently diagnosed in naturally infected rams, could be produced experimentally by the local application of cultures of *C. pseudotuberculosis* into the prepuce.

Personal observations indicate that many of the "abscesses" result from the use by farmers of contaminated hypodermic syringe needles when inoculating sheep for various diseases.

All these various possibilities were taken into consideration when planning this investigation.

MATERIALS AND METHODS

The culture of *C. pseudotuberculosis* used in the experiment was isolated from a scrotal abscess of a naturally infected ram. For purposes of infection a 24 hour-old culture, grown in peptone water, was used.

Thirty clinically normal 8 months old Dorper sheep were used. The 25 males were divided at random into five equal groups and the five females made up Group 1. Each group was stabled separately and maintained as a closed unit. Animals of this particular breed were selected for experimental purposes from a farm on which the incidence of lesions caused by *C. pseudotuberculosis* in Merino sheep was high and considerably lower in the Dorper flock[6].

The animals in the six groups were exposed to the following experimental routes of infection.

Group 1: A gauze swab was dipped into the bacterial culture and then inserted into the vagina for approximately 1 to 2 seconds.

Group 2: A 19 gauge hypodermic needle was dipped into the culture and then introduced subcutaneously into the medial femoral region and withdrawn immediately.

Group 3: Five ml of the culture was administered orally to each of the animals.

Group 4: A gauze swab was dipped into the culture and smeared onto fresh penetrating wounds about 5 cm long which were made with sterile shears in the region of both shoulders and both hind limbs.

Group 5: Each animal in this group received an intratracheal inoculation of 0,5 ml of the culture.

Group 6: A drop of culture was placed into the preputial orifice of each ram.

The peptone water culture of *C. pseudotuberculosis* was controlled by culture for purity, immediately before and after experimental use.

The sheep were clinically examined on days 5, 35 and 122 after experimental infection and were slaughtered on day 122. The following lymph nodes were collected from each animal for laboratory examination *Lnn. cervicales superficiales, mediastinales caudales, hepatici, mesentherici craniales, iliaci mediales, subiliaci, poplitei, scrotales, mammarici.* Abscesses at the site of injection and in the lung, together with vaginas from Group 1 and prepuces from Group 6 were subjected to laboratory examination.

Lymph nodes were sliced aseptically and the number of abscesses counted. The abscesses were bacteriologically cultured.

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RESULTS

The results of the clinical examination of the animals in the six groups were as follows:

Group 1: (Vaginal infection):
At days 5 and 35 all the animals showed: swollen vulva, hyperaemic vaginal mucous membrane and mucopurulent vaginal discharge. At slaughter these signs persisted in only three of the ewes.

Group 2: (Contaminated needle):
A hot, red subcutaneous swelling at the site of the introduction of the needle developed within the first 5 days in all five sheep. When examined on day 35, localized subcutaneous abscesses could be palpated at the site in all the animals. These persisted until slaughtered.

Group 3: (Oral infection):
Apart from an excessive amount of mucus noticed in the faeces of one sheep on day 5, no other signs were apparent.

Group 4: (Wound infection):
Five days after infection all the wounds were covered with a crust; there was no suppuration and all healed uneventfully.

Group 5: (Intratracheal infection):
Five days after infection all five sheep had a bilateral mucopurulent nasal discharge; in addition three showed an acute bilateral conjunctivitis. On day 35 the mucopurulent nasal discharge was still present in three sheep; no conjunctivitis was seen. At slaughter only one animal showed a nasal discharge.

Group 6: (Preputial infection):
The prepuces of the five rams in the group were red, hot, painful, swollen and a mucopurulent discharge was present on day 5; the inflammatory process was still present on day 35. The swelling persisted to the day of slaughter and the external orifice was covered with a partly dried mucopurulent exudate which when removed caused a small amount of haemorrhage. The skin of the prepuce was hyperaemic.

The results of the examination and culturing of lymph nodes are presented in Tables 1 and 2.

Bacteriological examination revealed that *C. pseudotuberculosis* was present in 52 out of 56 abscesses and that a mixed bacterial infection was present in four.

From the results in Table 1 it is evident that infection with the formation of abscesses occurred in the animals artificially infected by contaminating shearing wounds and needles and in those infected *per vaginam*, *per os* and by the intratrachael route. No abscess formation resulted from preputial infections. Table 2 illustrates that the route of infection plays an important role in the number of abscesses that develop.

Table 1: INCIDENCE OF ABSCESSES

LOCATION OF ABSCESSES	ROUTE OF INFECTION AND NUMBER OF ABSCESSES						TOTAL
	VAGINAL	SUBCUTANEOUS	ORAL	SHEARING WOUNDS	INTRA/ TRACHEAL	PREPUTIAL	
Lnn. cervicales superficiales				6	5		11
Lnn. mediastinales caudales	2			5	1		8
Lnn. hepatici	1						1
Lnn. mesentherici craniales	1						1
Lnn. iliaci mediales		1		1			2
Lnn. subiliaci		1	2	7			10
Lnn. poplitei				1			1
Lnn. scrotales (ram)		5		6			11
Lnn. mammarii (ewe)	5						5
Lung					1		1
Injection site		5					5
TOTAL	9	12	2	26	7	0	56

Table 2: EFFECT OF THE METHOD OF INFECTION ON THE FREQUENCY OF ABSCESS FORMATION

METHOD OF INFECTION	NUMBER OF ABSCESSES	PERCENT
Shearing wounds	26	46,5
Subcutaneous	12	21,5
Vaginal	9	16
Intratracheal	7	12,5
Oral	2	3,5
Preputial	0	0
TOTAL	56	100

DISCUSSION

Dorper sheep under natural conditions are only sporadically infected with *C. pseudotuberculosis* and it is obvious that the animals used in this experiment had very little, if any, resistance against the organism when experimentally infected by a variety of routes. There is no doubt that shearing wounds are the most important route; that other methods of infection play a role is also evident. It is interesting to note that all five animals infected with contaminated needles introduced subcutaneously, subsequently contracted local infections and abscesses in regional lymph nodes. Also of interest is the importance of vaginal infection; all five ewes developed mucopurulent vulvitis and vaginitis and caseous lymphadenitis of the supramammary lymph nodes. In addition all the

animals infected preputially developed a persistent mucopurulent prosthitis and it is interesting to speculate on the role that venereal infection may play in nature in *C. pseudotuberculosis* infection. This aspect warrants further investigation.

This experiment proved that there is a definite relationship between the route of infection and affected regional lymph nodes.

ACKNOWLEDGEMENTS

I have pleasure in thanking Prof. R.C. Tustin, Department of Pathology, Faculty of Veterinary Science, Onderstepoort, for his suggestions and assistance in the preparation of this report.

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INFORMATION INLIGTING

PREVENTING BEAK NECROSIS IN POULTRY

Scientists at the Agriculture Canada Research Station at Lethbridge, Alberta, have suggested that a simple change in ration from mash to pelleted feed may be all that is required to prevent beak necrosis, a poultry disease which, in extreme cases, results in mortality. Mortality percentages as great as 10 per cent had been experienced and the scientists believe that these might have been the result of beak necrosis.

The extent of the disease was studied by them in a typical broiler-breeder chicken flock at the Research Station, and about half were found to be suffering from varying degrees of beak necrosis, by the age of one year.

Using an electron microscope, the researchers conducted beak examinations and found that infection was caused by bacteria which multiply on particles of feed mash adhering to the beaks of the birds. In the early stages of infection, superficial rotting of the beak was observed. In severe cases, the lower part of the beak had rotted away.

The conclusion drawn was that beak necrosis can be prevented if a mash feed is replaced, as early as possible, by a pelleted feed.

("Preventing Beak Necrosis", News & Features, No. 1670, May 7, 1976, p. 7: Agriculture Canada, Ottawa, Canada.)

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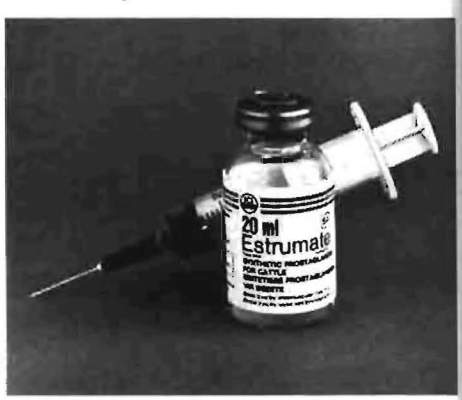

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ADMARK 1591

LABORATORY AND FIELD CONTROL OF CLINICAL MASTITIS IN DAIRY COWS AROUND BULAWAYO

R.W. BRYSON* and J.W. THOMSON*

ABSTRACT: Bryson, R.W.; Thomson J.W. **Laboratory and Field Control of Clinical Mastitis in dairy cows around Bulawayo.** *Journal South African Veterinary Association* (1976) **47** No. 3 (En) 201-203. Dept Veterinary Services, Box 572, Bulawayo, Rhodesia.

The organisms responsible for clinical mastitis in dairy herds around Bulawayo were identified and their antibiotic sensitivity was determined. Streptococci, staphylococci and coliforms were responsible for 37%, 28% and 29,5% of cases respectively. Antibiotic resistance increased over the 3 year period. The high incidence of coliform mastitis is discussed, as is the effect of dry cow therapy on peri-natal mastitis and the nature of the scheme of control. The laboratory is considered an essential adjunct to other control methods.

INTRODUCTION

A well equipped veterinary laboratory is in operation in Bulawayo and it was felt that information on mastitis could be obtained by this facility and farmers assisted in control of the disease. A free service was offered to all farmers supplying milk to the Bulawayo branch of the Dairy Marketing Board. The latter agreed to assist by receiving clinical mastitis samples sent in with daily bulk milk supplies. Thirty-five out of the eighty milk producers agreed to take part in the scheme and eventually about a hundred samples were submitted each month.

Each farm was regularly visited by a government veterinary surgeon at milking time to determine the cause of the mastitis problem in relation to the bacterial picture and to suggest methods of better control.

MATERIALS AND METHODS

Collections of Samples: Instructions on the lid of each holding box gave simple basic instructions for collecting an uncontaminated sample. Identification of cow and quarter and details of treatment was requested. Farmers were encouraged to start treatment immediately after sampling and not to wait for a laboratory report.

Laboratory examination: Diagnostic procedures followed those of Carter[1]. Samples were allowed to stand at room temperature for 5 to 6 hours before aliquots were taken for cell counting and culture; the balance of milk was incubated at 37°C for 12 hours.

Milk was plated onto MacConkey's agar, blood agar and Edwards media. Somatic cell counts were estimated by the direct microscopic method described by Prescott & Breed[2]. Bacterial identification in these smears was often possible and a direct antibiogram was then set up. In other cases this was carried out next day.

Bacterial identification was carried out by conventional methods. Usually the responsible pathogenic bacteria fell into one of several main groups. Recovery of pathogenic bacteria and a cell count of 500 000 or more was taken as criterium of the diagnosis of bacterial mastitis.

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RESULTS

Bacterial recovered: Table 1 shows the main types of pathogenic bacteria recovered between 1972-74.

Figures remained fairly consistent during this period and are similar to those recorded in United Kingdom by Howell[3].

Table 1: PERCENTAGE INCIDENCE OF BACTERIA ISOLATED FROM MASTITIC MILK SAMPLES 1972 — 1973 and 1973 — 1974.

Bacteria identified	1972 — 1973 Percentage		1973 — 74 Percentage	
Strep. agalactiae	12	33	26	41
Strep. dysgalactiae	17		11	
Strep. uberis	3		3	
Strep. faecalis	1		18	
Staph. pyogenes	24	27	25	29
Staph. epidermidis	3		4	
Coliforms	20	33	16	26
Klebsiella spp	13		10	
Pseudomonas spp		2		3
Corynebacterium pyogenes		4		1
Corynebacterium renale				
Candida spp.				
Yeasts				
Serratia spp.		1		—
Chromobacter spp.				
Micrococcus				
Total No. of milk samples examined:	471		894	
Total No. of isolates recovered:	353		330	

Antibiotic sensitivity of bacteria: A study of Table 2 reveals that resistance to practically all the antibiotics in current use gradually emerged. The most widely used is a combination of streptomycin and penicillin. This was effective against 83% of *S. pyogenes* isolated in 1972/73 and 59% in 1973/74.

In the case of streptococci this combination was effective against 70% of cases in 1972/73 and 87% in 1973/74.

Although chloramphenicol was consistently effective *in vitro* against most organisms it was often found that milk was being submitted from chronic mastitis cases which were beyond treatment. The futility of this had to be explained to farmers. The use of

Table 2: ANTIBIOTIC SENSITIVITY OF BACTERIA RECOVERED FROM MASTITIC MILK.

ISOLATE	PERCENTAGE INCIDENCE OF SENSITIVE ISOLATES 1972 — 73 / 1973 — 74.						
	PENICILLIN	STREPTOMYCIN	TETRACYCLINE	CHLORAMPHENICOL	SULPHADIMIDINE	NEOMYCIN	NITROFURANTOIN
Staph. pyogenes	52/59	83/59	85/70	100/100	4/5	95/84	95/70
Staph. epidermidis	100/70	90/80	100/80	100/50	90/50	90/70	90/100
Strep. agalactiae	73/100	40/5	92/100	100/95	20/5	27/5	92/56
Strep. dysgalactiae	80/92	90/40	100/92	100/92	20/25	80/50	90/70
Strep. uberis	80/*	40/*	100/*	100/*	20/*	20/*	100/*
Strep. faecalis	50/*	75/*	75/*	75/*	50/*	100/*	100/*
Klebsiella spp.	nil/nil	59/85	77/80	100/100	nil/nil	100/90	70/60
Strep. uberis, *Strep. bovis*, *Strep. faecalis*	*/100	*/15	*/100	*/100	*/15	*/15	*/100
Corynebacterium spp.	90/90	100/100	100/100	100/100	90/90	90/90	90/90
Pseudomonas aeruginosa	nil/nil	50/20	50/40	70/40	nil/nil	nil/40	nil/nil
Other coliforms	nil/nil	80/80	80/75	95/100	3/10	90/90	100/55

* VARIATION IN GROUPING 1972 — 73 / 1973 — 74.

chloramphenicol was discouraged for public health reasons.

Strains of *S. pyogenes* resistant to penicillin and streptomycin increased sharply from 17% in 1972/73 to 41% in 1973/74. In most cases these strains were susceptible to cloxacillin which was introduced towards the end of the period under survey.

DISCUSSION

The majority of dairy herds in the Bulawayo area are hand-milked. This undoubtedly is a factor in the high incidence of coliform mastitis where hygiene was often found to be suspect. Machine milked herds returned the highest incidence of streptococcal mastitis. Coliform mastitis was mostly caused by the *Klebsiella/Aerobacter sp.* The clinical picture produced has in the past been confused with mastitis produced by other organisms, but several distinguishing features emerged. Cows are affected shortly after parturition, the affected quarter being very swollen, oedematous and tender. The milk is grossly altered, consisting of a thin straw coloured odourless fluid containing large yellow clots. Fever and general systemic disturbance is evident. Inappetence, a temperature of 40°C or more with recumbency may precede death from toxaemia.

Vigorous and prompt therapy are usually successful. This includes frequent bathing and stripping of the quarter and parenteral and local administration of chloramphenicol.

We were able to confirm the observations of Schalm and co-workers[4] that clinical cure was followed by complete restoration to full milk production, although sometimes only in the following lactation. The inflammatory reaction is not followed by scar tissue formation as in other forms of mastitis, notably that produced by *Staphylococcus pyogenes.*

In one herd *Klebsiella* mastitis cases were occurring at the rate of 5 to 6 a week. A detailed investigation on the farm showed that udder washing water was grossly contaminated. This came from a well which was found to be heavily contaminated with coniform organisms including *Klebsiella spp.* and faecal *Escherichia coli.* The effect of chlorinating this water was quite dramatic in that coliform mastitis cases ceased almost at once. It was seldom possible to provide a completely pure water supply for dairy purposes. Generally borehole water was much cleaner than a well supply because of the reduced risk of external contamination.

Many farmers depend on chemicals to disinfect the small amounts of water used for udder washing and cleaning. In some cases the price of these disinfectants and claims made on their efficacy was such that farmers tried to economise unwisely and the water became grossly contaminated after the udders of a few cows had been washed. Such water was a frank source of infection in spite of the disinfectants used.

To make progress in any mastitis control scheme, regular personal inspection of the premises and the method of milking is considered essential.

We seldom saw a good system of hygiene on our first visit and it was pointed out to the farmer that unless he adopted a good routine, there was no point in submitting samples from the numerous mastitis cases which could have been avoided. Udder cloths were in use in almost every case and these were quickly prohibited. Other basic hygiene procedures were readily adopted when reasons were given for their use and dry cow therapy and teat dipping were soon in vogue.

Synthetic penicillins were widely used in dry cow therapy. It was found that the highest incidence of coliform mastitis was recorded in these herds and we tend to agree with the opinions expressed by Schalm *et al*[4]. Suppression of all susceptible bacteria by this method may reduce the leucocytes in the udder and facilitate the entry of non-susceptible bacteria, especially the coliforms, within a few days of calving. For this reason we recommended that only where there was a history of mastitis in the herd should universal dry cow therapy be practised, or alternatively for individual cows with a mastitis record.

Where practicable we recommended teat dipping of dry cows for the last few days before parturition.

After 3 years the scheme remains very popular as indicated by the increasing numbers of samples received and requests for on-the-farm assistance. The virtue of veterinarian/farmer contact on the farm cannot be overemphasised.

Monitoring the bacteria involved in mastitis on a farm will assist in hygiene control and indicate any developing antibiotic resistance.

Culling is an essential part of the control scheme and our current work is aimed at eliminating chronic cases confirmed by cell counts and bacterial identification.

ACKNOWLEDGEMENT

We are grateful to Mrs K. Pigott, Laboratory Technologist, for the many hours of work spent on the bacteriology and compilation of data.

Permission from Director of Veterinary Services, Rhodesia to publish this paper is acknowledged.

