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JOURNAL OF THE SOUTH AFRICAN VETERINARY ASSOCIATION

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

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Jl. S. Afr. vet. Ass. 47(4) 239-240 (1976)

EDITORIAL

THE PROBLEM OF PEOPLE AND THEIR PETS

Recent articles and correspondence in the veterinary, medical and lay press, as well as in programmes on radio and television, have focused attention on the many problems associated with the pet animals which people keep.

People have kept pets from the earliest times. Such animals provide their owners with many things. These include companionship and affection, often as a substitute for human companionship. Pets supply status and/or gratify the ego. They meet the need which people have to possess something to care for. They provide joy and recreation. They also serve as protectors of life and property, assist the handicapped, meet certain educational needs and even provide specific therapy for certain mental conditions.

The advantages and value of pet ownership must of course be balanced against certain other considerations. There are economic and environmental considerations, the need to comply with community laws and regulations, the attitudes and requirements of neighbours in particular and the community in general. To this must be added the important consideration of the health of animals and people.

Whether pet ownership is a right or a privilege may be debated. The owner must, however, always respect the rights of the community. No pet owner has the right to impose on his fellowman any nuisance, risk or health hazard.

That South African dogs do often carry a helminthic burden which is a hazard to man is clear from the results of survey covering 873 dogs from various parts of the country. Of these, 22,7% were positive for *Dipylidium caninum*, 2,2% for *Taenia multiceps*, 1,2% for *Echinococcus granulosus*, 16,3% for *Toxacara canis* and 21,3% for *Ancylostoma spp.* It is indeed fortunate that a range of extremely effective and safe anthelmintics are now available to the profession.

The numerical explosion in the cat and dog populations is cause for great concern, as this greatly multiplies the humane, social, community and zoonotic aspects of pets. It is this *irresponsible pet ownership* which is essentially responsible for the problems.

There can be little doubt that greater but reasonable control by local authorities is desirable in order that any anti-social behaviour by pet owners may be curbed or prevented.

As a profession we are intimately involved, and we certainly carry our own particular share of responsibility. This we assume and shoulder. It is our responsibility to strongly advocate and encourage the concept of responsible pet ownership, and support, assist and enable pet owners to meet their obligations in this regard. This includes surgical and hormonal control of breeding, advice on training and management in general, prophylactic immunisation against infectious and zoonotic diseases, regular anthelmintic treatment, etc. To be able to shoulder our responsibilities we must not only ensure that we keep

VAN DIE REDAKSIE

SONDE MET DIE HONDE

Onlangse artikels en briefwisseling in die veeartsenykundige, mediese en lekepers, asook programme op die radio en televisie, het die aandag gevestig op die veelvoudige probleme verbonde aan die aanhou van honde en ander troeteldiere.

Sedert die vroegste jare hou mense troeteldiere aan en wel om verskeie redes. Diere gee aan hul eienaars geselskap, soms as surrogaat vir menslike geselskap. Hulle verskaf ook status en/of bevredig die ego. Sommige mense vind dit ook nodig om iemand of iets te moet versorg. Diere verskaf ook genot en ontspanning. Maar diere dien ook as beskermers en bewakers van lewe en eiendom, is behulpsaam aan grestremdes, voldoen aan opvoedkundige vereistes en word ook as spesifieke terapie vir sekere geestestoestande beskou.

Vanselfsprekend moet die voordele en waarde van die besit van 'n dier opgeweeg word teen sekere ander oorwegings soos die ekonomiese en dié rakende die omgewing, die noodsaaklikheid om aan sekere gemeenskapswette en -regulasies te voldoen, en die houding en vereistes van bure in besonder en die gemeenskap in die algemeen. Hiertoe moet bygevoeg word die belangrike aspekte rakende die gesondheid van die dier en die mens.

Daar kan verskil van mening wees oor die vraag of die besit van 'n troeteldier 'n reg of 'n voorreg is. Dis egter onteenseglik waar dat die eienaar altyd die regte van die gemeenskap moet eerbiedig. Geen troeteldiereienaar het die reg om enige beslommernis, ongerief, risiko of gesondheidsgevaar op sy medemens af te dwing nie.

Dat honde in Suid-Afrika wel soms gesondheidskadelike helminte dra word duidelik afgelei deur die resultate van 'n opname van 873 honde uit verskillende dele van die land. Hiervan was 122,7% besmet met *Dipylidium caninum*, 2,2% met *Taenia multiceps*, 1,2% met *Echinococcus granulosus*, 16,3% met *Toxacara canis* en 21,3% met *Ancylostoma spp.*

Gelukkig is daar tans 'n reeks besonder veilige en doeltreffende wurmmiddels aan die professie beskikbaar.

Die aspekte van dierebesit wat gaan oor die humane, sosiale, gemeenskaps- en soönotiese word veelvoudig vergroot deur die numeriese bevolkingsontploffing onder katte en honde. In essensie is dit hierdie *onverantwoordlike besitters van diere* wat grootliks vir die probleem verantwoordelik gehou moet word.

Daar bestaan geen twyfel dat redelike beheer deur die plaaslike besture wel met voordeel uitgebrei kan word ten einde die antisosiale gedrag van diereeienaars aan bande te lê.

As beroep is ons intiem gemoeid, en moet ons ook ons besondere verantwoordelikhede dra. Dit wil ons ook doen. Dit is byvoorbeeld ons taak om die begrip van verantwoordelike dierebesit voor te staan en aan te moedig. Ons moet eienaars van diere ondersteun, help en instaat stel om hulle verpligtinge in dié verband na te kom. Dit behels chirurgiese en hormonale

ourselves up to date on the relevant information, but that we also speak out and do what is necessary in this regard. Part of our professional obligations is concerned with intensive public education. This should be directed at the public so that they become aware of the nature of the problem, and as pet owners to make them realise their commitments and responsibilities and to accept a *code of conduct*. This is based on *responsible pet ownership.*

The following points are essential to such a code:

Limit the number of pets; discourage casual ownership; accept the responsibility of ownership; prevent the environment and public places from becoming polluted with pet excrement; restrict entry into parks; accept compulsory leashing of dogs outside the owner's premises; accept compulsory identification of owner by means of collar and tag; respect antifouling regulations for pavements and public places; prevent pet animal proliferation and so make destruction of offspring unnecessary.

As a profession we must accept the prime responsibility for indicating steps to prevent the public health hazards associated with owning pet animals. We should never fail to educate the public in general and pet owners in particular on diseases transmissible from pets to man. We should eliminate parasitic zoonoses and helminths by effectively treating and preventing diseases like toxoplasmosis, ancylostomiasis, hydatidosis and similar diseases. We must ensure that dogs are vaccinated against leptospirosis, rabies, etc. Lastly, we must eliminate or control anthropod and fungal infections. We must also promote a satisfactory standard of meat hygiene to prevent many of the parasitic zoonoses such as echinococcosis. In addition we need to play our full part in preventing cruelty and neglect of animals and in controlling population growth.

beheer van voortplanting, advies oor afrigting en versorging in die algemeen, voorkomende immunisering teen besmetlike en soönotiese siektes, gereelde behandeling teen helminte, ens. Ten einde hierdie taak te kan behartig moet die veearts nie alleen op hoogte van sake bly nie, maar ook nie sal aarsel om daaroor te praat of daadwerklik op te tree nie. 'n Deel van ons professionele verpligtinge behels intensiewe voorligting van die publiek. Dit is nodig sodat hulle van die aard van die probleem bewus word.

Dit is nodig sodat eienaars van diere hulle verpligtinge en verantwoordelikhede sal besef en 'n *gedragskode* sal aanvaar. Dit berus weer op *verantwoordelike dierebesit.*

Die volgende punte is vir so 'n kode noodsaaklik:

Beperk die getal diere; ontmoedig toevallige besit van diere; aanvaar die verantwoordelikheid van dierebesit; voorkom besoedeling van die omgewing en openbare plekke met dierlike ontlasting; beperk toegang tot parke; aanvaar verpligte gebruik van leitoue wanneer honde buite die eienaar se perseel beweeg; aanvaar verpligte identifikasie van die eienaar d.m.v. plaatjies op halsbande; gee gehoor aan regulasies wat ontlasting op sypaadjies verbied; voorkom die aanwas van troeteldiere sodat die afmaak van die nakomeling onnodig word.

As lede van 'n verantwoordelike professie moet ons ook die voortou neem om die moontlike gesondheidskadelike fasette van dierebesit uit te skakel. Ons moet geen steen onaangeroerd laat i.v.m. die inlig van die publiek oor siektes wat van dier na mens oordraagbaar is nie. Protosoïese of helmintiese toestande soos toxoplasmose, ancylostomiase, en echinokokkose, moet voorkom of doeltreffend behandel word. Diere moet geïmmuniseer word teen soönoses soos leptospirose en hondsdolheid, en ektoparasiete en swambesmettings moet uitgeskakel of beheer word. Ons moet aandring op 'n bevredigende standaard van vleishigiëne om van die parasitêre soönoses soos echinokokkose te vermy. Daarby moet ons ons rol vervul in die voorkoming van verwaarlosing en mishandeling van diere.

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INFORMATION

INLIGTING

PREVENTING BEAK NECROSIS IN POULTRY

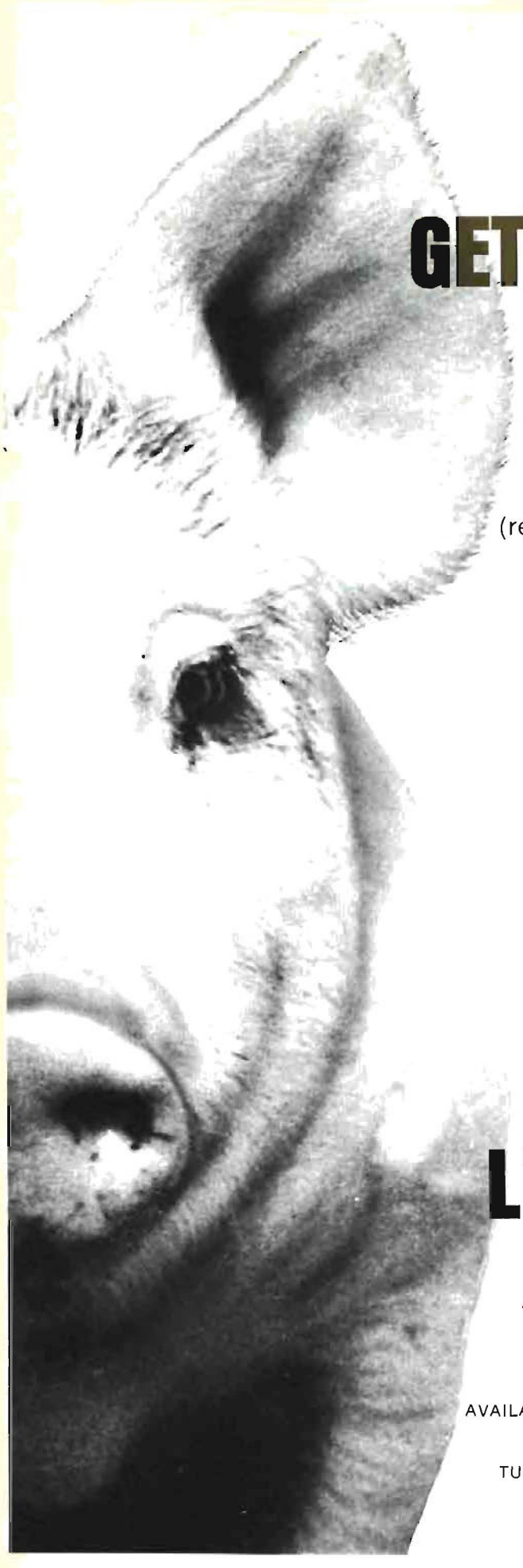
Scientists at the Agriculture Canada Research Station at Lethbridge, Alberta, have suggested that a simple change in ration from mash to pelleted feed may be all that is required to prevent beak necrosis, a poultry disease which, in extreme cases, results in mortality. Mortality percentages as great as 10% 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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71ste ALGEMENE JAARVERGADERING EN TWEE-DAAGSE GROEPSIMPOSIA, SAVV, BLOEMFONTEIN, SEPT.—OKT. 1977

71st ANNUAL GENERAL MEETING AND TWO-DAY GROUP SYMPOSIA, SAVA, BLOEMFONTEIN, SEPT.—OCT. 1977

PROFESSOR G.D.B. DE VILLIERS
VISE-REKTOR / VICE-PRINCIPAL
UNIVERSITEIT v.d. O.V.S. / UNIVERSITY of the O.F.S.

Dit is vir my baie aangenaam en 'n besondere voorreg om namens die Raad en Senaat van die Universiteit van die Oranje-Vrystaat u almal wat die 71ste Algemene Jaarvergadering en die Groepsimposia van die Suid-Afrikaanse Veterinêre Vereniging bywoon, baie hartlik welkom te heet op die kampus van die Universiteit van die Oranje-Vrystaat.

Eintlik is dit 'n historiese gebeurtenis aangesien dit die eerste keer in die bestaan van die S.A. Veterinêre Vereniging is dat die Algemene Jaarvergadering in die Vrystaat gehou word en dat die Universiteit van die Oranje-Vrystaat as gasheer optree. Ons wil vertrou dat die geriewe wat die Universiteit u aanbied u so sal bevredig en dat u simposia so suksesvol sal verloop dat dit nie die laaste keer sal wees dat u in die Vrystaat, hartland van die Suid-Afrikaanse boerderystreke, sal vergader nie.

This is not the first time that I address a gathering of veterinary scientists. On the 25th June, 1963, just about a year after being appointed Dean of the Faculty of Agriculture, I had to welcome the participants attending the Orange Free State, Lesotho and the Northern Cape branch of the S.A. Veterinary Association. On that day I expressed the view that if the authorities should decide on a second Faculty of Veterinary Science, Bloemfontein should be seriously considered for this purpose.

The shortage of veterinarians is in fact a universal occurrence and has already received serious consideration from world bodies such as W H O and F A O of the United Nations. At a meeting held by representatives from 32 countries in 1965, two fundamental considerations were evident: the continuing shortage of veterinarians in most areas and the paramount importance of maintaining standards of education while striving to overcome the shortage, whether by increasing the output of existing schools or by establishing new ones.

A few years ago the authorities in South Africa decided to enlarge the facilities at Onderstepoort so that about 90 entrants per year could be admitted. And just recently it has been made known that a Faculty for the training of non-white veterinarians will also be established.

Met 'n beesbevolking van slegs ongeveer 12 miljoen en 30 miljoen skape, 'n verwagte tekort van 2 miljoen beeste en 6 miljoen skape in 1980, en gelet op die feit dat die bees- en skaapvleisproduksie gedurende die afgelope 15 jaar met slegs 0,9 persent per jaar toegeneem het wat hoegenaamd nie voldoende was om tred te hou met die snel toenemende vraag na vleis nie, word die belangrikheid van die veeartsprofessie vir die veebedryf in Suid-Afrika baie sterk na vore gebring. Die ongelukkige toestand bestaan, veral op die platteland, dat plaasdiere ter waarde van miljoene rand tot op die huidige stadium min of geen veeartsenykundige toesig geniet nie.

Die betekenisvolle rol van die veearts rondom die probleem van die groeiende bevolking kan nie genoeg beklemtoon word nie. Sy taak sien ek nie net daarin om die kuddes gesond te hou nie, maar ook om 'n wesentlike bydrae te lewer tot produksievermeerdering deur hoër vrugbaarheid, verbeterde voeding en bestuurspraktyke te help bevorder.

Daar bestaan vandag veral 'n knellende tekort aan veeartse op die platteland. Onwillekeurig kan die vraag gestel word of dit slegs die gevolg is dat te min persone tot die veeartsenykunde toetree en of ander faktore, soos die ekonomie, ook 'n rol speel. In laasgenoemde geval kan die probleem miskien verlig of uitgeskakel word deur veeartse op die platteland met staatshulp in die vorm van 'n basiese salaris aan te stel, met die doel om staats- sowel as privaatwerk te behartig. Verbeterde geriewe soos grootdierhospitale en 'n versekerde basiese inkomste behoort die toestand aansienlik te verlig.

Indien opleiding die grootste probleem is, ontstaan die vraag onwillekeurig of die opleiding van veeartse in 'n plattelandse omgewing, dit wil sê waar die werklike behoefte bestaan, nie meer potensiële veeartse na die platteland sal trek waar die grootste leemte tans bestaan nie.

Die oprigting van 'n tweede Fakulteit van Veeartsenykunde op die platteland behoort nie die wêreld se geld te kos nie. Ook kan die oprigtingskoste aansienlik verminder word indien dit gekoppel word aan 'n bestaande landboufakulteit wat reeds oor die personeel en fasiliteite beskik om onderrig in sekere veeartsenykundige vakke te gee en ook oor proefplaasgeriewe beskik.

Die oprigting van 'n fakulteit op die platteland sal nie net meer veeartse oplewer nie, maar sal ook belangstelling stimuleer in aangeleenthede wat tiperend is van daardie gebied en sal veral navorsing aanwakker, veral as 'n mens in aanmerking neem dat ons land een van die lande is wat ryklik met veesiektes bedeeld is. Meer studente sal van ander provinsies getrek word met die nodige landbou-agtergrond.

Meer dosente in veeartsenykunde sal ook beter geleenthede vir gedagte- en inligtingswisseling skep en meer poste sal bevordering in plaas van bedankings aanhelp.

As iemand wat persoonlik gemoeid is met opleiding op 'n breë vlak is ek van mening dat die tyd aangebreek het om 'n meer progressiewe opleidingsbeleid in veeartsenykunde te volg in ooreenstemming met wat in die res van die gevestigde wêreld gebeur. Die

veearts sal in die toekoms 'n nog veel meer belangrike en onontbeerlike skakel in die produksiemasjien wees. Dit is die taak van almal gemoeid met die vooruitgang van die land, veral op landbougebied, om toe te sien dat reg geskied aan die veeartsberoep, sy probleme en knelpunte. U het vandag en môre die geleentheid om oor u probleme te besin en ons vertrou dat u besprekings vrugbaar en opbouend sal wees tot voordeel van ons hele land.

Congress Proceedings:

After the A.G.M. the Public Health, Rural Practitioners and Reproduction Groups consecutively presented a series of scientific papers. The occasion was concluded with the customary social occasion which was enjoyed by all.

Our new president Dr Awie Schutte, receives the chain of office from his predecessor Dr Basil Pappin.

Ons nuwe president, Dr Awie Schutte, ontvang die ampsketting van sy voorganger, Dr Basil Pappin.

Dr Jack Mason ontvang die SAVV Goue Medalje van die President.

Dr Jack Mason receives the SAVA Gold Medal from the President.

BOOK REVIEW

BOEKRESENSIE

VETERINARY REPRODUCTION AND OBSTETRICS

FORMERLY WRIGHTS' VETERINARY OBSTETRICS

G.H. ARTHUR

Fourth Edition. Baillière Tindall, London 1975. pp viii + 616, Plates 208, Figs 99, Tabs 28, Publ. Price R18,50

In the course of the 37 years since this book was first published many changes have occurred — not only its title and the authors but also its content. The latter has been considerably expanded and updated. The bibliography at the end of each chapter has been augmented and brought up to date and two additional chapters on "The Veterinary Control of Herd Infertility" and "Artificial Insemination" have been included. The book has been written essentially for the under-graduate veterinary student but it would also meet the requests for updated knowledge in this field by practising veterinarians. I recommend this book to these groups.

I look forward to future editions having sub-chapters on the "Veterinary Control of Herd Fertility" in sheep, horses, pigs and even dogs.

The book is divided into 5 parts, each dealing in the main, with:

1. The reproductive physiology of the different species together with the occurrence of pregnancy — its diagnosis and complications and finally parturition — essentially the physiology in the various species;
2. Pathological parturition. An excellent table on the causes of dystocia has been added to the text;
3. Surgical relief of dystocia as also with puerperal complications;
4. Infertility in its various forms in the different species; and
5. The physiology as well as the pathology of reproduction in male animals, and artifical insemination.

The photo on p 326 of the site of incision for equine Caesarian operation, is in my view indicative of the type of improvement which has been brought about in many places in this new edition. Likewise there is a new table on ovulatory dysfunction on p 396. On p 415 vibriosis is discussed and while the new edition does refer to *V. foetus venerealis* and *V. foetus intestinalis* it is a pity that no reference is made to the new name of *Campylobacter foetus*. The value of immunisation of bulls in an outbreak of vibriosis, is discussed.

J.S. v. H.

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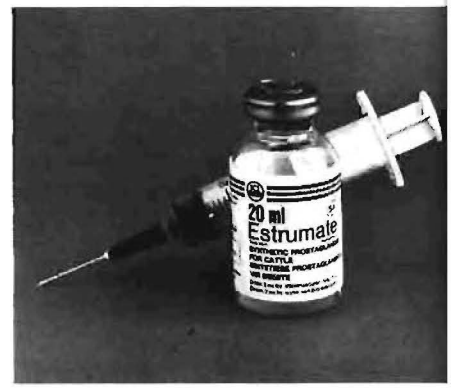

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LIVE MASS GAINS AND WOOL PRODUCTION OF MERINO SHEEP: THREE TREATMENT PROGRAMMES FOR PARASITE CONTROL

I.G. HORAK*, M.R. HONER** and J. SCHRÖDER**

ABSTRACT: Horak, I.G., Honer, M.R., Schröder, J. **Live mass gains and wool production of Merino sheep: Three treatment programmes for parasite control.** *Journal of the South African Veterinary Association* (1976) **47** No. 4, 247-251 (En) Faculty of Veterinary Science, 0110 Onderstepoort, Republic of South Africa.

A trial is described comparing the live mass gains and wool production of three groups of Merino sheep treated either at four-weekly intervals, strategically or not at all for the control of helminths and *Oestrus ovis* during a 19 month period. The group treated at four-weekly intervals had the greatest gain in mass during the first 7 months of the trial, but the overall gain in mass was not greater than that of the group treated strategically. At both shearings the four-weekly treated group produced more wool than did the strategically treated group. The latter group produced more wool in total than did the untreated group. The seasonal incidence of nematodes, cestodes and *Oestrus ovis* on the trial farm was determined by the slaughter of tracer lambs at regular intervals.

INTRODUCTION

Chemotherapeutic treatment is today the most common method of controlling helminth and *Oestrus ovis* infestations in sheep. Of the various dosing regimens practised, strategic drenching is probably the most valuable, the strategy being based on the seasonal incidence of the parasites or prevailing animal husbandry practices[1,3]. Because of the range of differing situations encountered in South Africa, a number of drenching programmes have been suggested, based on the seasonal occurrence of parasites and the husbandry practised in the various regions[1-14-17].

The beneficial effect of drenching sheep for nematode infestations has been well documented[3,9,15]. The effects of dosing for cestode infestations have been more variable, some trials demonstrating favourable results[5,16], while in others no benefits could be determined[5]. The control of *O. ovis* infestation by regular treatment has resulted in increased gains in the live mass of lambs[10].

The present paper describes a trial conducted in sheep on a farm near Tonteldoos in the Dullstroom district of the Eastern Transvaal Highveld. In it the live mass gains, wool production and faecal worm egg counts of two groups of Merino sheep treated for nematode, cestode and *O. ovis* infestation at four-weekly intervals or strategically are compared with those of a group of untreated control sheep.

MATERIALS AND METHODS

Selection of Trial Animals

On 18 May 1973, 523 unweaned Merino lambs, estimated to be over 10 kg in live mass and approximately 2½ months of age were selected from a flock of 560 lambs. These lambs were weighed, sexed and identified by means of numbered eartags. They and their dams were placed on a pasture containing mixed natural grasses, maize and oats, which had not previously been grazed by sheep.

A week later the lambs were treated with cambendazole (BONLAM : MSD) for suspected tapeworm infestation. On this occasion and another week later, the lambs were weighed, the total gain in mass calculated and the experimental animals selected.

The following criteria were used in the selection of 147 ewe and 123 ram lambs:

(i) Obviously healthy animals;
(ii) Gain in mass in excess of 0,5 kg during the previous 14 days;
(iii) Range in mass from 11,8 kg to 15,9 kg for ewe lambs and 11,8 kg to 16,4 kg for ram lambs.

The selected ewe and ram lambs were ranked separately according to their live masses and were allocated in sequence, starting from the lightest lamb, to one of the three groups of ewe and ram lambs. Each group contained 49 ewe lambs and 41 ram lambs. Each lamb now received an additional coloured eartag indicating the group it had been allotted to, the 3 colours being orange, white and pink.

Treatment

The group with orange eartags were treated with cambendazole and rafoxanide (RANIDE : MSD) at four-weekly intervals until they were 1 year old, when both anthelmintics were replaced by a combination of thiabendazole and rafoxanide (RANIZOLE : MSD) and the four-weekly treatments continued until the conclusion of the trial.

The group with the white eartags were treated strategically, as recommended in a dosing guide[1]. They were drenched with cambendazole on 8 August 1973 and 3 October 1973, thiabendazole (THIBENZOLE : MSD) on 28 December 1973, 15 May, 7 August and 3 October 1974 and with rafoxanide on 28 November 1973, 20 February, 17 April, 12 June and 27 November 1974.

The group with the pink eartags was kept as untreated controls.

Husbandry

The experimental lambs grazed as one flock with the remainder of the lambs, which were treated strategically with the same anthelmintics at the same times as the group with the white eartags. At two-weekly intervals faeces were collected for nematode egg counts and larval cultures from the same ten ewe and ram lambs from each group.

The egg counts were performed using the modification of the McMaster technique of Gordon and Whitlock[4], described by Reinecke[13].

The live mass of each of the experimental lambs was measured at four-weekly intervals. The ram lambs were castrated and all the lambs were docked

* Faculty of Veterinary Science, University of Pretoria, Box 12580, 0110 Onderstepoort, Republic of South Africa
** MSD Research Centre, Hennops River, Private Bag 3, 1685 Halfway House, Republic of South Africa.

and weaned during July 1973. They were shorn on 21 January 1974 and on 15 January 1975, at the termination of the trial. At shearing, greasy fleece masses were recorded, but the wool was not classed.

The live mass gains and fleece masses were analysed by analysis of variance for a completely randomized design.

All mortalities were recorded during the course of the trial, but because of the distance between the trial location and the laboratory, necropsies could not be performed. All data from the dead sheep were excluded from the trial results.

Nutrition

Immediately after weaning, the lambs were put into a small camp of kikuyu grass *(Pennisetum clandestinum)* where their diet was supplemented with lucerne hay and maize meal. During September 1973, they were put on an *Eragrostis* pasture and during October they were inoculated against enterotoxaemia and put out on natural pasture. In May 1974, they were shifted to a rested *Eragrostis* pasture from where they were moved back to the kikuyu camp during July and fed supplements. The lambs were again put out on natural grazing during October 1974.

Tracer lambs

At approximately four-weekly intervals, three lambs, free from helminth and *O. ovis* infestations, were grazed with the experimental lambs for a period of six weeks and then slaughtered for helminth and *O. ovis* recovery.

RESULTS

Live Mass Gains

The mean live mass gains are graphically illustrated in Figure 1.

On 21 January 1974, when the trial had been in progress for seven months, the mean live mass gain of the four-weekly treated group was significantly higher than that of the other two groups, which did not differ significantly ($p = 0,05$). The live mass gains of the two treated groups did not differ significantly over the total trial period due to more rapid gain in mass of the strategically treated group during the last year of the trial. The total gain in mass of the untreated group was significantly lower ($P < 0,05$) than that of either of the treated groups.

Wool production

The mean greasy fleece masses of the two wool clips are summarized in Table 1.

Table 1: THE MEAN GREASY FLEECE MASSES (kg) OF THE THREE GROUPS OF SHEEP

Treatment	First Clip	Second Clip
Four-weekly	2,63a	3,69a
Strategic	2,37b	3,54a
Untreated	2,42b	3,27b

Values with different superscripts within a column are significantly different (p=0,05) from each other

The first clip of the group treated at four-weekly intervals was significantly higher ($P < 0,05$) than that of the other two groups which did not differ significantly from each other. At the second shearing, the mean fleece masses of the two treated groups did not differ significantly, but were significantly higher ($P < 0,05$) than that of the control group.

