JOURNAL

OF

THE SOUTH AFRICAN VETERINARY MEDICAL ASSOCIATION

VOLUME
JAARGANG 35

1964

TYDSKRIF

VAN

DIE SUID-AFRIKAANSE VETERINÊR-MEDIESE VERENIGING

PROGRAMME

ANNUAL SCIENTIFIC CONGRESS AND FIFTY-NINTH ANNUAL GENERAL MEETING
ARTHUR'S SEAT HOTEL, CAPE TOWN

28TH SEPTEMBER — 2ND OCTOBER 1964

SUNDAY 27TH SEPTEMBER 1964

7.30 p.m. (onwards) —Arthur's Seat Hotel — Optional registration and Get-Together.
Film — To Catch a Rhino.

MONDAY 28TH SEPTEMBER 1964

8.00 a.m.	Registration.
9.00 a.m.	Convocation.
9.05 a.m.	Welcome to Guests, Delegates and Members by HIS WORSHIP, THE MAYOR OF CAPE TOWN.
9.10 a.m.	Expression of thanks to the Mayor by the Chairman of the Cape Western Branch — DR. A. A. ALBERTYN.
9.15 a.m.	Opening of Congress by MR. J. E. DE VILLIERS LOUBSER, Chairman of the Winter Rainfall Area Agricultural Union. Vice-President of the Cape Province Agricultural Union.
9.30 a.m.	Expression of thanks to Mr. J. E. de Villiers Loubser by the President of the Association DR. H. P. STEYN.
9.45 a.m.	Presedential Address.
10.00 a.m.	Opening of the Trade Exhibits by the Vice President — PROF. R. CLARK.

TEA

11.00 a.m.	Address by Dr. J. F. FIGUEROA, Vice President of the World Veterinary Association.
11.15 a.m.	Mycotoxicosis — L. ABRAMS.
12.00 m.	The Milkfever syndrome in the Western Cape Province — K. VAN DER WALT and R. CLARK.

LUNCH

2.00 p.m.	The Differential Diagnosis of Infectious Diseases occurring in Dogs in South Africa — J. D. SMIT.
2.45 p.m.	Lungworm in Dogs in the Western Cape Province — J. E. DORRINGTON.
3.30 p.m.	Blood transfusion in dogs with a reference to its effect in canine babesiosis — P. H. LE ROUX.
4.00 p.m.	Clinico-Pathological aspects of Biliary Fever — W. D. MALHERBE.

TUESDAY 29TH SEPTEMBER 1964

8.00 a.m.	Registration.
8.30 a.m.	A Study of the Influence of environmental factors on the Weathering of Merino wool — J. G. SNYMAN.
9.30 a.m.	DISEASES OF SHEEP IN THE WESTERN CAPE PROVINCE
9.30 a.m.	(i) Lupinosis in sheep in the Western Cape Province (With Illustrated Slide Projection) — L. F. NAUDE.
	(ii) Geeldikkop and Enzootic Icterus in sheep in the Western Cape Province— J. M. M. BROWN.

TEA

11.00 a.m.	(iii) Infertility and Reproduction in sheep in the Western Cape Province — K. M. VAN HEERDEN.
11.30 a.m.	(iv) The Relationship between Helminth infestation, Production, and Nutrition — G. L. MULLER.
12.00 m.	(v) Some common Bacterial Infections of Sheep in South Africa — P. W. THOROLD.

LUNCH

2.00 p.m.	Group Meetings and Formation of New Groups and Branches. Films.
8.00 p.m.	Conference Lecture — PROF. A. KIPPS — The Mechanism of Immunity.

WEDNESDAY 30TH SEPTEMBER 1964

7.30 a.m.	Instructional tour of Western Cape. Arranged and conducted by Mr. Smit M.P. and Dr. G. L. Muller. Leave Sea Point at 7.30 a.m. Braaivleis-lunch at Malmesbury by members of the Malmesbury Farmer's Association: Tour of K.W.V. Cellars.

THURSDAY 1ST OCTOBER 1964

8.00 a.m.	Registration.
8.30 a.m.	*Fifty Ninth Annual General Meeting.*

LUNCH

2.00 p.m.	Milk Control in the Western Cape Province — A. A. L. ALBERTYN.
2.45 p.m.	Georganiseerde Varkgesondheidsbeheer — G. F. VAN DER MERWE.
3.00 p.m.	The use of a Goitrogen in the Fattening Ration of Slaughter Cattle —H. J. J. TERBLANCHE.

FRIDAY 2ND OCTOBER 1964

8.30 a.m.	Hip Dysplasia in the Dog — With Illustrated Slide Projection — C. F. B. HOFMEYR.
9.15 a.m.	The Pathology of African Horesesickness — MAJOR R. M. McCULLY.
10.00 a.m.	Chemical Restraint of Animals — A. M. HARTHOORN.

TEA

11.15 a.m.	RESOLUTIONS.
11.30 a.m.	ADJOURNMENT.