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INFORMATION INLIGTING

BUTTER, MARGARINE AND HEALTH

The European Commission has told a Member of the European Parliament in answer to a written question that there is no scientific proof that butter is harmful to health. Similarly there is no evidence that margarine is better than butter from a dietary point of view, notably as regards the effects of polyunsaturated fatty acids. It is generally agreed, however, that excessive consumption of oils and fats is unwise.

In fact, the wave of advertisements attributing preventive or curative properties to certain margarines has prompted the European Commission to consult the *Scientific Committee for Food.* The Committee's view of these claims is as follows:

"At present there is not sufficient evidence to justify the conclusion that polyunsaturated fatty acids prevent or cure arteriosclerosis or coronary disease. There is general agreement that their consumption in specific dietary regimes in place of other fatty acids results in a lowering of the plasma levels of certain lipids in some human subjects with certain hyperlipidaemias. Even such established properties should not, in the opinion of the Committee, be used in advertising preventive or curative properties in relation to human disease. Only in very exceptional cases should such statements be authorised with the aim of informing the general public."

In its proposal for a general directive on food labelling the European Commission suggests that references to preventive or curative properties be banned, as both butter and margarine can be considered to be natural, thus healthy, products.

(Euroforum, Brussels; Nos. 3/76 and 6/76)

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ADMARK 1592

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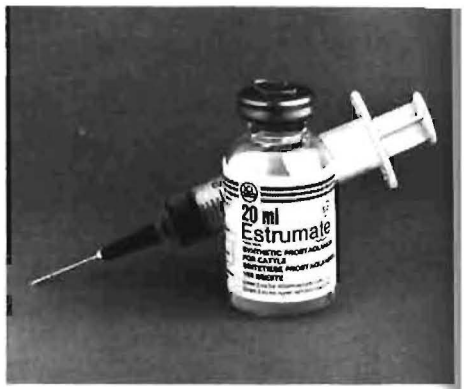

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THE INCIDENCE AND SOURCES OF PENICILLIN IN MILK SUPPLIED TO THE CITY OF JOHANNESBURG

R.C. COOK, K.W. KATZ and P.J. MEARA*

ABSTRACT: Cook, R.C., Katz, K.W., Meara, P.J. **The incidence and sources of penicillin in milk supplied to the City of Johannesburg.** *Journal of the South African Veterinary Association* **(1976) 47 No. 3,** 205-207 **(En) City Health Dept., P.O. Box 1477, 2000 Johannesburg, Republic of South Africa.**

The incidence of penicillin in bulk milk supplies to the city has varied from 1,2% to 2,6% over the past five years, and 3,2% of 366 pasteurised milk samples examined in 1975 were found to contain penicillin. Investigation of the sources revealed numerous instances of producers, milkers and veterinarians who had not acted responsibly in regard to the marketing of milk from treated cows. Details are provided. The legal and professional obligations of the veterinarian are emphasized. Reference is made to the dye marking of registered intramammary formulations for farmer treatment of mastitis.

INTRODUCTION

The presence of antibacterial residues in milk constitutes a public health problem, and also often results in starter failure in cultured dairy products causing financial loss.

Herd milk samples collected from the farm bulk tanks of licensed suppliers to Johannesburg are regularly subjected to microbiological assay for inhibitory substances using a disc of 13 mm diameter to which 0,1 m*l* of milk is added after treating the milk at 80°C for 5 minutes[1]. This disc is then plated on to pen-assay agar seeded with *Sarcina lutea* and incubated at 37°C for 24 hours. The quantity of penicillin in any positive sample is determined by measuring the zone of inhibition and correlating it to the diameter of the zone of inhibition around a known concentration of penicillin. The culture of *S. lutea* used is sensitive to 0,001 units of crystalline penicillin G potassium. This concentration gives a zone of inhibition of 20 mm or more in diameter.

The reason for concentrating on penicillin to the exclusion of other antibiotics is fourfold:—

1. Penicillin as such or in combinations, is still extensively used for the intramammary treatment of mastitis in spite of the availability of other antibiotics.
2. Many medical authorities in the field of allergies and paediatrics believe that of the various antibiotic therapies employed for bovine mastitis, penicillin residues are most likely to render the milk harmful to consumers (0,003 µ/m*l* penicillin has been known to cause allergies in humans).
3. The specific identification in milk of penicillin residue can be achieved because it can be specifically inactivated by penicillinase.
4. Penicillin in milk heated to 80°C for 15 minutes is not destroyed and therefore will not be destroyed by commercial pasteurization (72°C for 10 seconds).

The following data on antibiotic contamination over the past five years is of interest[2]:

Year	Herd Milks Tested	Inhibitory Substances	Penicillin Contamination
1971	2 345	63 (2,7%)	47 (2,0%)
1972	2 273	77 (3,3%)	59 (2,6%)
1973	2 238	49 (2,2%)	40 (1,8%)
1974	2 246	32 (1,4%)	27 (1,2%)
1975	2 259	44 (1,9%)	33 (1,4%)
Of 366 pasteurized milk samples tested during 1975, 12 contained penicillin residues (3,2%).			

Of 366 pasteurized milk samples tested during 1975, 12 contained penicillin residues (3,2%).

In 1974, milk producers were again circularized that antibiotics by any route of administration could be voided in the milk, and that milk from treated cows should be excluded for a period of 72 hours after the last treatment.

Since 1975, penicillin-contaminated milk supplies have been discontinued until retesting has established them free from contamination.

All suppliers whose bulk milks show inhibitory substances are visited by a veterinarian to determine the source of the inhibitory substance. The following sources of penicillin were revealed by the investigations:

1. Intramammary treatment of udder quarters by the farmer, then milked prior to the expiry of the excretion period and included in the bulk milk supply.
2. Quarters treated by the milker unbeknown to the farmer.
3. One quarter treated and milk from the remaining quarters of the udder included in the bulk milk supply.
4. Penicillin or penicillin compounds administered parenterally by the farmer for the treatment of mastitis and other conditions such as abscesses, retained afterbirth, footrot, etc.
5. Parenteral penicillin or penicillin compounds infused into the udder by the farmer.
6. Cows treated for footrot with procaine penicillin prescribed by the local medical practitioner.
7. Cows treated by veterinarians with intramammary preparations, without proper

*City Health Department, Box 1477, 2000 Johannesburg.

identification of the treated cows, and without warning of a degree of transfer from treated to untreated quarters.

8. Cows treated by veterinarians post-operatively with long acting penicillin, e.g. benzathine penicillin (NN' — dibenzylethylene-diamine).
9. Cows treated by a veterinarian with a combination of penicillin and dihydrostreptomycin by intra-uterine irrigation. 10 cows in milk were treated in a milk herd of 24 animals.
10. Cows treated with a combination of procaine penicillin and benzathine penicillin supplied to a farmer by a veterinary practitioner's receptionist to treat animals which had overeaten.
11. A cow treated by a veterinarian who left medicine with the farmer. The original label had been removed from the bottle and replaced by a gummed label on which only appeared "15 c.c. daily". A sample from this bottle proved positive for penicillin.
12. The use of dry cow therapy where the dry period was less than 4 weeks.

THE INTRODUCTION OF DYE-MARKED INTRAMAMMARY PREPARATIONS

In accordance with Act 36 of 1947, as from 1 January 1977 all stock remedies registered for intramammary therapy will contain a blue-green food dye marker for the presence of antibiotic substances. The dye will be synchronized with the excretion period of the antibiotic. This dye marking is intended mainly for the farmer and his milkers to detect discoloured foremilk and therefore not to milk such cow into the bulk supply. Dye-marked remedies will benefit the farmer, the industry and the consumer.

All undyed intramammary products registered under Act 36 of 1947 must be off the shelf by January 1, 1977. Unfortunately this has led to a highpowered selling campaign by representatives to the farmer and offer of bulk purchase discounts. Many representatives are implying that dye marking holds no benefit for the farmer but is just an added burden to his already numerous farming problems.

Even after the introduction of dye-marked intramammary preparations, farmers may still use undyed stock remedies registered under Act 36 of 1947 as stock remedies for intramuscular administration, as udder infusions, and will also attempt to obtain undyed intramammary preparations from veterinarians.

Dye marking will not stop inhibitory substances being found in milk, as made obvious from the investigated sources of penicillin-contaminated milk, but it is a most welcome step in the right direction.

THE OBLIGATIONS AND RIGHTS OF THE VETERINARIAN UNDER THE MEDICINES AND RELATED SUBSTANCES CONTROL ACT, 1965 (ACT 101 OF 1965)

It has to be emphasized that Act 101 of 1965 (as amended), and as read with Government Notices No. R352 of 21 February 1975, R2244 of 28 November 1975 and R1188 of 9 July 1976, as well as the Hazardous Substances Act 1973 (Act 15 of 1973), must be read by all veterinarians who sell or prescribe veterinary substances other than those registered under the Stock Remedies Act (Act 36 of 1947)[3]. These acts were excellently summarized by Naudé in his address to the S.W.A. Branch of the S.A.V.A. in Windhoek on 13 October 1975[4].

It can be anticipated that veterinary practitioners and other veterinarians will be requested to prescribe or supply unmarked intramammary formulations and other unregistered parenteral or oral antibiotics to farmers. The profession has a clear obligation to emphasize the purpose and value of the dye-marked intramammary stock remedies. Under no circumstances should the profession permit the evasion of the use of dye-marked remedies without good cause.

The veterinarian's obligation also goes further. Firstly, no substances which are scheduled in terms of Act 101 of 1965 may be sold, used or prescribed by a veterinarian for treatment of animals unless they are his patients and under his care and control[5]. This means inter alia that no veterinarian may have an "open shop", nor that any lay member of the veterinarian's staff may sell such substances even to a bona fide client. Secondly, where a veterinarian does treat, sell or prescribe antibiotics for the treatment of animals under his care, he has a distinct obligation to ensure that the owner of the animals is made fully aware of and properly informed about the method of use of the substance, including the excretion period of the substance in the milk and necessary precautions. Should a milk producer be prosecuted or suffer loss from the presence of antibacterial residue in his milk supply, or should the processing dairy suffer financial loss due to the presence of antibiotic residue, there is little doubt that the veterinarian can be held responsible if he did not properly instruct the producer at the time of treatment, sale or prescription of the antibacterial substance.

Verbal communication of the necessary information could be denied or confused by the milk producer, and therefore it is advisable to hand written instructions to the milk producer. This could be done by the manufacturer supplying suitably worded leaflets with his product, or alternatively, the veterinarian could produce his own.

OTHER RESIDUAL SUBSTANCES IN MILK

So far investigations in Johannesburg have been confined to penicillin residues, and other antibiotics have not been specifically identified. One cannot but wonder about the incidence of other residues such as hormones used in synchronizing oestrus and treating anoestrus, the corticosteroids and anabolic steroids, tick dips and mange treatments, teat dips and udder washes, as well as other chemicals used for cleaning and disinfecting the milking machines and bulk tanks.

CONCLUSION

Antibiotic contamination of the milk supplies, which is a public health hazard as well as a source of financial loss to the dairy industry, must receive increased consideration by veterinary practitioners. This investigation indicates without doubt that there are members of the profession who can rightly be held responsible for a considerable degree of the contamination of public milk supplies.

The veterinarian presently enjoys the privilege of unrestricted use of substances scheduled in terms of the Medicines Control Act (Act 101 of 1965). This

privilege is enhanced by the recent restrictions, dye marking and strict regulations enforced on stock remedies in terms of Act 36 of 1947. Veterinarians must always safeguard their privilege with proper responsibility for their professional obligations.

ACKNOWLEDGEMENT

We wish to thank the Medical Officer of Health, Johannesburg, for permission to publish this paper.

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TO THE EDITOR — AAN DIE REDAKSIE

Dear Sir,

Ovarian Autograft as an alternative to oophorectomy in cats and dogs

Oophorectomised bitches, and to a lesser extent cats, show a variety of symptoms which are attributable to the loss of gonadal hormones. Urinary incontinence is rarely seen in bitches after oophorectomy, obesity more often. Skin atrophy, seen as seborrhoea, poor hair quality and alopecia is very common. The majority of pet owners state that their animals lose vitality after being spayed. The administration of oestrogens and progestagens by injection or per os produces a satisfactory reversal of these unwanted changes.

In an attempt to find a solution to this problem the writer has investigated the effectiveness of a method of autotransplantation of the ovaries to visceral peritoneum, a procedure based on experimental work done by numerous workers such as Knauer (1895), Biskind (1941), Lipschutz (1953) and many others in more recent years.

A variety of surgical methods were tried and evaluated over a three year period, in cats and bitches. A small group of dogs were treated in this way. The levels of oestradiol, progesterone and thyroxine were monitored over a period of one year and compared to levels measured in spayed and entire bitches. Occasional determinations of testosterone and cortisol levels were done. The viability of the grafts were established after a year by inspection and biopsy. During the entire period the dogs were observed and their performance as army guard dogs was assessed. In addition to this group, a number of household dogs and cats were also operated upon and the conclusions drawn from all these cases were considered.

The author is of the opinion that autotransplantation of the ovaries is a practical method of abolishing oestrus behaviour and yet avoiding the effects of hypogonadism. A fully substantiated report is being prepared for publication in this journal.

Yours faithfully,

P.H. le ROUX
Hermitage Terrace,
Richmond,
Johannesburg.

INFORMATION — INLIGTING

USING WASTE PAPER AS BEDDING FOR ANIMALS
The Dangers that can be involved

Waste paper has been used for many years as bedding and litter for domestic pets. More recently, shredded newspaper has been introduced and used successfully as bedding for farm livestock. Since animals, particularly young ones, often chew and ingest the bedding, it is most important to be sure that only non-poisonous material is used.

Clean, white paper and the black ink used in newspaper printing are not toxic to animals. In fact, since ruminants, cattle and sheep, are able to digest the cellulose in paper, trials have been carried out using newspapers as a source of roughage and energy in their diet. In the United States of America levels of up to 8 to 12 per cent of newspapers have been used in cattle rations with no significant loss in production.

Coloured inks used in producing coloured newspaper pages or magazines may, however, contain considerable amounts of lead and other metals in the pigments, and many types of glossy paper are produced by using heavy metals and substances toxic to both animal and plant life. The likelihood of acute lead poisoning in puppies or calves resulting solely from eating coloured paper is probably slight unless the animal regularly consumes large quantities of coloured newspaper and magazines. But this source of lead, added to other sources, may be sufficient to result in illness or poisoning and, with other metals, can either build up in, or retard the growth of, plants which are manured with the discarded bedding.

Newspaper may become contaminated by many different substances which could be toxic to animals between the time it leaves the printing press and the time the waste paper is salvaged to be used for bedding.

When shredded newspaper for bedding is produced commercially, care is taken to see that only clean, black-and-white newsprint is processed. If farmers are preparing or using paper for bedding domestic or farm livestock, they should make sure that they, too, use only clean black-and-white pages and discard any obviously soiled paper and all colour-printed news pages or magazines.

(Ministry of Agriculture, Fisheries and Food, London; August 1975)

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Pneumonia, Virus Pig Pneumonia (VPP)
Bacterial Pneumonia, Respiratory Infection
(resulting from staph., strep., pasteurella, bordatella)
Mycoplasma Arthritis, Infectious Arthritis
(resulting from staph., strep., erysipelas)
Navel Infection (baby pig infection)
Soft Tissue Abscesses (due to staph., strep.)

Diarrhœa, Scours, Vibrio
Bacterial Diarrhœa, Coliform Diarrhœa
Bloody Flux, Hæmorrhagic Dysentery
Enteritis, Bloody Scours, Salmonellosis
Bacterial Dysentery, White Scours, Black Scours
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Jl. S. Afr. vet. Ass. 47(3) 209-210 1976

ATTEMPTED ARTIFICIAL INFECTION OF IMPALA, BLUE WILDEBEEST, BUFFALO, KUDU, GIRAFFE AND WARTHOG WITH HEARTWATER

D.V. GRADWELL*, C.A.W.J. VAN NIEKERK* and D.C. JOUBERT*

ABSTRACT: Gradwell, D.V.; van Niekerk, C.A.W.J.; Joubert, D.C. **Attempted artificial infection of impala, blue wildebeest, buffalo, kudu, giraffe and warthog with heartwater.** *Journal South African Veterinary Association* (1976) **47** No. 3, 209-210 (En) Veterinary Investigations Center, Skukuza 1350, Republic of South Africa.

Intravenous injection of *Cowdria ruminantium* infected blood produced no signs of disease in four impala, *Aepyceros melampus;* three blue wildebeest, *Connochaetes taurinus;* a buffalo, *Syncerus caffer;* a kudu, *Tragelaphus strepsiceros;* a giraffe, *Giraffa camelopardalis* and a warthog, *Phacochoerus aethiopicus*, a control sheep injected with the same blood reacted severely and showed typical lesions of heartwater at autopsy.

INTRODUCTION

A number of antelope species are known to be susceptible to heartwater. Neitz[4] demonstrated *C. ruminantium* in jugular endothelial cells of springbuck *Antidorcas marsupialis* found dead on the Springbuck Flats and concluded that this antelope is possibly susceptible to heartwater. Young & Basson[8] reported typical clinical signs and lesions as well as the presence of *C. ruminantium* in brain endothelial cells of a 3 month old eland *Taurotragus oryx* calf from the Addo National Park. Neitz[5] also showed that the black wildebeest, *Connochaetes gnou* and blesbok, *Damaliscus dorcas* can act as asymptomatic carriers while Grosskopf[2] showed that eland from heartwater free areas challenged with infective sheep blood only showed a slight rise in temperature between the 12th and 17th day post-challenge. Hofmeyr[3] reported the susceptibility to heartwater of exotic species e.g. blackbuck, *Antilope cervicapra* and suspected cases (no autopsy performed) in a mouflon, *Ovis mouflon;* barbary wild sheep, *Ammotragus lervia* and fallow deer, *Dama dama*. Young (personal communication) also incriminated heartwater as the cause of death in the latter three species after typical heartwater symptoms were seen. De Vos[1] states that a variable innate resistance to heartwater seems to be present in indigenous antelopes in enzootic areas. With this natural resistance in mind it was decided to challenge a number of animals in a heartwater endemic area.