Mortality

Ten lambs in the four-weekly treated group died, five in the strategically treated group and 16 in the

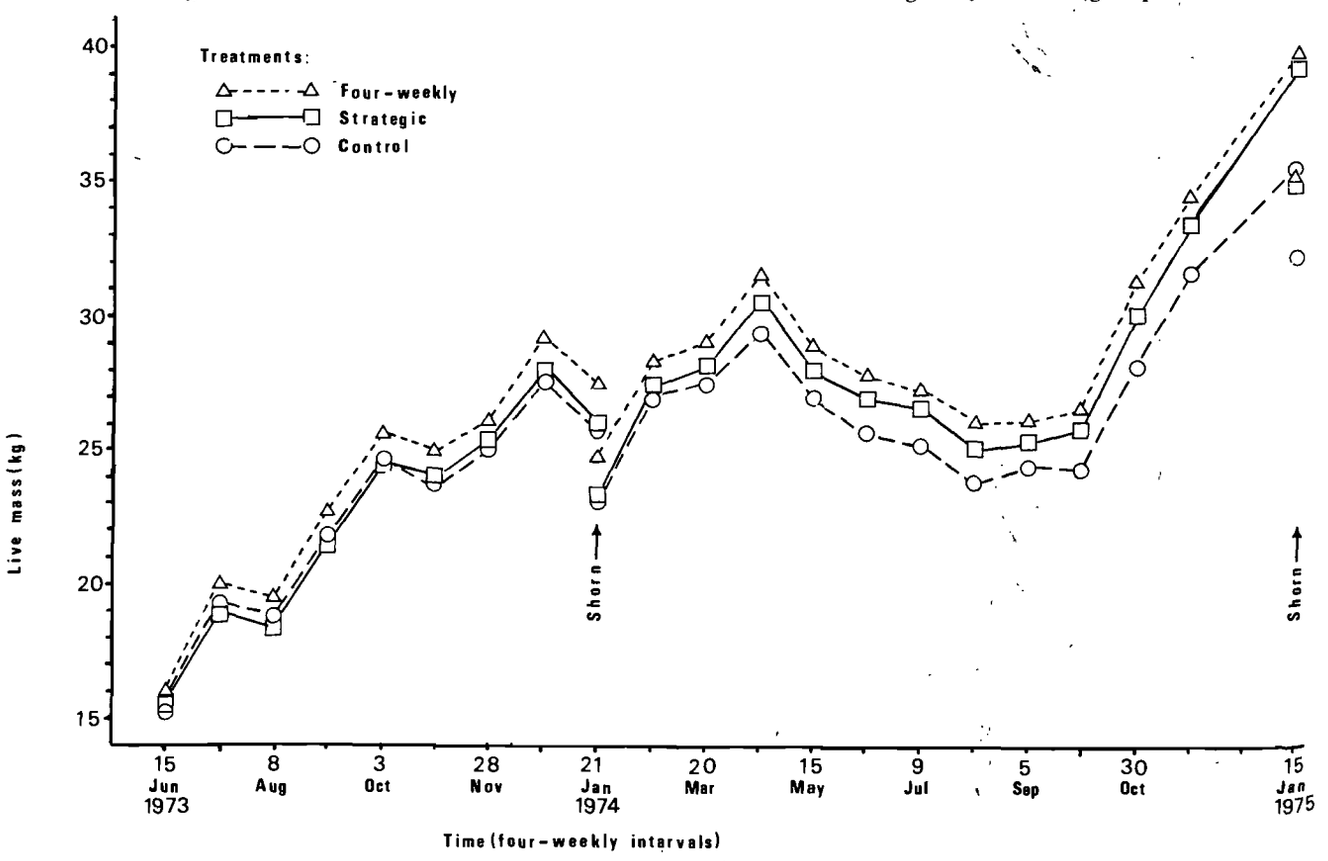

Fig.1: Live mass gains of Merino sheep on three dosage regimens

control group. As necropsies could not be performed, the role of parasitism in these deaths could not be determined.

Nematode egg counts

The mean monthly faecal egg counts, expressed as eggs per gram of faeces ("e.p.g."), are graphically illustrated in Figure 2.

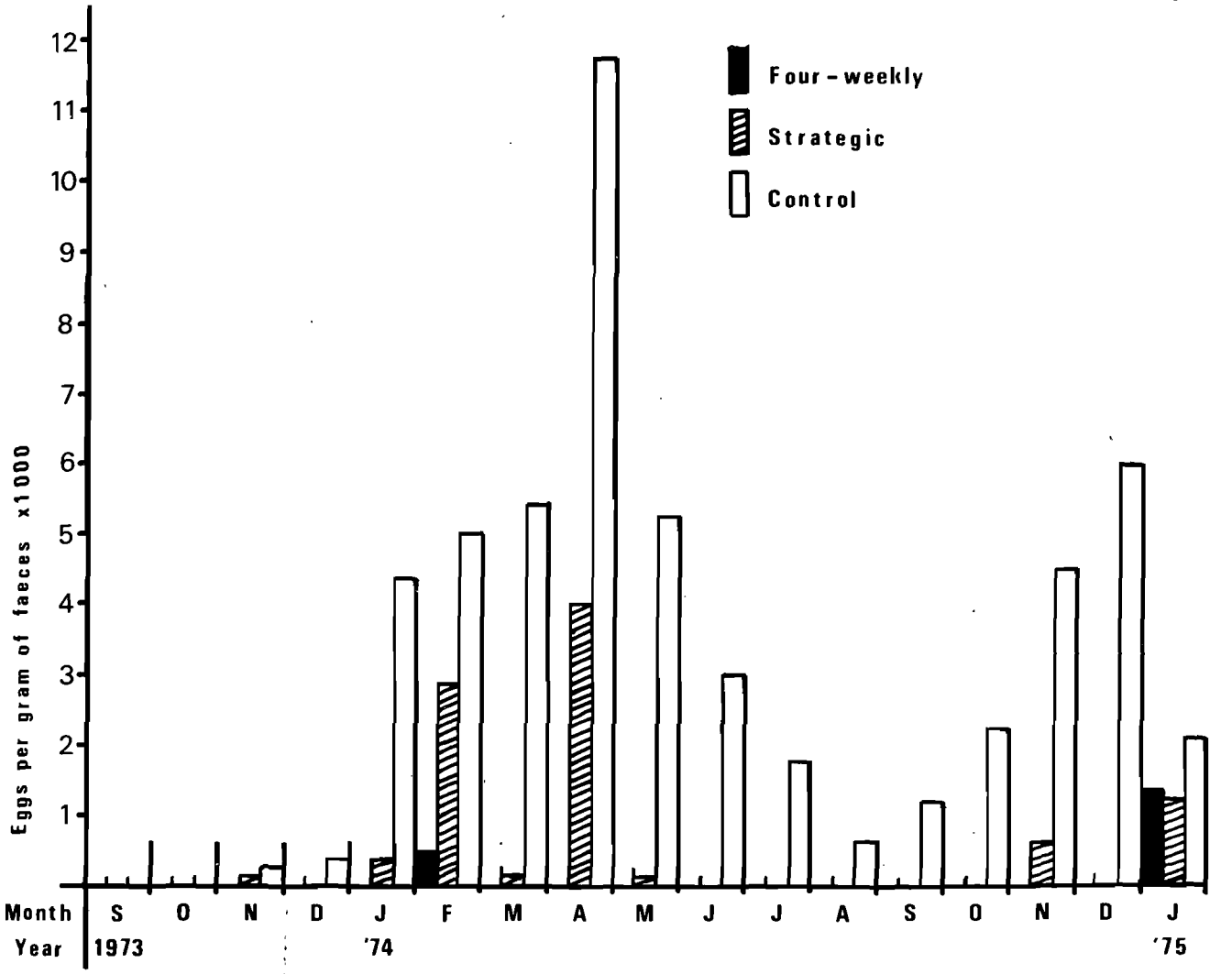

Fig. 2: Mean monthly faecal egg counts

Total faecal worm egg counts in the control group rose rapidly between December 1973 and January 1974 and reached a peak of 11 713 e.p.g. during April. Counts declined steadily thereafter to reach 584 e.p.g. during August, followed by a consistent rise to peak at 6 031 e.p.g. in December 1974 falling to 2 146 e.p.g. at the conclusion of the trial.

The total mean egg counts of the strategically treated group generally followed the pattern of those of the control group, but were considerably lower and fairly erratic due to the intermittent anthelmintic intervention.

The group treated at four-weekly intervals exhibited egg counts of consequence only during February 1974 and January 1975. The latter count occurred because no treatment was administered after 27 November 1974.

Haemonchus contortus was responsible for the major portion of the egg counts at all seasons of the year in all three groups. In both the strategically treated group and the control group *Ostertagia* spp. and *Trichostrongylus* spp. larvae were fairly regularly present in faecal cultures from December 1973 onwards.

Tracer lambs

The mean worm and *O. ovis* burdens of the tracer lambs are summarized in Table 2. Only data for significant genera are presented as these findings will be published separately in greater detail[7,8].

A number of tracer lambs failed to adapt to conditions on the pasture and died, thus accounting for the variation in numbers necropsied at each occasion.

In both years of the survey larger numbers of *H. contortus* were recovered from the lambs slaughtered during January than in the preceding five months. Burdens of fourth stage larvae and adult worms both reached a peak in the lambs slaughtered during April 1974. Fourth stage larvae were responsible for increasing proportions of the total *H. contortus* burden from January until June 1974 and constituted the major portion of this burden until October 1974.

Ostertagia circumcincta was recovered from all sheep slaughtered from January 1974 onwards. Fourth stage larvae were responsible for the major portion of the worm burden from autumn to spring, while adults predominated during the summer.

Trichostrongylus spp. *(T. axei, T. colubriformis* and *T. rugatus)* were recovered in the greatest number from the sheep slaughtered from March to June.

The sheep slaughtered from November to the beginning of May generally harboured the largest *Cooperia* spp. burdens *(C. pectinata* and *C. punctata).*

Oesophagostomum columbianum was recovered

Table 2: MEAN NUMBERS OF PARASITES RECOVERED FROM THE TRACER LAMBS

No. of Lambs	Date Exposed	Date Slaughtered	Mean numbers of parasites recovered: *H.contortus* 4th	*H.contortus* Adult	*O.circumcincta* 4th	*O.circumcincta* Adult	*Tric. spp.* 4th	*Tric. spp.* Adult	*Cooperia spp.* 4th	*Cooperia spp.* Adult	*O. col.* Larvae& Adult	*Trichur.* Larvae& Adult	*Moniezia* Scoleces	*O. ovis* Larvae
3	11 Jul 73	24 Aug 73	0	0	0	0	0	1	0	0	0	26	0	0
3	8 Aug	21 Sept	0	0	0	0	0	0	0	0	0	149	0	0
2	5 Sept	19 Oct	1	0	0	0	0	1	0	0	0	46	3	3
3	3 Oct	16 Nov	1	22	0	0	1	26	0	9	0	5	8	8
3	31 Oct	14 Dec	5	26	0	0	0	19	1	2	1	9	2	0
2	28 Nov	14 Jan 74	34	199	2	11	4	24	3	18	1	3	6	0
3	28 Dec	8 Feb	696	920	9	39	6	51	5	45	1	3	50	11
3	19 Jan 74	7 Mar	1 428	1 293	43	58	25	263	28	70	1	8	10	1
3	20 Feb	5 Apr	2 846	1 835	113	57	4	200	24	16	1	10	7	0
2	20 Mar	3 May	392	21	38	20	38	98	44	21	0	5	1	0
3	17 Apr	30 May	119	9	40	47	6	219	0	2	0	9	1	4
3	15 May	28 Jun	87	1	38	2	12	183	1	1	1	7	1	0
2	12 Jun / 9 Jul	9 Aug	27	3	36	4	0	84	1	0	0	7	0	0
1	9 Jul	6 Sept	36	2	52	13	0	24	0	1	0	1	0	0
2	7 Aug	23 Sept	60	8	42	13	1	13	0	0	0	5	1	0
3	4 Sept	21 Oct	25	14	10	43	1	3	0	1	0	9	6	4
3	3 Oct	18 Nov	0	86	1	43	6	58	37	50	0	10	3	4
2	30 Oct	17 Dec	5	48	0	9	6	20	8	54	0	3	2	19
3	30 Oct / 27 Nov	17 Jan 75	64	626	1	107	2	63	4	43	0	6	3	1

4th = Fourth stage larvae, *O. col.* = *O. columbianum*, *Trich.* spp. = *Trichostrongylus* spp., *Trichur.* = *Trichuris* spp.

from some sheep slaughtered from December 1973 to April 1974 and during June 1974.

Trichuris spp. were present in all groups of tracer lambs, peak burdens being recovered from the lambs slaughtered from August to October 1973.

Moniezia expansa were recovered from lambs slaughtered from October 1973 to June 1974 and from September 1974 to the conclusion of the trial in January 1975.

With the exception of *Trichuris* spp., the mean total helminth burdens did not exceed 100 worms in any group of lambs slaughtered before January 1974.

Oestrus ovis was erratic in its appearance in the tracer lambs. In both years of the experiment, however, the first infestations recorded after the winter months were recovered from the lambs slaughtered during October.

DISCUSSION

If the total production results of the three groups are compared, four-weekly treatment was little better than strategic treatment. If, however, the production of the lambs until March 1974 is compared, (they were one year old at this time), four-weekly treatment had definite advantages over both other regimens, which did not differ materially in either the live mass gain or wool production components (Figure 1, Table 1). Therefore regular, short interval drenching of lambs until they are one year old is recommended for maximum production and strategic drenching can be practised thereafter.

The faecal worm egg counts and worm counts of the tracer lambs indicated that the lambs were exposed to very low rates of infestation until January 1974, yet these low burdens during their first year of life were responsible for a significant loss of production in two groups despite the fact that the one group was treated at strategic intervals. The ability of the lambs to cope with the more severe exposure during the second year of the trial probably accounts in part for the success of the strategic drenching programme during this period of the trial. A further reason could be that the four-weekly treated group may have been nearing the top of its growth curve thus permitting the strategically treated group to catch up.

It has been demonstrated that *O. ovis* infestation has a depressing effect on live mass gain in Merino sheep[10], and that lambs under 15 months of age harbour larger burdens of *O. ovis* larvae than do older sheep[7]. Although the tracer lambs were not all infested, it can be assumed that the trial sheep were subjected to infestation during the experimental period and that this affected production.

The faecal worm egg counts due to *H. contortus* in the untreated control group closely followed the acquisition of *H. contortus* infestation by the tracer lambs from the start of the trial until April 1974. The drop in egg counts thereafter is due to self-cure and to ageing of the adult worm burden as very little further infestation was being acquired from the pastures.

The rise in egg counts due to *H. contortus* in October, November and December 1974 cannot be coupled to a recent intake of infestation as the tracer lambs were picking up very few larvae from the pastures. The rise is probably due to the maturation of the fourth stage larvae which had been acquired during the autumn and winter[2], and remained in this stage of development either as a result of immunity in the host[12], or as a result of hypobiosis, which is the overwintering mechanism for this nematode[2 12]. The increasing proportions of fourth stage larvae recovered from the tracer lambs slaughtered during autumn and winter, even though total worm burdens were decreasing, indicate that hypobiosis was indeed functioning in this nematode population.

The complete absence of *O. ovis* in the tracer lambs during the winter months and until October indicates that no infestation was taking place during these months. Since survival outside the host during the winter is not possible[7], the infestation had to overwinter in the heads of the trial lambs[7 11].

In the light of the faecal worm egg count and tracer lamb worm burden findings, the strategic dosing programme adopted during the trial can be re-evaluated as follows:

(i) The cambendazole treatment during early August is superfluous because few larvae are available for re-infestation during this period.

(ii) Treatment with cambendazole during October would assist in controlling tapeworm infestation. It would also eliminate those larvae overwintering in the fourth stage and thus prevent the spring rise in egg counts (Fig. 2).

(iii) Rafoxanide treatment at the end of November or early December is advisable as a prophylactic measure against *O. ovis* and early *H. contortus* infestations. It is imperative that following drenching the animals be shifted to a rested paddock to prevent re-infestation from the pasture. This would also delay the build up of *H. contortus* during the summer months.

(iv) A cambendazole drench during February is advisable to control tapeworm and nematode infestations in lambs. This can be combined with rafoxanide to control *O. ovis*.

(v) Treatment of adult sheep with rafoxanide during February is aimed at *O. ovis* and *H. contortus*. In view of the relatively high *Trichostrongylus* spp. and *O. circumcincta* burdens in February and March (see Table 2) a thiabendazole drench should be combined with rafoxanide to obtain a broad spectrum of activity.

(vi) Treatment with rafoxanide during April can be dispensed with because of the relatively low *H. contortus* infestations and absence of *O. ovis*.

(vii) During May, a drench with cambendazole (not presently advocated) would serve to eliminate the remaining tapeworm and round worm infestation in lambs. The drenching of adult sheep with thiabendazole at this time act against *H. contortus*, *Trichostrongylus* spp., *Ostertagia* spp. and *O. columbianum* (if the latter is present). To obtain the maximum benefit from this treatment it is essential for the sheep to be shifted immediately afterwards to a rested camp, to prevent re-infestation.

(viii) Rafoxanide, drenched early in June, is aimed primarily at the overwintering *O. ovis* infestation.

(ix) As very few nematode parasites are picked up during the months of July through November, treatment of adult sheep with thiabendazole in early August and October can be dispensed with. Lambs, should, however, be treated with cambendazole during October for recently acquired tapeworm infestations. (see (ii)).

(x) It must be stressed that should *H. contortus* infestation become a problem during December to April more frequent drenching during these months may be necessary.

In parasite surveys in the Transvaal, conducted in sheep on pastures irrigated throughout the year, the seasonal incidence and parasite species involved were virtually identical to those at Tonteldoos[6] [7]. The suggested drenching programme would thus probably be applicable for large areas of the Transvaal and Orange Free State in which wooled sheep are farmed.

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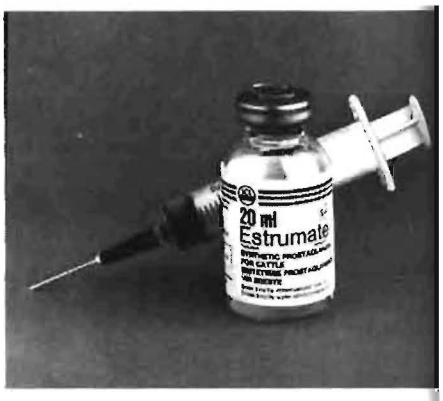

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IMMUNIZATION AGAINST COCCIDIOSIS IN FOWLS

F.W. HUCHZERMEYER*

ABSTRACT: Huchzermeyer, F.W.; **Immunization against coccidiosis in fowls.** Journal of the South African Veterinary *Association* (1976) **47** No. 4, 253-254 (En) Dept Infectious Diseases, Faculty of Veterinary Science, Univ. Pretoria, Box 12580, 0110 Onderstepoort.

Immunity plays a major role in the control of coccidiosis. With the presently practiced system of "protected exposure" under chemoprophylactic medication, problems are encountered because of seasonal variation in litter moisture and coccidial challenge. The alternative, "controlled exposure" or "vaccination" produces a more solid immunity and more reliable results. This was found to be the method of choice in coccidiosis control in replacement pullets in the semi-arid subtropical climate of Rhodesia.

INTRODUCTION

Economically coccidiosis is one of the most important diseases of the domestic chicken. In addition to the cost of chemoprophylaxis there are the losses caused by morbidity and mortality in outbreaks which still do occur as well as the cost of treatment of these outbreaks. An excellent review of the present knowledge of the control of coccidiosis is given by Reid[4]. The author's personal experience with coccidiosis control in Rhodesia is the basis of this paper[1,2].

Classification of outbreaks

In relation to the use of chemoprophylaxis outbreaks of coccidiosis can be divided into 3 groups:

1. Outbreaks in flocks where no coccidiostat is or has been used: This is at present mainly a backyard problem and of minor importance.
2. Outbreaks occurring while a coccidiostat is being administered: These "breaks" can be caused or triggered by a variety of factors such as resistance of the strain or species of *Eimeria* to the coccidiostat being used, increased challenge due to improvements in sporulation conditions or inadequate intake of the coccidiostat, usually caused by factors depressing feed intake[3]. Provided there is a challenge, there is also an increasing level of immunity which will support the protection given by the coccidiostat.
3. Outbreaks occurring after the administration of a coccidiostat has been discontinued: These involve older pullets and laying hens. In Rhodesia they are found to occur regularly at the beginning of the rainy season. They are considered a particularly serious problem, as not only is the mortality rate high but the laying performance of the flock is severely depressed for a considerable period. A study of the flock histories always showed that the birds have been reared during the dry season on clean litter and protected by a coccidiostat. The conclusion is drawn that they lacked immunity at a time of increased challenge. It was therefore thought advisable to review the currently practised rearing methods employing coccidiostat protection. From the point of view of immunization this can be classified as "protected exposure".

Protected Exposure

A coccidiostat is fed at a certain level and for a certain period to young birds reared on the floor. The coccidiostat interfering at a certain stage in the life cycle of the parasite theoretically allows the birds a certain degree of exposure to the parasite, which is sufficient to produce an immunity, and thus enables them to face challenges after the withdrawal of the drug. The main immunogenic stage is the second generation schizont, which in *Eimeria tenella* infections develops on the fourth day after infection. Coccidiostats such as clopidol, monensin, robenidine, amprolium, which act before this stage is reached, prevent the establishment of an immunity. Late acting coccidiostats are nicarbazin, nitrofurans, sulphonamides and ethopabate[4].

With the variation in resistance of strains and species of chicken *Eimeria* to the different coccidiostats, it can also be postulated that the birds acquire the best immunity to the species of *Eimeria* possesing the highest resistance to the coccidiostat being used, and *vice versa*. Thus most outbreaks of coccidiosis occurring after the use as chemoprophylaxis of amprolium plus ethopabate are caused by *E. tenella* and *E. necatrix*, which are controlled most effectively by this preparation.

This concept of protected exposure, however, depends on the constant presence in the litter of a sufficient number of sporulated oocysts of the locally important species. Suitable conditions in this respect prevail in the constantly humid moderate climates of the Northern Hemisphere, where the majority of the world's poultry is produced and where most of the poultry disease research is carried out. In the semi-arid subtropical zones with alternating dry and wet seasons there is a corresponding cyclic variation in litter conditions between dry and unsuitable, and moist and suitable for sporulation.

Birds reared in the dry season on clean litter and protected by a coccidiostat have little opportunity of being exposed to a sufficient level of infection to produce a protective immunity. In the subsequent wet season these birds may have to face a high challenge when oocysts are able to sporulate in large numbers. Realizing that this method did not produce the desired results under Rhodesian climatic conditions, we then considered the so-called "vaccination" or "controlled exposure".

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* Department of Infectious Diseases, Faculty of Veterinary Science, Univ. of Pretoria, Box 12580, 0110 Onderstepoort

Controlled Exposure

This method is widely used in the southern states of the United States of America. A small number of sporulated oocysts of the important species of chicken *Eimeria* is suspended in the drinking water and given once to chicks between 7 and 14 days old which are reared on litter and without the inclusion of a coccidiostat in the ration. This vaccine is commercially available as Coccivac, Sterwin Laboratories. Four days after this first exposure the most important part of the immunization process begins, *i.e.*, the litter management: In the floor area of one third of the house — usually a strip comprising one third of the width and extending the whole length of the house — the litter is kept slightly damp through daily watering with a hose pipe or a watering can in order to create suitable conditions for the sporulation of oocysts, which by this time will be excreted by the chicks. This litter management is continued for 4 to 8 weeks. If birds are transferred to another house — *e.g.*, at point of lay — a wheelbarrow load of infected litter is taken to the new house and scattered over the new litter. If that is not practicable, particularly when point of lay pullets are sold, a revaccination is recommended.

Except for a very mild depression in the growth rate for 2 weeks after the vaccination and for the occasional red dropping, no reaction is shown by the chicks. Oocyst counts in the droppings demonstrate an increasing rate of oocyst excretion over a period of 2 to 3 weeks after the vaccination, reaching peak levels of 20 000 oocysts per gram of fresh faeces (unpublished findings). Thereafter oocyst excretion returns to very low levels.

The immunity to the different species of *Eimeria* is species specific. There are also differences in the number of oocysts and frequency of infection required in order to establish an immunity, and in the ability of individual birds to produce an immunity. Generally a repeated infection with small numbers of oocysts produces the best results.

When a coccidiosis vaccine is used the birds are at first exposed to a very low level of infection. As this level rises with increasing production and sporulation of oocysts, immunity to the various species is increasingly stimulated in the flock until for each species and for each individual bird an equilibrium is established between challenge and immunity. Once established this equilibrium is more stable and less vulnerable to outside influences than that created by any other method of coccidiosis control.

DISCUSSION

It would be preferable to use attenuated strains of the various species of *Eimeria* for vaccination purposes, provided they are of equal immunogenic quality. Although some workers are progressing in this direction, there are no attenuated strains yet available for application in the field.

When, however, unattenuated strains of coccidia are used, as is the case with the presently available vaccine, it is due to the uniform spread and low initial dose, that an even level of initial immunity is stimulated throughout the flock. Thus the birds are ready for the second and increased challenge, when after the first prepatent period and the sporulation time the next generation of oocysts becomes infective and is ingested. It is in fact largely due to these intervals between increasing challenges that the birds are able to cope with the rising pressure of infection and respond with adequate levels of immunity.

With the ubiquitous presence of most chicken coccidia there is little danger of introducing new species or strains to the premises. There is no danger either of introducing other disease agents with the vaccine, as there is no evidence of infections being transmitted through oocysts.

Litter management consisting of partially moistening the litter produces several side benefits. The farmer becomes actively involved in the control measure and grows aware of the biological principles. The added moisture helps to combat dust problems. In my own practical experience there was no build-up of other internal parasites.

Even in experimental vaccination of broilers there was no ill effect on growth rates or food conversion[1]. Consequently there can be little danger in using this method on replacement pullets.

CONCLUSIONS

All methods of coccidiosis control are at least partially based on immunity.

Current, methods of protected exposure have several disadvantages:—

- The coccidiostat prevents to various degrees the production of immunity.
- The required presence of sporulated oocysts depends on climatic and other factors and exposure is fortuitous.

The advantages of controlled exposure are:—

- This method is particularly suitable for the local climatic conditions.
- The birds are exposed to all important species of chicken *Eimeria* under controlled conditions.
- The protection given is excellent.
- The cost of vaccination is a fraction of the cost of coccidiostat used for rearing up to 8 weeks.

ACKNOWLEDGEMENTS

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SUBCLINICAL SEPTIC BOVINE MASTITIS : CRITICAL EVALUATION OF INTRAMAMMARY IMMUNOGLOBULIN THERAPY

L.W. VAN DEN HEEVER*

ABSTRACT: Van den Heever, L.W. **Subclinical septic bovine mastitis : Critical evaluation of intramammary immunoglobulin therapy.** *Journal of the South African Veterinary Association* (1976) **47** No. 4 257-261 (En) Dept. Pathology, Faculty of Veterinary Science, University of Pretoria, Box 12580, 0110 Onderstepoort, Republic of South Africa.

A series of 152 quarters affected with subclinical staphylococcal and streptococcal mastitis was treated at random by intramammary infusion with one of 4 formulations containing inert solvent, specially prepared immunoglobulin (Ig) and antibiotics (penicillin G and streptomycin) in various combinations.

The effect of treatment was measured by assessing the conversion of mastitic quarters to a normal or mastitis-negative state over a post-treatment period of 28 days.

Considering the solvent as a placebo, solvent plus Ig had no significantly better effect than solvent only; solvent plus antibiotics gave results that were highly significant after 14 days even though about a third of the strains of *S. aureus* were resistant *in vitro* to one or both of the antibiotics; solvent plus antibiotics and Ig gave results that were highly significant during the whole 4 weeks after treatment.

The Ig solution appeared to be considerably more effective against staphylococcal than streptococcal mastitis, but the small numbers precluded statistical analyses.

It was concluded that use of the particular batch of Ig alone had no value in the treatment of the common forms of subclinical bacterial mastitis but that in combination with antibiotics the Ig had a somewhat better effect than was obtained with antibiotics alone.

INTRODUCTION

Antimicrobial substances in various formulations are generally used for therapy of septic bovine mastitis and their efficacy is more or less predictable if the microbiological agent and the stage and type of inflammatory reaction is known. Unfortunately, apparent or clinical recovery is not necessarily associated with elimination of the responsible microorganism, regardless of whether or not specific antimicrobial therapy has been applied. Self-cure also occurs and converts a mastitic quarter to an apparently normal state[4]. Such spontaneous recovery results in elimination of infection in some 19% of cases, while antibiotic therapy has achieved elimination in about 36% of cases[4]. In both cases streptococci are more easily eliminated from the quarter than staphylococci[4].

In consequence there is continual search for alternative and hopefully more effective substances for use in the therapy of mastitis. One of the most interesting to be considered in recent years was suggested by Botes, who reported extensively on his trials with immunoglobulin (Ig)[1]. Uncertainty regarding the relative respective value of Ig and antibiotics in the formulation led to the investigation which is the subject of this report.

Investigations into the value of intramammary therapy of mastitis are hampered by considerable variation in the type, extent, degree and aetiology of the mastitic reaction. Pairing of identical clinical cases for comparative purposes is rarely possible. In order to obtain an adequate number of cases of mastitis over a relatively short period, and to ensure satisfactory and confirmed data on the nature of the quarter milk prior to treatment, this investigation was confined to cases of subclinical septic mastitis caused by *Streptococcus agalactiae, Sc. dysgalactiae* and *Sc. uberis,* and by *Staphylococcus aureus.*

MATERIAL AND METHODS

1. *General:* Clinically healthy Friesland and Jersey type cows in commercial dairy herds were used for the investigation. Most herds were machine milked, and the machines were in good functional order. The milking process took place with due consideration of the usual sanitary procedures, and all teats were routinely immersed in an iodophor teat dip of 1000 ppm I_2 immediately after milking.

On the afternoon of Day 1 the herds were surveyed by visually examining the first three jets of milk from every quarter in a strip cup for physical abnormalities. The quarters were palpated and observed for tissue changes. Quarter milk samples (QMS 1) were aseptically drawn from apparently normal quarters into sterile containers, chilled during transport to the laboratory and examined on the following morning.

On Day 7 foremilk samples (QMS 7) were similarly collected from quarters which had been provisionally classified as affected with subclinical septic mastitis (SSM) after examining QMS 1. Assuming that QMS 7 would confirm the QMS 1 findings, the quarters were treated with one of four (I-IV) formulations. Only data from quarters classified as affected with SSM after examining both QMS 1 and 7 were further utilised. No more than 2 quarters per cow were treated.

Foremilk samples were subsequently drawn on Days 21, 28 and 35 from all treated quarters. For the purpose of assessing the hygiene of milking and of teat dipping relative to new cases of mastitis, completely normal quarters were also sampled on Days 1, 7, 21, 28 and 35 in the ratio of one normal to every 4 SSM quarters.