SOCIAL ARRANGEMENTS

MONDAY 28TH SEPTEMBER 1964

5.30 p.m. Mayoral Cocktail Party.

TUESDAY 29TH SEPTEMBER 1964

9.00 a.m. LADIES: Visit to Administrator's Residence — Leeuwenhof: Demonstrations on the preparation of savouries by Mrs. G. Faull and a Lecture by a beautician.

WEDNESDAY 30TH SEPTEMBER 1964

7.30 a.m. LADIES: Instructional Tour of Western Cape — leaving Sea Point 7.30 a.m.: Social Tour of K.W.V. Cellars.

THURSDAY 1ST OCTOBER 1964

9.00 a.m. LADIES: Visit to Kirstenbosch & Groot Constantia.
8.00 p.m. Buffet Supper and Dance at Arthur's Seat Hotel — Evening Dress.

FRIDAY 2ND OCTOBER 1964

2.00 p.m. Sport.
7.00 p.m. Informal Function and awarding of Sports Trophies — Cape Western Branch.

TRADE EXHIBITION

Monday 28th September, 1964 to Tuesday 29th September, 1964.

9.00 a.m. Opening of Congress — Morning tea Ladies Welcome.
8.00 p.m. Conference Lecture by PROF. A. KIPPS — *The Mechanism of Immunity.*

Redaksioneel op uitnodiging.

TWEE DISSIPLINES

Alhoewel die doel en strewe van die twee dissiplines Veeteelt en Veeartsenykunde fundamenteel dieselfde is, laat die samewerking tussen die persone wat die dissiplines beoefen soms veel te wense oor. Vir hierdie rede is dit vir my baie aangenaam om saam met u te kan verkeer. Ek wil dan ook die hoop uitspreek dat die werk wat op die gebied van die diere wetenskap gedoen word, in die toekoms 'n toenemende rol sal speel om aan die steeds groeiende mensdom, voedsel en klere te verskaf.

As daar gedink word aan die groeiende mensdom, en die benodigdhede vir beskermende voedsels vir die instandhouding daarvan kom mens tot die gevolgtrekking dat daar aan die diere wetenskappe waarby ek sekerlik die veeartsenykunde insluit, 'n groot uitdaging gebied word. U sal besef wat die uitdaging omvat indien dit bv. op die gebied van melkproduksie geillustreer kan word. Elke 24 uur vermeerder die populasie met ± 100,000 persone. Om aan die 100,000 persone 'n glas melk te verskaf benodig die daaglikse produksie van 8,000 produserende koeie. Om dus tred te kan hou, beteken dit dat daar elke dag 8,000 meer koeie in produksie moet kom.

Ensminger, een van ons vooraanstaande wetenskaplikes op die gebied van die dierewetenskappe sien die toekomstige ontwikkeling in 'n rewolusionêre lig. Hy sê o.a.: „To chart the future, one must dream; a fact that even the rugged individualists of the livestock industry is beginning to realize. To be sure, it is not expected that all of us have the same dreams; in fact, this would not be a good thing. Amongst the sure things about the livestock industry of the future . . . there will be momentous changes and these changes will take place at an accelerated rate . . . The biological sciences will arrive. In the present space age, the physical sciences, physics, mathematics and chemistry are having their day. In the next 50 years, however, the greatest advances will be in the biological sciences and many of these will come through the animal route. Now that the physical sciences have discovered how to destroy mankind, the biological sciences will unlock many of the secrets of the living organisms thereby making for greater health & happiness."

Die uiteindelike oogmerk van die dierewetenskappe is die verhoging van die biologiese sowel as die ekonomiese doeltreffendheid van produksie van die verskillende plaasdiere en hierby speel die algemene gesondheid van die diere natuurlik 'n fundamentele rol. Dierewetenskap is dus nie alleen 'n wetenskap wat beide die fundamentele en toegepaste aspekte van die produksie van plaasdiere omvat nie waar ook die bevindinge van verwante vakrigtings soos anatomie fisiologie, biochemie genetika mikrobiologie patologie ens. koordineer en toepas. Verder word die wetenskap in baie gevalle deur faktore van ekonomiese en sosiale aard beïnvloed. Die dierewetenskap omvat dus beide 'n fundamentele en toegepaste studieveld.

As gevolg van die besondere aard daarvan en die komplekse faktore wat dit beinvloed, behoort die vak in 'n noue verband met die praktyk te staan, veral omrede die toepassing daarvan die welsyn van 'n groot aantal individuele operateurs (boere) beïnvloed. Dit is 'n ongelukkige toestand dat die wetenskap en die praktyk in baie gevalle mekaar nog nie kon vind nie en as daar in aanmerking geneem word wat in die verlede sonder die hulp van die georganiseerde wetenskap tot stand gebring is, is ek nie so seker dat die skuld daarvoor altyd by die praktyk lê nie.