MATERIALS AND METHODS

Experimental animals: Four adult impala, three, three-year-old blue wildebeest and one each of the following species were used in the experiment; buffalo (3 years), kudu (16 months), giraffe (6 months) and warthog (17 months). The impala had been captured as adults and had been in the bomas for at least 3 years prior to the experiment. The blue wildebeest had been approximately 1 year old at the time of capture and had been in the bomas for more than 2 years. The buffalo was born in the bomas and reared by its mother, while the kudu was brought into our bomas at an age of about 4 days, the giraffe at an age of about 4 weeks and the warthog at an age of about 7 days, all of which were reared artificially on powdered milk until solid food was taken.

An old merino sheep which was also being held in our bomas with a number of other sheep was placed in isolation and used as a control.

Challenge material: Infective heartwater blood was obtained from the Veterinary Research Institute, Onderstepoort. The blood is taken from sheep at the height of the febrile reaction, following the inoculation of the Ball 3 strain of heartwater and is issued routinely for the immunisation of animals, especially calves, against the disease. The blood was kept frozen on dry ice until a few minutes before injection when it was placed in tap water to thaw. Injection took place within 48 hours of issue.

Challenge procedure: The impala, kudu, giraffe and sheep were caught manually without the use of capture drugs and the blood injected slowly intravenously via the jugular vein. The blue wildebeest, buffalo and warthog were darted using fentanyl and acetylpromazine or fentanyl and rompun combinations and when recumbent challenged intravenously via the recurrent tarsal, jugular and saphenous veins respectively, immediately followed by the antidote given intravenously. All the animals were on their feet within 3 minutes after challenge.

Rectal temperatures were taken at the time of challenge and daily for 35 days in the animals which could be handled without capture drugs. The animals that were too dangerous to handle without capture drugs were kept under close observation by the attendants as well as by the authors at least once a day. It was decided to dart them only if signs of disease were noticed for a thorough clinical examination.

RESULTS

At challenge the rectal temperatures of the four impala were 38,8°C, 38,8°C, 38,3°C and 39,2°C and at no stage during the 35 days thereafter did the temperatures rise above 39,2°C. No signs of illness were noticed at any stage. The kudu at challenge had a rectal temperature of 37,4°C and never went above 38,2°C at any stage. The temperature of the giraffe at challenge was 37,5°C and never rose higher.

The remainder of the animals showed no sign of inappetance or any other reaction at any stage.

* Veterinary Investigation Centre, Skukuza, Kruger National Park.

On day 9 after challenge the sheep showed inappetance, poor habitus and the rectal temperature rose from a mean of 38,5°C to 41,8°C and remained above 41°C until day 15 when it was slaughtered. Post-mortem examination revealed ascites, hydrothorax, hydropericard, tumor splenis and subepi- and subendocardial haemorrhages. Brain smears prepared by the method of Purchase[7] showed typical *Cowdria ruminantium* colonies in endothelial cells.

DISCUSSION

The higher temperatures recorded in the impala can be ascribed to severe struggling each time they were caught while the kudu and giraffe were tame enough to approach and take rectal temperatures without having to manually restrain them in any way.

The control sheep was kept under the same conditions in the bomas as the other animals which were separated by netting or treated pole fences. If the challenged animals had developed an immunity as a result of *C. ruminantium* infected ticks entering the bomas, the sheep should also have been infected. The sheep however was fully susceptible to heartwater. An active immunity in the captive wild animals is therefore unlikely unless ticks showed a preference for the wild animals above the sheep. This possibility can not be excluded with certainty in this experiment. The giraffe at 6 months of age was the youngest animal but probably too old to show the natural resistance observed in young domestic animals. The possibility that the animals had a residual immunity acquired before capture must also be borne in mind as Neitz[6] has shown that sheep remain immune for 4 years under laboratory conditions free from ticks. This possibility can only be eliminated in the buffalo which was born in captivity. The impala had been in captivity for over 3 years, blue wildebeest for just over 2 years and the warthog, kudu and giraffe for less, so they may still have had a residual immunity.

The reaction in the control sheep proved that the challenge material used contained viable organisms.

CONCLUSION

No reaction to infective heartwater blood could be produced in impala, blue wildebeest, buffalo, kudu, warthog or giraffe. From this experiment it would appear that the buffalo may have had an innate natural resistance but the other animals may still have had an acquired immunity resulting from infection prior to capture. In the impala and blue wildebeest this immunity must have been present for at least 3 and 2 years respectively without restimulation.

Very little trouble from heartwater can therefore be expected in these species in areas where heartwater may only occasionally occur during favourable climatic conditions.

ACKNOWLEDGEMENTS

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DOURINE IN SWAZILAND

Dear Sir,

I am writing with reference to the very interesting article on the prevalence of Dourine in Southern Africa in the June issue of your Journal. The Table 1 indicates that two cases occurred in Swaziland in 1971. The facts are that the animals concerned, both mares, were illegally imported and had been in the country less than one month when blood samples were taken which proved positive. The mares, together with a foal at foot, were destroyed. Ten mares which had had over the fence contact with one of the infected animals were blood sampled at monthly intervals for six months, all results being negative.

There are about 2 000 horses and 16 000 other equines in Swaziland and all entire mares and stallions exported to the Republic and elsewhere are subjected to the Dourine test performed at Onderstepoort. The consistently negative results together with an absence of observation of clinical symptoms suggest that the country is normally free of this disease.

Yours faithfully

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Mbabane, Swaziland.

Jl. S. Afr. vet. Ass. 47(3) 211-213 1976

THE YELLOW MONGOOSE *(CYNICTIS PENICILLATA)* AS A LATENT FOCUS OF RABIES IN SOUTH AFRICA

I.F. ZUMPT*

ABSTRACT: Zumpt, I.F. **The Yellow Mongoose** *(Cynictis Penicillata)* **as a latent focus of rabies in South Africa.** *Journal of the South African Veterinary Association* (1976) **47** No. 3 211-213 (En) Reg. Vet. Lab., P/Bag X5020, Stellenbosch 7600, South Africa.

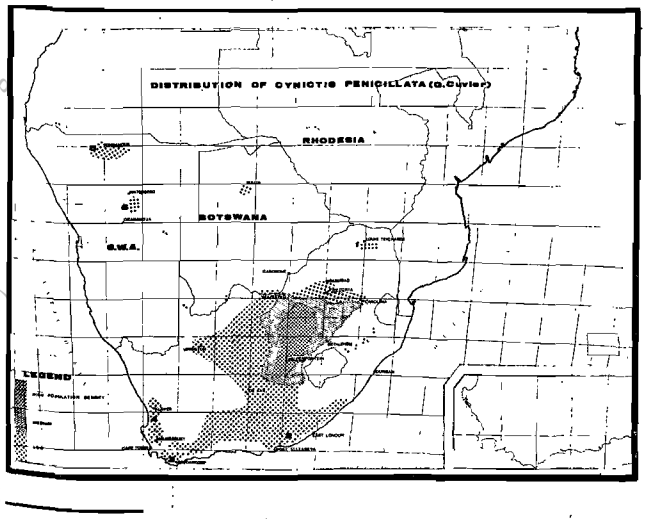
The role of *C. penicillata* as a latent focus of rabies in South Africa is discussed. A description of the colony life, feeding and breeding habits and the relationship of the Yellow Mongoose to the Suricate Meerkat and Ground Squirrel is given. Observations on the epidemiology and symptomatology of rabies in the Yellow Mongoose are reported and various methods of control are suggested.

INTRODUCTION

Since the first reported outbreak of rabies in South Africa was recorded in 1892[1,7], research into the epizootiology of the disease has been sporadic. The contributions by du Toit[1,2] and Snyman[1,5,6], however, have not been surpassed yet, and our present knowledge is mainly based on these findings.

Despite increased knowledge of the epidemiology and control of rabies, this world-wide epidemic appears to be on the increase in wildlife. In countries which had successfully eradicated the disease, wild animal movements have again disseminated it. For example, as a result of fox movements from Germany, rabies reappeared in Belgium, Luxemburg and France after an absence of some 36 years. In this paper the role of *Cynictis penicillata* as a latent focus of rabies in South Africa will be discussed, as it has become obvious that in spite of extensive projects little progress has been made in the eradication of the disease.

From 1964 to 1970, *C. penicillata,* together with *Suricata suricatta* Erxl (Suricate Meerkat) and *Xerus inauris* Zimmermann (Ground Squirrel), which live in close association with one another, were studied in the North Western Cape, Western Transvaal and Orange Free State. This paper is a summary of previously-published material[8, 9, 10, 11], field observations and field experiments carried out by the Zoological Unit No. 5 under my supervision. Only those aspects which might play an important role in the epidemiology of rabies will be discussed.



* Regional Veterinary Laboratory, P/Bag X5020, Stellenbosch.
Paper presented at the Biennial National Veterinary Congress, S.A.V.A. Durban, September 1975.

The Yellow Mongoose is widely distributed in South Africa (plate), but occurs in greatest numbers in the western and central Orange Free State and Western Transvaal[4,5]. This animal is diurnal, rarely nocturnal. Its colour is uniform, varying only in reddish or sandy shades, with a characteristic white tail tip. The average head and body length varies from 320 to 380 mm with a tail length of 180 to 250 mm. In many parts of the Republic it occurs in close association with the Ground Squirrel and Suricate Meerkat. In other areas the Yellow Mongoose, due to the absence of the squirrel, has different living habits and is thus a lesser danger as far as the dissemination of rabies is concerned.

Its daily activities are preoccupied with feeding and are largely dependent on ambient temperatures. The Yellow Mongoose can be seen regularly on warm evenings, but during winter will only become active after sunrise and return to its burrow in the late afternoon.

A typical colony or burrow varies from one or more single holes up to composite complex systems with forty or more entrance holes. In the study area an average of 22 entrance holes was established. The size of such a colony also varies, depending on type of soil and terrain, but usually does not extend below one metre with surface measurements of 7 x 7 metres.

In close proximity to such a colony, one or more neat "latrines" are situated, often with a urination-stone in the centre. These and the entrance holes can give a fair indication of activity of such a colony.

In the Lichtenburg-Delareyville study area comprising 142 475 morgen, an average of 0,14 colonies per morgen was established with an average of 3,9 mongooses per active colony. These figures were compiled from data of 362 carefully selected and opened colonies.

Feed Analyses[9] of 95 stomach specimens revealed the following: remnants of mice, locusts, termites, beetles, meat from unidentified sources, lizards, remnants of small birds, young Yellow Mongooses and, probably, dead Ground Squirrels. These animals are also often seen scavenging at picnic spots, rubbish heaps and animals killed on roads.

The **Home Range,** or that area over which Cynictis normally travels during the course of its daily routine, is not only influenced by the availability of food, but also by the proximity of other burrows, the presence of rivers, roads or railways. The home ranges have fluid borders and overlap with others; they vary from 600 to 3 000 metres in radius from a colony.

The **territory** or defended area of the Yellow

Mongoose is that area where the male competes for real estate, and not for a female as previously thought. Victory usually goes not to the stronger, but to the righteous, that is the owner of that territory. A territory is thus an area of space which an animal guards as its exclusive possession and which it will defend against all members of its own kind; this latter aspect will become of importance once rabies is involved.

The **breeding season** of *C. penicillata*[11] is fairly sharply demarcated, but varies slightly in different areas. In the North-Western Cape and Western Transvaal, the first signs such as love-play, inhibited biting and mock-fighting can be observed as early as August. This is also the time when the young of the previous season are gradually made to leave the home burrow, search for mates and for their own territories.

In a pregnancy study[11] it was found that the pregnancy period which starts as early as June has its peak during October and sharply tapers off to end in January. In this study no pregnant female was found suckling a litter, and it is doubtful whether more than one litter per year is born.

The gestation period is approximately 42 days; one to four young are born, of which only one or two are weaned at 10 weeks of age. These remain with their parents until shortly before the following litter is born. In a study of 41 litters, each was composed of one sex only, and although this is a small number, it does indicate at least that the greatest majority of litters are unisex. This again may play an important role in the epidemiology of rabies. At birth, the ♂ to ♀ ratio was found to be 1,6:1, whereas in adults this changed to 1,2:1.

Although litters are made to seek new territories at the end of their first year of life, no pregnant females were found in this age group. In captivity, the lifespan has been as high as 13 years or more. In a field survey[11], however, it was found that very few exceed the age of four years, the majority only reaching the age of one to two years.

The relationship of *C. penicillata* with *X. inauris* and *S. suricatta* is of interest. The Ground Squirrel is the architect and builder of the burrow systems. The Yellow Mongoose is a very poor burrower, but in his turn a vicious fighter and defender of a colony against danger (such as snakes). Although the mongoose is also very alert, the squirrel is the actual sentry, emitting a shrill whistle with approaching danger, such as humans, dogs or birds of prey. This symbiotic relationship is harmonious. Both species inhabit the same colony, but they have separate living areas within such a system.

The Suricate Meerkat is also found in the same areas as those mentioned above, but, as they usually are found in large groups numbering up to twenty to more, the inter-relationship is different. When such a group of Suricates invades a colony, they take it over and expel all other animals. They will remain in such a colony for a few days, deplete available food supplies and then move on to the next colony. The expelled mongooses and squirrels hide out in "escape-holes" or other vacated burrows until the Suricates have moved on. No actual fighting or biting was observed in any of these "take-overs".

Very little is known about the behaviour and habits of meerkats infected clinically with rabies and, for obvious reasons, no recorded experiments have been carried out in this country.

Animals known to be infected, whether showing clinical symptoms or not, lose their natural fear of man, may enter homesteads, gardens, kraals and other places usually avoided, and so become objects of curiosity to man and animal and bite on being touched or handled. Affected animals may appear tame, refuse to be chased away and can easily be caught or killed by dog or man. They may show various degrees of paralysis, the latter often characterised by incoordination or lameness of the hindquarters.

The concept that rabies survives in silent epidemics in maintenance hosts is becoming increasingly accepted[3].

In the epidemiology of rabies the fact that a litter of young *C. penicillata* has to move out of its parent territory during the mating season (e.g. August), but not later than when the peak of litters are born (e.g. October), is of great importance. If the greater majority of litters, or even all, are composed of one sex per litter, the distribution of animals will be great, as brother-sister-matings are thus impossible. During this period territory-defence is at its maximum, the search for a mate and the establishment of their own territory is vital as is the dwindling food supply from late winter to early spring in the study area. These and other stresses may trigger off clinical rabies in a mongoose population. Infected animals behave differently to non-infected ones, move over greater distances and thus come into contact with a considerable number of animals in established territories.

The ensuing fighting, in this case not inhibited, results in numerous wounds which are essential for the spread of rabies to susceptible populations. To what extent aerosol infection is involved is as yet not known.

Twenty-one colonies, where suspected rabid meerkats had entered, were opened within days to weeks afterwards. A number of reasonably fresh carcasses were found to be positive for rabies, and numerous remains of meerkats were collected. Only 23 live *C. penicillata* or 25,5% in the study area were counted in these 21 colonies. It thus appears that some animals may survive, whether these might have become latent carriers, or were refractory to rabies, or had had no contact with the invading rabid animal was not investigated.

CONTROL

Control measures, often controversial, should be applied in the light of the findings of modern biology. We need a critical re-examination of many of our attitudes towards animal control, as we often do not control, but only reduce the population, just to stimulate the increase in reproduction and survival in turn[3]. In the experimental areas where a complete and thorough eradication had been carried out, it was found that re-invasion and population shifts occurred almost immediately. Abnormally large litters were noticed and seemed to survive without great difficulty, and within three years the original population density was regained.

Experiments were conducted using various methods of extermination[5,6,7,8]. Very few animals were caught in a variety of live- and spring-traps, and only the occasional poisoned bait was removed from experimental areas by some animal or another. Besides the ineffectiveness of baits, the danger to domestic and other animals is a real one. Shooting, the use of

repellents, ploughing the infested areas and other methods studied were all expensive and only effective for a short period.

Poisonous gases, although efficient, are never very selective and often hazardous. Phosphine[8], cyanide gas, carbon dioxide and carbon monoxide were tested over a long time and over large areas. When proper extermination teams exist, the use of CO-gas, produced by petrol driven coal burners, is effective, cheap and reasonably safe to the operators. CO_2 was found to be an expensive and ineffective method, whereas phosphine and cyanide gas are not only expensive, but also dangerous if not handled correctly.

The question arises once again: Is the so-called total eradication beneficial in the control of rabies, is it economically justifiable, is it epidemiologically a sound practice? If not, what other lines of approach are there with our present knowledge? Some work on the aerosol immunisation of rabies carriers has been done[3], but seems to be impracticable at this stage. There may be hope in this direction, but until success is obtained an effective education- and extension-system should be adopted. Man, and especially children, should be taught not to touch or handle meerkats or any other rabies-suspicious animals. Endangered humans, dogs, stray cats and cattle (where warranted) should be vaccinated. Above all, funds and personnel should be made available to study all aspects of rabies epidemiology and control.

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BOOK REVIEW — BOEKRESENSIE

DERMATOPHILUS INFECTION IN ANIMALS AND MAN

EDITORS: D.H. LLOYD and K.C. SELLERS ACADEMIC PRESS. LONDON 1976

pp XVIII 322, Figs. 44, Tabs 44, Publ. Price £7,00

This book contains the Proceedings of a Symposium on *Dermatophilus congolensis* infection held in Nigeria. The papers presented deal very adequately with the epidemiology, clinical symptoms, pathology, bacteriology and immune response in domestic animals. Lesions and pathology of the infection in non-human primates and man are also discussed. It is felt, however, that more could have been said about the disease in sheep and horses.

The book is printed in type-written format and details of the degree of magnifications of the photomicrographs are not provided; even though an errata corrects this omission, it remains regrettable.

There is no mention of when the Symposium was held and readers are therefore left in some doubt about how up to date the work really is.

The book could nevertheless be said to be very useful as a reference work for veterinary students, veterinarians and bacteriologists.

A.L.L.