2. *Examination of Quarter Samples:* Every sample was examined as follows:
   (a) Bacteriological (BACT): 0,01 ml of milk was spread on to the surface of a pre-incubated blood tryptone agar (BTA) plate for 24h incubation at 37°C prior to examination for *Staphylococcus* and/or *Streptococcus* colonies[6]. *Staphylococcus* spp. were classified on the basis of coagulase production as demonstrated by the presumptive slide test using Difco Rabbit Serum, and *Streptococcus* spp. on the basis of aesculin breakdown and

* Div. Food Hygiene & Public Health, Dept. Pathology, Faculty of Veterinary Science, University of Pretoria, Box 12580, 0110 Onderstepoort.

the CAMP reaction on aesculin BTA plates[2,5]. Results were recorded as BACT-positive when either *S. aureus, S. epidermidis, Sc. agalactiae, Sc. dysgalactiae,* or *Sc. uberis* was isolated.

(b) Somatic Cell Content (SCC): The SCC/ml was determined by dual recordings of the number of cell particles/ml in fixed ("SOMAFIX")*, defatted ("SOLVENT")* and suitably diluted (1:50) aliquots of the milk sample, according to the method of the International Dairy Federation, using a Coulter Electronic Particle Counter Model ZB1 with an aperture of 100µ and a threshold value set at ±24µ. Cell counts in excess of 500 000/ml were recorded as "positive".

(c) The bovine serum albumin (BSA) content was established by means of the radial diffusion test described by Giesecke & Viljoen using standardised rabbit anti-BSA serum and recording precipitin rings of 8 mm diameter or more as "positive"[3].

3. *Diagnosis of SSM:* Quarters were classified as SSM-positive if the quarter and secretion appeared clinically normal, and both QMS 1 and 7 were BACT-, SCC- and BSA- positive.
4. *Treatment* was allocated without selection to emptied SSM quarters and consisted of a single intracisternal administration via the teat canal of 20 ml of a freshly reconstituted preparation of one of the following formulations**.

I Solvent only (=placebo) III Solvent and Ig
II Solvent and antibiotics IV Solvent and antibiotics + Ig

The solvent consisted of sterile pyrogen-free distilled water. The antibiotic combination consisted of 200 000 i.u. of penicillin G and 250 mg of streptomycin.

The Ig was derived from cow serum drawn after vaccination of the donor with killed cultures of various mastitis-organisms including *Staphylococcus* and *Streptococcus* spp. The purified Ig contained 92% of pure gamma globulin, the latter being free of Ig A and consisting of 70% Ig G, 20% Ig M and 10% 1OS. Before lyophilisation each dose consisted of 5 ml of an 8,3% protein solution.

5. *Evaluation of Efficacy of Treatment:* Where administration of a formulation failed outright in altering the SSM status, treatment was obviously unsuccessful. In instances where the status of the quarter had reverted to negative and normal (NN) i.e. milk was BSA-, SSC- and BACT-negative, treatment was classified as successful. Where the milk from a treated quarter had changed, but not become normal, the quarter's new status was classified according to the following scheme:

BSA	SCC	BACT	STATUS:
+	+	—	: Subclinical Aseptic Mastitis (SAM)
—	+	+	: Significant Teat Canal Infection (STCI)
—	—	+	: Insignificant Teat Canal Infection (ITCI)
—	+	—	: Non-specific Cellular Reaction (NSCR)
Others			: Inconclusive (INC)

6. All isolates of *S. aureus* from quarters due for treatment with preparations containing antibiotics were transferred to BTA plates and tested against Oxoid sensitivity discs containing 5 i.u. of penicillin G and 25 mcg of streptomycin respectively.

* Coulter Electronics SA (Pty) Ltd, Johannesburg
** Agricura Laboratories, Box 55, Silverton, Tvl

RESULTS

1. *Surveillance of normal untreated quarters during the trial period:*

Of 38 quarters, 38 (100%) were still normal on Day 7,
35 (92,1%) were still normal on Day 21,
32 (84,4%) were still normal on Day 28,
and 30 (79,0%) were still normal on Day 35.

Table 1: PRE- AND POST-TREATMENT STATUS OF SMM POSITIVE QUARTERS AFTER TWO (DAY 21), THREE (DAY 28), AND FOUR WEEKS (DAY 35) USING ONE OF FORMULATIONS I-IV

Formulation	Day of sampling	No. treated	No. (%) Status of Treated Quarters* SSM	X^2	P	SAM	STCI	ITCI	NSCR	NN	INC
I	QS 1+7	41		—	—						
	21		24(58.4)	—	—	4(9.8)	3(7.3)	4(9.8)	1(2.4)	4(9.8)	1(2.4)
(solvent only)	28		20(48.8)	—	—	2(4.9)	3(7.3)	7(17.0)	1(2.4)	4(9.8)	4(9.8)
	35		20(48.8)	—	—	1(2.4)	4(9.8)	6(14.6)	0	6(14.6)	4(9.8)
II	QS 1+7	33		—	—						
(solvent &	21		10(30.3)	30,4	0,001	5(5.1)	0	6(18.1)	0	12(36.3)	0
antibiotics)	28		10(30.3)	15,75	0,05	3(9.0)	2(6.0)	6(18.1)	0	9(27.2)	3(9.0)
	35		10(30.3)	15,03	0,05	4(12.1)	2(6.0)	5(15.1)	0	9(27.2)	3(9.0)
III	QS 1+7	40		—	—						
(solvent & Ig)	21		16(40)	3,53	NS	5(12.5)	4(10)	7(17)	1(2.5)	7(17.5)	0
	28		17(42.5)	8,58	NS	5(12.5)	5(12.5)	4(10)	2(5.0)	5(12.5)	2(5)
	35		20(50)	11,53	NS	4(10)	2(5)	5(12.5)	0	7(18.0)	2(5)
IV	QS 1+7	38		—	—						
(solvent & anti-	21		12(31.8)	47,04	0,001	3(7.9)	1(2.6)	4(10.5)	15(40)	0	
biotics & Ig.)	28		15(34.2)	28,58	0,001	4(10.5)	2(5.3)	1(2.6)	2(5.3)	13(34)	1(2.6)
	35		12(31.8)	28,13	0,001	4(10.5)	0	5(13.1)	3(7.9)	12(31)	2(5.3)

*See Methods and Material para 5 NS =Not Significant

2. *Incidence of subclinical septic mastitis (SSM) in 14 herds surveyed for trial purposes:* The survey involved 1255 cows and 4922 quarters; the percentage of SSM positive quarters/herd varied from 0 to 13%, with a mean of 3,5%.
3. *Status of SSM-positive quarters when examined two (Day 21), three (Day 28), and four weeks (Day 35) after treatment with one of four (I-IV) preparations.* These data are summarised in Table 1:

   The Chi-square (X^2) test was applied to the results provided in Table 1; it was assumed that none of the treatments had any therapeutic effect ("Null hypothesis") and that the expected frequencies of the reclassification of quarters after treatment would be the same as those treated with solvent (placebo) only. Data from each formulation was tested against those of the solvent only as recorded after the same post-treatment period. From this analysis it then appears that:
   1) The use of solvent plus Ig did not produce significant results at any stage up to Day 35 after treatment.
   2) The use of solvent plus antibiotic produced results which were highly significant ($p < 0,001$) at Day 21 and probably significant ($p < 0,05$) during the period 14 to 28 days post treatment.
   3) The use of solvent plus Ig and antibiotics produced results which were highly significant at all stages up to 35 days post treatment.
4. The comparison between the results of treating cases of subclinical staphylococcal and streptococcal mastitis is summarised in Table 2.
5. The *in vitro* sensitivity to penicillin G and streptomycin of *S. aureus* isolated from quarters prior to treatment with preparations containing these antibiotics is summarised in Table 3.

The numbers furnished in Tables 2 and 3 are too small to permit of statistical analysis and at most indicate a trend.

DISCUSSION

The results firstly indicate that the status of normal mastitis negative quarters in commercial herds is not static, even over periods of 5 weeks, when judged by tests designed to indicate either the bacterial infection, cellular reaction or the presence of specific inflammatory products in milk. "Normality" of a quarter in terms of its secretion is therefore somewhat difficult to establish as a permanent status within a commercial herd.

The results also indicate how few quarters (mean 3,5%) can be classified as being affected with *true* subclinical septic mastitis as determined by the presence of mastitis producing bacteria, elevated somatic cell numbers and of serum albumin. This contrasts sharply with the widely reported high incidence (up to 50%) of SSM when only elevated somatic cell counts and specific bacterial infection of the milk is taken into consideration[5], and casts considerable doubt on the validity of such diagnoses of "mastitis" in the more accurate sense. It is clear that when field trials of therapeutic substances are to be conducted, application of a strict definition of true

Table 2: COMPARISON OF RESULTS OF TREATMENT OF SUBCLINICAL STAPHYLOCOCCAL AND STREPTOCOCCAL MASTITIS WITH FORMULATIONS I-IV

Formulation	No. of Quarters Treated (on Day 7)		Status and No. (%) of Quarters 2 weeks after treatment (Day 21)			
			Complete Recovery*		Incomplete Recovery**	
	Staph.	Strep.	Staph.	Strep.	Staph.	Strep.
I (Solvent only)	18	23	0	4(17.4)	6(33.3)	8(34.8)
II (Solvent and pen.-strep)	12	20	1(8.3)	11(55)	5(38.4)	6(30)
III (Solvent and Ig.)	15	25	4(26.7)	3(12)	3(20)	14(56)
IV (Solvent, pen.-strep. & Ig.)	19	20	5(26.3)	9(45)	5(26.3)	6(30)

*Complete conversion to normal (BSA, SCC and BACT negative), **Conversion of status from SSM to ASM, STCI or ITCI.

Table 3: IN VITRO SENSITIVITY TO PENICILLIN G AND STREPTOMYCIN OF *S. AUREUS* ISOLATED FROM CASES OF SUBCLINICAL SEPTIC MASTITIS BEFORE AND AFTER TREATMENT WITH THESE ANTIBIOTICS ON DAY 7

No. of Isolates	No. of isolates resistant to penicillin G (p) and streptomycin (s) and to both (ps) after isolation on Days 7-35											
	Day 7			Day 21			Day 28			Day 35		
27	p	s	ps	p	s	ps	p	s	ps	p	s	ps
	6	8	1	8	9	2	11	8	4	11	9	4
Percentage	22,2	29,6	3,7	29,6	33,3	7,4	40,7	29,6	14,8	40,7	33,3	14,8

mastitis will undoubtedly make the search for suitable cases more difficult and costly than is the case where the less strict IDF definition is employed. Similarly there are no published reports on the evaluation of therapeutic substances on the basis of *true* subclinical mastitis as defined in this report.

Table 1 summarises the reactions of SSM quarters to treatment with four formulations as assessed after various intervals. As the primary purpose of this investigation concerns the relative value of therapy of true subclinical septic mastitis with Ig and antibiotics, it is appropriate to consider the success of treatment in the light of the failure rate, i.e. the percentage of quarters that retained the status of SSM despite treatment. Before doing so it is timely to consider the length of the interval between treatment and check sampling. Too early sampling is unsatisfactory because the responsible organism may be rendered dormant or otherwise be present in numbers too small to permit recovery by conventional methods; alternatively the secretion on that day may be sterile without the mammary tissue necessarily also being entirely free of infection. On the other hand a prolonged post treatment interval progressively increases the likelihood of new infections becoming established and leading to secondary mastitis. It is clearly desirable for the same micro-organism to be present before and on two consecutive occasions after treatment before it can be stated that treatment has failed. With this in mind, the following may be deduced from Table 1:

The "failure index", i.e. percentage of quarters which retained the status of SSM for up to 4 weeks after treatment, would place the four formulations in the following order as far as efficacy is concerned:
(a) Solvent and antibiotics: (Failure index 30,3%)
(b) Solvent + antibiotics + Ig: (Failure index 31,8%)
(c) Solvent + Ig: (Failure index 40%)
(d) Solvent only (Placebo): (Failure index 48%)

The "conversion to normal and negative (NN) index" indicates the percentage of treated quarters that have returned to a completely normal state. In terms of this measure of efficacy the four formulations would be placed as follows:
(a) Solvent + Ig + antibiotics: (NN-index 31%)
(b) Solvent + antibiotics: (NN-index 27,2%)
(c) Solvent + Ig: (NN-index 18,0%)
(d) Solvent only (Placebo): (NN-index 9,8%)

It is clear that whereas the particular batch of Ig was therapeutically slightly more effective than solvent only, it was distinctly inferior to penicillin-streptomycin in aqueous solution. The nature of the mastitogenic organism is, however, of some significance *(vide infra)*.

Table 1 also indicates that therapy may result in conversion to various intermediate states other than either complete return to normality or unchanged. The permanency of these intermediate states remains unknown, and no fixed pattern was evident during the four weeks of post-treatment observation.

Table 2 reflects a difference in the respective response of staphylococcal and streptococcal cases of SSM to treatment. Administration of solvent only (placebo) apparently resulted in complete recovery of 17,4% of subclinical streptococcal mastitis but no staphylococcal cases. This is in line with the rates of self-cure which have been reported[4]. Antibiotics in solution effected complete recovery of 55% and 8,3% of cases of subclinical streptococcal and staphylococcal mastitis whereas administration of Ig i solution resulted in the recovery of 12% and 27% respectively. The latter indicates a tendency to greater efficacy of Ig therapy in subclinical staphylococcal as opposed to streptococcal mastitis. Comparing the therapy of subclinical *Streptococcus* mastitis, Table 2 shows that combined Ig-antibiotic solution resulted in complete normalisation of quarters at a rate of 45% as compared with 55% when using only the antibiotic solution. In the case of subclinical mastitis caused by *Staphylococcus*, however, the Ig-antibiotic combination achieved normalisation of 26% of cases as opposed to only 8,3% after the use of antibiotics alone. This may in part be explained by the antibiotic resistance of some of the *Staphylococcus* strains but does indicate a favourable effect of Ig under such circumstances. In this regard reference to Table 1 indicates a tendency for Ig to have a favourable effect upon the course of mastitis over a long term when noting the progressive steady increase in the standard deviation (X^2) figures from 3,53 on day 21 to 11,53 on day 35.

This discussion would be incomplete without reference to antibiotic resistance of the staphylococci studied during the investigation. The results in Table 3 indicate that about 22% and 30% of the *S. aureus* isolated before therapy on Day 7 were resistant to either penicillin or streptomycin respectively, with only about 4% resistant to both these antibiotics. In the course of the subsequent four weeks of observation these figures rose progressively in all instances.

Antibiotic resistance is almost inevitable in strains of *S. aureus* isolated from cows in herds where mastitis therapy is regularly undertaken, and the figures in Table 3 are not unexpected. They do, however, partially explain the lower recovery rates of subclinical staphylococcalthan streptococcal mastitis when these were treated with antibiotics only.

CONCLUSIONS

From the results of this investigation it is concluded that:

1. Compared with placebo in the form of the solvent, single intramammary instillation of 5 ml of a specially prepared and purified bovine Ig containing 8,3% protein had no significant therapeutic effect on the common types of subclinical septic mastitis when assessed for 28 days after treatment. Ig administration was somewhat more effective in the local therapy of subclinical cases of staphylococcal bovine mastitis than in those caused by *Sc. agalactiae*, *Sc. uberis* or *Sc. dysgalactiae*.
2. Despite the *in vitro* resistance of about one third of the strains of *S. aureus* isolated, therapy of subclinical staphylococcal mastitis with an aqueous solution of 200 000 i.u. of penicillin and 250 mg of streptomycin was significantly more effective than Ig used alone and only slightly less effective than a combination of Ig and these antibiotics.
3. The single administration of a combination of Ig-penicillin-streptomycin for the therapy of subclinical cases of streptococcal mastitis offered no particular advantage over the administration of these antibiotics alone. In the case of staphylococcal mastitis, such a combination was, however, about four times as effective in converting quarters with subclinical mastitis to normality even though this amounted to a rate of efficacy which was only about half that obtained against streptococcal

mastitis.

4. Although Ig may possibly have a significant effect on subclinical forms of bovine mastitis when assessed over a long term after treatment, sole use of intramammary instillations of the particular batch of Ig investigated cannot be considered therapeutically effective in the treatment of the common forms of subclinical bacterial mastitis.

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BOOK REVIEWS

BOEKRESENSIES

THE PRINCIPLES OF HORSESHOEING

DOUG BUTLER

Doug Butler, Ithaca, N.Y. pp XV + 428, Figs. 425, Tabs several. Price not quoted

This book is divided into seven sections, with sub-headed chapters:

Section I. Perspective, includes chapters on the need for horse shoes, their necessary qualities, some historical notes and some definitions.

Section II on Horse Handling is interesting and instructive. The knots described are worthy of more general use. This animal management section will be found of value to all horsemen. Anatomy claims 70 pages. The tools, shoes and nails are well described. Shoeing of normal and abnormal feet, by hot and cold techniques are described and extensively illustrated.

Section V is devoted to Iron and Forge work, Section VI to Corrective Shoeing, and Section VII to Pathological Shoeing (an unfortunate term, for which read Surgical Shoeing).

If much equine infertility in South Africa derives from castration then it is also true that much lameness in horses derives from incorrect (or pathological) shoeing. The book under review covers the subject in a well balanced manner and the lavish illustrations would make much of it intelligible to the semi-illiterate. Many horse owners in South Africa would do well to read Page 153 and realise that in general shoes are required only where the wear exceeds the growth of hoof. The figures on page 186 showing principles of shaping the foot would make the book worth its price for many individuals. The T Square (page 178), the trimming (page 179) and the hoof tester described (page 375) are highlights in a generally sound and scholarly presentation. Most veterinarians in South Africa will continue to use arc welding to make bar shoes in preference to the more specialised fire welding described in Chapter 31. Veterinarians interested in horses and all farriers will find the book stimulating, instructive and helpful. Trainers and owners would also be rewarded by studying this book.

D.H.I.

THE EVALUATION OF FEEDS THROUGH DIGESTIBILITY EXPERIMENTS

BURCH H. SCHNEIDER and WILLIAM P. FLATT

The University of Georgia Press, Athens 1975. pp XXIII + 423, Figs 29, Tabs 19. Price not indicated

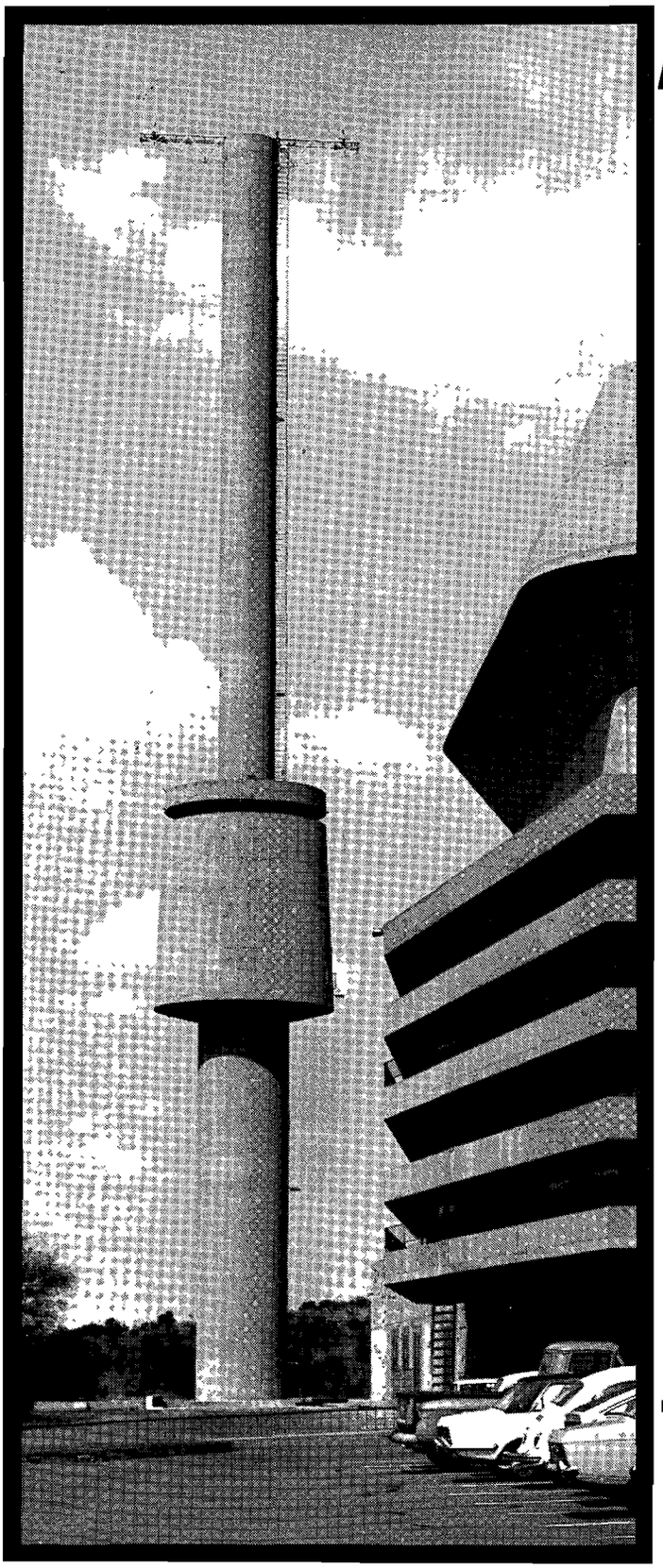
A valuable book to the student of nutrition and especially to the young researcher in animal nutrition. The basic principles concerning digestibility, its determination and calculation are clearly explained and appropriately illustrated with practical, yet uncomplicated examples. Basic techniques and some of the apparatus used on almost all aspects of digestibility experiments with animals, such as gathering faeces, methods of proximate analysis, etc., are methodically presented and elucidated with simple and clear illustrations.

There are two printing errors which the reviewer came across and feels obliged to point out:

pp 259, Fig. 25: Regression equation should read $Y = 29{,}0 + 0{,}5X$ and not $Y = 29{,}0 + 5X$, and

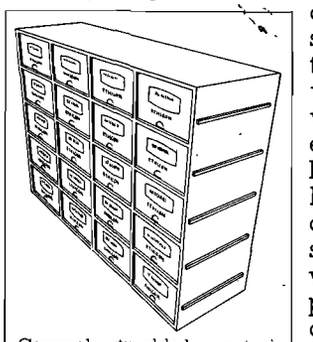
pp 261, Fig. 27: Regression equation should read $Y = 87{,}4 - 0{,}74X$ and not $Y = 874 - 74X$.

S.C.



'ONS HET KLAAR GEPRAAT'

Ons firma was een van die eerstes om die kobalt-60-pakkettebestralingsaanleg by die Nasionale Kernnavorsingsentrum op Pelindaba te benut – 'n aanleg wat met die besondere doel gebou is om 'n steriliseerdiens vir alle soorte wegdoenbare produkte te bied.
Hoewel Pelindaba in die spreektaal ‚ons het klaar gepraat' beteken, wil ons graag byvoeg dat gammastraling – wat allerweë as die uitnemendste in steriliseertegnieke aanvaar word – verseker dat elke afsonderlike naald en hegmateriaal deur ons voorsien absoluut vry van skadelike patogene is; en dit word alles sonder nadelige gevolge vir die naald of hegmateriaal verkry. Pelindaba se kerndeskundigheid is vir ons en vir u die waarborg van volkome



Gammabestraalde hegmateriaal en naalde beteken dat u op volkome sterilisering kan staatmaak.

VEILIGHEID ETHICON

4415/A

Jl. S. Afr. vet. Ass. 47(4) 263-264 (1976)

THE COMPARATIVE INCIDENCE OF SUBCLINICAL BACTERIAL BOVINE MASTITIS IN 17 HERDS WHEN USING TWO SETS OF DIAGNOSTIC CRITERIA

L.W. VAN DEN HEEVER and G.V.S. TURNER

ABSTRACT: Van den Heever, L.W.; Turner G.V.S. **Subclinical bovine mastitis: Comparison of results of two sets of diagnostic criteria.** *Journal of the South African Veterinary Association* (1976) **47** No. 4 263-264 (En) Div. Food Hyg. & Publ. Hlth, Dept. Pathology, Fac. Veterinary Science, Univ. of Pretoria, Box 12580, 0110 Onderstepoort, Rep. of South Africa.

On the single examination of 6804 quarter milk samples during a routine mastitis survey in 17 herds it was found that whereas 12,4% of quarters were diagnosed as subclinically mastitic when using the IDF standard set of diagnostic criteria, only 2,4% were positive when applying the additional criterium of a positive test for bovine serum albumin.

The remaining 10% of quarters were then classified as being affected with septic inflammation of the ducts. The ratio of true subclinical septic mastitis to duct and teat canal infections is recorded and the implications of the findings are briefly discussed.

INTRODUCTION

Whereas the diagnosis of clinical mastitis presents no particular problem, subclinical septic mastitis can only be accurately established by laboratory examination of quarter samples. Such diagnosis is essential for the evaluation of the mastitis problem within a herd and for the institution of a rational and practical programme for herd mastitis control. Subclinical septic mastitis is far more prevalent than the clinical forms, the ratio being perhaps as high as 20:1 or more[1].

Various parameters have been used to diagnose subclinical bacterial mastitis[2]. The most commonly accepted is the set of criteria proposed by Tolle[9] and accepted by the International Dairy Federation (IDF)[10]. According to this standard, aseptically drawn foremilk samples containing mastitogenic bacteria and more than 500 000 somatic cells/ml indicates mastitis in the quarter concerned.

Several authors have demonstrated that bacterial contamination of quarter milks obtained via the teat canal may result from bacterial colonisation of the epithelium of the ducts[3]. Others emphasize that a variety of factors other than mastitis may elevate the somatic cell content of milk[4-6]. This had led to expression of doubt regarding the validity of the IDF

Table 1: COMPARATIVE RESULTS OF EXAMINATION OF 6804 QUARTER MILK SAMPLES (QMS) FROM COWS IN 17 FRIESLAND-TYPE DAIRY HERDS

Herd	No. of QMS	No. of QMS with ECC and bacteria							Total IDF pos.	No. QMS IDF & BSA pos.
		S.a	S.e	Sc.a	Sc.d	Sc.u	Sc.	S + Sc		
A	208	13	15		3			3	34	
B	213	4	5	23					32	3
C	473	27	11		3		1		42	12
D	116	3	4	7	1		1		16	1
E	96	8	28	9			1	3	49	3
F	351	49	2	6	3	2	1	2	65	3
G	161	6		1	1				8	
H	315	12	7		2			1	22	5
I	589	4	40	41		1	10	1	97	1
J	259	3	5		2				10	3
K	166	1	11	3					15	1
L	109	4		2					6	1
M	474	6	3	3	3				15	1
N	492	23	5		1		1	3	33	5
O	919	20	5	54	11	3	10	1	104	26
P	887	6	16	109	9	2	8	2	152	56
Q	976	48	11	59	17	11	9	7	162	39
TOTAL	6804	237	168	317	56	19	42	23	862	160
%		3.5	2.5	4.7	0.8	0.3	0.6	0.3	12.7	2.4

Legend:
ECC = Elevated somatic cell content (over 500 000/ml)
S.a = *Staph. aureus*
Sc.a = *Streptococcus agalactiae*
Sc.u = *Sc. uberis*
Sc. = other streptococci
S.e = *S. epidermidis*
Sc.d = *Sc. dysgalactiae*

Dept of Pathology, Faculty of Veterinary Science, University of Pretoria, Box 12580, 0110 Onderstepoort

criteria, and to a call for differentiation between "udder infection" and "mastitis" and a re-appraisal of the term "mastitis" as commonly used[2 4]. "Mastitis" must of necessity include inflammation of the secretory epithelium of the mammary gland, and it has been suggested that the presence of bovine serum albumin (BSA) in the secretion results from the increased vascular permeability which follows epithelial damage and characterises such inflammation[2 4 5]. Accordingly it has been suggested that by additionally requiring a positive BSA test it would be possible to distinguish between those milks derived from a quarter with true subclinical septic mastitis on the one hand and the quarter with septic galactophoritis or a significant teat canal infection on the other.

Quarter milk sample surveys, involving 17 random commercial dairy herds consisting of Friesland type cows, presented the opportunity of comparing the incidence of subclinical bacterial mastitis as diagnosed by the use of the IDF criteria for the diagnosis of subclinical mastitis, with those obtained when these IDF criteria were combined with the results of a monovalent test for the presence of BSA.

MATERIAL AND METHODS

Quarter samples of foremilk were aseptically drawn into sterile tubes after discarding the first three jets of milk. Samples were chilled and immediately transported to the laboratory.

After thorough mixing of the contents of the tubes to ensure even dispersion of suspended matter, 0,01 ml of milk was plated onto the surface of blood tryptose agar (BTA) plates. After incubation at 37°C for 18-24 h the plates were examined for staphylococcal and streptococcal colonies. Staphylococci were classified on the basis of the slide coagulase test, using Difco coagulase serum. Streptococcal isolates were streaked onto CAMP-Aesculin BTA plates for further classification.

The somatic cell content of milks was determined by means of a Coulter Electronic Particle Counter Model B after fixing ("Somafix")* defatting ("Solvent")* and suitable dilution, as described by Tolle[9].

The monovalent BSA test was performed as described by Giesecke and Viljoen[5], and precipitin rings of 8 mm diameter or more were considered positive.

The results are summarised in Table 1.

DISCUSSION

Even when applying the IDF standard set of criteria, the incidence of 12,7% of subclinical septic mastitis in the 6804 quarters examined is lower than expected. Reports from other countries indicate that as many as 50% of cows in average herds may have such subclinical mastitis in at least one quarter at any time. The 17 herds concerned in this survey had not experienced any particular clinical mastitis problem and the survey was of a routine nature. The situation may perhaps be very different in problem herds or in a larger sample of the dairy cow population in South Africa.

The incidence of subclinical septic mastitis is however significantly lower when requiring that quarter milk complying with the IDF diagnostic standard should also contain excessive quantities of serum albumin before the diagnosis of true subclinical mastitis is recorded. The 10% of quarters producing mastitogen infected milk of high somatic cell content but negative to the BSA test could then only be classified as being affected with galactophoritis or a significant teat canal infection. Based on this assumption it would appear that in the herds concerned, the ratio of true subclinical septic mastitis to significant teat canal infections lies between 1:4 and 1:5. It remains uncertain whether this ratio would apply in herds where a clinical mastitis problem was being experienced or where the incidence of subclinical septic mastitis was higher than in the herds in this survey.

Concomitance of teat canal infections and milking malpractices may for example lead to a relatively higher proportion of true subclinical mastitis in the herd concerned.

These results indicate that the difference between the incidence of true subclinical septic mastitis and bacterial colonisation of the inflamed duct system, which ends at the teat orifice, is considerable and should receive greater consideration. The difference may be of significance in assessing the relative economic effect of such conditions, and then there is also the question of their treatment and prevention. These findings confirm to some extent the contention of Pearson who has emphasized that "researchers in the field of mastitis must define carefully what, in fact, they are studying or what they are advising when measures of control are being advocated"[7].

* Coulter Electronics (Pty) Ltd.

ACKNOWLEDGEMENTS

The cooperation of the milk producers concerned and the technical assistance of Mariana Erasmus, Maria Beyer, Sandra Marais and Linda Halland is gratefully acknowledged.

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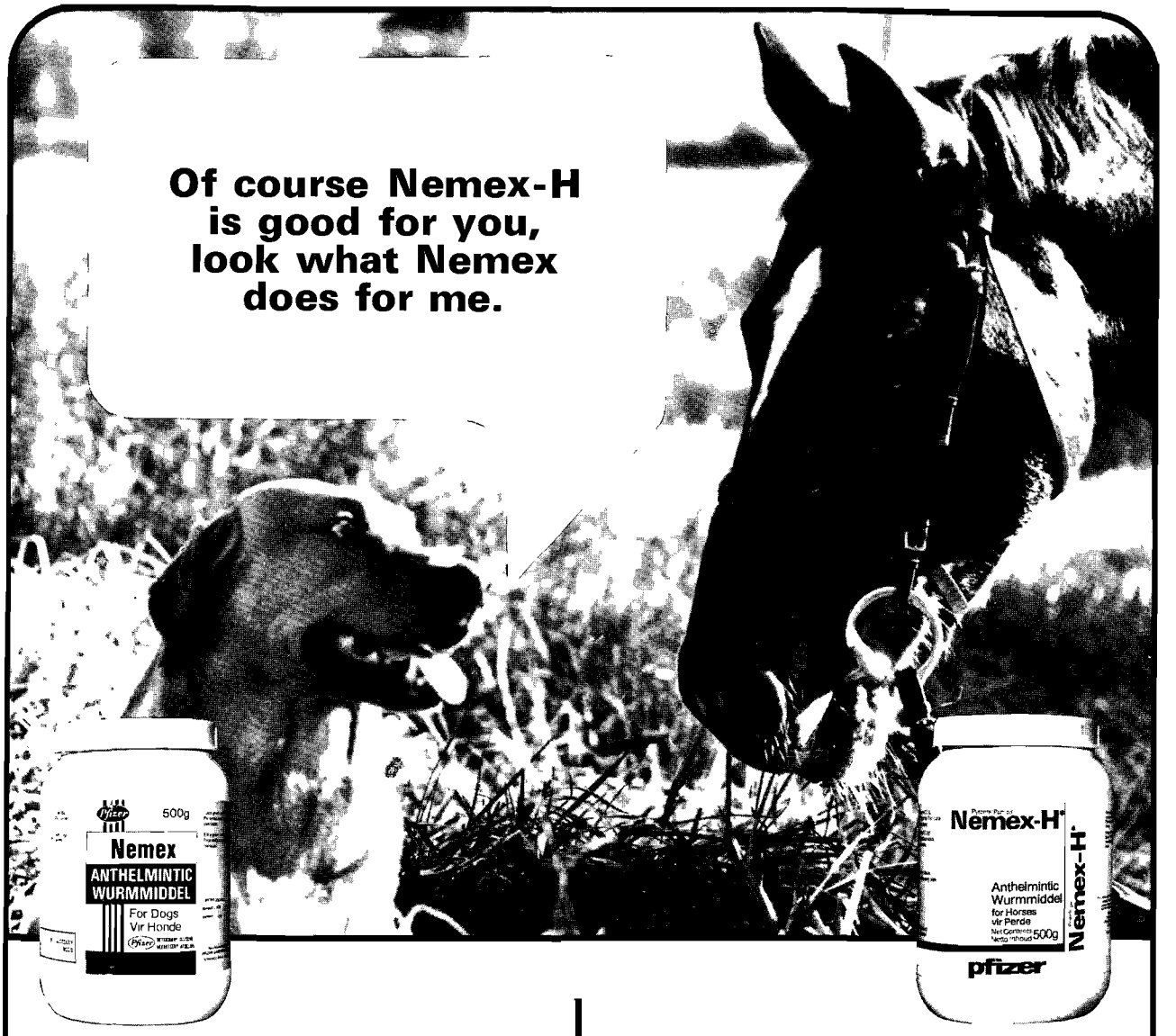

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Jl. S. Afr. vet. Ass. 47(4) 267-275 (1976)

CLINICAL COMMUNICATION KLINIESE MEDEDELING

CANINE SYSTEMIC LUPUS ERYTHEMATOSUS
THE DISEASE, CLINICAL MANIFESTATIONS AND TREATMENT

D.J. MOORE*

ABSTRACT: Moore, D.J. **Canine systemic lupus erythematosus: The disease, clinical manifestations and treatment.** Journal of the South African Veterinary Association (1976) **47** No. 4, 267-275 (En) P.O. Box 46149, 2119 Orange Grove, Rep. of South Africa.

The similarities between human and canine systemic lupus erythematosus are enumerated, using the incidence of symptoms reported for man as a guide to the possible incidence of clinical signs in the dog. Differences between the human and canine diseases are listed. Four clinical cases of the disease are reported. Diagnostic procedures were confined to the observation of clinical signs and serological and haematological methods.

INTRODUCTION

Systemic lupus erythematosus (SLE) is a chronic disease affecting connective tissue, cells and many organ systems, either individually or in combinations. The clinical course may be fulminating or indolent but is usually characterized by periods of remission and exacerbation[38]. A hallmark of the disease is the presence of large numbers of antibodies to nuclear components[24 26].

The term *lupus* has been used in human medicine to describe morbid cutaneous conditions for at least seven centuries. Rogerius (c.1230) is credited with using the term, being a Latin derivative *(lupus* — wolf) to describe erythematosus ulcerations about the face — a disease which eats away, bites and destroys[9].

The canine analogue, which mimics the human disease to a remarkable degree, was first described by Lewis, Henry, Thornton and Gilmore in 1963[17].

AETIOLOGY

SLE is an autoimmune or immune complex disease of unknown cause[8 9 24]. Autoimmunity is an immune response, either humoral or cellular, resulting in self tissue injury[1 39]. The response may reflect:

(1) Clones of tolerant lymphocytes becoming self-reactive after mutation[8 37].
(2) A reaction to self antigens sequestrated anatomically from the immune system during foetal development and appearing later as a result of tissue breakdown[1 8 37].
(3) A reaction to self antigens which cross-react with exogenous antigen[1 8 37].
(4) A reaction to self antigens which become altered by exogenous antigen or hapten[1 8 37].
(5) Deficiency of the immunologic regulating system[7 37].

The present concept of the development of autoimmune disease requires a brief review of immunobiology.

Fig. 1 is a schematic summary of the development of humoral and cellular immunity, in which the lymphoid stem cells are derived from the yolk sac, liver and eventually bone marrow during ontogeny[38 42].

The stem cells pass to the thymus and bursa of Fabricius equivalent (Peyer's patches, tonsils, appendix or bone marrow) and develop under the localized milieu into T and B lymphocytes[38].

T lymphocytes are long-lived and circulate as small uncommitted lymphocytes until they come into contact with antigen, after which they migrate to the lymphoid tissue and develop into short-lived committed lymphocytes and memory cells[1 38]. The latter are responsible for the characteristics of the secondary immunologic response[38]. Both types of cells are located in the paracortical areas of the lymph nodes, periarteriolar lymphocyte sheaths of the spleen and diffuse lymphoid tissue of Peyer's patches[38]. The committed T lymphocytes are responsible for cellular immunity[1 7 38 39 42 45].

B lymphocytes migrate to the lymphoid organs and remain in the lymph follicles and white pulp of the spleen and cortical areas of the lymph nodes[38]. On exposure to antigen they develop into plasma cells and memory cells[38 45]. The former are responsible for antibody production[38 45].

In both humoral and cellular immunity macrophages process the immunogenic material after phagocytosis and combine the antigen with macrophage RNA which then acts as the immunogenic stimulating factor[38].

Antigenic stimulation of B cells can be either direct (thymus-independent antigen) or indirect (thymus-dependent antigen) and varies in relation to the inducing antigen[1 7]. Indirect stimulation requires the co-operation of activated helper T cells which produce a factor and induce B cells to respond to thymus-dependent antigens[1 7]. The primary helper factor is antigen specific and is a soluble non-immunoglobulin having a molecular weight of approximately 50 000 daltons[7]. Thymus-independent antigens stimulate B cells without the participation of helper T cells[7].

Antibodies are principally gamma globulins and differ in primary amino acid sequence thus

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* Veterinary Practitioner, P.O. Box 46149, 2119 Orange Grove.

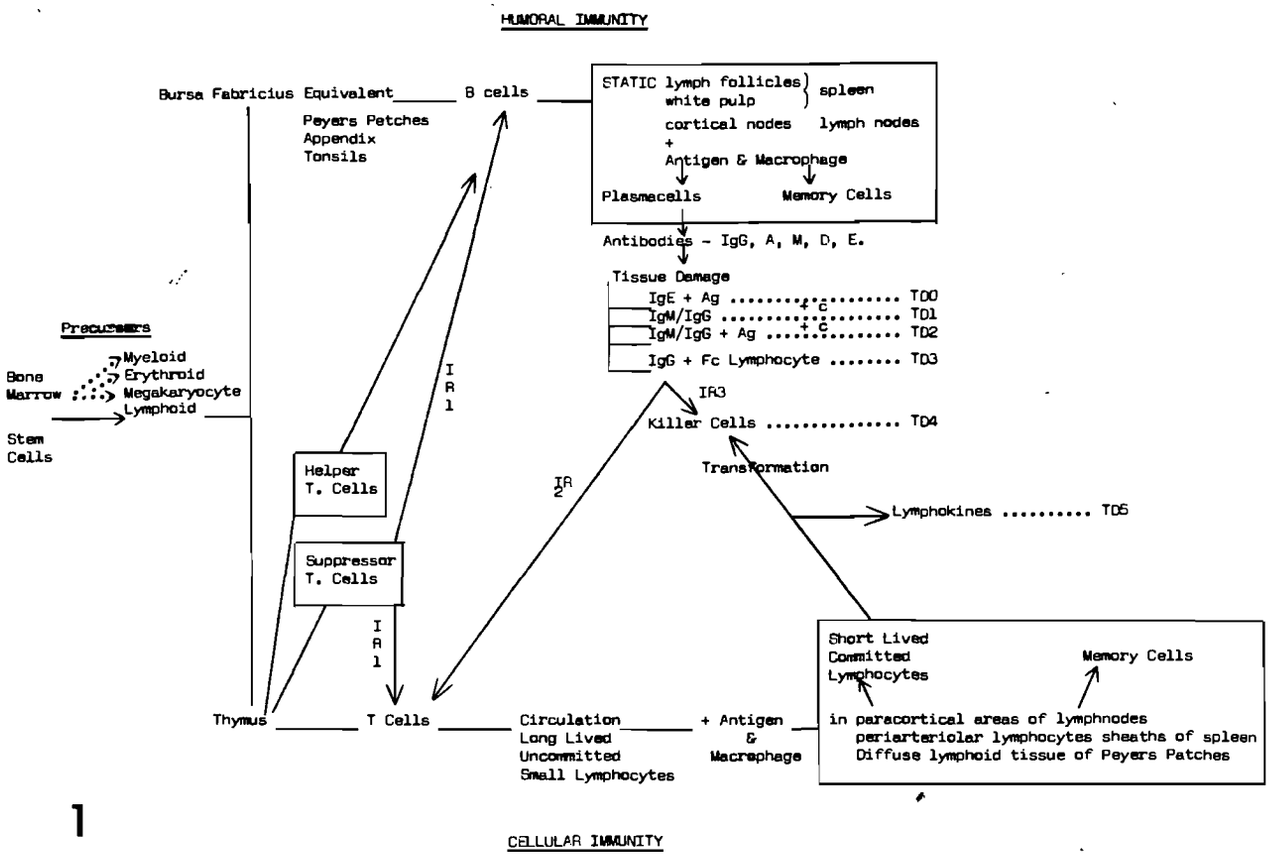


Fig. 1: A schematic summary of the development of humoral and cellular immunity.

characterizing them into different classes with distinct chemical structure[38]. These are designated by the letters G,A,M,D,E[38]. The dog lacks antibodies of the immunoglobulin (Ig) D class[37].

Antibodies may induce tissue damage (TD) directly by reacting, once localized on the cell surface, with antigen without the participation of complement (C) as in TDO, or directly by reacting with cell surface antigens utilizing complement (TD1) or indirectly via immune complexes and utilizing complement (TD2) or via antibody mediated cell-dependent lymphocytotoxicity (TD3) where cells sensitized with antibody but not damaged directly can be detected and damaged by B cells or macrophages with receptors for the Fc immunoglobulin determinants[39].

Specifically stimulated T cells may cause cell damage either directly (TD4) or via the release of mediators (TD5)[39].

As well as inducing tissue damage, antibodies are responsible for immunoregulation (IR) or blocking of cell mediated immune response[1 7 39]. The immunoregulation takes place either directly via the action of IgG or IgG-antigen complexes acting on T cells or indirectly via the stimulation of Suppressor T cells[1 7 39]. This type of immunoregulation is now known to be responsible for some of the normal phenomena of tolerance to self antigens (IR 2 + 3)[1 7 39].

A small percentage of T cells (Suppressor T cells) act in a controlling capacity so that if B cell or T cell activation does occur to an autoantigen resulting in autoantibody production, these controlling T cells are able to inhibit this response (IR1)[1 7 39].

A deficiency of this controlling T cell population could therefore account for the persistence of autoantibody production[1 2 7 14 28 39 41 42].

The mechanism of induction of a deficient controlling T cell population is one of much conjecture and the following observations have been made in SLE:—

(1) A genetic predisposition may be indicated as the disease often occurs in related individuals[1 7 8 9 24].
(2) C-type RNA viruses have been isolated and these attach specifically to lymphocyte membranes and may possibly be the cause of the antilymphocyte antibody found in this disease[16].
(3) Thymic defects, indicated by histopathological changes, have been noted[8].
(4) Thymic hormone deficiency[8 36].
(5) Inherited deficiencies of the complement system occur in patients[11].
(6) The disease occurs more frequently in females[4 8 9 19].
(7) Certain drugs are known to induce the disease[8 9 24].

Thus genetic factors and viral infections play a significant role.

PATHOGENESIS

The serum of patients with SLE contains many autoantibodies of the IgG, IgM and IgA classes which are directed against nucleoprotein, DNA, RNA (collectively termed antinuclear antibodies or ANA) lymphocytes, thrombocytes, erythrocytes and many other intracytoplasmic constituents such as mitochondria, lysosomes and soluble protein[9 24].

Antigen-antibody complexes circulate and although the bulk of the material is removed by the reticuloendothelial system, small amounts of the immune complexes are entrapped by vascular and glomerular basement membranes. The complement system becomes activated and results in membranolysis and release of vasoactive amines which cause increased vascular permeability and chemotax-

is of leukocytes. The invading polymorphonuclear leukocytes release their proteolytic enzymes which aggravates the local tissue destruction. This results in narrowing of the capillary lumens, hypoxia, tissue damage and fibrinoid necrosis[9 24].

PATHOLOGY

A vasculitis and fibrinoid desposition is the most common finding in inflammatory sites which involve many organs. By immunofluorescent methods fibrin and serum proteins, including immunoglobulins, complement and DNA have been detected in fibrinoid[9].

Haematoxylin bodies (defined as haematoxylin stained round or oblong masses in areas of inflammation) are thought to represent degenerated nuclei that have interacted with ANA[9].

CLINICAL MANIFESTATIONS

The prevalence of SLE in man is estimated to be 2 to 3 per 100 000 and 77% of patients have a five year survival[24], indicating a rare disease with a grave prognosis. Some patients have spontaneous remissions, others respond favourably to treatment while in some the course is unresponsive to currently available therapy[24].

Articular manifestations exhibited as a migratory polyarthralgia are the cardinal symptoms of the disease in 92% of human patients[9 24] as well as being the most common clinical sign in the dog[18 19]. Warmth, redness, pain and swelling of especially the carpus, elbow, tarsus and knee are shown[8 9 18 24]. Myalgia, stiffness in gait and reluctance to exercise are frequently present[18]. Joint deformity is rare[18 24] although aseptic necrosis of especially the femoral head may occur[24].

Fever during the acute phase of the disease is present in 84% of human patients[9 24] and is frequently present in the dog[17 18 19].

Fatigue, malaise, anorexia and progressive weight loss usually occur although some patients may have no systemic manifestations[8 9 12 18 24].

Cutaneous manifestations in the form of a facial eruption with a butterfly distribution over the malar areas and bridge of the nose consisting of erythema, atrophy, telangiectasis and keratotic plugging occur[9 18 24]. The lesions are worsened by ultraviolet rays[9 24]. Small infarcts and ulcers in the skin, nasal and oral mucous membranes may occur[9 18 24].

A generalized hair loss or patchy alopecia with short broken hairs and grey pink hyperkeratotic lesions are also described[9 18 24].

A haemorrhagic tendency with petechiae in the skin and mucosae occurs in 17% of human patients[12] although it may be more frequent in the dog[18] and is due to antibodies directed toward various clotting factors particularly Factor X and thrombocytes[9 15 17 18 46].

Lymphadenopathy either localized or generalized occurs in 58% of human patients[9] and is frequently present in the dog[18] and is thought to be due to increased activity of the immune system[9 24].

Hepatosplenomegaly is encountered frequently[9 18 24].

Renal manifestations are the most serious complication of the disease and 53% of human patients exhibit glomerulonephritis in the form of azoturia, cellular casts, hyaline casts, proteinuria, haemoglobulinuria and haematuria[12 14]. Eventually renal failure supervenes. Dogs exhibit similar renal failure[18 29 34]. The renal lesions have been classified into focal glomerulonephritis, diffuse glomerulonephritis and membranous lupus nephritis[8 9 12 18 24 29 37].

The following photomicrographs of human biopsy material are presented because of their similarity to the canine lesions and because permission for biopsy was not granted for any of the clinical cases to be discussed below.

Fig. 2 shows a normal glomerulus consisting of a mass of anastamosing capillaries with thin basement membranes and endothelium surrounded by Bowman's capsule with a thin basement membrane and parietal (capsular) epithelium and visceral (glomerular) epithelium. Erythrocytes can be seen in the capillaries.

Fig. 3 shows focal glomerulonephritis with segmental involvement of the glomerular tufts and increased cellularity in the affected areas. Only a small number of glomeruli are involved. Proliferation of the endothelial cells occurs with focal necrosis and infiltration of polymorphonuclear leukocytes. At this stage proteinuria will be present but not azoturia[9].

Fig. 4 shows a diffuse glomerulonephritis which has all the features of the focal disorder but involvement is more severe with widespread interstitial and tubular inflammation along with the more uniform glomerular involvement. Tubular atrophy is common, together with fibrosis of Bowman's capsule and thickening of the glomerular basement membranes and infiltration of lymphocytes and plasma cells. Renal failure is the rule with azoturia, hypertension, oedema and haematuria[9].

Fig. 5 shows membranous lupus nephritis with the capillary basement membrane thickened unevenly and some capillaries obstructed. Tubular atrophy and interstitial mononuclear cells are present. A typical nephrotic syndrome results in hypoproteinaemia, weight loss and generalized oedema[9]. Cardiopulmonary manifestations in the form of a polyserositis and including a pleuritis and pericarditis with effusions is present in 60% of human patients[9] but has not been described in the dog. A myocarditis and non-bacterial verrucous endocarditis may occur and is the cause of cardiac murmurs occasionally heard[9 24].

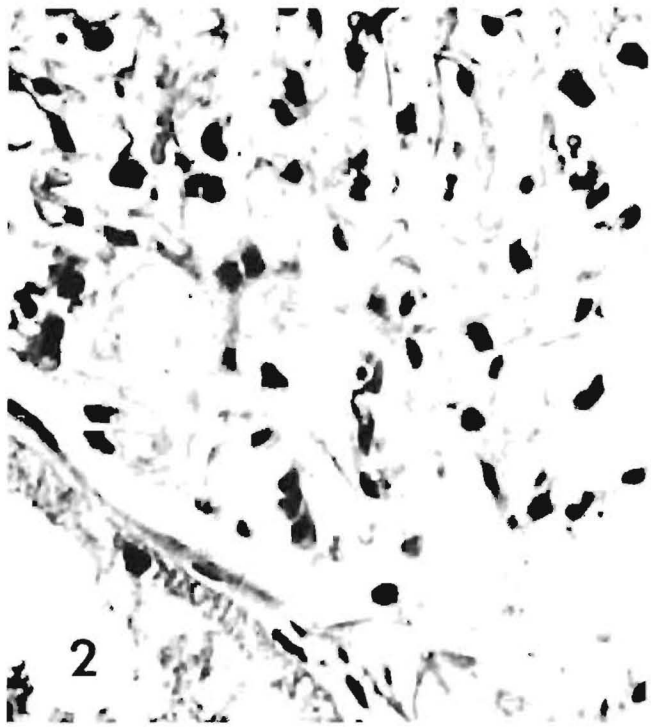
Neurologic manifestations may include convulsions, ataxia, paralysis or alterations in mental function and occur in 26% of human patients[24] and have been described in the dog[19]. These symptoms occur as a result of ischaemic or haemorrhagic events[24]. Blindness due to retinal haemorrhage and vascular thrombosis occur infrequently[24]. Retinal lesions have been described in the dog[19].

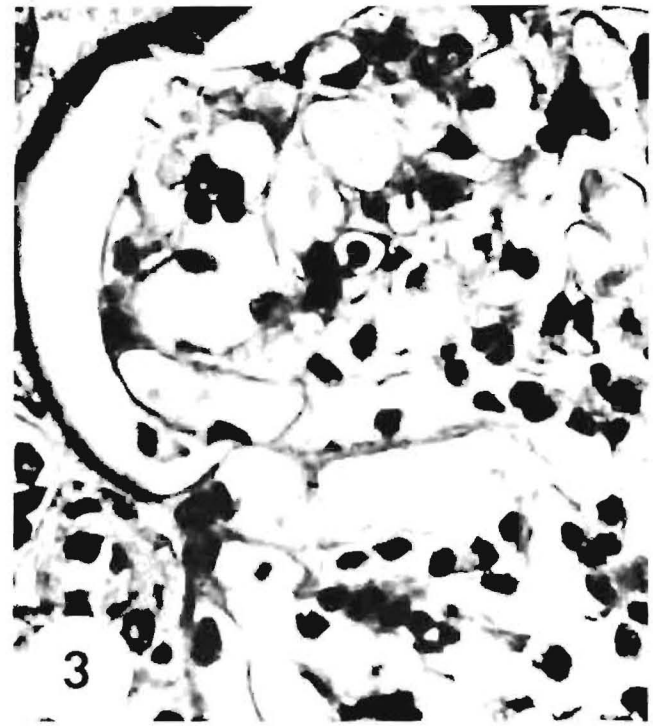
LABORATORY FINDINGS

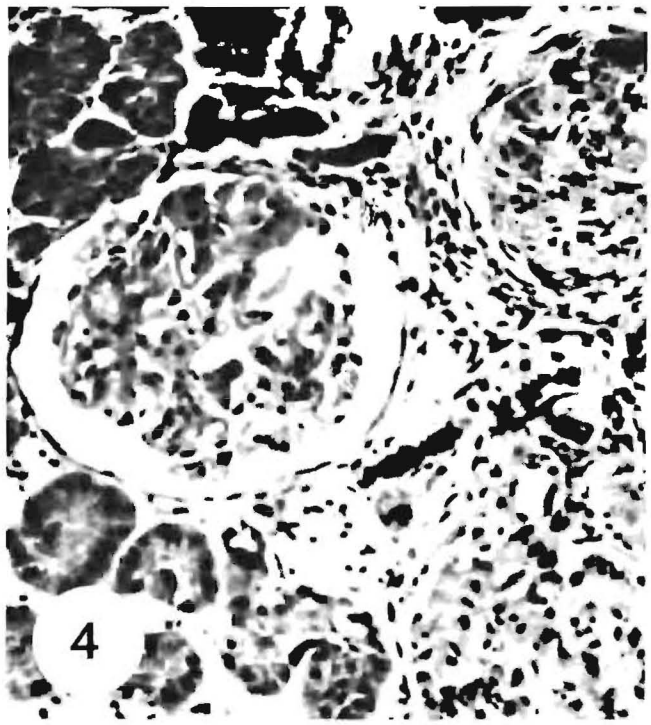
The Haemogram or Complete Blood Count (CBC)

A mild normochromic, normocytic anaemia is present in 78% of human patients[9] and is frequently present in the dog[18 19].

A leukapoenia with the decrease in lymphocytes greater than the decrease in granulocytes is a typical finding in man[9 24] and is due to autoantibodies directed toward lymphocytes[20], possibly altered antigenically by C-type RNA viruses[16]. Human patients with active SLE have decreased numbers of cir-







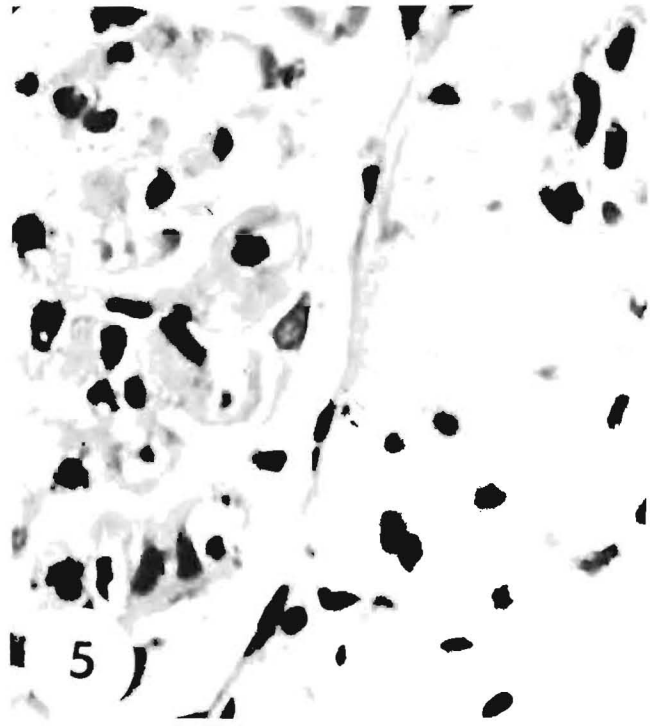


Fig. 2: Photomicrograph of the kidney demonstrating the normal glomerular histology. 8HE × 800.

Fig. 3: Photomicrograph of the kidney demonstrating focal glomerulonephritis. HE × 800.

Fig. 4: Photomicrograph of the kidney demonstrating diffuse glomerulonephritis. HE × 400.

Fig. 5: Photomicrograph of the kidney demonstrating membranous Lupus Nephritis. HE × 800.

culating T and B lymphocytes and increased numbers of lymphocytes that lack either B or T markers ("Null Cells")[5 35 36].

Dogs usually exhibit a normal or slightly elevated leukocyte count[17 18 19] and this is possibly an expression of reduced resistance with resultant secondary infection.

Thrombocytopaenia is present in 15% of human patients[24] and the incidence in the dog is usually considerably higher[18 23 34 46]. The cause of the thrombocytopaenia is autoantibodies directed towards platelets[15 46].

Coomb's (antiglobulin) Test

The Coomb's Test determines the presence of serum autoantibodies directed against erythrocytes (indirect Coomb's Test) or the presence of incomplete autoantibodies coating erythrocytes (direct Coomb's Test)[10 37]. A positive direct Coomb's Test is usually present in autoimmune haemolytic anaemia[3 23 34] and may be present in SLE[9 18 24 34 46], immunologically mediated thrombocytopaenia[46] and rheumatoid arthritis[21].

A positive direct antiglobulin test is present in 14% of human patients[24] but may be considerably higher in

the dog[18 34 46].

Because of the difficulty in standardizing canine antiglobulin, papain has been employed to modify the erythrocyte membrane to expose incomplete antibodies[13]. The Papain Test is possibly more sensitive in dogs than the direct antiglobulin test[13].

Antinuclear Antibodies (ANA)

ANA are serum autoantibodies of the IgG, A and M classes directed against nucleoprotein, DNA, RNA, histone and nucleoli[26]. ANA lack species specificity and these autoantibodies present in human and canine SLE patients can react with nucleoprotein (antigen) from a wide range of vertebrate species[9]. This forms the basis for the tests used to detect ANA.