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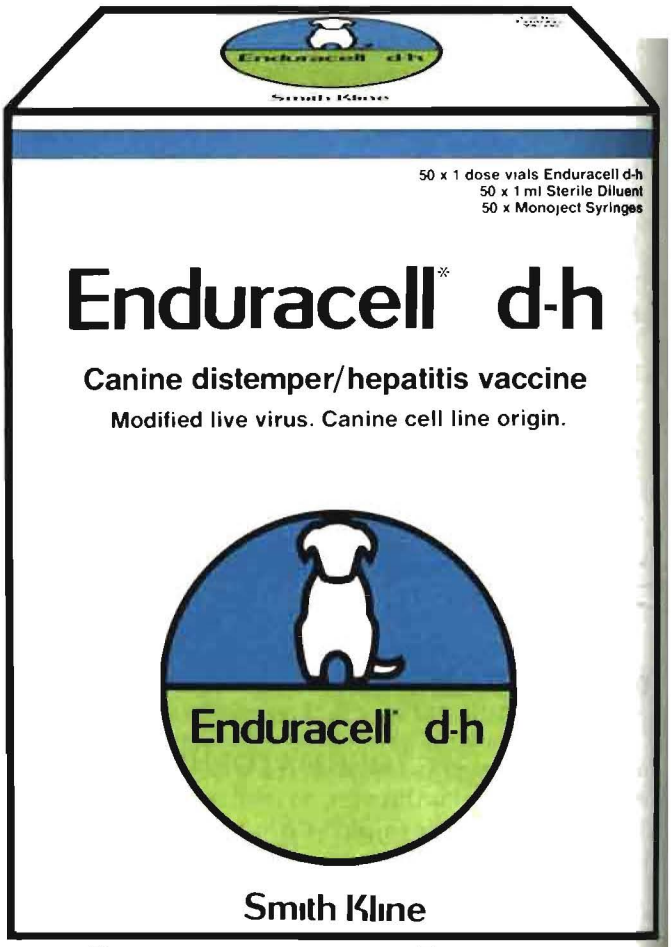

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Jl. S. Afr. vet. Ass. 47(3) 215-218 1976

THE INCIDENCE OF EPISTAXIS IN RACEHORSES IN SOUTH AFRICA

G. PFAFF*

ABSTRACT: Pfaff, G. **The Incidence of Epistaxis in Racehorses in South Africa.** *Journal South African Veterinary Association* (1976) **47** No. 3 215-218 (En) P.O. Umtentweni 4235, Republic of South Africa.

In South Africa 2,41% of horses bleed in a race. If all raced until they were 7 years old probably another 2,12% would bleed. Many others bleed after the race or during exercise or at rest. The incidence of epistaxis is significantly greater in geldings than in females and uncastrated males ($P<0,001$). There is an age distribution of bleeding which is highest at 4 years and lowest at 2 years of age ($P<0,001$).

There is no evidence that epistaxis occurring in a race in South Africa is the result of, or is associated with, disease of the lungs. "Softness" of the thoroughbred might be a causal factor. In many cases genetic factors seem to play an important part. Epistaxis is more prevalent at coastal than at inland racing centres ($P<0,001$).

INTRODUCTION

INCIDENCE OF EPISTAXIS

(a) Australia and Malaysia:

The meagre information on the incidence of epistaxis is reviewed in an excellent and exhaustive article by Cook[1]; his figures are based on two publications and three personal communications. According to Cook[1], Bourke stated that in Victoria, Australia, 0,3% of flat-racers and 13,0% of jumpers bled; Bourke estimated that for the period 1969-1973 the incidence on the race-course amongst horses of all ages in Victoria was 0,8%. Choy, quoted by Cook[1], stated that in Singapore and Malaysia from 1970 to 1972, 2,5% of the 800 flat-racers bled; these 800 were mostly Australian horses more than 3 years old. Presumably the figures of Bourke and Choy refer to bleeding during or within minutes after a flat-race.

(b) England:

The English Jockey Club takes no action in connection with epistaxis; bleeders in that country are not reported to, nor recorded by, any Racing Authority, and consequently nothing is known of the incidence of epistaxis in England.

In 1913 Robertson[2] stated that in England "only one runner out of every 400 each year, on the average, breaks a blood-vessel in public". He did not give the source of his information.

Cook[1] analysed in great detail 174 bleeders referred to him in the 10 years 1963-1973; 124 bled while at rest and 50 after "competitive exercise". These 50 ranged in age from 1 to 13 years; the majority were aged 6 to 9 years. 36 of the 50 were race-horses and of these 44% bled during the race or exercise, and 56% after the race, the average interval after the race being 47 minutes. Cook[1] states that "when blood appeared after the race it usually coincided with the moment when the horse lowered its head to the ground" — thus supporting his contention that the blood comes from the lungs. Of the 36 racehorses that bled, only four were flat-racers. Of the 50 bleeders, 39 were geldings, but "as there are more male horses in training than female, these figures do not indicate any predisposition based on sex".

(c) South Africa:

Pfaff[4] recorded that in the 2 years 1948-1949, 49 (1,2%) of 4015 horses bled during a race.

CONTROL OF RACING IN SOUTH AFRICA

Racing in South Africa and Rhodesia is under the control of the Jockey Club of South Africa. All runners are examined by the Veterinary Officials before the race and again immediately after the race while being off-saddled in the parade ring; they are released from observation at the very latest 10 minutes after the race. The Officials report any bleeders and these are suspended from racing, the suspension being notified in the Weekly Racing Calendar: the period of suspension depends on the severity of the haemorrhage and any previous suspension for the same reason.

An initial mild bleeding would earn 1 month's suspension. For a severe initial bleeding, the suspension might be up to 3 months, and for a second haemorrhage the period would be 3 to 6 months. A horse that bleeds for the third time would be suspended permanently from racing. A horse that bleeds at rest or at exercise would also be suspended, but trainers, knowing this, do not report bleedings. Very few horses are kept in training after a second bleeding: owners and trainers prefer not to persevere with a horse which has been suspended for 3 to 6 months and which they know, will be permanently suspended the next time he bleeds.

The Racing Calendar of the Jockey Club, known as "The Annual Volume", gives details of all races run during the year; it also contains an "Index to the performances of horses" and this index gives the name, age, sex and breeding of every horse that raced during the year.

The analyses which follow are based on these two Jockey Club publications.

RESULTS

From January 1962 to December 1975 the Jockey Club of South Africa suspended 404 horses for bleeding. Of these 352 bled once, 45 bled twice and six bled three times. So there were in all 460 suspensions.

Footnote: In this article "racehorse" refers to thoroughbreds and "racing in South Africa" to flat-racing; for convenience a horse that bleeds from the nose is called a "bleeder" and epistaxis is often referred to as "bleeding".

Thoroughbreds foaled South of the Equator take their age from the 1st August, so that a horse foaled in December 1974 officially becomes one year old on 1st August 1975. In this article the age used is the "official" and not the "actual" age.

A colt is an uncastrated male under five years of age, and a horse is an uncastrated male five years and over. A filly is a female under five years of age and a mare is a female five years and over.

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In addition, during the last 2 years, four deaths from bleeding came to my notice — one during a race and three at exercise. This list of bleeders is incomplete because it seems that in the earlier years bleeding received scant attention in Rhodesia and Port Elizabeth. In Rhodesia no bleeders were reported until 1970, and until 1967 few were reported from Port Elizabeth.

An analysis of the 404 horses that bled 460 times is as follows:—

Sex: Of the 460 bleedings, 341 were in males (102 colts, 31 horses and 208 geldings); and 119 were in females (106 fillies and 13 mares).

Age: The ages at which the 460 bleedings took place: 41 at 2 years; 118 at 3 years; 142 at 4 years; 79 at 5 years; 48 at 6 years; 32 at 7 years and over.

Breeding: The 404 bleeders were the produce of 181 sires and 369 mares:—

94 horses each sired one bleeder
41 horses each sired two bleeders
17 horses each sired three bleeders
11 horses each sired four bleeders
2 horses each sired five bleeders
7 horses each sired six bleeders
4 horses each sired seven bleeders
1 horse sired eight bleeders
2 horses each sired nine bleeders
1 horse sired twelve bleeders
1 horse sired fifteen bleeders

Of the 369 mares, 338 each produced one bleeder; 29 each produced two, one produced three and one produced five.

Geographical: Of the 460 bleedings, 139 were in Durban, 113 in Port Elizabeth, and 67 in Cape Town; these three centres are at sea level. In Johannesburg, at an altitude of some 5400 feet, there were 116 and in Rhodesia 25.

It is only in Johannesburg that there is a marked difference between summer and winter temperatures. In Durban the climate verges on the subtropical and from December to March it is trying for both man and beast.

In 1975 the approximate number of horses in each centre was: Johannesburg 1800, Durban 1100, Cape Town 800, Port Elizabeth 700 and Rhodesia 600. There are now four race-courses in Johannesburg (including the one near Vereeniging); three in Durban (including the one in Pietermaritzburg); two in Cape Town, two in Port Elizabeth and two in Rhodesia. To count the number of race-meetings and the number of runners at each meeting over 13 years, would be a tedious and laborious business, but considering the number of meetings allotted by the Jockey Club to each Race Club each year, and the size of the fields at each centre, it is fair to say that during the period 1962 to 1975 in Johannesburg there was 50% more racing than in Durban, while Durban had 40% more racing than Cape Town, Port Elizabeth and Rhodesia.

Seasonal: The 460 bleedings represent an average of 38 per month. The actual figures were:—

38 in January	47 in July
22 in February	42 in August
32 in March	32 in September
46 in April	45 in October
40 in May	36 in November
48 in June	32 in December

An x^2 analysis of the above showed significance at $P<0,05$. This suggests that seasonal differences probably exert an effect on the incidence of epistaxis.

The severity of bleeding: This can to some extent be gauged by the period of suspension, summarised in Table 1.

Table 1: DURATION OF SUSPENSION OF HORSES DUE TO EPISTAXIS.

Horses suspended	Period of Suspension			
	3 months	6 months	More than 6 months	Total number of suspensions
Geldings	84	26	9	208
Colts	45	6	2	102
Horses	13	1	5	31
Fillies	44	7	2	106
Mares	7	1	0	13

These figures suggest that in addition to geographical and seasonal differences, significant sex and age differences might exist among racehorses in South Africa, suspended because of epistaxis. It appears that:—

1. Geldings are more likely to bleed than horses and mares.
2. The tendency to bleed increases up to the age of 5 years — but four and five-year-olds probably do more racing than horses of other ages.
3. Bleeding is more likely to occur at the Coast than at an altitude of 5400 feet.
4. The worst months for bleeding are the cooler months of April to July — but these are also the months when most racing takes place.

To get more accurate information on the incidence of epistaxis, particularly in regard to age and sex, a more detailed analysis was done. All two-year-olds of the six seasons from 1962 were followed through to the end of their racing careers, that is to the 31st July 1973. The result of this analysis is shown in Table 2. More horses raced as three-year-olds than at any other age, and the 5292 that raced as three-year-olds is regarded as the total number of horses involved. This is an approximation because a few would have raced as two-year-olds and not again, and a very few might not have started until they were four-years old, but 5292 must be very nearly the correct number of horses.

The total number of fillies and mares was 2318 and if the total number of geldings is taken as 1210 (the largest number at any age) then the total number of colts and horses was 5292 minus 2318 and 1210 = 1764. On this basis, when subject to an x^2 test, the figures in Table 2 show that:—

1. The incidence of epistaxis in geldings is significantly higher than in colts, horses, fillies and mares.
2. In all sexes, the tendency to bleed increases up to the age of 4 years ($P<0,001$). The incidence is lowest at 2 years of age ($P<0,001$).
3. 2,41% of all horses at all ages bled.

The percentage of bleeders at each age, tempts one to speculate on what might have happened had all raced until the age of 7 years.

Had they done so, and if among those that did not race after they were three-year-olds, there was the same percentage of bleeders as among those that did race, there might have been the following additional bleeders:—

As 4 year olds	1,1% of 1568	= 16
As 5 year olds	1,05% of 2820	= 29
As 6 year olds	0,82% of 3954	= 32
As over 6 year olds	0,79% of 4536	= 35
		112

On this basis, if all had raced until they were seven-year-olds, the total number of bleeders would probably have been 128 + 112 = 240 out of 5292 = 4,53%.

either a recent history of nasal catarrh or discharged blood-stained mucus at exercise;" "70% exhibited an occasional cough." In age they ranged from 1 to 13 years "but the majority were 6 to 9 years old." 32 were engaged in hurdle, steeplechase, and Point-to-Point races; seven were hunters and three showjumpers.

All the South African bleeders recorded in this article were apparently healthy with no history of recent respiratory disease. While suspended from racing they continued in training and again raced soon after the suspension was lifted. 352 bled only once (though had

Table 2: BLEEDERS IN THE SIX CROPS FROM 1960 to 1965, WHICH RACED FROM AUGUST 1962 TO JULY 1973 i.e. UNTIL THE 1965 CROP WERE SEVEN-YEAR-OLDS.

	Geldings			Colts and Horses			Fillies and Mares			Total		
Age	Number	Bleeders	%	Number	Bleeders	%	Number	Bleeders	%	Number	Bleeders	%
2	322	2	0,62	2 414	6	0,24	2 318	4	0,17	5 054	12	0,23
3	1 088	7	0,64	1 932	8	0,41	2 272	17	0,74	5 292	32	0,60
4	1 210	20	1,65	984	11	1,11	1 530	10	0,65	3 724	41	1,10
5	1 092	20	1,83	618	2	0,32	762	4	0,55	2 472	26	1,05
6	792	6	0,75	342	4	1,17	204	1	0,49	1 338	11	0,82
Over 6	516	5	0,96	186	1	0,53	54	0	0	756	6	0,79
Total	1 210	60	4,96	1 764	32	1,81	2 318	36	1,55	5 292	128	2,41
Mean	Annual %		1,09			0,64			0,52			0,76

NOTE: The total number of horses involved is taken as 5292, i.e. the number that raced as three-year-olds.

DISCUSSION

These analyses show that in the 13 years from 1962 to 1975, 404 horses bled, and of the six crops of 5292 that commenced racing in August 1962, 128 or 2,41% bled. It must be emphasized that these are the numbers suspended for bleeding in a race: horses that bled during a race but were not detected, and those that bled after a race, or at work, or at rest, are not included. There is good reason to believe that many did in fact bleed: in the parade ring attendants have been noticed trying to conceal bleeding, and I know of many horses that bled at work or at rest that were not reported, and so not suspended.

One trainer told me "I don't mind my horse bleeding at work on Tuesday because then I know he will not bleed in his race on Saturday." Another trainer, speaking of a very successful sire, now dead, remarked "the trouble with him was that so many of his progeny bled at work". Very recently a horse pulled up normally after a race, but the following night bled profusely. Three weeks later he again bled profusely during the night. Within 8 weeks after the second haemorrhage he won two races. Also very recently a horse bled profusely at work. Three weeks later he won at odds of 3 to 1 on — an indication of the trainer's opinion of epistaxis!

The large number of bleeders in Port Elizabeth is puzzling, especially as there were so few reported before 1967. The explanation might be that horses not good enough for the main centres gravitate to Port Elizabeth, and horses that bleed at work in other centres might be quietly disposed of to Port Elizabeth.

From his study of 50 horses which broke bloodvessels at exercise, Cook[1] concludes that these horses were bleeding from the lungs, and that "their problem was associated with a pre-existing pulmonary disease — possibly broncho spasm linked with the early stages of chonic bronchitis and pulmonary emphysema". 52% of his horses "had they all continued to race some might have bled again), whereas Cook's 50 bled "from one to twenty times, but the average was five". Only four of Cook's horses raced on the flat, whereas all the 404 South African bleeders were confined to flat races. Moreover many South African horses, not included in this survey, won within a few weeks or even days of bleeding profusely at work. Obviously Cook[1] was dealing with a population of horses markedly different from that considered in this survey.

That geldings are twice as likely to bleed as females and uncastrated males is puzzling; it might shed some light on the cause and treatment of the condition, but it does not support the view that bleeding during a race is associated with pulmonary disease. Chronic pulmonary disease is known to be uncommon in horses of 2 to 5 years of age.

Of the four horses that died in 1974-1975, two — a four-year-old colt from Durban and a three-year-old New Zealand filly — died in Cape Town; the other two, both six-year-old horses, died in Durban. One six-year old horse died during a race; the other three died after slow work. All were reported to have ruptured bloodvessels in the thoracic cavity. The lungs were not examined histologically, and consequently no conclusions can be drawn regarding the presence or absence of pulmonary disease. The colt and the two horses were among the thirty best in the country.

Cook states that "the possibility that bleeding may be a factor influencing the occurrence of epistaxis is not denied". "Softness", or increased susceptibility, resulting from in-breeding and the strain of racing when immature, may well explain the widespread nature of epistaxis in South Africa. For 40 years I have been hearing older trainers bewailing the fact that "horses are getting soft". Some 75 years ago Mat Dawson, the celebrated trainer, remarked to Lambton[2] that "horses for years have had too great a strain put on them in their two and three-year-old days, con-

sequentlv every succeeding generation becomes less robust".

In 1948 Persse[3] wrote, "It seems to be the general experience nowadays to find horses nothing like so robust as they were 50 years ago; and for this I blame the long-continued practice of in-breeding."

The excessive use of vitamins, hormones and other "tonics" might explain why certain trainers have so many bleeders.

In 1913 Robertson[5] affirmed that the tendency to break bloodvessels is inherited as a recessive character, but that a horse is not likely to break a bloodvessel unless he carries the factor in a double dose.

The number of bleeders that trace to certain mares and to certain sires suggests that genetic factors might play an important part in epistaxis. Briefly the evidence is:—

On the female side, 31 mares each produced more than one bleeder. The mare Miracle, by Jubic out of Hay Presto by Marcius, produced five bleeders. Tropic Shore, by Marcius out of Cocoa by Double Up II, produced three bleeders. Muscovite by Fairthorn out of Muscovy was a bleeder, and his half sister Diza, by Herculaneum out of Muscovy, produced two bleeders. The imported mare Nestona, by Nearco out of Keystone by Umidwar, produced Babble by Dialogue, and Near Truth by True Cavalier. Babble was a bleeder and she produced Indaba and Bridge Drive, both bleeders. Near Truth produced Soho, a bleeder.