The immunofluorescent technique is positive in 99% of human patients with SLE[26]. However, positive reactions can occur in other diseases such as rheumatoid arthritis, Sjögren's syndrome, dermatomyositis, periarteritis nodosa[9], thus decreasing the specificity of the test. The indirect immunofluorescent test utilizes a source of nucleoprotein (antigen) such as mouse liver, to which the patient's serum (antibody) is added. Fluorescent antiglobulin specific for the species being tested is added[37]. Depending on the pattern of fluorescence when viewed under ultraviolet light, ANA can be detected[26].

The Latex Nucleoprotein Test is positive in 82% of human SLE patients[6]. The test reagent is polystyrene particles coated with deoxyribonucleoprotein (derived from foetal calf thymus) to which is added the patient's serum. If ANA is present macroscopic agglutination occurs[6]. This test is available in kit form (SLE Latex Test Kit — Lederle Diagnostics) and is a suitable simple screening procedure and gives results similar to the LE Cell test[6] although this is disputed by some[9].

Lupus Erythematosus (LE) Cell Test

The LE cell is a granulocyte leukocyte (usually a neutrophil) containing a large homogeneous intracytoplasmic inclusion which displaces the segmental nucleus against the cell membrane[9]. These cells are present in 60 to 80% of human SLE patients[24] and are frequently present in canine patients[18 19 34]. LE factor is an autoantibody of the IgG class which reacts with nuclear material and fixes complement thus forming an immune complex[9]. This complex is then phagocytosed by a granulocyte leukocyte resulting in an LE cell[9].

The test is conducted on blood or bone marrow which is incubated, traumatized and centrifuged[37]. Smears are made from the buffy coat to detect LE cells. LE cells can sometimes be detected in other diseases such as rheumatoid arthritis, Sjögren's syndrome and scleroderma[9].

Rheumatoid Factor (RF) Test

RF is an autoantibody mainly of the IgM class directed against native IgG[9]. RF is found in 20% of human SLE patients[24]. RF is found in 84% of human rheumatoid arthritis patients[33] and can sometimes be found in osteoarthritis, fibrositis, periarteritis nodosa and scleroderma[33]. RF has been found in dogs with SLE and rheumatoid arthritis[21].

The Rose-Waaler sheep red blood cell agglutination test is used to detect RF[21 37]. Sheep erythrocytes are coated with subagglutinating doses of rabbit or canine anti-sheep erythrocyte serum[37]. The serum to be tested is to be mixed with the sensitized sheep

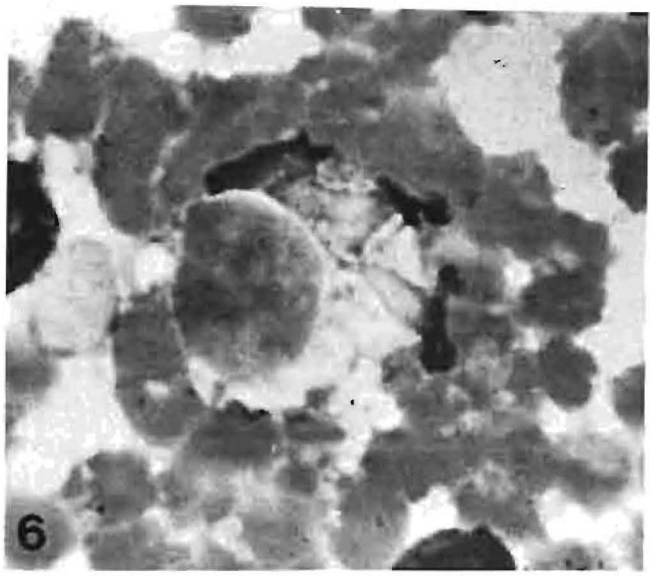


Fig. 6: LE Cell demonstrating a neutrophil with a three lobed nucleus compressed by a homogeneous LE body. HE × 1500.

erythrocytes, incubated and examined for agglutination[37]. The Latex Particle Test utilizes latex particles coated with human IgG[33]. The patient's serum is added to the particles and if RF is present macroscopic agglutination occurs. This test is available in kit form (RA-Test-Hyland) and is a simple screening procedure to detect human RF, as the test correlates well with results obtained using the Rose-Waaler test[33]. However, canine RF does not cross react well with human IgG and thus the sensitivity of the test in dogs is questioned[21 37] unless canine IgG is used as antigen[37].

Double-stranded DNA Antibody Test

The Double-Stranded DNA Antibody Test is currently the most specific test available for demonstrating SLE as all patients with the disease exhibit antibodies to double stranded DNA[26 30 41]. The test determines antibodies to double stranded (native) DNA in contrast to single stranded (denatured) DNA. Antibodies to double stranded DNA occur in SLE, rheumatoid arthritis and Sjögren's syndrome whereas antibodies to single stranded DNA occur in a variety of diseases[41]. The test is most accurate when utilizing the radioactive technique described by Pincus, Shur, Rose, Decker and Talal[30], and is available in kit form (anti DNA Kit-The Radiochemical Centre).

Complement (C')

Complement is an intrinsically linked system of eleven enzyme precursors found in the serum and other body fluids which can be activated to interact in a sequence of restricted proteolytic reactions which potentiate inflammation[25 38].

A serum hypocomplementanaemia, usually measured as the C3 component, is found in 75% of human SLE patients[24] and is due to the activation and consumption of C' by immune complexes[11 14 24]. Complement levels in the dog are infrequently determined and little information is available as to its level in SLE[21].

Serum Proteins

The serum proteins are separated electrophoretically into albumin, alpha, beta and gamma-

globulins[21 37]. The gammaglobulin fraction contains antibodies and is raised in inflammatory processes and immunoproliferative disorders[21]. An increase in gammaglobulin fraction is found in 60 to 77% of human patients[24]. Hypergammaglobulinaemia is found in canine SLE although the time of sample collection during the course of the disease may affect the level[18].

DRUG INDUCED SLE

The following drugs have induced the disease in a small percentage of human patients: hydrazaline, procainamide, several anticonvulsants e.g. dilantin, phenothiazine derivatives, alphamethyldopa and levodopa[24]. Drug induced SLE patients do not develop antibodies to double stranded DNA[41] and the symptoms abate on drug withdrawal[24]. In some patients the administration of sulphonamides and penicillin have been associated with exacerbation of SLE[24]. Drug induced SLE has not been reported in the dog.

TREATMENT

A cure for SLE is currently not available. However, abundant evidence indicates that appropriate therapy may suppress flare-ups and prolong life[24].

The arthralgias, myalgias and fever may respond adequately to rest and salicylates[24].

Central nervous system involvement, cardiopulmonary manifestations, renal disease, haemolytic anaemias, clotting defects, leukopaenias and thrombocytopaenias are indications for corticosteroid therapy[24]. Prednisolone is the drug of choice dosed at 2mg/kg live mass[24 34]. Once improvement has occurred the dose should be reduced to maintain control of symptoms[24]. If all clinical symptoms disappear therapy may be discontinued, although human patients are invariably maintained on a low maintenance dosage[24]. Spontaneous recovery occurs infrequently[24].

Diffuse and membranous glomerulonephritis do not respond well to therapy and cytotoxic agents such as azothioprine or cyclophosphamide are used at a dosage rate of 1 mg/kg live mass[24]. They are used in conjunction with corticosteroids.

Patients with SLE are prone to bacterial infections and the patient should be monitored to detect this[24]. The dog is particularly prone to secondary infections during the course of SLE and broad spectrum antibiotics should be administered routinely once the diagnosis has been made[18 34].

Exacerbations of SLE frequently occur during the last trimester of pregnancy or in the immediate post partum period and as the drugs used may be teratogenic they should be used judiciously[24].

A new approach to therapy has been proposed since indications are that SLE may in fact be an immune deficiency syndrome resulting from a lack of Controlling T lymphocytes.

Thymic hormone (Thymosin) administration to active SLE patients resulted in a decrease in "Null Cells" and an increase in T cells in the peripheral circulation as well as abatement of symptoms[36].

Levamisol, a broad spectrum anthelmintic, is now recognized as an immunomodulator as it improves the hosts' defence mechanisms by increasing leukocyte phagocytosis as well as stimulating T lymphocyte response[22 31 32 40 43 44]. Administration of levamisol to active SLE patients has a similar effect on "Null Cells" and T-cells as Thymosin, described above.

An attempt is also being made to reconstitute the affected patients' immune systems by adding self regulatory antibody produced by an immunologically fully competent individual[2].

An interesting adjuvant to treatment of SLE is the use of tranexamic acid. Plasmin (fibrinolysin), a serum enzyme is capable of activating the complement system and thus potentiating the pathogenesis of SLE. Tranexamic acid inhibits plasmin and its use in SLE should not be overlooked[27].

MATERIAL AND METHODS

Laboratory tests consisting of white blood cell counts (WBC), red blood cell counts (RBC), haemoglobin level (Hb), mean cell volume (MCV), mean cell haemoglobin (MCH), mean cell haemaglobin concentration (MCHC) and platelet number are determined by electronic counter.

The haematocrit (Ht) was determined by a standard michohaematocrit technique.

Blood urea nitrogen was determined using a photometric microtechnique utilizing the Unimeter (Bio-Dynamics) system.

Urine sediment examination was achieved using a mid-stream urine sample which was centrifuged at 1000 rpm for 10 minutes. The sediment was stained with Sedistain (Clay Adams) and examined microscopically. Urine protein estimation was achieved using Multistix (Ames).

ANA was determined using the ANA Latex Test Kit (Lederle) as a screening procedure only.

LE cells were determined from clotted blood employing the modified two hour sieved-clot technique of Dubois[9].

Incomplete erythrocyte antibodies were determined using the papain technique of Jones and Dale[13].

Rheumatoid factor was determined utilizing the human RA test (Hyland) as a qualitative test only.

Anti DNA antibodies were determined utilizing the anti-DNA kit (The Radiochemical Centre) and were regarded as positive if anti DNA antibody concentration was greater than 25 units/ml.

CASE HISTORIES AND TREATMENT

The following cases are presented with histories and clinical and laboratory findings to illustrate the diverse organ involvement of SLE. All the patients have responded to therapy.

Case 1:

An 8 month old female German Shepherd suddenly began limping severely on her left foreleg, after which her right hind leg became painful. She was unable to stand and was anorexic when presented. The carpus and tarsus of the affected legs were oedematous, warm and very painful. The mucosae were pale and the rectal temperature was 41°C. A blood smear for *Babesia canis* was negative although it revealed a neutrophilia with a left shift.

Table 1 is a summary of the laboratory findings before 1974.12.11 and after 1975.07.20 treatment. The significant deviations from normal in the initial report were a leukocytosis with left shift (WBC 25 000/mm³), slight anaemia (RBC 5,33 Mil/mm³, Hb 13,7g%, Ht 35,9%), thrombocytopaenia (platelets 70 000/mm³),

Table 1: LABORATORY DATA OF CASE 1

	Laboratory Report 11.12.74	Normal Values (average)	Laboratory Report 20.7.75
WBC	25 000/mm³	11 500/mm³	11 800/mm³
RBC	5.33 Mil/mm³	6.8 Mil/mm³	8.2 Mil/mm³
Hb	13.7 g%	15 g%	19 g%
Ht	36.9%	45%	53.3%
MCV	68 fl	70 fl	67 fl
MCH	24.7 pg	23 pg	23.3 pg
MCHC	36.3%	33%	35.7%
Platelets	70 000/mm³	350 000/mm³	276 000/mm³
ANA Latex Test	Positive	Negative	Negative
LE Cell Test	Positive	Negative	Negative
Papain Test	Not Done	Negative	Negative
RA Latex Test	Not Done	Negative	Positive
Bun	40 mg%	Less than 20 mg%	10 mg%
Urine Sediment	Hyaline Casts Cellular Casts Proteinuria Haematuria	Negative	Negative

LEGEND REFERRING TO TABLES 1, 2, 3 & 4.

WBC = Leukocyte count; RBC = Erythrocyte count; Hb = Haemoglobin; Ht = Haematocrit; MCV = mean corpuscular volume; MCH = mean corpuscular haemoglobin; MCHC = mean corpuscular haemoglobin concentrations; Neut = Neutrophil Count; Mono = Monocyte Count; Lympho = Lymphocyte Count; Eosin = Eosinophil Count; Baso = Basophil Count; ANA Latex Test = Anti Nuclear Antibody Latex Test; LE Cell Test = Lupus Erythematosus Cell Test; RA Latex Test = Rheumatoid Arthritis Factor Latex Test; BUN = Blood Urea Nitrogen; Anti DNA = Double-Stranded DNA antibody Test.

positive ANA latex test, positive LE cell test and nephritis (BUN 40 mg/100 ml and urine sediment with hyaline casts, cellular casts, proteinuria, haematuria).

The treatment consisted of oxytetracycline at the dosage rate of 10 mg/kg live mass as well as prednisolone at the rate of 2 mg/kg live mass, both drugs being administered parenterally for 4 days.

The clinical response was dramatic and within 24 hours of the initial treatment the patient's temperature was 38°C and the dog was eating and able to walk without limping. The dog was discharged from hospital after 4 days and maintained on dexamethazone tablets (0,04 mg/kg live mass) for 3 weeks. Treatment was then stopped and the dog has remained in good health. The subsequent laboratory report indicates that all findings were within normal range except the test for rheumatoid arthritis factor was positive.

Case 2:

A 3 year old Scottish Terrier started losing weight after having her first litter and this became marked once the litter was weaned. Eventually she developed painful hindquarters and began limping severely on her hind legs. A gradual loss of appetite progressed to anorexia. Generalized lymphadenopathy and hepatosplenomegaly were present. The rectal temperature was 40°C and a blood smear for *B. canis* was negative.

Table 2 is a summary of the laboratory findings before 1975.03.14 and after 1975.08.15 treatment. The significant deviations from normal in the initial report were a leukocytosis (WBC 18 500/mm³), neutrophilia with left shift, thrombocytopaenia (platelets 60 000/mm³), positive ANA Latex Test, positive LE cell test and nephritis (BUN 35 mg/100ml and urine sediment containing cellular casts and protein).

The treatment instituted and the dosage rates used

Table 2: LABORATORY DATA OF CASE 2

	Laboratory Report 14.3.75	Normal Values (average)	Laboratory Report 15.8.75
WBC	18 500/mm³	11 500/mm³	12 300/mm³
RBC	6.67 Mil/mm³	6.8 Mil/mm³	5.7 Mil/mm³
Hb	15.6 g%	15 g%	13.9 g%
Ht	43.6%	45%	37.8%
MCV	68 fl	70 fl	70 fl
MCH	23.5 pg	23 pg	24.7 pg
MCHC	35.9%	33%	34.5%
Dif. Count			
Neut	87%	70%	Not Done
Mono	6%	5%	Not Done
Lympho	7%	20%	Not Done
Eosin	0%	4%	Not Done
Baso	0%	1%	Not Done
Platelets	60 000/mm³	350 000/mm³	200 000/mm³
ANA Latex Test	Positive	Negative	Negative
LE Cell Test	Positive	Negative	Negative
RA Latex Test	Not Done	Negative	Negative
Bun	35 mg%	Less than 20 mg%	15 mg%
Urine Sediment	Cellular Casts Proteinuria	Negative	Proteinuria
Anti DNA	Not Done	Negative	Positive

were identical to these used in Case 1. The clinical response was not as dramatic and recovery was protracted. The dog was discharged from hospital after 4 days. Thereafter oral dexamethazone tablets were administered and although the patient improved steadily she is maintained on 0,5 mg dexamethazone orally per day as a decrease in dose results in recurrence of symptoms. The subsequent laboratory report 1975.08.15 indicated that the only abnormal findings were a positive anti-DNA antibody test and proteinuria.

Case 3:

A 7 year old Labrador began losing weight and became lethargic anorexic and limped intermittently. Her haircoat became dull and she began shedding hair excessively. On presentation her rectal temperature was 39,5°C and a blood smear for *B. canis* was negative.

Table 3: LABORATORY DATA OF CASE 3

	Laboratory Report 8.8.75	Normal Values (average)
WBC	10 700/mm³	11 500/mm³
RBC	6.93 Mil/mm³	6.8 Mil/mm³
Hb	17.4 g%	15 g%
Ht	46.8%	45%
MCV	70 fl	70 fl
MCH	24.6 pg	23 pg
MCHC	36.1%	33%
Dif. Count		
Neut	66%	70%
Mono	16%	5%
Lympho	16%	20%
Eosino	2%	4%
ANA Latex Test	Positive	Negative
LE Cell Test	Positive	Negative
RA Latex Test	Positive	Negative
Bun	30 mg%	Less than 20 mg%
Urine Sediment	Proteinuria	Negative

Table 3 is a summary of the laboratory findings before treatment. The significant deviations from normal were a monocytosis, positive ANA Latex Test, positive LE cell test, positive RA Latex Test, and nephritis (BUN 30 mg/100ml and proteinuria).

The treatment instituted was identical to Case 1 and the dog was discharged from hospital after 4 days. The dog's condition improved considerably on oral dexamethazone therapy which was stopped after 3 weeks.

Case 4:

A 1 year old male Staffordshire Bull Terrier began limping and became anorexic and febrile at intermittent intervals. The dog was polydipsic. Eventually pruritic cutaneous erythematosus plaques developed on the abdomen and sternum. On presentation the dog's temperature was 40°C and a blood smear was negative for *B. canis.*

Table 4 is a summary of the laboratory findings before treatment. The significant deviations from normal were a positive ANA Latex Test, positive DNA test and nephritis (urine sediment revealed cellular casts, hyaline casts, haematuria and proteinuria).

The treatment instituted and the dosage rates used were identical to these used in Case 1. The clinical response was dramatic and the dog was discharged from hospital after 4 days. Oral dexamethazone therapy was stopped after 3 weeks and the dog has remained in good health.

Table 4: LABORATORY DATA OF CASE 4

	Laboratory Report 19.8.75	Normal Values (average)
WBC	9 400/mm³	11 500/mm³
RBC	6.2 Mil/mm³	6.8 Mil/mm³
Hb	12.5 g%	15 g%
Ht	40.3%	45%
MCV	67 fl	70fl
MCH	24.6 pg	23 pg
MCHC	35.4%	33%
Dif. Count		
Neut	79%	70%
Mono	4%	5%
Lympho	15%	20%
Eosin	2%	4%
ANA Latex Test	Positive	Negative
LE Cell Test	Negative	Negative
RA Latex Test	Negative	Negative
Anti DNA	Positive	Negative
Bun	18 mg%	Less than 20 mg%
Urine Sediment	Cellular casts Proteinuria Hyaline Casts Haematuria	Negative

DISCUSSION

The four case reports presented reveal that no breed predisposition was shown. Their ages ranged from 8 months to 7 years and three of the animals were female.

The dogs had similar disease patterns. All 4 patients exhibited lameness, fever, anorexia, positive ANA Latex Test and nephritis. A positive LE cell test was shown by 3 dogs. Both dogs tested for anti-DNA antibody were positive; Case 2 had an anti-DNA antibody concentration of 80 units/ml while Case 4 had a concentration of 132 units/ml. Weight loss was shown by 2 dogs. Thrombocytopaenia was present in both dogs on which platelet counts were performed. Rheumatoid Factor Test was positive in 2 of the 4 dogs tested. Leukocytosis was present in 2 dogs and anaemia in one. Lymphadenopathy and hepatosplenomegaly was shown by one dog.

The clinical symptoms, serological and haematological findings warranted a diagnosis of SLE. All the dogs responded dramatically to therapy and are alive and well. Only Case 2 is receiving a maintenance dose of dexamethazone.

It is hoped that this report will familiarize colleagues with an unusual canine disease and enable a definite diagnosis to be made where previously this may not have been possible.

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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 5 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 5 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)

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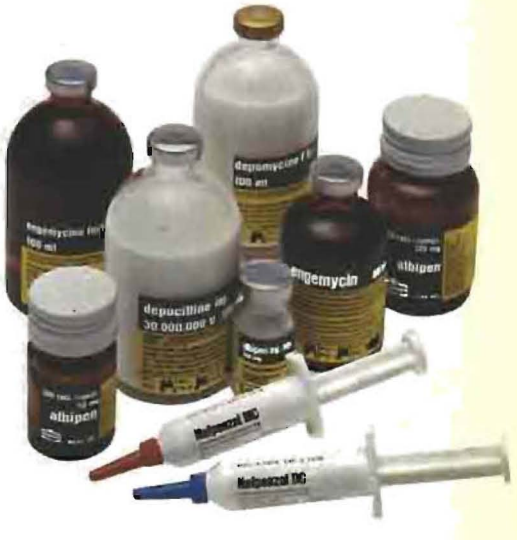

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LEHRBUCH DER VETERINÄR-PHYSIOLOGIE

A. SCHEUNERT und A. TRAUTMANN

6, Auflage

Verlag Paul Parey, Berlin und Hamburg. 1976. pp xxiv + 968, Figs 388 (2 colour). Tabs 146. Publ. Price DM 220

This 6th edition of the standard German language veterinary physiology text has been completely revised by a panel of 21 authorities. This has resulted in the addition of new chapters on general physiological principles and on behavioural physiology, and some rearrangement of other sections.

The main chapter headings encompass the physiology of the following areas: general principles; endocrinology; digestion; metabolism and energetics; thermo-physiology; the liver; blood, lymph and other body fluids; circulation; respiration, the kidney, water and electrolyte balance; reproduction, milk secretion, movement; the nervous system (including the organs of special sense); and behavioural physiology.

This is an excellent book. Despite the very wide field covered, the authors have managed to strike a fine balance between what is essential in order to understand basic principles and what might be unnecessary detail. There has been a judicious use of illustrations which ably supplements the text. A major emphasis exists with regard to integrated function which adds considerably to the value of the book. The main emphasis is of course on the domestic animals and the fowl, but many references to other animals are present. There are a number of very comprehensive tables of physiological norms which give the book added value as a source of reference.

The book is printed in clear style and the text is free of mistakes. The index is comprehensive and in addition most section, are concluded by a short bibliography. Literature references up to 1975 are included. The price of the book (± R80) is rather high.

This book is perhaps the best of the currently available veterinary physiology texts, and an English language edition would be well received.

J.K.

Jl. S. Afr. vet. Ass. 47(4) 281-287 (1976)

CLINICAL COMMUNICATIONS — KLINIESE MEDEDELINGS

A SYMPOSIUM ON CANINE BILIARY FEVER*

PAPER 1 : RELAPSES AND IMMUNITY IN CANINE BABESIOSIS

R.D. BIGALKE**

ABSTRACT: Bigalke, R.D., **Relapses and immunity in canine babesiosis.** *Journal of the South African Veterinary Association* (1976) **47** No. 4 281-287 Veterinary Research Institute, 0110 Onderstepoort, Republic of South Africa.

Canine and bovine babesiosis are compared with particular reference to the occurrence of relapses and some immunological aspects of the diseases. Intact dogs resemble splenectomized cattle in respect of the relatively frequent occurrence of clinical relapses of babesiosis. The possible reasons for this are discussed. Attention is drawn to the fact that a sterile immunity of variable duration follows on auto sterlization of some *Babesia* infections and that antigenically difference strains of the parasite occur in the field.

One is sorely tempted to state that biliary fever of dogs *(Babesia canis* infection) is very similar to redwater of cattle and to confine this discussion to the latter disease. The reason for this is that much is known about redwater whereas biliary fever has not received particular attention. For the purposes of this discussion it will be necessary to speak about both diseases, but the comments on canine babesiosis will be rather hypothetical.

1. RELAPSES

If a dog is successfully treated for biliary fever and is re-presented with similar symptoms, say 2 weeks later, there are 2 possible explanations to consider: (i) a relapse has occurred, or (ii) re-infection with either homologous or heterologous strains has taken place. In by far the majority of cases this phenomenon is probably due to a relapse, even though it is quite possible for re-infection to occur within that period.

Clinical relapses have not been observed by the author in intact (non-splenectomized) cattle even after successful treatment with a non-sterilizing drug. On the other hand, parasitic relapses, representing the peaks of low-level fluctuations in the numbers of parasites, which probably occur on account of antigenic variation of the parasite, are invariably observed. These peaks may be detectable by examination of blood smears, but the parasites are apparently not sufficiently plentiful to cause clinical signs of redwater.

Splenectomized cattle treated with non-sterilizing drugs, however, usually show one or more clinical relapses requiring therapy. The same drug used to treat the primary reaction can be used to control such a relapse. Eventually such cattle also become immune to show the low-level fluctuations in parasite numbers referred to above.

Why do splenectomized cattle relapse?

The obvious explanation is that splenectomy has interfered with their immune mechanism.

Some dogs are rather similar to splenectomized cattle in that they show relapses. What then is the explanation for this phenomenon in dogs?

An obvious prerequisite for a relapse to occur is that the infection must not have been sterilized by therapy. This is not the only explanation since not all dogs in which the infection has not been sterilized by therapy will relapse. On the other hand, it is also true that many dogs do not relapse because their infection has been sterilized. If a drug were available which would, at a specified dose, invariably sterilize the infection, relapses would not be a problem. Unfortunately no such drug exists.

In the case of cattle it is known that diminazene, phenamidine isethionate and quinuronium sulphate, at prescribed doses, almost invariably sterilize *Babesia bigemina* infections, whereas infection with *B. bovis* is not sterilized. Euflavine sterilizes neither infection, and imidocarb sterilizes both at a dose of 3 mg/kg.

In dogs diminazene sometimes sterilizes at a dose of 4 mg/kg, but 10,5 mg/kg of the drug is apparently necessary to ensure sterilization. In many cases 2 daily doses of 10 mg/kg of diminazene will kill the dog concerned.

There is some evidence that phenamidine not infrequently sterilizes the infection at the recommended dose of 15 mg/kg. We know, however, that 2 doses of 20 mg/kg will often be fatal.

The author is of opinion that dogs relapse to biliary fever because their immune mechanism is unable to cope with the infection for one or more of the following reasons:—

(a) Antigenic variation, alone or in combination with one or more of the following, is almost certainly involved.
(b) Sometimes the dog is probably to blame. This may be due to a deficient immune mechanism or a severe stress of some sort, like an intercurrent infection with *Ehrlichia canis.*
(c) Another possible explanation is that the drug or the form of therapy used is at fault, i.e., it does not suppress the infection to such an extent that the defensive mechanism of the animal can take over the control.
(d) The parasite may be to blame, i.e., drug-resistant: it is often necessary to use another drug to cure the patient.

All of this is rather hypothetical at this stage, but it is certainly true that the small animal clinician requires a formidable array of babesicidal drugs on his shelf. If, however, the case presented is a chronically

*Clinicians Group, Witwatersrand Branch, SAVA, November, 1975. Edited by L. Bomzon.

**Veterinary Research Institute, 0110 Onderstepoort

relapsing patient, which is looking fairly well, viz. eating, and the temperature is close to normal, it may be wise to withhold specific treatment and to keep the animal under close observation, which includes regular monitoring of bloodsmears for parasites, and symptomatic treatment only.

2. IMMUNITY

PREMUNITY

The old concept that immunity to babesiosis is only operative in the presence of the parasite in the body is no longer quite true.

In cattle it is known that auto-sterilization occurs in the case of infection with *B. bigemina* after a variable period of subclinical infection. With *B. bovis* infections the carrier state is more permanent. Animals which have undergone auto-sterilization are immune to infection with a homologous strain for a variable period. In other words a sterile immunity exists in babesiosis. But it is also true that a carrier is still better off from the point of view of immunity to reinfection than an animal with a sterile immunity. The main reason for this is that the premune bovine is often also protected from the clinical effects of infection with heterologous strains. It is also important to remember that exposure to the living parasite over a relatively long period is essential for the development of an effective sterile immunity.

Although little information is available the situation seems to be very similar in dogs. Non-sterile immunity, i.e. premunity, may be short (less than 1 month) or long (15 months or more) and is followed by a sterile immunity to challenge with homologous strains.

STRAIN DIFFERENCES

It is known that antigenically different strains of bovine babesias exist in the field. Their presence can be demonstrated very clearly under laboratory conditions in splenectomized cattle, but they do not appear to be very important in the field.

Although experimental evidence is lacking, antigenically different strains of *B. canis* almost certainly occur in nature. This would explain why some dogs contract biliary fever annually or even more than once a year. An alternative or even complementary explanation for this phenomenon would be that the infection is either sterilized by treatment or that auto-sterilization occurs very soon after infection, with the result that no, or a very poor, immunity is elicited against reinfection.

PAPER 2: THE BLOOD GROUPING SYSTEMS OF DOGS

D.J. MOORE*

Canine red blood cell antigens are divided into 8 groups and are designated by capital letters of the alphabet. The groups are A, B, C, D, E, F, G, Tr. These letters or groupings have no relationship to blood grouping systems in other mammalian species since the system of designation is based on the order of discovery of the antigen. For example, the first antigen discovered was designated Do for dog, but this was later changed to A as more antigens were identified.

The blood group antigens are glycolipids within the red blood cell membrane. Similar antigens occur in soluble form in various tissue fluids and secretions and are glycoproteins.

All the blood group antigens are capable of stimulating antibodies (isoantibodies) specific for that antigen, if introduced into a recipient negative for the antigen. The severity, however, of the antigen-antibody reaction is dependent upon the interaction of this complex with various plasma proteins. The antibodies are the gamma-globulins or immunoglobulins. They are categorised in the dog as IgG, IgA, IgM and IgE. Only IgG and IgM are capable of fixing complement and thus inducing haemolysis.

Naturally occurring isoantibodies occur in less than 15% of all dogs. The predominant isoantibodies found are the B and D antibodies and these cause a weak transfusion reaction. The A antibody, the predominant cause of most transfusion reactions does not occur naturally. Its presence in the blood occurs following a previous incompatible blood transfusion.