On the male side, of the 310 bleeders by horses that sired more than one bleeder each, 45% trace back to Isonomy: 9,5% were by sons of Persian Gulf, 11,6% by sons of Nearco, and 24% by sons and grandsons of Fair Trial. Nearco and Fair Trial were grandsons of Phalaris, who traces back to Isonomy through the mare Arcadia. So of these 310 bleeders 35,6% trace back to Arcadia. Arcadia was by Isonomy out of Distant Shore by Hermit. Gallinule was by Isonomy out of Moorhen by Hermit. Phalaris and Humorist were both by Polymelus. Gallinule and Hermit were celebrated bleeders and Humorist bled to death 18 days after winning the Derby. Robertson referred to Humorist as "the bloodvessel breaker", and wrote that "so far as soundness in the thoroughbred was concerned, his death was the best ending".

These very brief remarks on the breeding of bleeders in South Africa suggest that heredity might be an important factor in epistaxis, and are a reminder of Robertson's words: "Hermit, with his grandson Gallinule, despite their undoubted claims to rank as pillars of the Stud Book, are very largely responsible for the incidence of bloodvessel breaking".

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BOOK REVIEW

BOOKRESENSIE

THE VETERINARY ANNUAL

EDITED BY C.S.G. GRUNSELL and F.W.G. HILL. SIXTEENTH ISSUE.

WRIGHT-SCIENTECHNICA, BRISTOL 1976.

pp XIV ± 317. Figs. 99. Tabs 27. R10,50.

The sixteenth issue of this well-known publication, while not as bulky as its immediate predecessor of last year, nevertheless contains an excellent assortment of papers on a great diversity of subjects. These papers are contributed by 56 authors with special knowledge of the fields they are writing about, as judged by the frequent references to their own work. There are articles on all species and preventive medicine, in its broadest sense, is well to the fore. The editors mention their objective "to give some overall coverage of advances in the various aspects of small animal veterinary science over a timespan of 5-6 years". In this issue there are 11 papers in this category, including the following subjects: bone tumours in the dog, skull fractures, Canine middle ear disease, a review of surgical repair methods in Canine hip dislocation, treatment of radius-ulna growth disturbances, patterns of pulmonary disease on thoracic radiographs, Canine bladder diseases and oestrus control in bitches and queens.

Articles on farm animal problems include the following: 2 papers on dairy cattle preventive medicine, colibacillosis in calves, viral diarrhoea in calves, thrombosis of the posterior vena cava in cattle, perinatal lamb mortality, arthritis in the pig, nervous disorders in the pig, equine abortion and the acquired resistance of horse strongyles to anthelmintics.

The general and review articles include subjects such as anaesthetic emergencies, influence of heat stress on the developing fetus ("hyperthermia acts as a typical teratogen"), veterinary attention for reptiles, animal production and grass, reproduction and infertility, helminthology and anthelmintics and the current situation on the significance of Mycoplasma infections in cattle.

There is an enormous amount of diverse and up-to-date information available in this book, and it is unhesitatingly recommended to all who are anxious to remain well-informed on the latest advances.

R.K.L.

Jl. S. Afr. vet. Ass. 47(3) 219-222 1976

PARENTROVITE AS A SUPPORTIVE THERAPY FOR LOCOMOTORY STRESS IN TSESSEBE

A.M. HARTHOORN AND LYNDA M. HARTHOORN*

ABSTRACT: Harthoorn, A.M.; Harthoorn, Lynda M. **Parentrovite as a supportive therapy for locomotory stress in tsessebe** (1976). *Journal South African Veterinary Association* (1976) **47** No. 3. 219-222 (En). Nature Conservation Division P. Bag X209, 0001 Pretoria. Republic of South Africa.

Parentrovite was administered to tsessebe as supportive therapy against artificially induced locomotory stress. An attempt was made to judge the efficacy of this treatment from enzyme levels such as that of LDH. Other parameters such as systemic blood pressure and the ECG were also recorded. No significant differences were established between treated and untreated animals although LDH levels were considerably higher in the untreated than in the treated group. The small numbers of tsessebe available and the artificial nature of the exercise induced made an evaluation of the effectiveness of Parentrovite in the treatment of locomotory or of capture stress uncertain.

INTRODUCTION

Deaths in wild animals occurring after capture are recognised as a major obstacle to certain conservation practices such as the redistribution of diminished species.

A high proportion of deaths during the capture of the Hunter's hartebeest *Damaliscus hunteri* resulted in the investigation that first documented the occurrence of capture myopathy in wild animals[5]. Treatments on the whole have been ineffective and Young[6] stated that prophylactic and subsequent symptomatic treatment of capture myopathy with various medicaments, including vitamin E and selenium containing preparations and/or vitamin B_{12}, calcium borogluconate, systemic antibiotics, detoxicants, corticoids and antihistamines, proved to be ineffective.

MATERIALS AND METHODS

Six tsessebe bulls *Damaliscus lunatus,* weighing 93 to 123 kg, with a mean weight of 105,5 kg were used. They were subjected to forced exercise on a specially constructed exercise track situated at Percy Fyfe Nature Reserve (Longitude 29,25 E, Latitude 23,54 S, Altitude 1 450 m).

The animals were stressed individually at a mean speed averaging 22,2 km/h for a distance of exactly two kilometres, the range of average speeds being from 17,27 km/h to 25,70 km/h. A light motor vehicle was used to chase the antelope around the exercise track. The track consists of a figure '8' course enclosed by a 2,45 m high fencing lined with reeds to form a continuous thick mat. The larger track or section of the figure '8' has a circumference of 125 m so that eight laps constitute a distance of 1 kilometre. Large gates designed to blend in with the walls, allow access to the main track and permit the animal to be deflected into the smaller track at the end of the required number of laps. The smaller track is equipped with a crush and exits that lead either to the pens or to the inner area where measurements of physiological parameters may be made under cover.

Immediately upon termination of the exercise, measurements were made of body temperature, heart and respiratory rates. The electrocardiogram was taken with the use of a *Devices* electrocardiograph, systemic blood pressure was checked by auscultation using a manometric cuff on the front limb at the distal part of the humerus. Blood samples were taken from the recurrent tarsal vein at 15 minute intervals during the hour after exercise and at bi-weekly to weekly intervals thereafter, the samples being drawn with the animal standing in the crush. The blood samples were used to determine enzymes and haematocrit. The enzymes were estimated using the standard methods described in the *Boehringer Mannheim* and *Dr Lange* test kits and determined on a *Lange LP 3* spectrophotometer.

Parentrovite† is composed of thiamine hydrochloride BP 35 mg; riboflavin 0,5 mg; pyridoxine hydrochloride BP 7 mg, and nicotinamide 23 mg, for each millilitre, and was administered at a dose of 20 ml for each animal, followed by the same dose 3 days later at the first repeat sampling, each dose given intramuscularly. Three animals were treated and three were used as controls.

RESULTS

(a) *Creatine phosphokinase (CPK)*

The difference between the treated and untreated groups in respect of CPK was small (i.e. 140 and 160 mU/ml) and this figure in fact reflects the starting difference between the two groups. The time taken for all animals to reach normal levels was almost identical, although there was a sharper fall in the treated group (Fig. 1).

(b) *Glutamic pyruvic transaminase (GPT)*

The reaction to *Parentrovite* as shown by GPT levels indicate no advantage from the therapy (Fig. 2). In fact the GPT levels in the treated animals were higher than those in the untreated group. However, none of the animals showed marked rises in GPT, the maximum level being 65 mU/ml.

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Paper delivered at the South African Veterinary Association Congress held at Durban 8 - 12th Sept. 1975.

† Beecham Veterinary Products, Crawley, Sussex.

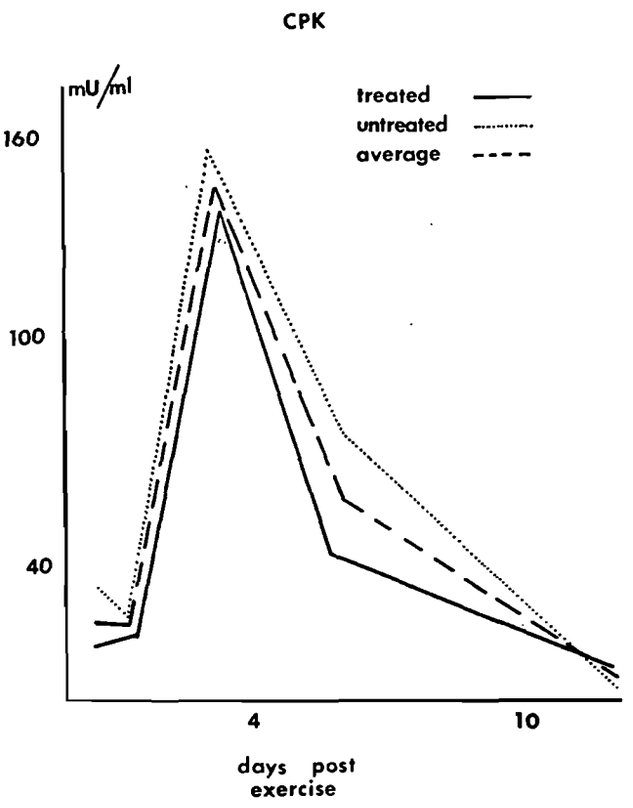

FIG. 1 : CPK LEVELS IN TSESSEBE AFTER FORCED EXERCISE

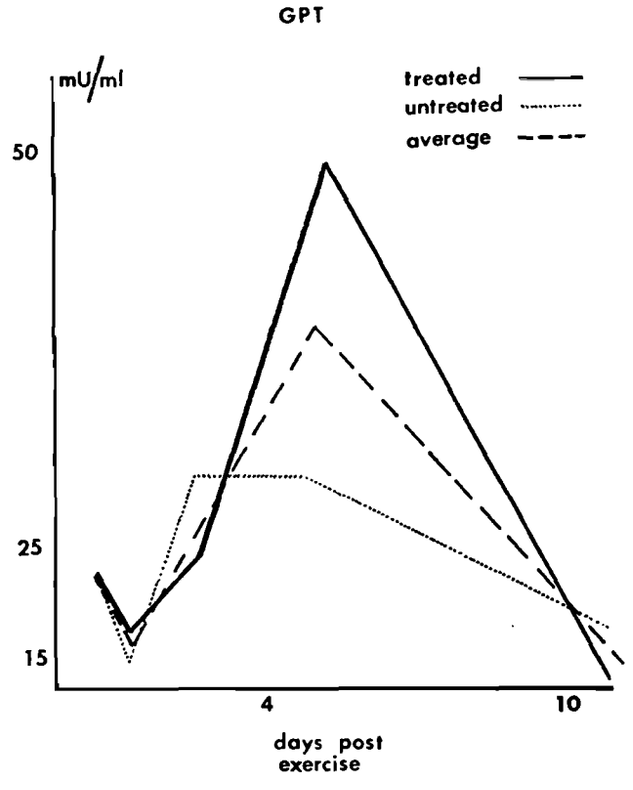

FIG. 2 : GPT LEVELS IN TSESSEBE AFTER FORCED EXERCISE

Table 1: MEANS AND RANGES OF ENZYME LEVELS IN TSESSEBE AFTER FORCED EXERCISE.

	x	low range	high range	s
AFTER EXERCISE				
GOT treated	79,67	61	106	19,15
GOT untreated	84,00	76	89	5,72
GPT treated	20,67	15	30	6,65
GPT untreated	21,00	17	25	3,27
CPK treated	17,00	16	19	1,41
CPK untreated	37,33	11	68	23,47
LDH treated	458,33	376	529	63,00
LDH untreated	344,67	313	395	35,98
15 MINS POST EXERCISE				
GOT treated	76,67	57	112	25,04
GOT untreated	72,67	59	91	13,47
GPT treated	16,67	13	23	4,50
GPT untreated	15,33	11	21	4,19
CPK treated	19,67	11	25	6,18
CPK untreated	36,67	11	54	18,52
LDH treated	425,00	407	445	15,58
LDH untreated	341,00	222	413	84,76
1 DAY POST EXERCISE				
GOT treated	123,00	98	159	26,09
GOT untreated	130,33	45	173	60,34
GPT treated	21,67	19	25	2,49
GPT untreated	28,33	18	42	10,08
CPK treated	139,67	99	186	35,74
CPK untreated	158,67	32	236	90,29
LDH treated	416,33	224	629	159,74
LDH untreated	916,67	620	1 447	375,88
4 DAYS POST EXERCISE				
GOT treated	155,67	15	240	100,12
GOT untreated	196,00	122	235	52,35
GPT treated	50,00	29	64	15,12
GPT untreated	28,00	21	35	5,72
CPK treated	42,33	20	68	19,74
CPK untreated	75,67	16	142	51,65
LDH treated	515,33	360	770	181,52
LDH untreated	682,33	360	883	213,73
37 DAYS POST EXERCISE				
GOT treated	109,00	68	170	43,98
GOT untreated	75,00	68	85	7,26
GPT treated	13,00	11	15	1,63
GPT untreated	14,33	13	15	0,94
CPK treated	14,00	9	19	4,08
CPK untreated	6,00	2	13	4,97
LDH treated	371,33	357	385	11,44
LDH untreated	392,33	284	470	78,97

x = mean
s = standard deviation

(c) *Glutamic oxaloacetic transaminase (GOT)*

The starting points of GOT levels were almost identical while the peaks showed a marked difference, i.e. from 155 in the treated group to 195 mU/ml in the untreated (Fig. 3). The recovery of the treated group was somewhat slower than that of the untreated group. There seems to be a considerable amount of discrepancy in the reactions in respect of GOT to *Parentrovite*, and this is reflected in the ranges (Table 1).

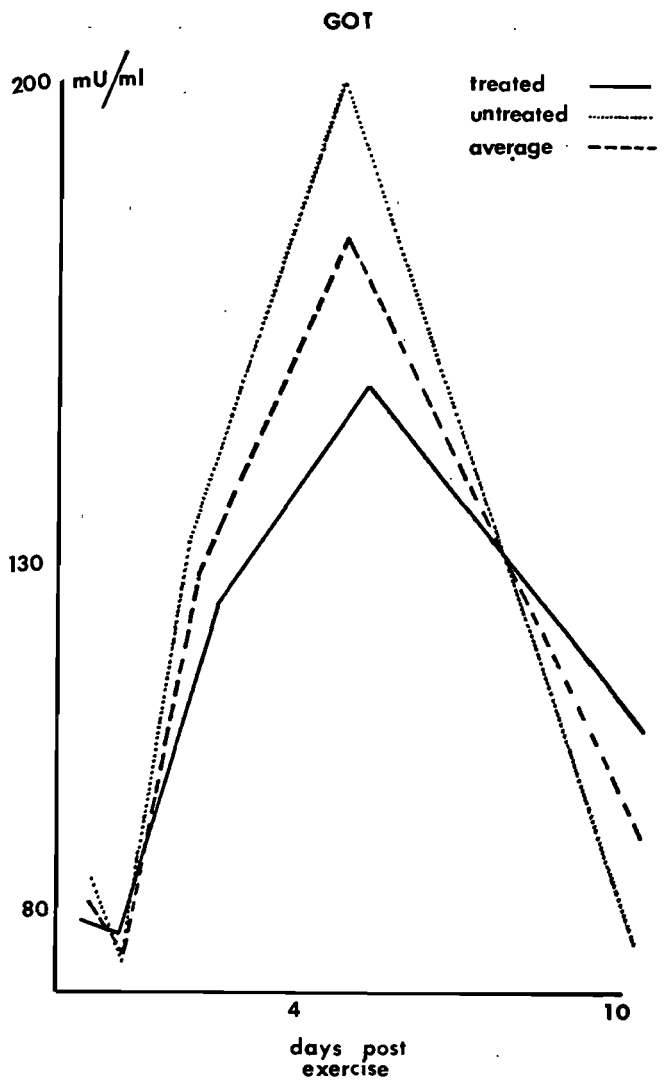

FIG. 3 : GOT LEVELS IN TSESSEBE AFTER FORCED EXERCISE

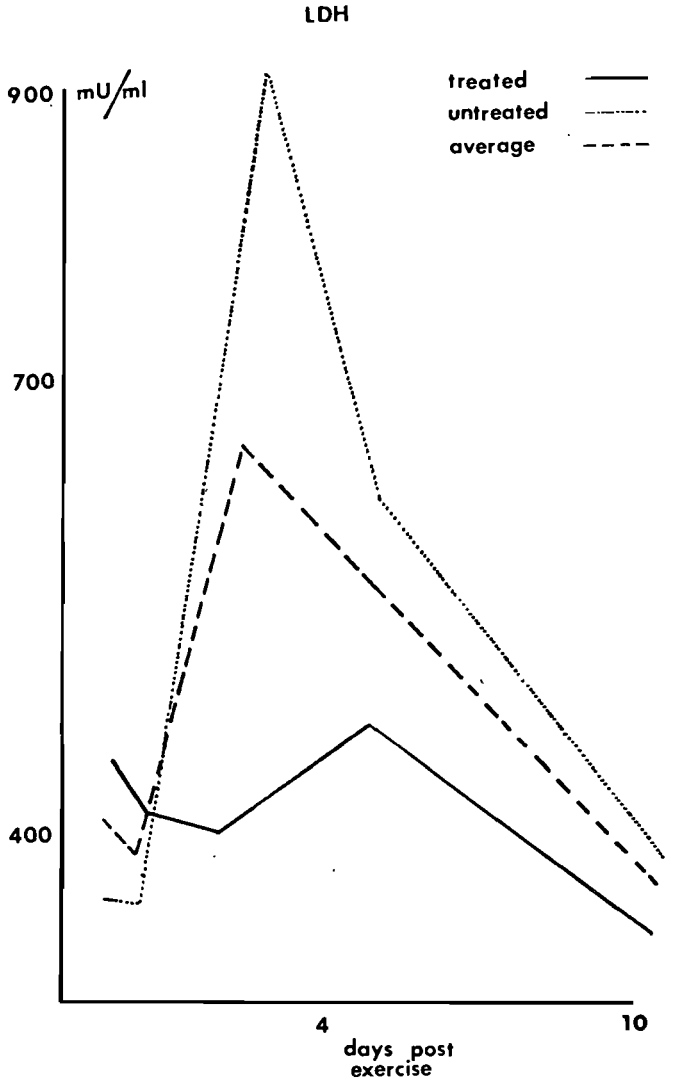

FIG. 4 : LDH LEVELS IN TSESSEBE AFTER FORCED EXERCISE

(d) *Lactate dehydrogenase (LDH)*

The results for *Parentrovite* therapy on LDH appear to be significant (see Fig. 4). The sample size was, however, too small to reflect statistical significance. High mean levels exceeding 900 mU/ml in the untreated group compare with 416 mU/ml in the treated group 1 day after exercise, the latter commencing at a higher level. Recovery of these levels in the untreated group was slower. The ranges show little diversion from the mean values (Fig. 4).