Blood groups A and to a lesser extent, blood group E are capable of inducing a transfusion reaction. The other blood group antigens stimulate agglutinin production and cause weak transfusion reactions.

*Veterinary Hospital, P.O. Box 46149, 2119 Orange Grove.

Table 1: THE INCIDENCE OF ANTIGENIC TYPES

Antigen	Incidence%
A	62.6
B	5.5
C	98.4
D	22.3
E	73.1
F	99.4
G	?
Tr	50

After any incompatible transfusion, isoantibodies can be detected 7-10 days later and cause destruction of the transfused red blood cells. No systemic manifestations of such a transfusion are evident unless antigen A is involved.

The ideal donor should be A negative, i.e., have no A antigens. The incidence of isosensitisation using random donors and recipients is 25%. The incidence of transfusion reaction occurring on a repeat transfusion is 15%. Table 1 shows the incidence of each antigenic type. Table 2 summarises the incidence of antigen combinations.

Antibodies in a sensitised individual destroy incompatible erythrocytes in 2 ways:— opsonisation and haemolysis. Opsonisation leads to extravascular haemolysis and this occurs when the red blood cells coated with antibody promotes phagocytosis by the reticulo-endothelial system and leukocytes. Haemolysis leads to intravascular haemolysis and this occurs when complement is fixed following an antigenic red blood cell-antibody reaction.

Transfusion reactions may be divided into 2 types:— immunologically — mediated or non-immunologically — mediated reactions. The symptoms of the immunologically-mediated transfusion

Table 2: THE INCIDENCE OF KNOWN ANTIGENIC COMBINATIONS

Antigen Combinations	Incidence %
A1C	29,6
C	27,2
A2C	13,9
A1CD	9,2
CD	6,8
A2C2	3,9
A1BC	2,9
A1	1,6
A1BCD	1,3
BC	1,0
A1D	0,5
D	0,5
A2	0,3
A1BD	0,3

reaction include dyspnoea, emesis, faecal and urinary incontinence, prostration, pyrexia, convulsions, haemoglobulinuria and haemoglobulinaemia. The onset of symptoms are within minutes of commencement of a transfusion.

The reaction is not usually fatal and recovery occurs within 12-24 hours. However, haemorrhagic tendencies can exist in the post-transfusion phase due to a leukopaenia and thrombocytopaenia. Acute renal tubular necrosis or lower nephron nephrosis, as occurs in man, does not occur in the dog.

Urticaria and erythema can also be seen following a transfusion reaction. These signs are mediated by the IgE antibodies and occur following sensitisation of the recipient to various protein fractions in the blood of the donor. These reactions are mild and spontaneous recovery usually occurs.

The non-immunologically — mediated transfusion reactions are of 2 types. Firstly, circulatory overload leading to dyspnoea, pulmonary oedema and coughing can occur following a too rapid rate of infusion or over-infusion. Secondly, citrate overload due to the chellating effect of citrate with calcium causing tremor and tetany.

Blood typing is only done in experimental laboratories as antiserum to the blood factors is not

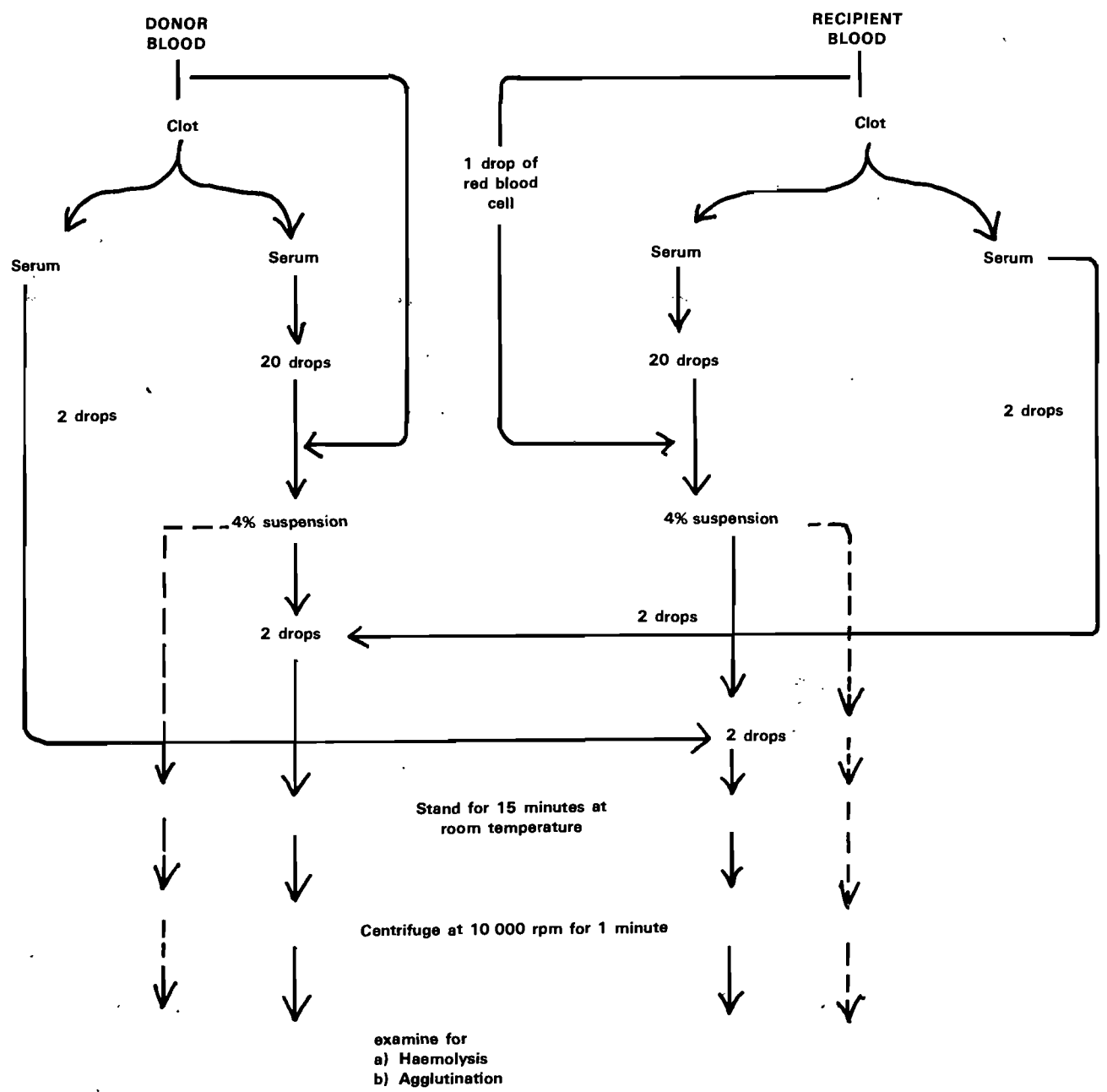

Figure 1: The method for crossmatching of blood

available commercially. Cross-matching is only of value in the case of a retransfusion since A isoantibodies do not occur naturally. The cross-matching procedure is quite simple and outlined in Figure 1. The tests should be done in small test tubes to detect haemolysis and agglutination.

Blood is taken from the donor and recipient and allowed to clot. The serum of each specimen is then divided into two equal aliquots. Red blood cells from each clot are mixed with one of its own serum specimens. Two drops of the donor cell suspension is added to 2 drops of the recipient serum. Two drops of the recipient cell suspension are added to the 2 drops of the donor serum. The tubes should be allowed to stand for 15 minutes at room temperature. The specimens should then be centrifuged for 1 minute at 10 000 rpm. The specimens should then be examined for haemolysis. This is then followed by an examination for agglutination by agitating the tube. If no macroscopic agglutination can be seen, a drop of the specimen should be examined microscopically under low power. Both donor and recipient cell suspensions should be examined in a similar manner after centrifugation to act as a control.

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PAPER 3 : FLUID THERAPY IN CANINE BABESIOSIS

C. BUTTON*

INTRODUCTION

Babesia canis infection of the dog's erythrocytes leads to an increased rate of reticulo-endothelial removal of red cells, or, more rarely, to intravascular haemolysis, haemoglobinaemia and haemoglobinuria.

A stage is reached where anaemic hypoxia of tissues results in anaerobic tissue metabolism, production of metabolic acids, shock and death. Organ failure, e.g. liver or kidney failure, may complicate the picture.

Medical therapy, if it is to be successful, must be prompt, vigorous and specific. In this paper, fluid therapy for biliary fever (BF) will be discussed. It need hardly be said that severely affected dogs require a blood transfusion as well, since only red cells can carry oxygen in significant amounts. Additional supportive therapy and intensive nursing are also required if the critically ill patient is to survive.

Four aspects of fluid therapy in biliary fever are:

1. Patient selection.
2. Fluid volume, route and rate of administration.
3. Fluid composition.
4. Recognition of sequelae of biliary fever in which fluid therapy is essential for survival.

1. PATIENT SELECTION

The following classes of patient require fluid therapy:

(a) All shock cases.
(b) All patients after blood transfusion.
(c) All older dogs, particularly those with a history of renal disease.
(d) All patients that are clinically dehydrated.
(e) All patients with intravascular haemolysis and haemoglobinuria. Although the canine kidney is relatively resistant to haemoglobin-induced nephropathy, haemoglobin casts do on occasion lead to renal "shut down".
(f) Patients with vomition and/or diarrhoea.
(g) Patients with polyuria resulting from ischaemic nephrosis.

2. FLUID REPLACEMENT

(a) FLUID VOLUME

A review of the patient's history to determine the approximate duration of the illness, whether or not the patient is still drinking, or is vomiting or has diarrhoea; a knowledge of the patient's usual body mass; clinical examination of the skin fold for elasticity, the eye for sunkenness and mucous membranes for dryness; and measurement of total serum proteins with a refractometer** should allow the clinician to place his patient in one of the following categories:

(i) Mildly dehydrated (approximately 5%), requiring 50 ml fluid replacement per kg body mass.
(ii) Moderately dehydrated (approximately 10%), requiring 100 ml fluid replacement per kg body mass.
(iii) Severely dehydrated (approximately 15%), requiring 150 ml fluid replacement per kg body mass.

Packed cell volume, usually a reliable means of determining the degree of dehydration, is obviously useless in biliary fever.

Patients suffering acute or peracute BF and shock may not be clinically dehydrated, and yet they may require fluid therapy to fill an expanded vascular bed. The volume of fluid required in such cases is less easily determined, but will, in general, lie between the above limits of 50 and 150 ml per kg body mass.

(b) ROUTE OF FLUID ADMINISTRATION

(i) Intravenous. For the majority of BF cases ill

*Dept of Medicine, Faculty of Veterinary Science, University of Pretoria, 0110 Onderstepoort

**"T.S. Meter", American Optical Instruments, Optolabor, Box 31208, Braamfontein, Johannesburg.

enough to warrant fluid therapy, the route of choice is intravenous. The use of "in-dwelling" intravenous cannulas is becoming more common and is certainly to be advocated for patients requiring fluid therapy over a period of days. Cannulas can be heparinized, plugged, taped down and re-used on successive days.

(ii) Intraperitoneal. This route is practical for non-emergency situations and for times when assistance is limited, e.g. after-hours and over weekends. It has the advantage that large volumes (approaching 150 ml per kg body mass) can be given RAPIDLY through a wide-bore needle. The patient is provided with a "24 hour depot" from which it can draw fluid.

When using this technique, the following precautions should be observed:

(a) The abominal site should be shaved, surgically prepared and infiltrated with local anaesthetic.
(b) The urinary bladder should be emptied and the spleen moved away from the site of puncture.
(c) The site of puncture (midventral or lateral) should be a safe distance away from the kidneys.
(d) Hypertonic solutions should not be administrered by this route.

Fluids for intraperitoneal use should be warmed to approximately 38°C to minimize the pain of administration and further hypothermia in shocked patients.

(iii) Subcutaneous. This route is painful, involving multiple skin punctures; unsightly because of large subcutaneous blebs; and impractical because it is virtually impossible to infuse the volumes required, e.g. a 20 kg dog which is 10% dehydrated requires 2 *l* of fluid.

(iv) Rectal. Administration of fluids is seldom practised by this route but might be used on selected cases.

(v) Oral. The oral route of fluid administration is practical in the convalescent patient. Large volumes of fluid can be given by this route using a pharyngotomy or stomach tube. Nutritional requirements for protein, fat, carbohydrates, vitamins and minerals can also be supplied by this route.

(c) RATE OF INFUSION

Infusion rate is important in intravenous administration of fluids. In general, veterinarians are unnecessarily conservative. It is essential to adjust the rate of fluid infusion according to the mass of the patient. Finco stated that fluids could be administered at the rate of 90 ml/kg body mass per hour*, so an 11 kg dog could receive 1 litre per hour. The rate of infusion should obviously be tempered by common-sense clinical judgment and the rate reduced in old patients and in those with myocardial aesthenia, hypoproteinaemia etc. Speedy administration of blood and fluids is especially important in the shock phase of BF, and a central venous pressure line provides a reliable means of monitoring the rate of infusion in critically ill patients. Once the current deficit has been corrected, volumes for maintenance and contemporary loss should be given more slowly.

3. FLUID COMPOSITION

(a) ELECTROLYTES

It is the author's experience that serum sodium and potassium levels are not seriously deranged in severely affected and shocked dogs with BF. Plasma bicarbonate is consistently lowered and chloride raised (see Table 1). The treatment of the metabolic acidosis of BF will be discussed in some detail below (3b).

Serum potassium, which could be expected to be raised because of the acidosis, red cell breakdown and poor renal function, is, in most cases, within the normal range or even slightly lowered. It seems logical and safe, therefore, to administer polyionic solutions which resemble body fluids in their electrolyte balance. One such fluid is Plasmalyte B**, which has the following electrolyte composition: Na^+ 130, K^+4, Mg^{++}1.5, Cl^-109 and HCO_3^- 28 m eq/*l*.

Lactated Ringer's solution and fluids containing acetate, proprionate etc. as alkalinizing agents are less desirable in shock cases of BF, since they have to be metabolized to exert their effect, and since the biochemical pathways responsible for such metabolism are known to be inactive in BF shock owing to tissue hypoxia. Fluids containing these agents could certainly be used for the treatment of less severe, non-shocked BF or recuperating patients.

(b) ALKALINIZING AGENTS

As is now fairly well known, dogs with advanced BF and shock usually suffer a metabolic acidosis with varying degrees of respiratory compensation. Anaemia, poor tissue perfusion and "stagnant hypoxia" result in anaerobic metabolism and the production of lactic and other metabolic acids. In most patients, the metabolic acidosis is surprizingly well compensated (see Table 1) but when the blood's buffers are exhausted and the respiratory response is maximal, pH falls and death must follow. Before this happens the clinician must administer base to replenish the body's store of buffer and raise the blood pH towards normal. It is neither necessary nor desirable to correct the metabolic acidosis of BF completely. A mild acidosis is advantageous to an anaemic animal since it shifts the oxygen/haemoglobin dissociation curve to the right, reducing the affinity between O_2 and Hb and thus allowing greater release of oxygen by haemoglobin at the tissue level.

Without the aid of an acid base analyser, a conservative approach of 1 to 4 m eq bicarbonate per kg body mass is recommended. 1 m eq per kg is given in mildly affected cases, 2 in moderate, 3 in severe and 4 in very severely affected semi-comatose, hypothermic patients. This schedule can be monitored and modified by noting changes in the rate, depth and force of respiration. It prevents lethal acidaemia while other measures (blood, fluid, etc) are improving circulation and tissue oxygenation and the lactic acid load is being metabolized.

Sodium bicarbonate containing fluids used include:

(i) Plasmalyte B (28 m eq per *l*).
(ii) 4,2% Sodium bicarbonate solution (0,5 m eq/ml).
(iii) 8,5% Sodium bicarbonate solution (1,0 m eq/ml).

The usual procedure in the Department of Medicine is to start treatment by setting up a blood transfusion and then slowly to inject the requisite amount of 8,5% $NaHCO_3$ solution intravenously through the rubber tubing at the end of the blood infusion line (over a period of about 10 minutes). When the blood transfusion is complete, the calculated volume of fluids is administered.

*The usual fluid administration set delivers 15 drops per ml fluid.

** Baxter

Table 1: SERUM ELECTROLYTES AND ACID BASE PARAMETERS IN NORMAL DOGS AND DOGS SURVIVING OR SUCCUMBING TO SEVERE BILIARY FEVER

	Normal ± SD	Survivors ± SD	Fatalities ± SD
Na meq/1	141,1–152,3[7]	139 ± 14,8(18)	147 ± 8,1(3)
K meq/1	4,4 – 5,7[7]	3,8 ± 0,7(18)	410 ± 1,5(3)
C1 meq/1	105,2 – 114,8[7]	119 ± 7,2(15)	—
Art. HCO_3 meq/1	20,0 ± 2,5(20)*	11,2 ± 4,3(19)	6,6 ± 1,1(4)
Arterial pH	7,4 ± 0,02(20)*	7,4 ± 0,1(19)	7,0 ± 0,2(4)
Art. PCO_3 mm Hg	3411 ± 3,4(20)*	22,1 ± 10,1(19)	15,0 ± 9,0(4)
Art. base excess	–3,9 ± 2,1(19)*	–14,1 ± 5,2(19)	–21,6 ± 2,8(4)
Art. lactate meq/1	1.5 ± 0,8*	4,4 ± 4,9(20)	16,1 ± 2,9(5)

[7] = Reference; *Author's figures, normal large breed dogs bled 4h after feeding and 20 min after being held quietly on a table. Number of dogs in brackets.

(c) CALORIES

The provision of energy by means of parenteral fluid therapy in the acute BF patient is relatively unimportant, and it is in any case impractical to attempt to meet the patient's full caloric requirements parenterally. The BF patient is catabolizing body protein and fat at a relatively rapid rate and short term persistence of this situation should not be too harmful. Glucose, fructose and invert sugar all have a caloric value of about 4 kilocalories per gram. A 10 kg dog requires about 700 kilocalories per day for maintenance, equivalent to 170 g of glucose or 3,5 *l* of a 5% dextrose solution. Were this given at the optimum rate of 0,9 g per kg body mass per hour, it would take about 19 hours to complete the infusion.

Ten per cent dextrose and more concentrated solutions tend to produce blood glucose levels exceeding the renal threshold (approximately 180 mg glucose per 100 ml blood) and lead to urinary wastage and diuresis. They also tend to cause phlebitis and should be given with care. Hypertonic solutions should never be given intraperitoneally unless one's purpose is to draw fluid from the circulatory system.

Parenteral use of ethanol (5,6 kilocalories per gram) and lipids (9 kilocalories per gram) has been practised elsewhere to meet energy requirements.

Parenterally administered amino acid solutions are largely wasted unless the patient's caloric needs are met first.

The practical route of energy administration in the recuperating dog, is, of course, oral. If the patient will eat unaided, so much the better. If not, force feeding or gavage can be used. A blender is required to reduce most foods to a suitable consistency for tube feeding.

4. SEQUELAE OF BILIARY FEVER REQUIRING FLUID THERAPY

(a) RENAL "SHUT DOWN" (ANURIC OR OLIGURIC RENAL FAILURE)

Blockage of renal tubules by desquamated renal tubular cells and/or haemoglobin casts is an infrequent complication of more severe cases of BF. Typically the patient suffering from this complication is an older dog presented 2 or 3 days after an acute attack with a history of persistent vomiting. The patient is usually dehydrated, anuric or oliguric and uraemic.

The procedure used in an attempt to save such patients is both intensive and expensive and is, in general, unrewarding.

The patient must be hydrated, and acid-base and electrolyte imbalances must be corrected. Overhydration must be avoided at all costs as excess fluids have no renal "escape route" and may lead to fatal pulmonary oedema or acute heart failure. Clinical judgment, monitoring of the total serum protein concentrations or the central venous pressure will aid in assessing the volume of fluid needed to rehydrate a patient. During the infusion, the patient should be catheterized and urine flow should be measured over a period of 15 minutes. Next, a test dose of the osmotic diuretic "Osmitrol" (mannitol 20%)* should be infused at 0,25 to 0,5 per kg over a period of about 5 minutes. If effective, an increased rate of urine flow should result in 15 to 30 minutes, in which case up to 2 g per kg of mannitol can be infused slowly, preferably under control of a central venous pressure monitoring line. Solutions of hypertonic dextrose (10 or 20%) may be used instead of mannitol when the latter is not on hand.

If the test dose fails to stimulate urine flow, a diuretic eg. 2 - 4 mg/kg of Lasix** (furosemide) should be injected intravenously. If the diuretic fails as well, peritoneal dialysis can be used to keep the patient alive while maintenance volumes of fluids and test doses of mannitol and furosemide are given daily. Peritoneal dialysis is time consuming and expensive, but unless haemodialysis becomes available it remains the patient's only chance of survival. Disposable dialysis infusion sets, peritoneal catheters and drainage bags are available. For renal "shut down" the less dehydrating "Dineal"*** with 1,5% dextrose warmed to 38°C should be used. Enough fluid to mildly distend the abdomen should be left in the peritoneal cavity for 1 hour and then drained. Lavages should be repeated up to five times daily. "Dineal" with 4,5 or 7% dextrose is used to draw fluid into the peritoneal cavity of overhydrated renal failure patients and is usually not required for BF renal "shut down".

(b) POLYURIC RENAL FAILURE

The "polyuric syndrome" may occur in patients surviving BF induced shock or renal "shut down". It is presumed to result from renal tubular epithelial (RTE) necrosis secondary to hypoxia (ischaemic or "anoxic" nephrosis).

Clinically, the patient remains depressed, extremely dehydrated and polyuric, and it may be polydypsic. The chief components of the urinary sediment are RTE cells and casts, reflecting the renal damage occurring after BF. The urinary specific gravity is low,

* Baxter
** Hoechst
*** Saphar Laboratories

usually in the "fixed range" of 1,008 to 1,012, reflecting the inability of the renal tubules to concentrate (or dilute) the glomerular filtrate. Blood urea nitrogen may be normal to moderately raised due to pre-renal (dehydration) and renal (tubular nephrosis) causes.

The "polyuric syndrome" must be treated by administering large volumes of polyionic solutions, eg. Plasmalyte B, parenterally if progressive dehydration, uraemia and death are to be avoided. The prognosis is fair, particularly in younger dogs. Re-epithelialization of renal tubules can occur provided the basement membrane remains intact. Immature RTE cells concentrate urine less effectively than mature cells, so that recovery from this syndrome takes time and is accompanied by increasing urinary specific gravity and decreased dehydration and fluid needs. Once the patient is drinking reasonable quantities of water, parenteral fluid and electrolyte therapy may be replaced by oral electrolyte tablets, eg. Enteren tablets*. The provision of a diet containing a low percentage of high quality protein, anabolic steroids, vitamins and unlimited access to fresh water is also advocated.

*NaCl 500 mg, $NaHCO_3$ 400 mg, aluminium hydroxide 100 mg, and casein 100 mg.

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BOOK REVIEWS — BOEKRESENSIES

THE PRACTICE OF EQUINE STUD MEDICINE

P.D. ROSSDALE and S.W. RICKETTS

Baillière Tindall, London 1974. pp viii + 421, Tabs 75, Figs 110 (4 colour plates). Price £10.50

The authors have written this book for clinicians and students concerned with the problems which affect horses at stud. As a handbook on the desk of the busy equine practitioner it will often be referred to and I believe it will fill a need long felt.

In recommending this book most strongly also to general practitioners and to graduate researchers in the field of genesiology, I feel confident that it would lead to a better understanding of the diversified and complex processes constituting the reproductive cycle.

Since the book is written in a style which is easy to read and understand, I believe that the stud owner and stud master will profit by acquiring it and by referring to it from time to time. This will give him a clearer insight into the problems of the veterinarian and his efforts to correct and improve reproductive efficiency in the stud.

Dr Peter Rossdale is well known for his work on the newborn foal. However, his many years of equine stud practice becomes evident throughout the book, since he writes with clear and concise confidence which comes from successful practice. In producing this book he and Dr Ricketts have made a major contribution to the professional library of the veterinarian.

The comprehensive and up to date bibliography at the end of each chapter will be of great value to the research worker in this field.

The book is divided into seven chapters viz: 1. Events leading to conception; 2. Gestation; 3. Birth; 4. Adaptive period; 5. Conditions and afflictions of the older foal; 6. General medicine; and 7. Clinical and further examinations.

The photographs and microphotos are of excellent quality and lucid line drawings and graphs, together with a fair number of tables, are used with discretion.

J.S. v. H.

REPRODUCTIVE DISORDERS IN PIGS

A.E. WRATHALL

Review Series No. 11 of the Commonwealth Bureau of Animal Health, 1975. Obtainable from the Commonwealth Agricultural Bureaux, Farnham Royal, Slough SL2 3BN, England. Price £4.00

Despite recent advances in the fields of reproductive physiology and pathology, disorders of reproduction still constitute a major source of loss to pig producers. In this book the author presents an up to date, critical review of the world literature on patterns, causes and mechanisms of reproductive failures in pigs, and also describes methods for their effective diagnosis and control. There are seven chapters; the first, a general one, covers pathological reactions during pregnancy and prenatal development, and the others deal successively with genetic, nutritional, environmental, toxic, infectious and management factors affecting reproduction. There are 225 pages of text and 1360 references.

Jl. S. Afr. vet. Ass. 47(4) 289-290 (1976)

CLINICAL NOTE **KLINIESE MEDEDELING**

THE DETERMINATION OF PLASMA BICARBONATE LEVELS AS AN AID IN THE TREATMENT OF CANINE BILIARY FEVER

R.N. PRYCE*

INTRODUCTION

The determination of plasma bicarbonate levels is a measure of metabolic normality with regard to the acid base balance of the body. If an abnormal pH exists in body fluids, cellular chemistry cannot proceed at its optimum rate and recovery from a disease process becomes further complicated and protracted, or therapy is rendered completely unsuccessful.

DETERMINATION

The method used for bicarbonate determination is a modification by Bittner of the original method of van Slyke in which plasma or serum is added to a known excess of hydrochloric acid. The amount of acid neutralised by the serum is proportional to the bicarbonate ion concentration. The amount of acid neutralised is calculated by means of an Oxford titrator and the level of bicarbonate in the serum is determined in mEq/*l*.

IMPORTANT POINTS FOR THE COLLECTION AND HANDLING OF SAMPLES FOR BICARBONATE DETERMINATION

(1) Blood should be collected anaerobically in tubes and filled to the top from clean arterial or venous puncture ("Venoject" tubes are ideal). If collected in a syringe the needle should be bent over to avoid contact with air and thus alter the pCO_2 — bicarbonate balance.
(2) The sample should be submerged in ice water until analysed.
(3) The sample should be centrifuged with stopper in place to prevent atmospheric contact as undue exposure will falsely depress values.
(4) Heparinised plasma or serum may be used but not oxalate, citrate or EDTA plasma.
(5) Jaundiced serum or plasma produces no error.
(6) A haemolysed sample will give inaccurate results.

DISCUSSION

There are varying degrees of metabolic acidosis with most cases of biliary fever.

Firstly, the poor tissue perfusion and hypoxia leads to cellular anoxia and the anaerobic metabolism of glucose to lactic acid, inducing a primary metabolic acidosis.

Secondly, the glomerular filtration rate is decreased with a low concentration of Na ions in the filtrate. With the decreased resorption of Na ions, fewer H ions are exchanged and thus more H ions remain in the body, inducing a renal metabolic acidosis. The other renal electrolyte balances affected, which are minor, are glutamine — glutamic acid and Na acid phosphate exchanges. The main reasons for this renal malfunction are haemoglobin overload and hypoxia.

Thirdly, anaemia and a decrease in red blood cells results in reduced removal of CO_2 by means of the chloride shift. This CO_2 combines with H_2O in the presence of carbonic anhydrase to form H_2CO_3 (carbonic acid).

CALCULATION OF DEFICIENCY OF BICARBONATE

Having determined the plasma bicarbonate level the following formula is used to calculate the bicarbonate deficit or amount of bicarbonate required to return the system to homeostasis:—

Total deficit in mEq/*l* = body wt (kg) × 0,6 × (25 — plasma bicarb mEq/*l*)[1]. In a 20 kg dog with a bicarbonate value of 10 mEq/*l* the amount of bicarbonate required to correct the deficit is:—

20 × 0,6 × (25 — 10) = mEq/*l*.

This is equivalent to 180 ml of the 8,5% bicarbonate solution or 360 ml of the 4,2% bicarbonate solution which is packed in 500 ml bottles, and over 6 *l* of Plasmalyte B. This consideration is that of bicarbonate alone in correcting the acid base balance over a period of 48 hours.

The preferred method of administration is to give half of the calculated amount fairly rapidly over 12 - 24 hours and then to reassess the position by means of a further determination. In an acidotic condition and also due to anaemia, hyperpnoea is usually present. This also helps as a compensating mechanism. If hyperpnoea is absent one can expect the amount of bicarbonate required to be slightly increased.

RULE OF THUMB[1]

One way of working out the quantity of bicarbonate required without determining bicarbonate levels is by degrees of clinical severity:

Less severe — 3mEq bicarbonate per /kg.
Severe — 6mEq bicarbonate per/kg.
Very severe — 9mEq bicarbonate per/kg.

A very severe case in a dog of 20 kg requires 180 mEq administered as above.

The other very important and easy way of administering bicarbonate is orally in drinking water; the amounts given are not so critical, but the animal has to be drinking.

* Box 65001, 2010 Benmore.

CONSIDERATIONS FOR THE READILY AVAILABLE SOLUTIONS

(1) *Bicarbonate solutions* — 8,5% sodium bicarbonate in water which contains 1 mEq/ml (Saphar).

4,2% sodium bicarbonate in water which contains 0,5 mEq/ml (Saphar).