(e) *Electrocardiogram (ECG)*

Electrocardiogram results show no particular abnormalities relating to any one animal. All animals show tachycardia with a tendency to extra systoles. High T-waves indicate a hyperkalaemia which was reflected in red discolouration of the serum indicating either an intravascular haemolysis or liberation of myoglobin from the muscles.

(f) *Haematocrit*

All haematocrit values show some decline during the first 15 minutes after capture, i.e. from approximately 50 to 46 per cent (Table 2). This decline is considerably less than that recorded for other animals, such as sable antelope immobilised with fentanyl-azaperone-xylazine, and probably reflects initial adrenergic discharge. The fall was followed by gradual rise over the subsequent days, although this is probably not significant and none of the animals exhibited symptoms of shock.

Table 2: HAEMATOCRIT VALUES IN TSESSEBE AFTER FORCED EXERCISE.

Animal	imm. post capture	15 mins later	1 day later	4 days post exercise	10 days post exercise
1*	—	—	51	50	48
2**	48	48	47	50	47
3**	54	45	48	54	54
4**	53	50	—	45	50
5*	45	—	56	44	46.5
6*	46	42	47	43	44.5

* treated
** untreated

(g) *Systemic blood pressure*

Blood pressure showed no changes from normal values. There was a fall in systemic pressure soon after capture and fluctuation related to periodic struggling. Samples values are shown in Table 3. A fall in systemic blood pressure as noted in zebra captured by chasing[2] was not observed.

Table 3: BLOOD PRESSURE MEASUREMENTS ON TSESSEBE AFTER FORCED EXERCISE.

Animal	minutes after exercise	value mm Hg
1	40	160/95
2	20	150/100
3	24	170/100
4	5	165/105
5	11	155/90
6	5	160/97

DISCUSSION

Derivatives from the B group of vitamins form co-enzymes which play an important part in the processes of biological oxidisation such as the oxidative carboxylation of alpha-keto acids such as pyruvic acid. When there is a deficiency of these co-enzymes, pyruvic and lactic acid accumulate in the body tissues and fluids while they cannot be metabolised.

Deaths of wild animals during the acute stage of capture myopathy have been ascribed primarily to an accumulation of lactic acid and the effect of the resulting low pH on the heart and blood vessels[1,2]. On this basis the treatment of animals after capture with those factors likely to enhance the biological oxidation and breakdown of lactic and pyruvic acids would appear to be a rational therapy.

The exercise on a track as described here (in contrast to capture in the wild as mentioned above) proved to be sub-lethal so that criteria other than survival had to be established to gauge the effect of therapy. High values of the various enzymes such as LDH and GPT have been described as occurring as a result of capture stress[4,3] being generally accepted as reflecting damage to skeletal muscle, heart and the various parenchymatous organs of the body. Marked differences in enzyme levels between the two groups were seen only with regard to GPT (Fig. 1) and LDH (Fig. 2).

A great deal of work remains to be done in this context involving a greater number of animals run at a faster speed. It is rarely possible to extrapolate from one species to another, from domestic to wild, and even from truly wild and captive species. Wild animal species readily susceptible to capture stress are also those of increasing rarity in this country and therefore difficult to procure for experimental purposes. Further experiments are, however, now projected.

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TO THE EDITOR — AAN DIE REDAKSIE

EQUINE ELECTROCARDIOGRAPHY

Dear Sir,

During the SAVA congress held in Durban in September 1975 Dr D.W. Howell presented a short paper in which was discussed the desirability of standardised reports on electrocardiographs (ECG). The proposals found favour with those present and it was agreed that a standard technique of reporting, and the use of a standard form, be adopted by members of the Equine Practitioners Group.

A form along these lines has now been printed and provides for inclusion of the following points:

1. Identification of the horse, including recording of its age in months.
2. The heart rate per minute.
3. Arrythmias, if present.
4. Heart score (Steele technique).

Copies of the standard form are available from the undersigned

Yours faithfully

Secretary
Equine Practitioners Group,
S A V A
Box 4107
1451 Alrode.

Jl. S. Afr. vet. Ass. 47(3) 223-226 1976

DISCOLOURATION OF WOOL: 1 GREEN DISCOLOURATION

VAN TONDER E.M.*, KELLERMAN G.E.* AND BOLTON T.F.W.*

ABSTRACT: Van Tonder E.M., Kellerman G.E., Bolton T.F.W. **Discolouration of Wool : 1 Green Discolouration.** *Journal of the South African Veterinary Association* (1974) **47** No. 3 223-226 (En) Regional Veterinary Laboratory, Middleburg, 5900 Republic of South Africa.

Since 1974 green bacterial discolouration of Merino wool was frequently encountered in the Karoo and Eastern Cape areas. The occurrence of this condition coincided with the extraordinary wet conditions that prevailed during this period. *Pseudomonas aeruginosa* was recovered in almost pure culture from affected wool of 24 out of 25 different sheep. The role played by this particular organism was confirmed by the successful reproduction of this condition after exposure of unaffected sheep to cultures of *Ps. aeruginosa* isolated from green wool.

INTRODUCTION

Various deficiencies and discolourations in wool have been classified by Henderson[4]. These discolourations vary tremendously according to the aetiology which might include hereditary, nutritional, chemical, ecto-parasitic, mycotic and bacterial agents. In most outbreaks, whether confirmed or suspected to be of bacterial origin, high atmospheric temperatures and humidity following thorough wetting of the fleece are essential[1, 2, 7, 8, 11, 13, 14, 20, 21].

Discolourations ascribed to bacterial organisms are of a wide range, depending on the type of organism involved. These include unidentified chromogenic bacteria[13] (yellow), *Pseudomonas aeruginosa*[1, 2, 13, 21] (green and brown), *Chromobacterium violaceum*[4, 13] and an unidentified chromogenic bacterium[13] (violet or purple), *Serratia marcescens*[13] and *Ps. aeruginosa*[1, 11] (red), *Chromobacterium coeruleum*[13], *Ps. indigofera*[4] and possibly also *Arthrobacter atrocyaneus*[4] (blue) and *Bacillus vulgatus*[4, 13, 20], a fluorescent bacillus[13] and an unidentified micrococcus[4] (pink).

Green discolouration of wool was first described by Stuart[16] in Australia, who proved it to be caused by chromogenic organisms which he did not identify by name. He also proved that two pigments were involved: a blue pigment soluble in chloroform and a light green or yellow pigment soluble in ether. Subsequent to a preliminary report by Seddon & McGrath[14] on green discolouration in wool, Seddon[13] published a full account of investigations which not only incriminated *Ps. aeruginosa* as a causative agent but also identified pyocyanin as the responsible pigment. Supportive evidence has since been published in the same country[7, 8] as well as in the United States[21] and New Zealand[2].

Records of fleece discolouration indicate that green staining of wool is uncommon in South Africa and occurs only during very damp seasons, in the moist belt of the Province of Natal and other areas with similar climatic conditions occur. No additional information could be obtained on more detailed investigations carried out in this country.

During the past 3 years climatic conditions in the Karoo and Eastern Cape have been extremely favourable for the discolouration of fleeces. A green type discolouration was commonly encountered in wooled sheep and as many as 50% of fleeces in a particular flock were affected.

In order to evaluate the position in this country in relation to the situation elsewhere, a systematic investigation was carried out on specimens of abnormally stained wool. This report covers the results of bacteriological examinations on green stained specimens.

MATERIALS AND METHODS

Investigation of natural cases

Specimens of abnormal wool from farms in the Karoo Midlands were classified according to the discolouration displayed and kept in the dark in separate plastic containers. Representative samples were submitted to the Wool Research Section of the Agricultural Research Institute, Grootfontein, Middelburg, Cape for determination of their physical properties.

Bacteriological Examination: The distal end of a complete staple of discoloured wool was introduced into a tube containing peptone water and severed below the tip, but well above the discoloured band; this allowed the coloured part of the staple to drop into the peptone water. The tubes were incubated at 37°C for 2 and 4 hours. After thorough stirring with a sterile platinum loop, cultures were plated out onto 5% horse blood tryptose agar plates. The plates were incubated aerobically and anaerobically (Gaspak†) for 24 to 48 hours at 37°C. Standard procedures for purification and identification of bacterial cultures were followed.

For differentiation of *Ps. aeruginosa* from other gram-negative chromogenic organisms and closely related *Pseudomonas* spp. the production of blue-green and green-fluorescent pigments on King's media[6], and Kovac's oxidase test[5, 15, 16], growth at 42°C[3] ⊅ [5] ⊅ [9], the production of sodium 2-ketogluconate[3] ⊅ [5] ⊅ [15] and simple tests on pigmented cultures or specimens of wool[2] ⊅ [13] ⊅ [19] were employed.

Tests on Pigmented Wool: Samples of green coloured wool were exposed to light for some weeks and afterwards alternatively treated with strong solutions of hydrochloric acid and ammonia as described by Seddon[13], Frazer & Mulock[2] and Wilson & Miles[19].

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† Gaspak, Baltimore Biological Laboratory, Division of Bioquest, Cockeysville, Md.

Experimental Reproduction of Green Discolouration

Two two-tooth Merino rams with about 7 months growth of wool were used. Both sides of the neck as well as a portion of the back behind the withers were thoroughly wetted with distilled water every day for 2 days. On the third day a marked area about 10 cm in diameter on the left side of the neck of the one and on the back immediately behind the shoulders of the other ram, was contaminated with freshly reconstituted freeze dried cultures of *Ps. aeruginosa* isolated from coloured wool. These cultures were freeze dried as fairly dense suspensions in 0,2 ml amounts, but were reconstituted in 1 ml volumes which corresponded approximately to Brown's opacity tube Nr. 2. Two ml of the reconstituted suspension was used per sheep and carefully deposited by means of a needle, close to the skin in small quantities at different sites.

On the following day the marked areas were continuously wetted to maintain dampness of the fleece while preventing the excessive run-off of water and consequent spreading of the contamination. The right side of the neck as well as the area on the back of the one and both sides of the neck in the other sheep were situated well away from the contaminated areas. These were kept as wet but uncontaminated controls.

Both sheep were kept together in an open pen and paddock system and observed daily. Specimens for bacteriological examination were taken on days 19 and 28 after contamination.

RESULTS

Natural Cases

From January 1974 excessive rains were experienced in the Eastern Cape and Karoo sheep farming areas. The official records kept at the Agricultural Research Institute, Grootfontein, Middelburg, Cape could possibly be regarded as indicative of the change in climatic conditions. In Table 1 the total rainfall, the average maximum and minimum atmospheric humidity and temperature recordings for succeeding spring, summer and autumn months are compared.

Apart from the increase in rainfall, a change in the distribution pattern also occurred. In previous years the normally low rainfall occurred mostly in the form of sudden heavy thunderstorms followed by relatively long intervals of hot dry weather. The rainfall experienced during the preceding 3 years has not only increased but also occurred in spells of soft showery rain and cloudy weather lasting for days, followed by relatively short intervals of clear weather. Comparison of the mean relative humidity figures reveals an increase during this period, on a seasonal basis. On the other hand the mean maximum and minimum ambient temperatures did not show marked differences although there was a tendency for these figures to be lower during the seasons of excessive rain.

All specimens consisted of Merino wool of at least 5 months growth. The affected sheep were mostly of the more developed types. The affected areas were comparatively small and confined to the upper parts of the body; the sides of the neck and chest were more commonly affected. None of the cases examined were complicated by other conditions like myiasis, lumpy wool or other forms of external parasitism.

The discolouration varied from a bright to a dull green, sometimes accompanied on the distal side by a light to rusty brown band.

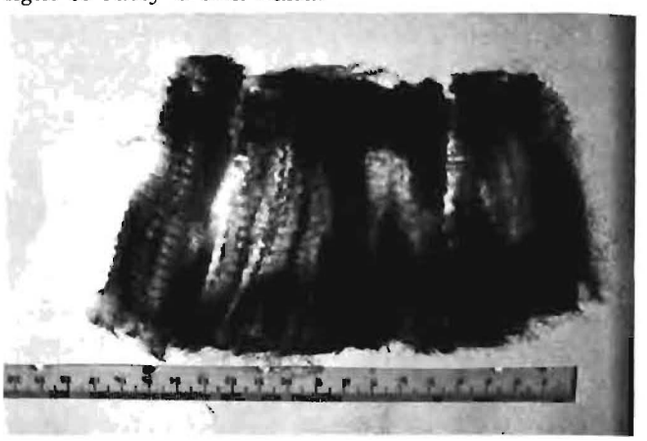

Plate 1: Green stained wool from a natural case

Tests on specimens of the affected wool proved that the green discolouration of the fibres could not be removed by ordinary scouring processes but that the tensile strength and physical soundness of the fibres were unaffected.

Smears prepared from the sediment of saline washings of the pigmented wool showed the presence of numerous bacterial organisms of which gram-negative rodshaped types predominated.

Ps. aeruginosa was isolated from 24 of the specimens taken from 25 sheep. A striking feature was the purity of the original cultures, despite the fact that wool is expected to be grossly contaminated by a variety of micro-organisms. In general, isolations were more readily and frequently obtained on solid media subcultured from the 4-hour as compared to the 2-hour peptone water cultures. In a few instances, the colonies assumed a dirty green colour after 24 hours on the blood-tryptose agar and potato agar cultures. The

Table 1: TOTAL RAINFALL, AVERAGE MAXIMUM AND MINIMUM HUMIDITY AND AMBIENT TEMPERATURES FOR SUCCEEDING SPRING TO AUTUMN SEASONS AT GROOTFONTEIN, MIDDELBURG, CAPE

Recording	1970/71 Sept-Apr	1971/72 Sept-Apr	1972/73 Sept-Apr	1973/74 Sept-Apr	1974/75 Sept-Apr	1975/76 Sept-March
RAINFALL (mm)	257,8	314,3	239,1	602,2	328,1	479,0
HUMIDITY (%)						
Av. max.	85,5	85,5	81,9	84,6	87,3	86,2
Av. min.	26,8	28,5	24,9	34,4	28,6	29,3
TEMPERATURE (°C)						
Av. max.	26,2	25,7	27,1	25,1	25,3	25,4
Av. min.	9,7	9,4	9,9	9,8	8,6	7,4

majority of isolates were of a greyish-white to greyish-brown colour after 24 hours, turning to a dirty green after 72 to 96 hours. On King's medium A[6] and peptone sugar media a green to bluishgreen colour developed within 24 hours in all cases. All isolates produced pyocyanin on King's medium A[6] and a green fluorescent pigment on King's medium B[6]. The presence of pyocyanin was confirmed by extraction with chloroform and acidulated water and by the colour reaction in alkaline medium[2, 13, 19].

Specimens of green coloured wool exposed to direct sun slowly changed to a light to rusty brown colour on the exposed surface, while the under side remained unchanged. Alternative treatment with hydrochloric acid and ammonia brought about a colour change from red or pink to the original green[2, 13, 19]. These changes were however not very dramatic.

Experimental Cases

The two sheep exposed to cultures of *Ps. aeruginosa* developed a light green discolouration in the wool on the contaminated site, close to the skin surface, while the control areas remained unaffected throughout the period of observation. On day 19 after exposure the colour was well developed and dark green (Plate 2).

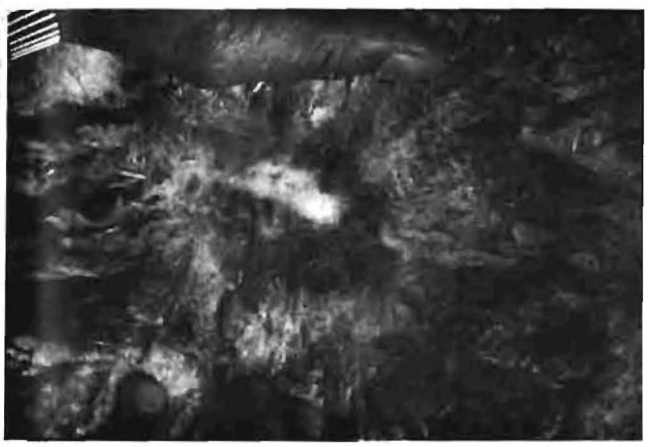

Plate 2: Experimentally produced green discolouration of wool

Ps. aeruginosa was recovered from the affected wool of both sheep on days 19 and 28 after exposure.

DISCUSSION

Green discolouration of wool is relatively uncommon in South Africa[1]. The close association between wet weather and bacterial staining has been well established[2, 4, 7, 8, 13, 21] and is undoubtedly responsible for the widespread high incidence in this country since 1974.

The average maximum and minimum atmospheric temperature tended to be lower during the 3 years under study, suggesting that hot weather is not essential for the development of this condition. This would seem to differ from other observations[7, 8, 13, 21], and it appears significant that *Ps. aeruginosa* can multiply within a wide temperature range, bearing in mind that skin temperature is regulated largely by the body. The condition was successfully reproduced during the winter of 1975 when the average maximum and minimum temperatures were 17,6°C and 15,9°C and 0,8°C and 0,6°C for June and July respectively.

Observations made on the type of sheep affected, the length of wool, the predilection sites, the intensity of this particular discolouration and the occasional transition to a brown colour are in accordance with earlier reports[2, 4, 7, 8, 13, 21].