These are ideal for replacement of large amounts of bicarbonate and can be mixed with other fluids except those containing calcium.

(2) *Plasmalyte B* or Ringer's bicarbonate — a solution similar to plasma with a pH of 7,4; it has bicarbonate at a level of 28 mEq/*l*. It is an ideal solution where fluid volume, as well as other electrolytes, is required and is fine for correcting a mild acidosis.

(3) *Ringer's lactate* — This was originally formulated before the sterilisation of bicarbonate was possible[2]. Lactate is converted into bicarbonate by the liver and thus for immediate effect Ringer's lactate is unsuitable, especially when the liver is not functioning at full capacity, as in biliary fever. Thus in the short term one may aggravate the situation with lactic acid, and in the long term with overdosage one may reverse the situation and cause an alkalosis, once the lactate has been converted to bicarbonate. An accumulation of excess lactate greater than 4 mEq/*l* may be fatal. Thus in the more severe case of biliary fever it would be advisable to avoid lactate if possible, especially when a rapid change in acid base balance is required.

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INFORMATION — INLIGTING

PLASTIC INSERT BOOSTS GAINS

A new idea for boosting gains in beef heifers or gilts has been announced by a California company, Agrophysics Incorporated. The method involves the insertion of an inert plastic object, 17,8 cm in length, into the vagina (next to the cervix) by means of a speculum. Used in conjunction with DES (diethylstilboestrol) and Synovex H hormone implants, the device, known as the Agrophysics Heifer Device, is said to result in faster and more efficient gains. Retention in beef heifers is 100%. According to the company, no apparent physical damage is caused to the animal, and no residue problems result from its use.

University and feedlot trials support the results of the manufacturer's trials. Although results have been consistent, they are not regarded as being spectacular, however. Heifers carrying the device and implanted with the 2 hormones apparently gain an average of 4% faster and 6% more efficiently in the feedlot than the controls. Heifers fitted with the device and implanted with both drugs gained 1,3 kg per day. The cost of production for a trial group of animals carrying the device and implanted with the hormones was R0,06/kg less than for those not fitted with the device.

The company discovered the growth promoting features of the device accidentally 4 years ago, during research with gilts at the University of Missouri. One in four gilts attained sexual maturity at about 160 days of age, and early breeders produced large litters. The researchers also found that the device boosts gains in gilts by as much as 12%. Work with swine has been deferred and concentrated, instead, on beef heifers.

The reason for the effectiveness of the device has not been established. It is believed that it may trigger some change in hormonal secretions, providing a natural growth stimulant. The device is expected to be available commercially before long and it is anticipated that it will sell for less than R1,75. Pre-clearance is not required by the U.S. Food and Drug Administration.

(*Farm Journal,* February, 1976, 230 W. Washington Square, Philadelphia, Pennsylvania).

BOOK REVIEW — BOEKRESENSIE

OUTLINES OF AVIAN ANATOMY

A.S. KING and J. McLELLAND

Baillière Tindall, London 1975. pp 154, Figs 67. Publ. Price R6,00.

This book represents indeed only the outlines of avian anatomy. The authors claim no more. They are well known for their research in this field and they have succeeded in producing a book that is the answer to the undergraduate's prayer. Despite its modest size and price it contains a wealth of information — all ready available knowledge that is required by the student and the practitioner. Comparisons are made within the different species and the main differences in mammals are pointed out. From the functional point of view the anatomy of each system is described succinctly and in clear and simple language. Reference is made to applied aspects. The line drawings are clear, uncomplicated and effective. The elaborate respiratory system is presented in a most refreshing and stimulating manner. On the venous side the location of the brachial vein used for venepuncture could perhaps be illustrated in future editions.

The reviewer recommends this book to students and veterinarians without reservation.

J.M.W. le R.

CASE REPORT **GEVALVERSLAG**

OSTEOSARCOMA OF THE AXIAL SKELETON IN A DOG

J. SCHRÖDER*

ABSTRACT: Schröder J. **Osteosarcoma of the axial skeleton in a dog.** *Journal of the South African Veterinary Association* (1976) **47** No. 4, 293-294 (En) Fac. Veterinary Science, Univ. Pretoria, Box 12580, 0110 Onderstepoort, Rep. of South Africa.
A case of primary osteosarcoma of the axial skeleton in a dog is described. The relative rarity of this site is stressed.

INTRODUCTION

A 2 year-old Alsatian bitch was presented manifesting signs of a 'stiff neck' and clinically evident atrophy of the shoulder muscles, which had been apparent for the previous 6 weeks. Radiological examination by Prof. C.J. Roos, Department of Surgery, Faculty of Veterinary Science, University of Pretoria, revealed ventral displacement of the trachea by a soft tissue mass which was attached to the ventral aspects of the last three cervical vertebrae (Fig. 1).

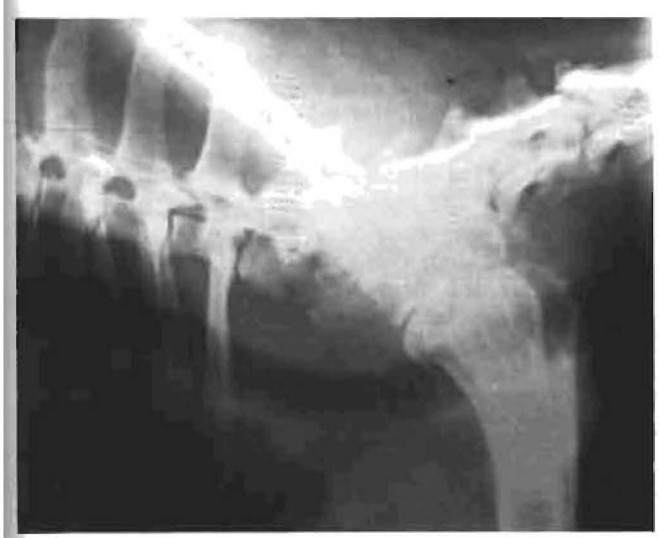

Fig. 1: Lateral radiograph of the thorax showing ventral displacement of the trachea by a soft tissue mass at the thoracic inlet.

The lesion varied from 10 to 15 mm in thickness and was apparently intimately related to the periosteum. The precise nature of this mass could not be determined but neoplasia was strongly suspected.

Due to the relative surgical inaccessibility of the site and the poor prognosis, euthanasia and an autopsy were performed.

PATHOLOGICAL FINDINGS

The muscles over both scapulae and humeri, and the caudal neck region were markedly atrophic. The tissue mass seen in the radiograph proved to be a neoplasm which occurred on the ventral aspect of the vertebral column at the thoracic inlet (Fig. 2). It was a soft, pale yellow-brown mass, 17 × 5 × 3 cm in size which was firmly attached to, and covered the ventral surfaces of the bodies of the fifth cervical to the first thoracic vertebrae.

Fig. 2: Tumour mass on the ventral aspect of the vertebral column at the thoracic inlet. The first four ribs on the left hand side have been removed, while the rest are being reflected for better exposure.

The spleen and enlarged iliac and mesenteric lymph nodes, when sectioned, all displayed numerous disseminated nodular foci resembling lymphoid tissue, all of which being about 1 mm in diameter.

On completion of the necropsy the affected part of the spinal column was removed, most of the muscles dissected away, and the specimen again radiographed. This radiograph clearly illustrated the typical 'sun-ray' periosteal reaction of an osteosarcoma.

Specimens for histopathological examination were taken from the neoplasm, lungs, liver, spleen, kidneys and mesenteric lymph nodes and fixed in 10% formalin prior to being processed, sectioned and stained with haematoxylin and eosin (HE).

HISTOPATHOLOGY

Sections of the tumour revealed a broad peripheral zone of relatively cellular reactive tissue resembling that described by Ling, Morgan and Pool[3], intermingled with fascicles of striated muscle fibres. The

* Formerly Dept Pathology, Fac. Veterinary Science, Univ. Pretoria.
Present address: MSD Research Centre, Private Bag 3, 1685 Halfway House.

greater part of the tumour mass itself showed marked necrosis — evidenced by the presence of neutrophils, macrophages. and cellular debris — and contained small. poorly circumscribed groups of anaplastic tumour cells with little, unmineralized matrix.

The neoplastic cells were spindle-shaped or stellate, with a light to moderately basophilic cytoplasm. Their nuclei were large (average diameter 7,5 mu), round to oval, and vesicular with small but mostly clearly visible, nucleoli. The interstitial matrix was lightly eosinophilic and fibrous, resembling collagen. There was no detectable sign of osteoid formation, or mineralization of the matrix (Fig. 3).

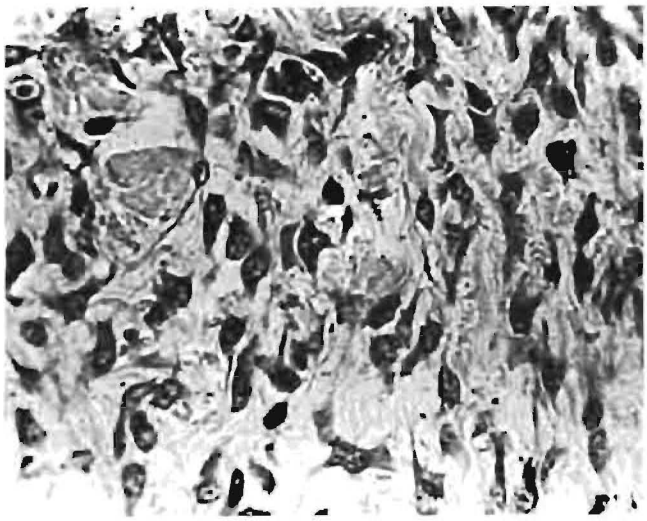

Fig. 3: Osteosarcoma. Cell detail. HE × 400.

In the section of the spleen that was examined, every Malpighian body was to a greater or lesser extent infiltrated by groups of cells arranged roughly spherically. The nuclei of these cells resembled those described above, and their cytoplasm was lightly basophilic. In this case the extracellular substance was homogeneous, lightly eosinophilic, and some areas contained lacunae with nuclear remnants (Fig. 4). This matrix showed signs of mineralization (moderate basophilia) in some foci.

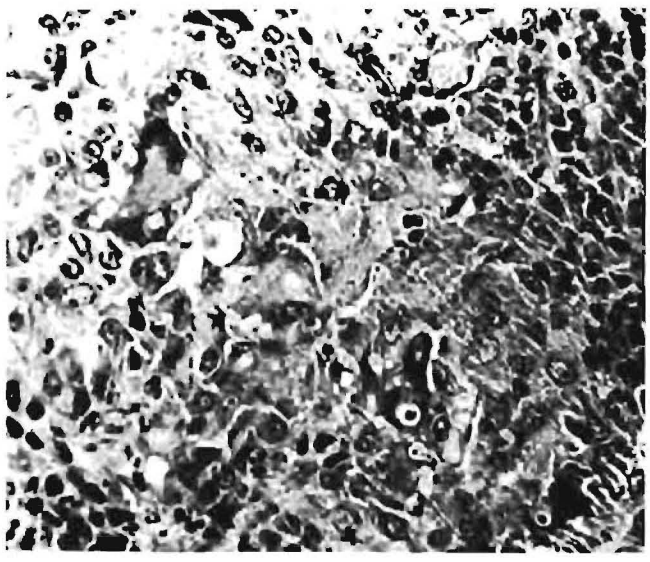

Fig. 4: Spleen, metastatic tumour. Lacunae containing nuclear remnants in interstitial matrix HE × 400.

The lymphoid tissue, particularly that of the cortex of the mesenteric lymph nodes was also infiltrated and largely replaced by poorly circumscribed, more or less confluent islands of cells with pyknotic to vesicular nuclei and the same type of matrix as in the spleen. This ground substance contained several lacunae with the remains of cellular nuclei. A light neutrophil infiltration was especially evident at the periphery of these metastatic tumours.

Numerous small disseminated foci of tissue similar to that in the spleen and lymph nodes, but less striking in appearance, were also found in the lungs.

DISCUSSION

Primary tumours of bone are relatively rare in all animal species[2]. Of these, osteosarcoma is most common in the dog[2 3 5], followed in incidence by chondrosarcoma and fibrosarcoma[5].

Less than 30% of all bone tumours[3] (less than 6% of osteosarcoma in one survey[2]) primarily occur in the axial skeleton. The appendicular skeleton is mostly affected by osteosarcoma. The usual predilection sites are the head of the humerus and distal extremities of the radius and ulna (33%), and the proximal and distal extremities of the femur and tibia[3 5].

Although Jacobson[2] uses the term 'osteogenic sarcoma', 'osteosarcoma' is preferred, as these tumours are mostly osteogenic and osteolytic at different sites and stages of growth.

Dogs most often affected are the large breeds (dogs over 20 kg in mass become increasingly prone[1]) over the age of 2 years (half the cases in dogs between 5 and 9 years of age[5]).

Another unusual feature of this case was that secondary lesions in the spleen and iliac and mesenteric lymph nodes were considerably larger and therefore probably older than those in the lungs, which would indicate that the first emboli passed through the pulmonary vasculature without colonisation there.

It seems feasible, as no evidence of pressure on the spinal cord was found, that the muscular atrophy seen was due to partial denervation as a result of pressure on the nerves constituting the brachial plexus (last 3 cervical and first 2 thoracic) at their emergence from the spinal column.

ACKNOWLEDGEMENTS

For his encouragement and aid in the preparation of the text, the author is grateful to Prof. R.C. Tustin of the Department of Pathology. I am indebted to Miss C. Gouws for making the histological sections and to Mrs B. Blake for typing the manuscript.

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NUWE GRADUANDI — NEW GRADUATES

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Oorkant verskyn 'n klasfoto van die finale-jaar studente van 1976:

Tydens 'n gradeplegtigheid op 20 November 1976 is die graad B.V.Sc aan 36 nuwe kollegas toegeken;

Al die ander kandidate kan verwag om vroeg in die eerskomende jaar ook aan die vereistes te voldoen.

Die volgende meriete-toekennings is gemaak deur die genoemde instansies tydens 'n afskeidsfunksie wat deur die dosente van die Fakulteit aangebied is:

A photograph of the final year class of 1976 appears opposite.

The degree B.V.Sc was awarded to 36 new colleagues at a graduation ceremony on the 20th November;

The other candidates will be meeting the necessary requirements fot the degree early in the new year.

The following merit awards were made by the donors mentioned below at a "farewell" given by the teaching staff of the Faculty:

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CASE REPORT **GEVALVERSLAG**

CANINE TETANUS

LEA STOGDALE*

ABSTRACT: Stogdale, L. **Canine tetanus.** *Journal of the South African Veterinary Association* (1976) **47** No. 4, 299-302 (En) Dept. Med., Fac. Vet. Science, Univ. Pretoria, Box 12580, 0110 Onderstepoort, Rep. of South Africa.

A report of tetanus in a dog is presented. The symptoms were typical and the dog was successfully treated. The differential diagnoses and the therapy of tetanus are discussed with emphasis on the dose of antitoxin required.

INTRODUCTION

Tetanus is an acute, non-febrile, infectious disease characterized by hyperaesthesia, tonic muscular spasms and convulsions. It is caused by the dissemination of toxin produced by *Clostridium tetani* multiplying in a localized infection. All species of domestic animals are affected but their susceptibility differs[25].

The susceptibility of a species to the pathogenic effects of *Clostridium tetani* infection is dependent upon the effects of the toxin and not on the growth characteristics of the bacterium[28]. The susceptibility to the effects of tetanus toxin is calculated by the amount of toxin per gram of body mass required to prove fatal when injected. With the mouse being taken as the standard, the horse is 12 times more susceptible and the dog is 50 times more resistant[28]. Thus it is not surprising that cases of clinical tetanus are relatively rare in the dog when compared with the horse[24].

The symptoms of mild tetanus in the dog are well described by Ripps[23]. The clinical signs observed were a drawing back of the lips, rigid, erect ears, a general body stiffness with an awkward gait, "lockjaw", eating, but with some difficulty, and protruding nictitating membranes. Tetanus in the dog is rarely seen by veterinary practitioners and when it is diagnosed treatment may not be attempted as conventional therapy is very expensive. This case report records the clinical symptoms of mild tetanus in a dog, the differential diagnosis and treatment. Discussion is centered on a rational and practical approach to therapy.

CASE HISTORY

A 3 year old, cross-bred male dog, weighing 30 kg, was presented at the Faculty of Veterinary Science at Onderstepoort for veterinary examination.

Thirteen days previously, (day 1) a lay person had castrated the dog using tranquilization, local anaesthetic, an aseptic technique and sterilized equipment; the wound was sutured post-operatively. The dog showed no adverse after-effects. On day 9, the owner noticed that the dog was depressed and that the castration wound was septic. Non-veterinary assistance was sought, a diagnosis of nephritis was made and 5 ml of a penicillin-streptomycin combination was injected intramuscularly. Sulphonamide powder was sprayed onto the septic wound which was open and discharging.

This therapy was repeated on days 10 and 11. On day 11 the dog began to show stiffness of his limbs and he ceased eating. On day 12 the owner noticed that the stiffness had increased and that the dog had an altered facial expression. The ears were "pricked" and the third eyelids were partially covering each eye. By day 13 the owner was sufficiently concerned to seek professional veterinary advice.

CLINICAL EXAMINATION

The dog appeared bright and very alert. The temperature was raised to 40,3°C and the mucous membranes were very congested. The popliteal lymph nodes were enlarged; the other peripheral lymph nodes were of normal size. The castration wound was swollen with a fibrous reaction and oedema. There was a serous exudate from the incision which had not healed. Blood smears examined for *Babesia* were negative.

The outstanding clinical sign was the comical facial expression. The ears were continually drawn together towards the midline; the interscutular and frontal muscles were forcibly contracted causing longitudinal "wrinkles" on the forehead. The lips were pulled back in a "grin" and saliva ran from the mouth. The dog's face gave an impression of attention and laughter. The skeletal musculature was in generalized tonic contraction and the jaw tone was increased but the mouth could be opened with effort. The extensor reflex was increased but flexion could be obtained. The knee jerk reflex was greatly exaggerated; other neurological tests were normal. The dog could still walk but with a stilted gait and he had difficulty in negotiating steps. The tail was stiff and extended straight out behind the animal. Tactile stimulation caused mild clonic muscle spasm of the legs and prolapse of the nictitating membranes.

DIAGNOSIS

Based on the history and symptoms a clinical diagnosis of tetanus was made. The important clinical features were the scrotal wound and swelling, the slow onset of signs (stiffness being noticed for 3 days), the tonic muscle tone and the characteristic facial expression.

Presented at the Witwatersrand Branch, SAVA, Johannesburg, 20th July 1976.

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

Differential diagnoses considered were:

(1) rabies	(6) cerebral babesiosis
(2) distemper	(7) diamidine poisoning
(3) cerebral toxoplasmosis	(8) strychnine poisoning
(4) spinal meningitis	(9) trauma to the head
(5) lead poisoning	(10) puerperal tetany

General diagnostic tests performed were haematology, serum enzymes and electrophoresis. The significant findings were a mild leucocytosis of 19,200 cells/cu mm with a neutrophilia of 84%. Serum enzyme values for liver and muscle damage were normal, as was the blood urea nitrogen level. Electrophoresis showed a slight increase in alpha 2 globulin indicating tissue damage and an increase in gamma globulin showing that there was an immune response in progress[14].

(1) *Rabies:* an acute fatal, infectious disease which causes encephalomyelitis. It is characterized by emotional changes and neurological disturbances with reflex excitability, paralysis and death[6]. The transmissability of rabies to humans, its fatal outcome and unpleasant preventative requirements, make early diagnosis essential.

The alert expression, the salivation and the protrusion of the nictitating membranes with external stimulation were suggestive of rabies. The owner claimed, however, that there was no change in the dog's character: he was his same friendly self but was less able to express himself. The patient's muscular tone was increased as opposed to the flaccid paresis to paralysis of dumb rabies and he was showing no vicious tendencies. Rabies was, therefore considered unlikely: the animal was hospitalized and no signs suggestive of this fatal disease developed.

(2) *Distemper:* the nervous complications may follow an inapparent systemic infection especially in an adult dog with some degree of immunity[6]. The neural manifestations include myoclonus (contractions of a portion of a muscle, an entire muscle or a group of muscles[9]), inco-ordination, circling, epileptiform or clonic convulsions and coma[6].

The tonic muscular contractions were unlike the usual symptoms of neurologic distemper and so this viral disease was discarded as a possible diagnosis.

(3) *Cerebral toxoplasmosis:* chronic infection by *Toxoplasma gondii* in a resistant host may affect the brain as well as all other organs. If the animal's resistance is reduced, the organisms multiply and cause necrosis[12]. Clinically there is fever, depression, weight loss, respiratory and intestinal signs and nervous system disturbances with tremor, inco-ordination and paralysis[18].

The patient's symptoms were confined to the neuro-muscular system. Serum was collected for the Sabin - Feldman test which was negative for toxoplasma antibodies.

(4) *Spinal meningitis:* usual signs are fever, muscular rigidity, hypersensitivity, hyperexcitability and abnormal spinal fluids[18].

This was considered unlikely as there was no peripheral hyperaesthesia. Cerebrospinal fluid was collected under general anaesthesia and cultured for bacteria and fungi. There was no micro-organism growth on the culture media.

(5) *Lead poisoning:* neurologic symptoms in dogs are not characteristic. There is anorexia, emaciation, mental irritability, muscular tremors, ataxia and intermittent convulsions[12].

Although the patient's symptoms were unlike those of lead toxicity, faeces were collected for lead estimation. The result was negative.

(6) *Cerebral babesiosis:* a deviant form of canine babesiosis caused by *Babesia canis* in which a wide variety of nervous symptoms are observed. Most commonly there is depression, delirium, epileptic-type fits, tonic convulsions with collapse and death. There is usually an accompanying parasitaemia[2,3].

The peripheral blood smears were negative at the initial examination, as were the smears made on subsequent days.

(7) *Diamidine poisoning:* a therapeutic chemical used in the treatment of babesiosis. Occasionally with overdosage cerebral haemorrhage occurs resulting in tonic and clonic convulsions of increasing severity, progressing to coma and death[19].

This animal had never been treated with any diamidine-containing drugs.

(8) *Strychnine poisoning:* rapid onset, usually within 2 hours of ingestion, with intermittent convulsions (clonic) and marked sensitivity to external stimuli. The convulsions become increasingly frequent, severe and prolonged until death from respiratory muscle paralysis occurs[7].

The continual character of this dog's muscular contraction and the slow onset of the symptoms differentiated this case from strychnine toxicity.

(9) *Trauma to the head:* pressure on the nervous tissue by displaced bone or haemorrhage may cause convulsions which initially are tonic but progress to clonic before unconsciousness intervenes. Epileptic convulsions are also seen with these lesions[6].

The patient's clonic muscle contractions made such a lesion seem unlikely. There were no external signs of trauma and cranial radiographs were negative for osseous displacement.

(10) *Puerperal tetany:* occurs in the post-whelping period and is characterised by tonic and clonic convulsive reactions which increase in severity[6].

The sex of this dog dictated that this possibility was only considered briefly.

TREATMENT AND COURSE

As the symptoms of tetanus were mild, and energetic therapy could be undertaken immediately, the prognosis in this case was considered to be good.

The dog was given a general anaesthetic and the

scrotal wound was thoroughly debrided. All the fibrous and necrotic tissue was removed and 1 million units of crystalline penicillin ("Crystapen" Glaxo Allenbury) was injected into the area.

It was decided to use the conventional dose of tetanus antitoxin of 100 000 units[10 6]. Initially the dog was tested for hypersensitivity to antitetanus horse serum by injecting 0,2 ml subcutaneously and observing him for half an hour for any adverse reaction. Since no reaction was observed, 10 vials of 10 000 units per vial of tetanus antitoxin ("Tetanus antitoxin", Institute for Medical Research) were injected intravenously, slowly. Only one dose was given since adequate plasma concentrations of antitoxin are present for at least 14 days[17] after administration crystalline penicillin ("Crystapen" Glaxo Allenbury), 1 million units per dose was injected intravenously four times a day on the day of admission and on the following day. Thereafter, 20 000 units per kilogram of procaine penicillin ("Aquacillin" A S Ruffel) was injected intramuscularly each day for 5 days. The patient's condition was carefully assessed each day with a view to supportive therapy.

The mild muscular tetany remained static for 2 days and then gradually decreased until by day 20 (8 days after admission and treatment) all the clinical signs of muscular contraction had disappeared. The temperature decreased to normal on the second day of therapy. The dog was able to take in fluid throughout the illness although lapping was difficult initially. After 4 days of treatment the patient commenced eating. By day 20, or 8 days after treatment was initiated, the dog was normal in all respects: he was very lively with no signs of stiffness. The facial expression was natural and the ears were mobile and relaxed. The dog was eating and drinking normally.

DISCUSSION

Tetanus in the dog was reviewed by Loeffler[15] in 1962 and by Mason[16] in 1964. Since these two extensive reviews very few cases of tetanus have been reported in the canine. Table 1 summarizes the case reports of tetanus in the dog published since Mason's review.

Table 1: CASE REPORTS OF TETANUS PUBLISHED SINCE 1964

Reference	Number of cases	Symptoms	Tetanus antitoxin treatment	Response
Mason[16]	55	Typical*	100 000 - 220 000 units once or daily for up to 5 days	29 of 55 dogs recovered, (53%)
Zontine & Uno[30]	1	Typical*	52 500 units, once; route unspecified	Recovery
Ong[20]	1	Typical*	5 000 units twice daily for 4 days, intravenously. Then 2 500 units twice daily for 4 days, intravenously. Total, 60 000 units.	Recovery
Kaiser[13]	1	Typical	60 000 units, intravenously. 30 000 units, subcutaneously	Recovery

*Typical symptoms include stilted gait, wrinkled forehead and a "grin"-type facial expression, skeletal muscle rigidity, trismus and protrusion of the nictitating membrane.

Two facts emerge from this summary. Firstly, since 1964 only successfully treated cases have been reported, as opposed to the situation prior to this time. The recovery rate of 58% in the 55 cases reviewed by Mason compares favourably with the overall average recovery rate of 45 to 55% in human cases[27]. However, with modern intensive care, the mortality in humans can be reduced to 10% or less[28]. Secondly, there appears to be a great variation in the dose, route of administration and regime of tetanus antitoxin therapy. Considering the cost of this treatment it would seem logical to examine this aspect very carefully and critically.

Weinstein[27] in his review of current concepts of tetanus in humans stresses the following principles of treatment:

(1) neutralization of any circulating toxin before it reaches the nervous system by the early administration of specific antitoxin;
(2) removal of the source of toxin by surgical excision of the area infected by the tetanus bacilli, if this is possible;
(3) penicillin parenterally and locally to kill the vegetative form of *Clostridium tetani* and so prevent any further toxin production;
(4) place the patient in a very quiet environment and avoid disturbances;
(5) hypnotics, sedatives, anaesthetics and neuromuscular blocking agents as required to control convulsions;
(6) maintenance of fluid, acid-base and electrolyte balance;
(7) ensure air passage patency and
(8) intensive and constant nursing care.

In cases of mild tetanus, such as the subject of this report, the treatment may be restricted to neutralization of the toxin and removal of its source, penicillin injections, sedation and provision of a quiet, dark cage. In these cases the cost of the tetanus antitoxin may become the decisive factor as to whether or not to proceed with therapy.

The dose of antitoxin recommended in the veterinary literature varies from 10 000 to 100 000 units[10 6]. In discussing the amount of antitoxin to be administered it must be remembered that therapeutic antitoxin can only neutralize toxin before it becomes fixed to nervous tissue[28 29]. Mason, Robertson and Austin[17] showed that heterologous antitoxin persisted in the blood stream for 42 days and that adequate levels for therapeutic efficacy were present for at least

14 days. Brown, Mohamed, Montgomery, Armitage and Laurence[4] established the therapeutic value of tetanus antitoxin in man but determining the optimal dose is extremely difficult. Wilson and Miles[28] discuss the efficacy of various doses of antitoxin. They conclude that a dose of 10 000 to 20 000 units should be adequate for a human and Adams[1] advises a single dose of 10 000 units given intravenously. Weinstein[27] recommends 100 000 units of horse-serum if homologous hyperimmune globulin is not available for the treatment of tetanus in humans. Mason[16] converts a human dose of 100 000 units for a 60-80 kg man to a per kilogram dose for a dog of 1 000 units. Thus a medium sized dog weighing 20 kg would need to receive only 20,000 units of antitoxin. The efficacy of a moderate dose of tetanus antitoxin is substantiated by case reports in dogs[16], cattle[11 21 22 26] and horses[5 8]. A dose of 10 000 to 20 000 units of tetanus antitoxin makes the treatment of tetanus economically feasible without reducing the chance of recovery.

The intravenous route is usually used for the administration of tetanus antitoxin. The intrathecal or intraspinal route has been suggested[6] but this has not proved superior clinically and horse serum is toxic when injected into cerebrospinal fluid. In human clinical practice the intrathecal route has now been abandoned[28]. Intravenous and local injection around the infected area is recommended both in human and veterinary medicine[10 16 27]. The tetanus antitoxin should be injected as soon as the condition is diagnosed and attention must be directed to supportive therapy and nursing. It is the latter that largely determines the outcome of clinical cases of tetanus.

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OUT OF THE PAST — UIT DIE VERLEDE

AN OUTLINE HISTORY OF VETERINARY SCIENCE

A.J. MORREN

Veterinary medicine as a science may be said to have begun in France in the 18th Century when, in 1762, Louis XV established the first veterinary school in Lyons. The instinctive treatment of animals by prehistoric and primitive peoples, and the gradual development of such treatment into some form of art such as was practised by the traditional horse-doctor, the cow-leech and the farrier, is as old as man. and veterinary practice of some sort is as old as civilisation itself. From its early beginnings in ancient India, the veterinary art rose to great heights of achievement during the Byzantium period, only to pass into almost complete disuse during the medieval age. From that period, when it was confounded by religious superstition and abysmal ignorance, it had its renaissance in Italy and was set on a course of development which has resulted in the status enjoyed by veterinary science at the present day.

Little real information is available concerning the practice of animal care in prehistoric times, but much can be inferred from Stone Age cave paintings dating back some 20 000 years. They indicate clearly the early association of man with his dog, and show some appreciation of the position, size and shape of the vital organs of the animals hunted. The spear, for example, is directed to the heart of the mammoth; a trepanning operation, presumably to release the demon of disease, is depicted there, and the possession of flocks as an index to wealth no doubt provided an incentive to Stone Age man to care for and preserve his worldly goods. Bacteria, among the first species of living things, have left traces of disease in the fossil remains of that remote period and hint at the struggle of primitive man against a hostile environment.

The earliest identification of veterinary art in antiquity is found in the papyrus of Kahun (c. 1900 B.C.), among the edicts of Hammurabi, and the sacred writings of the Vedic period (1800 - 1200 B.C.) in India. The Hindu attributed the origin of medicine to Brahma and held as a sacred religious duty the care equally of man and beast, since both were considered to have the same destiny. Traces of animal hospitals are still extant, and prescriptions for the treatment of animals issued by King Asoka included blistering, firing, cautery, blood-letting and the administration of the simple herbal drugs by means of a stomach tube through the nose, — all considered, a fairly broad spectrum of animal therapy.

The fragments of papyri relating to medical practice in Egypt would indicate that the Egyptians were better engineers than physicians. Post mortem examination was not practised, and embalming was motivated by reverence for the dead, not by any desire to extend a knowledge of anatomy and pathology. Mummies of men and animals reveal the presence of rickets, tuberculosis, arthritis and arteriosclerosis, and the setting of fractures. There is no doubt that its geographic position and its climatic conditions made Egypt specially vulnerable to the importation of diseases which ravaged the country, apparently unchecked, from time to time.

Persian manuscripts contain one of the earliest references to any specific training of physicians, but not to veterinarians as such. Medicine, being essentially a magico-religious practice in the hands of priest physicians, was closely regulated and the fees for the treatment of man and animals were prescribed by law.

Hippocrates of Cos, the greatest of the philosopher-physicians in the classic period of medicine in Greece (470 - 146 B.C.), is regarded as the founder of modern medicine. In contrast to previous systems of mystical medicine from which Greece was emerging at the time of the birth of Hippocrates, the Hippocratic system placed emphasis upon a high standard of ethics, accurate observation, clarity and honesty in recording case histories, and rational treatment. Actually there is little reference to veterinary practice in the writings of Hippocrates, but it is clear that he must have been familiar with the anatomy and, to a lesser extent, the pathology of domestic animals.

Medicine in ancient Rome was marked by mysticism and superstition, and professional and veterinary practice was regarded as socially infra dig. The dearth of physicians led to an influx of Greek practitioners, mostly quacks, to whom were relegated the diagnosis and treatment of disease. A few Roman names, however, emerge. Cato (234 B.C.) essentially conservative and opposed to Greek innovations, in his *de Agricultura* presented cabbage as a panacea for all ailments, human and animal: Varro, a true farmer and advocate of the simple rustic life, had enlightened, humanitarian views on animal husbandry, and, anticipating the germ theory by 1800 years, recognised contagion as a vital factor in disease. If we consider the decline of medicine in Rome we cannot but marvel at how far ahead of his time was one of Varro's stature. Virgil's Georgics (30 B.C.) is a manual of agriculture and animal husbandry in verse, but, like the writings of Columella 1st century A.D., added little to the veterinary art since Varro's time. The medical doctrine of Galen (131 - 200 A.D.), a Greek practitioner in Rome, supported humoural pathology, and his knowledge of anatomy, derived from animals (since human dissection was outlawed), influenced medical and veterinary practice for many generations.

The decline of Rome may be attributed, among other causes, partly to the decline of farming. When the centre of government shifted to Byzantium, medicine was still in the hands of Greeks and under their influence reached new heights of achievement. The Hippiatrika, a compilation of the original com-

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munications of 156 veterinary practitioners, including Absyrtus, became a veterinary classic, and dealt with surgical techniques, the digestive trouble of horses, bleeding, phlebitis, anthrax, acupuncture, castration and rabies, etc. Vegetius, in the 4th century A.D., was clearly influenced by Absyrtus, but his designation of *father of the veterinary art* may have originated from his enlightened outlook upon the practice of veterinary medicine and from his plea for the art to be restored to the once high esteem it enjoyed in Ancient Greece. After Vegetius, the veterinarian and his title disappeared from the scene and the farrier became the sole guardian of animal health.

The first half of the millenium between the fall of Rome and the end of the Byzantium period may be termed the Dark Ages. Men became slaves to convention, a conformity dictated largely by the prevailing religious philosophy and by the church as the instrument of that philosophy.

Physicians were denied the right to heal since disease was regarded as a punishment for mortal sin, prayer and fasting providing the sole therapy. There is no evidence of any measures taken to control the devastating plagues which swept over Europe during the period and left it prone before the Mahommedan invasions of the 11th and 12th centuries. The Arabs absorbed the scientific heritage of the peoples they conquered, and, though there was little originality in their research, they contributed the first literature on the physiology of horses, smallpox and pediatrics.

New concepts of freedom of thought and of human dignity, and a philosophy for the living gradually replaced the depressing aura of the Dark Ages. Veterinary science, however, lagged behind the great culture movements of the Renaissance until the end of the 16th century when Ruini made a notable contribution to veterinary art with his famous *Anatome a del Cavallo* (1598). At the same time, William Harvey's masterpiece on the circulation of the blood, described as the greatest single work in medicine, placed research beyond the realm of speculation and showed how an hypothesis can be proved by the experimental method. Other significant contributions to the veterinary art about this time were: John Fitzherbert's *Boke of Husbandrye,* which definitely established the horse-leech as a professional entity: George Tuberville's *The Noble Arts of Venerie or Hunting,* which attempts to differentiate between the different types of rabies and is perhaps the first treatise on canine pathology: Thomas Blundeville, who produced the first printed veterinary work in English (1566), and it is unfortunate perhaps that nowhere does he employ the terms veterinary or veterinarian and failed in what might have been, and probably unconsciously was, a primary object in placing the veterinary art, in contrast to farriery as such, on a higher plane in England. Other names were to follow: Thomas de Grey, in the vanguard of those who saw the necessity of an entirely new type of veterinary practitioner: the Frenchman, Jacques de Sollysel, celebrated for his work on glanders: Hermannus Boerhaave of Holland: Albrecht von Haller, the prodigious Swiss physiologist, the founder of modern physiology: Edward Jenner, one of the greatest benefactors of mankind through his work on vaccination for smallpox, some aspects of whose work had veterinary implications.

There is a touch of irony in the fact that, although at the beginning of the 18th Century there was not a single veterinarian with sufficient knowledge or experience to meet the challenge of an outbreak of pestilence, this was the century which was to become most eventful in the history of veterinary medicine. The succession of devastating plagues which swept Europe in the early part of the century underlined the necessity for the establishment of some organised effort of control and this led to the establishment of the first veterinary colleges.

The founding of the Royal Veterinary School at Lyons in 1762, already referred to, was a turning point in the history of veterinary medicine. With veterinary skill now in the hands of educated men, the profession acquired a new status, its development closely paralleling that of the physician. The method and techniques of one were readily adapted to the other, and, with the recognition of the close inter-relation of many human and animal diseases, human and animal medicine came to be regarded as complementary.

In 1791 the London Veterinary College was founded by Charles Sainbel, a former student of the Lyons Veterinary School, the Edinburgh Veterinary College in 1823 under the direction of William Dick, and in 1844 the Royal College of Veterinary Surgeons was created by Royal Charter by Queen Victoria. From this period veterinary medicine as a science has had an uninterrupted history, schools and university colleges of veterinary medicine being progressively established in most European and Commonwealth countries, in America, and in South Africa.

Veterinarians qualifying at these institutions have found their services sought after to an increasing extent. Notable employers of their skills in the 19th and early 20th centuries, when the horse was indispensable to warfare, were the armed forces of various countries. The early history of veterinary activity in South Africa is therefore closely associated with the names of veterinary officers originating from the British Army.

The opportunity for the greatest contribution to the development of veterinary medicine as a science in South Africa was heralded in 1896 when an outbreak of rinderpest from the north devastated the country. The Transvaal Republic was fortunate in this emergency in having available the services of Arnold Theiler, a dynamic Swiss veterinarian, who was summoned by President Kruger to assess the position and institute measures for the control of the scourge. Largely through his endeavours, the disease was brought under control and finally eradicated. The catastrophic losses involved convinced the authorities of the need for veterinary research, and Theiler was commissioned to establish a research laboratory. He selected a site for it on the farm, *De Onderstepoort,* 11 km to the north of Pretoria, and the premises were occupied in 1908. Under the direction of Sir Arnold Theiler and his successors, Onderstepoort researchers have made great progress with the solution of animal diseases. Their numerous achievements in the field of veterinary research have made Onderstepoort world-renowned and a respected centre of reference. Its constant ally through the years has been the Department of Veterinary Services which is responsible for the implementation of measures aimed at the control of scheduled diseases.

Theiler saw the necessity of training veterinarians in South Africa and, through his initiative, a Faculty of Veterinary Science was established in 1920. Large numbers of veterinarians have thereby become available to this country and the standard of service and expertise has reached a very satisfactory level.

Veterinary medicine, after its varied and interesting history, has made great strides towards resolving the question of animal pathology. Its achievements have been largely cumulative and have progressively provided the veterinarian of today with the background and perception necessary for dealing with the problems of tomorrow.

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TOEKENNING

S A V V-GOUE MEDALJE : VIERDE TOEKENNING

In 1975 het die Raad voorstelle ontvang vir hierdie hoogste toekenning. Ooreenkomstig die Raad se aanbeveling is die medalje namens die SAVV deur die President, Dr B.H. Pappin, tydens die opening van die Tweejaarlikse Nasionale Veterinêre Kongres te Durban in Sept. 1975 te Durban toegeken aan

EMERITUS PROF. DR DOUW GERBRAND STEYN

ter erkenning van sy "UITMUNTENDE DIENS AAN DIE PROFESSIE".

AWARD

S A V A GOLD MEDAL : FOURTH AWARD

In 1975 Council received proposals for this premium award. On Council's recommendation the president, Dr B.H. Pappin presented the SAVA's Gold Medal on its behalf to

EMERITUS PROFESSOR DR DOUW GERBRAND STEYN

at the opening of the Biennial National Veterinary Congress held in Durban in Sept. 1975, "FOR DISTINGUISHED SERVICE TO THE VETERINARY PROFESSION".

Prof. Dr D.G. Steyn

Gebore op 7 Maart 1898 te Colesberg, het hy te Steynsburg gematrikuleer en in 1919 die graad B.Sc aan die Universiteit Stellenbosch behaal. Hy lê daarna die Staatseksamen in Veeartsenykunde aan die Universiteit van Weenen, Oostenryk in 1924 af, en word in 1925 die graad Dr Med Vet (Pharm) deur dieselfde Universiteit toegeken. In 1925 word hy navorser en hoof van die seksie Farmakologie en Toksikologie van die Navorsingsinstituut vir Veeartsenykunde te Onderstepoort. In 1934 het die Universiteit van Pretoria aan hom die graad D V Sc

Born in Colesberg in 1898, he matriculated in Steynsburg and obtained the B.Sc. degree from Stellenbosch University in 1919. In 1924 he successfully took the State Examination in Veterinary Science at the University of Vienna, where he obtained the degree M.Med.Vet (Pharm) a year later. In 1925 he became Research Officer and Head, Section Pharmacology and Toxicology of the Veterinary Research Institute at Onderstepoort. In 1934 the University of Pretoria awarded to him a D V Sc for his work on the Toxicity of Plants in South Africa. From 1934 to 1946

toegeken vir sy werk oor die Giftigheid van Plante in Suid-Afrika. Vanaf 1934 tot 1946 was hy Professor in Farmakologie en Toksikologie aan die Fakulteit Veeartsenykunde van die Universiteit van Pretoria. In 1946 word hy aangestel as Professor in Farmakologie aan die Fakulteit Geneeskunde van dieselfde Universiteit. In 1963 bereik hy die gebruiklike pensioneerbare ouderdom maar neem 'n betrekking in die Lewenswetenskapafdeling van die Atoomkragraad te Pretoria. In 1965 word hy aangestel as wetenskaplike adviseur van die Minister van Gesondheid, en in 1968 word hy farmakologiese adviseur vir 'n farmaseutiese onderneming. In 1975 word hy aangestel op die personeel van die Nasionale Botaniese Navorsingsinstituut om te help met die opstel van 'n Gifplantbeheersentrum.

Sy prestasie as navorser kan gemeet word teen die 121 wetenskaplike artikels wat uit sy pen in plaaslike en buitelandse tydskrifte verskyn het. Hy is ook die outeur van vier bekende boeke. Sy 54 artikels in die Onderstepoort Joernaal handel oor onderwerpe soos gifblaar en tulpvergiftiging, vermeersiekte, geilsiekte, kaalsiekte by bokkies en lammers, dunsiekte by perde, nier- en blaasstene, die giftigheid van seringbessies, krimpsiekte, crotalariose, *Lathyrus*vergiftiging by mens en dier, vygies en bietou, jaagsiekte by perde, Kimberley-perdesiekte, gousiekte, seneciose by mens en dier, kropgeswel by mens en dier, fluor, swamme, blousuurvergiftiging, fenol ens. ens. Baie van die toestande was reeds bekend, en die veeverliese was sorgwekkend. Sy ondersoeke was grondig en met 'n globale benadering tot plantvergiftiging en inagneming van alle faktore wat die voorkoms en mate van giftigheid kan beïnvloed kon hy dikwels die regte diagnose, voorkoming en behandeling stel. Bepaling van die giftige bestanddele van die gifplante was vir hom 'n verdere uitdaging. Sy uitgang was egter dat dit nie behandeling maar wel voorkoming was wat nodig is om die probleem van gifplante te oorkom. Die toepassing van sy aanbevelings t.o.v. uitroei van gifplante en die bewaring en opbou van veldweiding het ten gevolge gehad 'n geweldige afname in veeverlies a.g.v. plantvergiftiging.

As farmakoloog en toksikoloog het hy wêreldberoemdheid verwerf en is sy mening oor netelige probleme aangevra deur wetenskaplikes en andere in baie dele van die wêreld bv. die VSA, Brittanje, Ierland, Duitsland, Frankryk, Swede en Nieu-Seeland. As gevolg van sy plaaslike en internasionale bekendheid het hy deskundige getuienis gelewer in 'n groot aantal hofsake betreffende vergiftiging van mens en dier. In die verband is hy ook gekonsulteer deur firmas, o.a. oor die bekende talidomiedtragedie van 1967.

As opvoedkundige was hy sowel leermeester as leier. Hy het die vermoë besit om sy eie geesdrif oor te plant op ander en om die belangrikheid van die vakgebied vir mens en dier te laat besef. Dis ook dus geen wonder dat hy by sowel die Veeartsenykundige as die Geneeskundige Fakulteite van U.P. uitmundende diens gelewer het. Hy was 'n alomaanvaarde deskundige op sy gebied. Hy was vir 18 jaar eksterne eksaminator in farmakologie en toksikologie aan verskeie mediese, farmasie- en veeartsenyskole in die R.S.A. Hy is 'n lid van die SAVV en 11 ander hoogaangeskrewe plaaslike en internasionale wetenskaplike verenigings, waarvan twee op uitnodiging, en is erelid van die Henry Doubleday Research Society in Engeland en lid van die Raad van die Association Francais pour l'advancement des Sciences de l'Homo in Frankryk. Hy

he was Professor of Pharmacology and Toxicology at the Faculty of Veterinary Science, University of Pretoria. In 1946 he became Professor of Pharmacology at the Faculty of Medicine of the same University. He reached retiring age in 1963 but took a position with the Life Sciences Division of the Atomic Research Board in Pretoria. In 1965 he was appointed as technical adviser to the Minister of Health, and in 1968 he became pharmacological adviser to a pharmaceutical concern. In 1975 he was attached to the staff of the National Botanical Research Institute to assist in compiling a Poisonous Plant Control Centre.

He is the author of four well-known books on toxicology. His contribution as research worker is perhaps best measured against the 121 scientific articles which he has had published in local and overseas journals. Of these, 54 appeared in the Onderstepoort Journal and dealt with subjects such as "gifblaar" and "tulp" poisoning, "vermeersiekte", "geilsiekte" (HCN), alopecia in goat kids and lambs, "staggers" of horses, kidney and bladder calculi, the toxicity of *Syringa* berries, *Cotyledon* poisoning, crotalariosis, *Lathyrus* poisoning of animals and man, "vygies" and "bietou" and wild lucerne, poisoning of equines, Kimberley horse disease, "gousiekte", seneciosis in animals and man, goitre in animals and man, fluorine, fungi, prussic acid poisoning, phenol, etc. Many of these conditions were already well known, and stock losses in some instances assumed alarming proportions. He instituted renewed investigations and took a global view of plant poisoning by considering all the factors which could influence the incidence and degree of toxicity. By so doing he was frequently able to arrive at the correct diagnosis and advise on treatment or prevention. Determination of the toxic principles of poisonous plants constituted to him a further challenge. He adopted the attitude that it was not treatment but prevention which was required to overcome the problem of plant poisoning of livestock. Application of his recommendations concerning the eradication of poisonous plants and the conservation and improvement of pastures resulted in a dramatic decrease of stock losses due to poisoning.

He became world renowned as pharmacologist and toxicologist and his opinions on contentious matters were sought by scientists and others in the USA, Britain, Ireland, Germany, France, Sweden, New Zealand and many other parts of the world. As a result of his local and international reputation he was called upon to give expert evidence in numerous court cases concerning poisoning of animals and man. In this regard he was also consulted by commercial concerns on matters which included the thalidomide tragedy of 1967.

As an educationalist he was a born teacher and leader. He possessed the ability to transmit his own enthusiasm to others and to impress them with the importance of his subject to the well being of both man and animals. It is therefore little wonder that he was so successful at both the medical and veterinary faculties of UP. He was a recognised expert in his field. For 18 years he was external examiner in pharmacology and toxicology at several medical, pharmacy and veterinary schools in the R.S.A. In 1941 he was awarded the Senior Captain Scott medal from the S.A. Biological Society. He also received the Havenga prize for medical research from the S.A. Akademie vir Wetenskap en Kuns.

He is a member of the SAVA and at least 11 other esteemed local and international scientific societies,

was ook streeksverteenwoordiger van die International Association of Forensic Toxicologists (London).

Hy het gedien op 'n verskeidenheid van Komitees en Kommissies wat ondersoek ingestel het na onderwerpe soos waterbesoedeling, opleiding van aptekers en drogiste, middels, voedselverwerking, insektemiddels, spoorelemente, kropgeswel, slukdermkanker in die Transkei, volksgeneeskunde, sterftes tydens algemene verdowing, hoë kostes van geneeskundige dienste en geneesmiddels, fluoridasie van openbare watervoorrade, en beveiliging van die mens teen vergiftiging.

Vir sy kollegas in beide die menslike en dierlike geneeskunde was hy altyd toeganklik en altyd meer as bereid om hulp en inligting te verskaf. Teenoor die boeregemeenskap was hy ewe diensvaardig. Meer as 75 artikels in die populêrwetenskaplike en landboupers is daarvan ten dele bewys. Hy het talle boereverenigings toegespreek en was alombekend in landboukringe.

Teenoor sy medemens is hy altyd sowel vriendelik as hoflik. 'n Man met 'n aangenamer persoonlikheid, voorbeeldiger karakter en vaster beginsels sou kwalik gevind kan word. Hy was by uitstek altyd onselfsugtig, en sy aktiwiteite oor 50 jaar was basies geïnspireer deur sy inherente begeerte om die welvaart van sy land en medemens te bevorder.

Hoewel hy sy werksfeer in latere jare tot die mediese professie uitgebrei het, was dit nooit ten koste van sy eie veterinêre beroep nie, en het hy nog onlangs op versoek van die SAVV 'n lywige dokument oor "Die Gevare en Nadelige Effekte van die Wangebruik van Antibiotika by Diere" opgestel.

Hierdie seun van die Karoo en van Suid-Afrika het sy plek as mens en as veearts volgestaan op 'n wyse wat elke lid van hierdie beroep trots maak om met hom geassosieer te wees. Hy het baie duidelik getoon oor watter wye veld die veearts die welsyn van die mens kan bevorder, en is voorwaar 'n waardige ontvanger van die hoogste eer wat die veeartsenyberoep aan hom kan bring.

of which two on invitation. He is an honorary member of the Henry Doubleday Research Society in England and a member of the Board of the Association Francais pour l'advancement des Sciences de l'Homo in France. He was also appointed Regional Representative of the International Association of Forensic Toxicologists (London).

He has served on numerous Committees and Commissions which investigated matters such as water pollution, training of chemists and druggists, drugs, food processing, insecticides, trace elements, goitre, oesophageal cancer in the Transkei, folk medicine, deaths during general anaesthesia, high costs of medical services and medicine, fluoridation of public water supplies, and the safeguarding of man against poisoning.

His colleagues in veterinary and human medicine have always found him to be approachable and more than willing to assist and furnish information. To the farming community he was equally helpful. More than 75 of his popular scientific articles appeared in the agricultural press; he addressed innumerable farmers meetings, and was very well known in agricultural circles.

Towards his fellow man he is always friendly and courteous. A man with a more pleasant personality, exemplary behaviour and firm principles would indeed be hard to find. He is essentially unselfish by nature, and his work over a period of 50 years was basically inspired by his inherent desire to promote the welfare of his country and its peoples.

Although he latterly extended his activities into the medical field, this was never at the expense of his own veterinary profession. Quite recently he acceded to a request from the SAVA to prepare a lengthy document on "The Hazards & Harmful Effects of the Abuse of Antibiotics in Animals".

This son of the Karoo and of South Africa has made his mark — both as a man and as a veterinarian. Every member of this profession can be proud to be associated with him. He has so clearly shown how broad is the field in which the veterinarian can promote the welfare of man. He is truly worthy of receipt of the highest honour which the veterinary profession can bestow.

BOOK REVIEW

BOEKRESENSIE

VETERINARY PHYSIOLOGY

J.W. PHILLIS, EDITOR

Wright — Scientechnica, Bristol 1976. pp. X 882, Figs 288, Tabs 80, Price £17.00

"Veterinary Physiology" is certainly a book which will find a place on the shelves of those who teach that subject. In contrast to most of the standard texts which are based entirely on human physiology, this book is essentially concerned with physiology of the domestic animals. It is therefore possible to find tables which give data from a wide variety of domestic animals in this book. Illustrations of anatomical features are all of domestic animals. The chapters on lactation are particularly welcome as this aspect of physiology is of great importance to the veterinarian and is not adequately dealt with in most human physiology texts.

In the preface the editor states that "The text has been written bearing in mind the constraints imposed by the recent trends to shorter curricula in the preclinical or basic sciences in veterinary schools". This is a most welcome aim as courses in physiology, given at the undergraduate level, are tending to become excessively voluminous. The authors have tried to adhere to this aim. In some cases however the desire to shorten the text appears to have led to a reduction in the amount of explanation of principles rather than a restriction of the amount of material covered.

The reviewer questions the advisability of including a short section on metabolism and bioenergetics. This has been done at the expense of basic knowledge of biochemistry. As an example it is noted that whole sections of metabolism are covered without any chemical structures being given. Despite these criticisms this book should be a valuable aid in teaching veterinary physiology. The high price of the book may, however, prevent it being generally prescribed for veterinary students.

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INFORMATION

INLIGTING

SCOURS PROTECTION FOR UNBORN CALVES

Professor Gabel H. Conner of the Department of Large Animal Surgery and Medicine in Michigan State University's College of Veterinary Medicine has reported that work done by Gay and Conner during 1971 to 1975 has stimulated interest in the immunologic capabilities of the foetus. These investigators showed that a bovine foetus to which an *Escherichia coli* bacterin was administered, either intra-amniotically or intramuscularly, during the last 3 months of the gestation period posessed serum antibodies against *E. coli* at the time of birth. Also, colostrum-deprived calves vaccinated prenatally survived oral challenge by live organisms which killed calves that had not been immunised prenatally. In addition, Gay showed that when the foetus was vaccinated in utero with a single serotype of *E. coli*, the result was heterogenetic protection against neonatal colisepticaemia.

Subsequent research by Professor Conner has shown that vaccination of the calf before birth is effective in preventing one form of diarrhoea, namely scours. The intrauterine vaccination of the foetus can be accomplished without performing surgery on the cow, and the procedure (Fig. 1) takes less than 10 minutes. With practice, it could probably be performed in half this time, under field conditions, according to Professor Conner.

The researcher says that it appears that vaccination of the bovine foetus can become a practical, routine, clinical procedure. Professor Conner found that when an *E. coli* antigen (bacterin) is deposited in foetal fluids 3 to 4 weeks prior to birth, the newborn calves possessed antibodies and were also protected against an oral challenge dose of virulent *E. coli* (the same organism as in the antigen). In the tests, newborn calves were deprived of colostrum and instead maintained on reconstituted, dried, cow's milk. Those that had been vaccinated *in utero* did not have diarrhoea and survived an oral challenge, whereas those not vaccinated were found to have diarrhoea and died within 2 to 4 days after being exposed to the challenge inoculum.

To prepare the antigen (immunising inoculum), a culture (*E. coli* 026:K60:H11) was grown overnight on tryptocase soy broth in a shaking water bath at 37°C. After determination of the purity of the culture, cells were collected by centrifugation, and washed twice with sterile saline. To kill the cells, 0,4 per cent formalin was added and the suspension incubated in a shaking water bath (at 37°C) overnight. The formalin-killed cells were then washed twice with sterile saline and a total cell count was obtained by comparing the optical density of the cell suspension to a series of optical tubes (Burroughs Wellcome Company). The suspension was then diluted with sterile saline with 0,01 per cent merthiolate to a concentration of 1,5 or 2 x 10^{10} cells per cc of inoculum. Two to three cc of the inoculum is used, so that a total of approximately 30 to 60 billion* cells are deposited in the amniotic fluid.

Knowledge gained from this research is now being applied in several local dairy herds, under farm conditions. Prenatal vaccination is being performed in herds which have previously sustained losses as a result of colibacillosis (diarrhoea, septicaemia), a disease of the newborn, usually resulting in death within the first 10 days after birth. Insufficient field data for a definitive evaluation are available at present, but immunisation before birth appears to protect calves from colibacillosis.

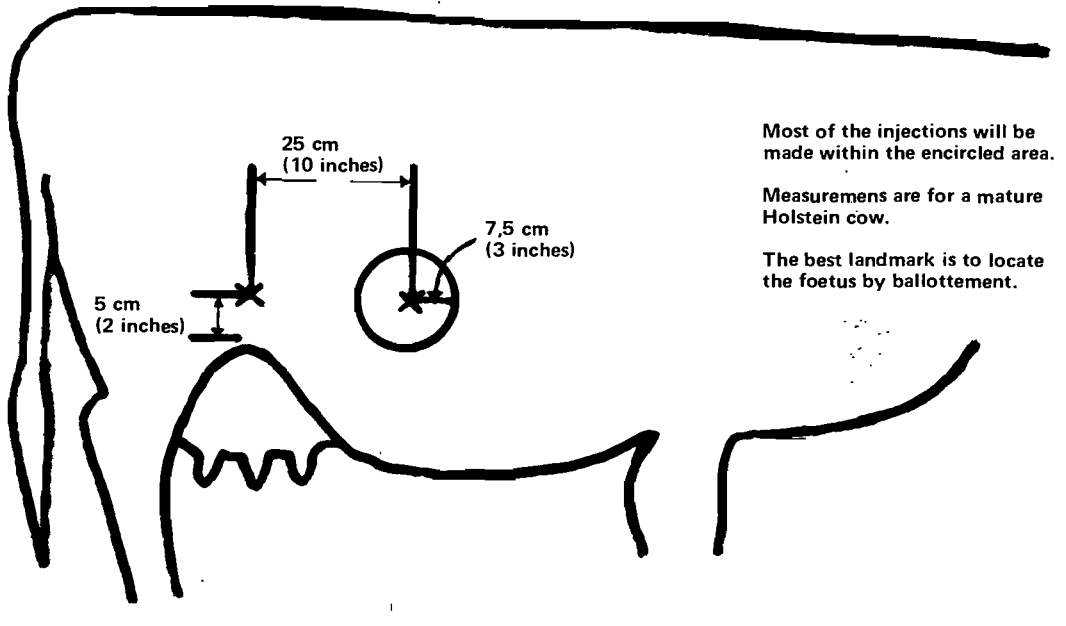

Fig. 1: *In Utero* vaccination of the bovine foetus

. Recommended times to deposit bacterin (antigen) into the amniotic fluid are three to four weeks before parturition;

2. Clip hair over site and cleanse (surgical scrub);
3. Infiltrate a local anaesthetic into the skin, muscles, and peritoneum at the site where needle-puncture will be made;
4. Insert a 12 gauge x 2" (2,65 mm dia. x 50,8 mm) needle through the skin, muscles and peritoneum. (It is easier to

1 billion = 1 000 million

Book Reviews

AUTHOR INDEX FOR VOLUME 47, 1976. SKRYWERSINDEKS VIR JAARGANG 47, 1976

Bold page number indicates senior or sole author.
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