The physical soundness of the fibres were not affected. The fact that the pigment cannot be removed by scouring is of great economic significance, as it severely affects the usefulness of the fibre from the processors point of view[1, 14, 16, 21].

The isolation of *Ps. aeruginosa* from specimens, almost without exception and in virtually pure culture, is of aetiological significance. Successful reproduction of green wool discolouration in two sheep by application of *Ps. aeruginosa* isolated from natural cases confirm this. Similar isolations and confirmatory experiments have incriminated this organism as a causative agent[2, 13, 14, 16, 21]. Pyocyanin and not the green fluorescent pigment is responsible for the green discolouration[2, 13]. The change of colour when wool is exposed to daylight or to acid or alkali can be ascribed to photo-chemical conversion of pyocyanin to hemipyocyanin[2, 4] or pyoxanthose[13].

The ease with which pure cultures were obtained in this investigation from wool exposed to gross natural contamination can be attributed to the bactericidal properties of pyocyanin and other substances produced by *Ps. aeruginosa*[10, 12].

Acknowledgements

Miss E. Rubidge is thanked for technical assistance and the Director of Veterinary Services for permission to publish this report.

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REVIEW **OORSIG**

TOXOPLASMOSIS AS A PUBLIC HEALTH HAZARD

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ABSTRACT: Turner G. V. S. Toxoplasmosis as a public health hazard. *Journal of the South African Veterinary Association* (1976) **47** No. 3, 227-231 (En). Faculty of Veterinary Science, University of Pretoria, Onderstepoort 0110, South Africa.

Recent advances in the epidemiology and life cycle of toxoplasmosis are reviewed. Cats play a kev role. **Toxoplasma** has a coccidian-type entero-epithelial cycle with oocyst production in the feline host. An extra-intestinal cycle occurs in both feline and non-feline hosts. The worldwide distribution and the public health significance of toxoplasmosis as a zoonosis is discussed, with particular reference to available data regarding Southern Africa.

INTRODUCTION

The aetiological agent of toxoplasmosis, *Toxoplasma gondii,* is a protozoan parasite belonging to the Order Eucoccida. *T. gondii* was first reported simultaneously in 1908 by Nicolle and Manceaux[12] in a North African rodent, *Ctenodactylus gondii,* and by Splendore[23] in São Paulo in the rabbit.

T. gondii has since been isolated in the tissue of many species of mammals and birds, and the parasite is known to have a world-wide distribution with the exception of Antarctica[4 8 10 12 19 20 28 30 33].

The first description of a possible congenital infection in man was in 1923, when Dr Joseph Janku, an ophthalmologist in Prague, described parasitic cysts which he found in the retina of an eleven month old child with congenital hydrocephalus and microophthalmus. Transplacental transmission was first described by Cowen, Wolf and Paige in 1939 [5 23 28].

Human toxoplasmosis is divided into two main forms: Congenital and Acquired Congenital infection, the first form to be described, has various clinical manifestations including the following: Hydrocephaly; microcephaly; icterus; convulsions; chorioretinitis; cerebral calcification; blindness; mental retardation; epilepsy; deafness and various other neurological abnormalities [5 14 20 23 31].

The acquired form also covers a wide clinical spectrum including: Lymphadenopathy; ocular lesions; fatal acute fulminating pneumonitis; myocarditis and encephalomyelitis [5 10 14 23]. The lymphoglandular form is a frequent manifestation of an acquired infection. There is also a high incidence of asymptomatic infections in man [5 10 14 16 23 30].

In animals toxoplasma infections are also known to cause various symptoms, for example, abortion storms in sheep; ocular lesions in cats; myelomalacia in horses [6 16 26]. Asymptomatic infections also occur.

With regard to human toxoplasmosis, congenital toxoplasmosis continues to be of the utmost importance and concern as a zoonosis and clinical entity. For example, in the United States, an estimated 3 000 babies are born with the disease each year. It has been calculated that of these babies, 5 to 15% die, 8 to 10% have marked brain and ocular lesions, 10 to 13% have moderate to marked visual damage and 58 to 72% are clinically normal at birth but some of these develop active retinochoroiditis in childhood or young adulthood. The total annual cost of neonatal toxoplasmosis in the United States has been estimated at approximately forty million dollars — including hospitalisation, institutionalisation and special education[20].

It is now well established that toxoplasmosis is a true zoonosis manifesting itself sporadically in humans and with cats playing a key role in the epidemiology of toxoplasmosis in mammals, including man, and birds [9 10 12 13 16 33 35 36 37 38].

In order to evaluate the epidemiology and public health significance of toxoplasmosis it is necessary to have a clear understanding of the life cycle of *T. gondii.*

LIFE CYCLE

The most recent advance in the study of toxoplasmosis was when *T. gondii* was shown to have coccidian affinities with an entero-epithelial cycle and oocyst production in cats and other felines [12]. Only domestic cats and certain other members of the family Felidae have been shown to produce Toxoplasma oocysts[9 11 12 19 20 23 24 28]. In addition, *Toxoplasma* has highly successful tissue-invasive (extra-intestinal) forms which are capable of proliferation in many hosts[12 16 19].

Two cycles in separate biotypes and three stages of Toxoplasma are known and can now be linked into a life-cycle[12]. In cats, the entero-epithelial stage is generally similar to that in other coccidia[7, 12, 16, 17, 19, 23, 28] and leads to gametogony and oocyst production with sporogony. Two additional stages occur in the extra-intestinal tissues: Tachyzoite-forming groups ("trophozoites") occur during the acute infection and bradyzoites within the tissue cysts are found during chronic infection[12 19 20].

The three known stages of *Toxoplasma* are bradyzoites (tissue cyst stage), tachyzoites (proliferative forms in tissue during acute infection) and sporozoites (in sporulated oocysts).

Entero-epithelial cycle:

The feline host is infected by the carnivorous ingestion of bradyzoites (eg tissue cysts in raw meat,

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rodents or birds) or tachyzoites (eg rodent with acute *Toxoplasma* infection) or by the ingestion of sporulated oocysts originating from feline faeces.

This constitutes the entero-epithelial stage where the multiplication takes place in the epithelial cells of the small intestine and the ileum with the formation of gametocytes, microgametes, macrogametes and finally oocysts. The oocysts are detached from the intestinal epithelium and are discharged with the faeces. Sporulation occurs in from one to five days depending on the temperature and availability of oxygen. An infected feline host sheds oocysts for seven to twenty days during primary infection[6 7 11 12 18 19 20 28].

The appearance of these stages, together with the nature of the oocysts, indicates that *T. gondii* is a coccidian parasite related to the genus *Isospora*[17]. The oocyst of *Toxoplasma* (b) is much smaller than the oocysts of *I. felis* (a) (Fig 1).

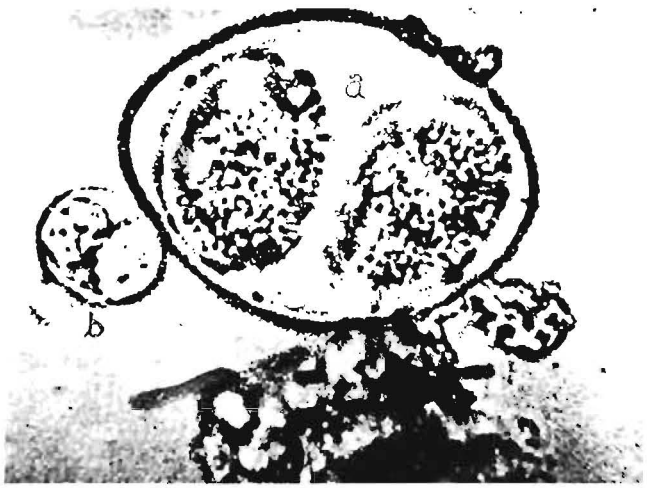

Fig. 1: Oocysts of *Isospora felis* (a) and *Toxoplasma gondii* (b) x 1 500
(Kind permission J W Plant *et al*[26])

Extra-intestinal (tissue) cycle:

These stages, which also occur in cats, appear to constitute the entire cycle in non-felines[7 10 12 19 28]. In non-feline hosts infection is usually from the ingestion of sporulated oocysts of feline origin or the ingestion of tissue cysts containing bradyzoites or transplacental infection of the foetus with tachyzoites after ingestion of encysted bradyzoites or sporulated oocysts by the pregnant female. Tachyzoites then spread from cell to cell and are disseminated to various organs in macrophages, lymphocytes, granulocytes and in free forms in the circulation[12 16 19].

In the acute visceral infection, as seen in many hosts, the tachyzoites develop within a vacuole in a multitude of cell types. From about 8 to 16 or more organisms accumulate in the host cell before it disintegrates and new cells are infected. These are the "tachyzoite-forming groups" of the acute infection in which rapid replication occurs.

The severity of the infection is determined by the degree of cellular necrosis caused either directly by the number of proliferating tachyzoites or indirectly by hyper-sensitivity, or by both. Cyst formation then begins and appears to coincide with the development of immunity[12 19 20].

Tissue-cysts are characteristic of the chronic infection and occur mainly in the brain, heart and skeletal muscle. Cysts may persist for months and it appears that they may persist for the life of the host[6 12 16 19 20, 28].

EPIDEMIOLOGY

Serological surveys carried out in various parts of the world confirm the high prevalence of *Toxoplasma* infection in man and animals. More than a third of the population in most parts of the world have antibodies indicating past infection with *T. gondii*[20 30].

It has been noted that *Toxoplasma* antibodies in humans tend to vary in different parts of the world, with positive dye test percentages in human groups varying from 0% in Eskimos to 83% in parts of Nigeria [3 14 37]. Of 806 samples of human sera collected in South Africa 37% were positive for *Toxoplasma* antibody[22].

Age groups have repeatedly shown a classic distribution of incidence in serological surveys, the number of positive reactions being low in the first decade of life and increasing with age[16 22].

Based on the new concept of the life cycle, an extensive transmission scheme for *T. gondii* can now be drawn up. Basically the three modes of transmission are carnivorism, faecal contamination and transplacental.

Oocysts are the key to understanding the epidemiology of *Toxoplasma* infections in nature, since they can infect mammals and birds. The oocysts are non-infective when unsporulated, with sporulation taking 1 to 5 days[12 23]. Millions of oocysts may be shed in a single stool[13 23].

It has been shown that oocysts excreted in cat faeces may remain infective for a year in warm, moist climates and longer in cooler climates[12 13 23 40]. Oocysts have been isolated from naturally contaminated soil. It is therefore likely that yards and gardens around the house provide good sites for the persistence of oocysts[29].

Fig. 2: Life cycle of *Toxoplasma gondii* (Adapted from Frenkel[12])

Toxoplasma oocysts are found to be resistant to most common detergents, acids and alkalis, but are

killed by boiling water, dry heat (66°C), 10% ammonia and by incineration[1, 10, 11, 12, 16, 20, 23, 28].

Serological surveys have indicated that *Toxoplasma* infection is prevalent in livestock throughout the world. Approximate seropositives for each species on a world wide basis are: Cattle 22%, sheep 39%, goats 22%, horses 19% and dogs 33%[33]. *Toxoplasma* cysts have been isolated from consumer cuts and diaphragm muscle specimens of lamb, pork and beef[33].

The mode of transmission of *T. gondii* in herbivorous animals remains largely unexplained. There are, however, indications that faecal contamination of pasture and concentrates by infected cats may play an important role in infecting herbivores.

It is generally accepted that freezing is not a reliable way of killing *Toxoplasma* tissue-cysts[12, 19, 23]. Tissue-cysts in meat are killed by heating the meat throughout to 60°C[10, 23]. Cats can become infected with *T. gondii* by eating infected birds, small mammals (eg rodents), raw or undercooked meat or offal or by faecal contamination from cats that are excreting *Toxoplasma* oocysts[1, 10, 16, 35, 38].

Data collected indicates that common species of domestic rats eg *Rattus rattus* and *R. norvegicus* are chronic carriers of the tissue form of the parasite and probably serve as a reservoir of infection for the cat[39]. It has also been shown that mice can acquire *Toxoplasma* infection from ingesting oocysts from cat faeces[8].

Immunity to the intestinal stages in cats is apparently not absolute. When the antibody titre related to a first infection had fallen, cats could successfully be re-infected, with renewed oocyst production. Some cats with antibody will pass oocysts under experimental conditions, and not all cats that shed oocysts develop antibody[9, 16]. Cats usually have lower titres of antibody than dogs, mice or humans. For these reasons, serological data are diagnostically less useful in cats[10].

Numerous *Toxoplasma* antibody surveys in cats have been performed throughout the world. The prevalence of antibody has been found to range from 5 to 85%[38]. Cats younger than a year of age have been shown to already have a high antibody titre[32]. The prevalence of *Toxoplasma* was higher in stray cats than in domiciled cats, strays probably having more opportunities to become infected by feeding on garbage (eg scraps of raw meat) and hunting rodents and birds[9].

Infected raw or undercooked meat can serve as the source of human toxoplasmosis[4, 12, 23, 33]. It is thought that most of the infections in man result from the accidental ingestion of oocysts originating from cat faeces[9, 16]. Oocysts in cat faeces are usually buried superficially in soil, often close to human habitations. From 10 000 to 100 000 oocysts have been isolated from a gram of contaminated soil. After digging in contaminated soil, between 7 and 13 mg of soil can be removed from under the fingernails, which might contain 10 to 100 oocysts. Thus infected garden soil, children's sandpits, cat litter pans etc. can be a source of human infection[10, 13].

Laboratory workers appear to be at risk when working with faeces of infected cats[5, 8, 16, 24]. Being the owner of a cat appears to increase the risk of infection[1, 20, 22, 25]. In serological surveys, no significant difference was however noted between veterinarians and the normal population controls[1, 30].

In certain instances human infection appears to have been related to the handling or consumption of raw or undercooked meat[10, 19, 21, 28, 30]. The most widely quoted example is an epidemic of acute lymphoglandular toxoplasmosis involving five medical students. The students all ate rare hamburger at the same place on the same night, and evidence indicated that this was the way in which the infection was acquired[21]. *T. gondii* has frequently been isolated in hares and rabbits in all parts of the world. Rabbit handlers and hare trappers have been shown to have a high prevalence of antibodies[15].

It is conceivable that some cases of human toxoplasmosis result from the ingestion of infected raw eggs or infected chickens. *Toxoplasma* has been isolated from chicken eggs and various organs and tissues of the chicken[10, 12, 23].

It is also possible that coprophagic arthropods such as flies, cockroaches, certain snails and slugs which normally feed on faeces are involved as transport hosts of the parasite. Viable oocysts have been isolated from these transport hosts[2, 13, 19, 34]. The mechanical transmission of oocysts by filth flies from cat faeces to milk has been demonstrated experimentally[39].

T. gondii has also been isolated from the milk of cows, goats, sheep, pigs, dogs, cats, rabbits, guinea-pigs and mice with naturally occurring or experimentally induced infections[12, 20].

The possibility of the transmission of *Toxoplasma* by blood transfusions must not be ignored. An asymptomatic infection in a donor with a late parasitaemia can play a role here[19].

It seems clear, from the present data, that the cat is a necessary link in the maintenance of *Toxoplasma* infection as a zoonosis[19]. However, the question still remains as to the relative importance of other routes of infection to man.

Increasing numbers of cases of toxoplasmosis in the compromised host are being recorded. This is in patients undergoing therapy for malignancies, in patients on immunosuppressive agents and in people suffering from an immunodeficient disease. Reactivation of a dormant acquired latent infection or a primary generalised infection may occur[12, 28].

The occurrence of habitual abortion in women due to *Toxoplasma* infection is rather contentious. The foetus is generally only infected during primary infection of the mother. Premunition appears to be of practical importance in preventing infection of the foetus during successive pregnancies in women[12]. From the majority of the literature it appears that there is no valid evidence to indicate that abortion in two successive pregnancies is either common or habitual[12, 16, 19].

A mother with a positive titre to *Toxoplasma* at conception should not be in danger of infecting the foetus *in utero* with *T. gondii*[16, 19]. However, occasional cases of congenital toxoplasmosis have occurred in lambs of ewes that had high antibody levels of *Toxoplasma* at mating[16].

Te Groen[31] mentions that Langer succeeded in isolating *Toxoplasma* in 23 of 70 women with habitual abortions, repeated miscarriages, premature births and stillbirths, or from their foetuses in whom other causes had been excluded. He also described a number of cases, where patients with poor obstetrical histories plus positive *Toxoplasma* sera delivered healthy infants after being treated solely for Toxoplasmosis.

THE PUBLIC HEALTH HAZARD

In spite of the fact that toxoplasmosis is a relatively uncommon clinical disease, it continues to be a significant public health problem in terms of the severity of the disease. This is confirmed by the reported cases of toxoplasmosis in human foetuses and neonates, children, adults, cancer patients, transplant and transfusion patients, and ophthalmic patients[20].

The world-wide distribution of this zoonosis, together with the recent knowledge of the role played by oocysts shed by felines and tissue-cysts in slaughter animals as a source of human infection, makes *Toxoplasma* infection a potential hazard for any seronegative human. Jacobs[19] sums up the problem by saying "The man going blind from progressive toxoplasmic retinochoroiditis is not comforted by the fact that his condition occurs less frequently than other causes of loss of vision".

The danger of *Toxoplasma* infection in the young seronegative pregnant woman should always be of great concern. As a rule toxoplasmosis does not manifest itself in the form of an epidemic. Observations from France record that about 40% of women who contract toxoplasmosis while pregnant will produce infected offspring[16].

The recent revelations concerning the life cycle of *T. gondii* and the clearer understanding of the epidemiology of toxoplasmosis should be of great interest to the veterinarian, gynaecologist, paediatrician, neurologist, ophthalmologist and the public health official.

It now seems clear that *Toxoplasma* infection can be traced directly to man's use of animals as pets or for food.

The cat shedding oocysts appears to play the main role in the epidemiology of human toxoplasmosis. The ingestion of tissue-cysts and the other routes of infection may not be as common a source of infection. People coming in contact with contaminated soil, cat litter pans, children's sandboxes etc. can expose themselves to infection. From the occupational aspects, those at greatest risk appear to be cat owners, laboratory workers, slaughtermen, butchers and housewives. Cultural patterns concerning meat preparation and eating habits may play a role here as well.

On this basis, a scheme for the prevention of toxoplasmosis in humans, especially pregnant women, and in cats could include the following:

1. Heat meat throughout to 60°C before eating.
2. Wash hands after handling raw meat.
3. Feed cats only dry, canned or boiled food.
4. Flush cat faeces down the toilet; scale litter pans daily; incinerate disposable litter trays daily. *It is imperative that cat faeces be disposed of daily, whether in the home, hospital, kennels or in the zoo that keeps Felidae.*
5. Cover children's sandboxes when not in use.
6. Wear gloves when handling litter pans and potentially contaminated soil.
7. Control cockroaches, flies, stray cats and rodents.
8. Avoid newly acquired cats to the household of a pregnant woman.
9. In the laboratory avoid contamination of hands, centrifuge, microscope, benches etc. with cat faeces. Gloves should be worn at all times by those handling cat faeces.

The periodic faecal examination and serological surveillance of cats is not all that effective. The short duration of excretion of oocysts and the uncertainty of differentiating the oocysts from those of *Isospora* may add to the frustration of the practitioner trying to make a diagnosis.

Serological data can be misleading in cats.

Presence of antibody to *Toxoplasma* in cats indicates previous exposure to *Toxoplasma* and are thus less likely to be excretors of *Toxoplasma* at the time of examination and in the future. However, this is not necessarily true in each case. Thus basically a seropositive cat is a safer cat to have in the household.

Daily contact with pet cats during clinical practice does not necessarily increase the risk of exposure to infection. The veterinarian is more likely to become infected from contaminated laboratory apparatus, litter pans in the small animal hospital or from handling material infected with tissue-cysts.

We live in a "sea" of *Toxoplasma* and in the future the wide *Toxoplasma* infection rate in both man and animals may have to be reduced by the introduction of legislation to possibly prevent the feeding of raw meat or offal to cats, the control of feral and stray cats, the development of a *Toxoplasma* coccidiostat, and the development of an effective vaccine for young children and animals.

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INFORMATION — INLIGTING

EFFECT OF PESTICIDES ON HOST DEFENCES

Although a significant quantity of information is available concerning the toxicological properties of pesticides, there is a dearth of information about their potential effects on host defences, the mechanisms which assist humans in resisting a wide variety of infectious diseases. Hypersensitivity reactions occasionally develop as a result of the activation of these host defence mechanisms through exposure to a number of substances, including pesticides; since little is known, there is a need for data especially in regard to the effects of exposure to low dosages of pesticides such as might be encountered from residues in foods.

Accordingly, researchers of Pennsylvania State University's Agricultural Experiment Station initiated studies aimed at determining the effects of varying acute and chronic oral doses of five common pesticides, using experimental mice selected on the basis of uniformity. The pesticides tested were ametryne, carbaryl, chlordimeform, DDT and parathion.

The experiments entailed the use of "sensitive and specific techniques", and the testing of two components of host defences by means of what are described as "the most modern and sensitive procedures available". The defences tested were: the ability of an animal to form antibodies, and to form activated white blood cells.

Results thus far indicate that administration of relatively high acute doses of the five pesticides can cause a marked depression in antibody and activated white blood cell production ability. Lower acute doses did not cause any significant effects. These results would be comparable to cases of accidental acute pesticide poisoning, suggesting a probable reduction in the human's host defences.

The chronic administration of small quantities of the various pesticides revealed no significant effects except in the case of parathion, which resulted in a significant depression of the above host defence components.

The effects of small doses of pesticides over long periods of time still remain to be determined. The researchers plan to do this and also to test the effects of exposure to pesticides in aerosol containers, as humans frequently encounter this type of exposure. They say that complete and systematic evaluation of the consequences of pesticide exposure on all host defences appears to be warranted, and that an added incentive for investigation would be the possibility of discovering chemicals which may be used to manipulate host defences to advantage.

("Can Pesticides Alter Host Defences?", Science in Agriculture, Vol. XXIII, No. 2, Winter, 1976, p. 16: Pennsylvania Agricultural Experiment Station, Agricultural Administration Building, University Park, Pennsylvania 16802).

AWARD

THE S A V A GOLD MEDAL FOR DISTINGUISHED SERVICE TO THE PROFESSION : THIRD AWARD

After consideration in 1974 of a strongly supported proposal Council unanimously decided to award the SA Veterinary Association's highest award to

DR JACK GORDON BOSWELL

The medal was presented to him at the Annual General Meeting held at Kempton Park in October 1974 by the Vice-President Prof. C F B Hofmeyr.

TOEKENNING

DIE S A V V SE GOUE MEDALJE VIR "UITMUNTENDE DIENS AAN DIE PROFESSIE" : DERDE TOEKENNING

Na ontvangs van 'n sterk gemotiveerde voorstel het die Raad in 1974 eenparig besluit om die S A Veterinêre Vereniging se hoogste toekenning te maak aan

DR JACK GORDON BOSWELL

Die medalje is tydens die Algemene Jaarvergadering in Oktober 1974 te Kempton Park deur die Vise-president Prof C.F.B. Hofmeyr aan Dr Boswell oorhandig.

Dr Jack Gordon Boswell

After graduation with the degree B V Sc at the University of Pretoria in 1930, he left for England and there studied under such illustrious persons as Sir Frederick Hobday and Professors John Wright and John Hickman before successfully undertaking his M R C V S examinations.

On his return in 1936 he established himself in private practice in Johannesburg. At that time practically all members of the profession were State employed and private practice was a hazardous venture. From a humble and often struggling start he soon proved that the public were prepared to pay for veterinary services of a high standard. As a result of hard work, strict adherence to ethically correct

Na verwerwing van die graad B V Sc aan die Universiteit van Pretoria in 1935 is hy na Engeland waar hy o.a. onder beroemdes soos Sir Frederick Hobday en professore John Wright en John Hickman homself verder bekwaam het en die eksamens vir M R C V S met sukses afgelê het.

In 1936 vestig hy hom in die privaatpraktyk in Johannesburg. Destyds was feitlik alle lede van die beroep in Staatsdiens, en privaatpraktyk was 'n waagstuk. Vanaf 'n beskeie en aanvanklik sukkelende begin het hy bewys gelewer dat die publiek bereid was om vir 'n goeie standaard van veeartsenydiens te betaal. Sy praktyk het gegroei as gevolg van harde werk, streng etiese en korrekte optrede teenoor

behaviour towards both his clients and colleagues and the exemplary standard of service he rendered to the animal industry as a whole, his practice grew. His reputation as man and as veterinarian was one on which he and his colleagues could be proud. As early as 1949 he installed the first X-ray apparatus used in a private veterinary practice. He also pioneered the use of A I and a bull station was set up on his premises at Sandown. He was adept at surgery and keen on demonstrating the operative techniques for Caslick's operation on mares and caeserian section on cows at a time when such operations were rarely performed.

His reputation as practitioner extended over a large part of the country and assistants and partners soon joined him. Later very many of them were to branch out on their own and establish practices throughout the country. He was always ready and willing to encourage, lead and inspire his colleagues in this direction. Today more than half the profession are self-employed practitioners. After 38 years of private practice he not only has the longest experience in this field but may justifiably be referred to as the "Father" of private practice in South Africa. He is held in great respect by his colleagues and the public.

But he was not only a busy practitioner. With a scholarship from the S A Jockey Club he proceeded to the U S A and U K to study infertility in the mare. He was twice invited to address international meetings on equine practice. He taught animal management and veterinary science at the University of the Witwatersrand for many years. He served as nominee of the Administrator on the Transvaal Peri Urban Areas Board. He became Charter President and District Governor of the Rotary Club and was chairman of the "Veld & Vlei Adventure School". He advised large mining companies both here and in Rhodesia regarding farming operations and was official Veterinary Surgeon to the Witwatersrand Agricultural Show Society. He also served as Official Veterinary Surgeon to the Turf Clubs of Germiston, Newmarket, Turffontein and Vaal.

Over and above the guidance and inspiration of his younger colleagues intending to practise, he also served the Association as member of Council for 10 years. He was behind the founding of the Association's oldest branch (Witwatersrand). He was primarily instrumental for recognition of the honorary title for veterinarians now universally accepted by the public and the profession. The traditional annual cricket match between Onderstepoort students and Boswell's team of graduates will also not be forgotten.

His contribution to the promotion and establishment of private practice in South Africa, and the distinguished service which he rendered to the profession as practitioner, has rightfully earned him the profession's highest award.

kollegas en kliënte, en 'n voorbeeldige dienslewering aan die dierenywerheid, groot en klein. Hy het as mens en as veearts 'n reputasie opgebou waarop hy en al sy kollegas trots kan wees. Hy het reeds in 1949 die eerste X-straal-apparaat in 'n privaat veeartsenykundige praktyk laat installeer. Hy was ook 'n pionier op die gebied van K I en 'n eerste "Bulstasie" is in 1949 op sy perseel te Sandown opgerig. Hy was 'n behendige chirurg en het graag Caslick se operasie op merries en die keisersnee op koeie gedemonstreer in 'n tydperk toe sodanige operasies nie algemeen onderneem is nie.

Sy reputasie as praktisyn het oor 'n groot gebied gestrek, en assistente en vennote moes noodwendig bykom. Van hulle het weer op verskeie plekke elders in die land privaatpraktyke gestig. Hy het aangemoedig en gehelp, en vandag is meer as 50% van die professie selfgeemplojeerd. Na 38 jaar van deurlopende praktyk is hy nie alleen die praktisyn met die langste ondervinding nie maar word ook met reg die "Vader" van privaatpraktyk genoem. Onder sy kollegas en die publiek is hy ongetwyfeld 'n man van aansien.

Maar hy was nie net 'n besige praktisyn nie. Hy het van die Jockey Club of S A 'n beurs ontvang om onvrugbaarheid by merries in die V S A en V K te bestudeer. Hy is tweekeer uitgenooi om buitelandse kongresse oor renperdpraktyk toe te spreek. Hy was vir etlike jare dosent in diereversorging en veeartsenykunde aan die Universiteit van die Witwatersrand. Hy was vir 12 jaar die Administrateur se genomineerde op die Transvaalse Raad vir Buitestedelike Gebiede. Hy was o.a. "Charter President" en "District Governor" van die Rotariërsklub en Voorsitter van die "Veld en Vlei Adventure School". Hy was vir baie jare adviseur vir die boerderyondernemings van groot mynmaatskappye in S.A. en Rhodesië. Vir die Witwatersrandse Skougenootskap het hy gedien as Amptelike Veearts, en ook vir die Renperdklubs te Germiston, Newmarket, Turffontein en Vaal.

Wat sy professie betref het hy ook sy plek volgestaan. Afgesien van die inspirasie en leiding wat hy aan voornemende jong praktisyns gegee het, het hy ook 10 jaar op die Raad van die S A V V gedien. Hy was die dryfveer agter die stigting van die Vereniging se oudste Tak (Witwatersrand). Hy was ook grootliks verantwoordelik vir die ere-betiteling wat die veearts en die publiek vandag as vanselfsprekend aanvaar. Die jaarlikse krieketwedstryd tussen Onderstepoort studente en Boswell se span van gegradueerdes sal ook nie vergeet word nie.

Sy bydrae tot die bevordering van die privaatpraktyk, en die uitmuntende diens wat hy as privaatpraktisyn aan die professie gelewer het, maak dat hy terdeë die allerhoogste onderskeiding verdien.

BOOK REVIEW

BOEKRESENSIE

VETERINÄRMEDIZIN UND INDUSTRIEMÄSSIGE SCHWEINEPRODUKTION

HARTWIG PRANGE UND JOST BERGFELD GUSTAV FISCHER VERLAG, JENA 1975 ppxx + 491 Figs 72 Tabs 175
Publ. Price DM 60,20

As the title indicates, this book details the veterinarian's role in industrialised pig production systems and his integration into the industry. Different aspects such as reproduction, breeding and performance testing, hygiene requirements, housing environment, nutrition, herd diagnostics, principles of assessing and reducing animal losses and health control are comprehensively dealt with. There is an adequate index.

A thorough knowledge of German is required to appreciate this text and particularly some of the rather involved tables presented.

S.K.E

Jl. S. Afr. vet. Ass. 47(3) 235-236 1976

OUR INCOMING PRESIDENT

ONS INTREDENDE PRESIDENT

DR A.P. (AWIE) SCHUTTE

Dr Awie Schutte, our incoming President, needs very little introduction to the many members who have heard his sincere and thought-provoking lectures at very many Branch and Group meetings throughout South Africa. His popularity as a speaker at our Congresses stems, not only from his professional knowledge and experience, but also from his warm personality and his willingness to serve his chosen profession. We, who have got to know him through his activities on Council and in committee work regard him as a willing horse, a man who knows exactly

Vir die groot getal lede wat al geluister het na die opregte en goeddeurdagte voordragte wat dr Awie Schutte tydens 'n veelvoud van Tak- en Groepvergaderings gelewer het, het ons pasverkose President eintlik geen voorstelling nodig nie. Sy gewildheid as spreker by ons kongresse spruit nie slegs uit sy beroepskundigheid en ondervinding nie maar ook uit sy vriendelike geaardheid en sy gewilligheid om sy gekose beroep te dien.

Diegene wat hom leer ken het as gevolg van sy werksaamhede op die Raad en verskeie komitees beskou

where he is going, who takes opposition in his stride and always seems to reach his goal. As chairman of the Production/Reproduction Group, as our representative on the A I Board and the Animal Production Advisory Council he has made a great impact on the cattle industry of our country and his many visits overseas in this connection have labelled him a world authority in his field. A great achievement was his Group's presentation last year of the refresher course on Reproductive Disorders and Fertility of Ruminants which was very well attended and resulted in the publication of an excellent proceedings book. As a member of the Educational Committee he has been intimately involved in preparing the memoranda submitted by the Association to different State departments and the University of Pretoria and the recent submission on the Specialist Register. He is at all times an avid protagonist of our Association and through his experience on Council and in the Executive Committee is au fait with all the activities of the Association.

After graduation (B.V.Sc. University of Pretoria) in 1959 he entered private practice. In 1961 he took a position in the Reproduction and A I section of the Veterinary Research Institute at Onderstepoort. In 1963 he became lecturer in the Department of Genesiology of the Faculty of Veterinary Science of the University of Pretoria. In 1964 he was awarded the degree M.Med.Vet (Gyn), and eventually proceeded to Belgium after winning the Agricura and B.P. scholarships for post graduate study. He worked at the Clinic for Reproductive Disorders at the University of Gent and the Veterinary Research Institute in Brussels, and in 1969 received the degree "Speciaal Doctoraat" from the University of Gent. He also undertook study tours to the USA, Israel and other parts of Europe. He returned to continue lecturing at the Faculty of Veterinary Science of the University of Pretoria until 1971, when he transferred to the Reproduction Section of the Veterinary Research Institute at Onderstepoort. He has 31 scientific publications to his credit and has read 35 papers at scientific meetings.

He lives in Pretoria with his wife Kinnie and their two daughters. I feel confident in handing over the presidency of our Association to such a competent, dedicated and enthusiastic colleague.

B.H. PAPPIN

September 1976. Outgoing President (1974 - 76)

hom as 'n baie gewillige perd, 'n doelgerigte man wat homself nie deur teenkanting laat stuit nie en altyd sy doelwit skyn te bereik. As voorsitter van die Produksie- en Reproduksiegroep en as ons verteenwoordiger op die K I Raad en die Adviesraad vir Diereproduksie, het hy reeds groot bydraes tot welvaart van ons beesnywerheid gemaak. Sy verskeie buitelandse reise in hierdie verband het hom as 'n deskundige van formaat in hierdie werksfeer bekendgestel. Die besonder suksesvolle verloop van sy Groep se besonder goedbegewoonde opknappingskursus in 1975 oor Probleme in verband met Reproduksie en Vrugbaarheid van Herkouers, en die daaropvolgende uitgawe van die handelinge daarvan, was een van die grootste bydraes. As lid van die Komitee oor Opleiding was hy intiem betrokke by die opstel van die omvattende memoranda wat die Vereniging aan verskillende Staatsinstansies en die Universiteit van Pretoria gestuur het. Die jongste voorlegging oor 'n Register van Spesialiste kom ook van sy hand. Hy is ten alle tye 'n vurige voorstander van ons Vereniging en is as gevolg van sy ondervinding op die Raad en die Uitvoerende Komitee ten volle bekend met al die werksaamhede van die SAVV.

Nadat hy in 1959 die B.V.Sc.-graad aan die Universiteit van Pretoria verwerf het, het hy vir tweejaar privaat gepraktiseer alvorens hy die Seksie Reproduksie en K.I. aan die Navorsingsinstituut vir Veeartsenykunde te Onderstepoort aangesluit het. In 1964 is die graad M.Med.Vet (Gyn) aan hom toegeken. Daarna is hy met die hulp van die Agricura- en B.P.-studiebeurse na België. Vir twee jaar was hy werksaam by die Kliniek vir Voortplanting Steurnisse aan die Universiteit van Gent en by die Navorsingsinstituut vir Veeartsenykunde te Brussels. In 1969 ontvang hy die "Speciaal Doctoraat" van die Universiteit van Gent. Sy studiereise het hom ook na Europa, Israel en die VSA geneem. Hy was vanaf 1963 tot en met 1971 aan die Departement Geslagskunde van die Fakulteit Veeartsenykunde van U.P. verbonde en was onder ander breier van die Onderstepoort-studenterugbyspan.

Altesaam 31 gepubliseerde artikels het uit sy pen gevloei en hy het 35 lesings en 31 voordragte by wetenskaplike byeenkomste gelewer.

Hy is 41 jaar en woon in Pretoria met sy vrou Kinnie en hulle twee dogters. Hy beklee tans die pos van Hoof van die Seksie Voortplanting aan die Navorsingsinstituut vir Veeartsenykunde te Onderstepoort.

Dis met die volste vertroue dat ek die Presidentskap van ons Vereniging oorhandig aan 'n toegewyde, bekwame en entoesiastiese kollega.

B.H. PAPPIN

September 1976. Uittredende President (1974 - 76)

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