'n Groot aantal rasse plaasdiere en produksiesisteme is al tot stand gebring en ontwikkel voordat die epogmakende bydraes van Mendel, Darwin, Morgan en Johanssen die lig gesien het. Allan Frazer sê in hierdie verband: „I feel

therefore, that the traditional knowledge based on practical experience which forms the solid basis of our Animal Sciences through the criticisms of certain scientists, a trifle too wise in their own conceit, is being ignored or even somewhat unfairly derided and that the newer knowledge based on science is being applied too uncritically and with too great a haste".

Daar is vandag persone wat die standpunt huldig dat alle biologiese verskynsels, as dit reg verstaan word, gereduseer kan word in terme van fisika, wiskunde en chemie, dat die verskil tussen 'n kristal en 'n dier eerder in die gekompliseerdheid as in die aard daarvan gesoek moet word en dat daar geen fundamentele verskil tussen lewendige en lewelose liggame is nie. Is dié opvatting reg? Is die biologiese wetenskappe en die landbou-wetenskap in besonder nie geneig om in die moderne tyd té basies en ontoepaslik te word nie. Is dit nie miskien die rede dat die wetenskap en die praktyk mekaar nie altyd kan vind nie.

Alexis Carrel, die persoon wat met sy werk op 'n kuikenhart wat hy buite die liggaam in 'n voedingsmedium aan die lewe gehou het, aan die brandpunt van ewigdurende lewe, die misterie van greoi en die lewe self geraak het, verklaar: „One cannot understand the living body by studying the dead. In reality an organ separated from its nutritive medium no longer exists. In order to apprehend this inner world as it is, we must study the organs of living animals and even as they are seen in surgical operations and not simply those of cadavers. All living cells depend absolutely on the medium in which they are immersed. They modify this medium unceasingly and are modified by it. In fact, they are inseparable from it". Toynbee laat homself as volg uit: „We are sufficiently on our guard against the socalled "Pathetic Fallacy" of imaginatively endowing inanimate objects with life. We now fall victims to the inverse "Apathetic Fallacy" of treating living creatures as though they were inanimate".

Daar bestaan 'n misterieuse verskil tussen die wetenskappe wat te doen het met lewelose materie en die wat te doen het met lewendige wesens. Die hoër vorme van dierelewe soos die waaraan ons plaasdiere behoort, is 'n onverdeelbare geheel van 'n uiterste kompleks. As sodanig bestaan daar geen tegniek waardeur dit gelyktydig in die geheel en afsonderlike dele daarvan sowel as in die verhouding tot die omgewing verstaan kan word nie. Om die dier, veral die produserende dier, te probeer verstaan is dit nodig om verskillende tegnieke van variërende aard aan te wend waardeur verskeie verwante wetenskappe tot hulp geroep word. Hierdie verskillende wetenskappe kom gewoonlik tot variërende gevolgtrekkinge van wat die aard en wese van hulle gemeenskaplike studiemateriaal eintlik is. Dit neem slegs uit die dier dit wat hulle gespesialiseerde tegnieke moontlik maak en dikwels wanneer die abstraksies saamgevoeg word tot 'n eenheid, is die resultaat nog steeds arm aan konkrete feite en laat dit 'n leemte na wat te belangrik is om oor die hoof te sien. Die dier, soos deur verskillende spesialiste (fundamentaliste?) verstaan, is ver verwyder van die konkrete dier waarop die mens staatmaak vir doeltreffende biologiese en ekonomiese produksie van die belangrike, die sg. „beskermende" voedsels.

Die dier waarin die basiese wetenskappe belangstel, is niks anders nie as 'n komplekse skema bestaande uit ander skemas wat uit die tegnieke van studie van verskillende wetenskappe opgebou is. Dit is terselfdertyd 'n karkas wat deur anatomiste, vleiskundiges en biochemikers ontleed en bestudeer word; 'n wonderlike samestelling van selle, weefsels en voedingsmedia waarin die fisioloog en histoloog belangstel (selfs 'n meganistiese en 'n vitalistiese fisioloog sien die dier uit verskillende oogpunte); 'n chemiese kompleks waaruit die weefsels en vloeistowwe van die liggaam bestaan; 'n uiters komplekse samestelling van genetiese materiaal; 'n medium waarop talle mikro-organismes, interne en eksterne parasiete hul funksie verrig en hul heenkome vind en 'n organisme wat aan siektetoestande, wat sy aard, produksie en gedrag modifieer, blootgestel word. Verder is die dier 'n voorwerp waarop die omgewing (fotoperiode, temperatuur, vogtigheid, bestuur en voeding) gedurigdeur 'n invloed uitoefen en nietemin trag om sy genetiese potensialiteite onder wisselende omgewingstoestande tot uiting te laat kom. Dit is 'n biologiese eenheid wat onverpoosd nutriente moet inneem en doeltreffend verwerk tot verskeie produkte sodat dit aan sy medeskepsel, die mens, 'n diens kan verrig.

Maar, die dier is ook 'n wese met 'n eie geestesstatus soos openbaar word deur natuurlike gewoontes, individuele en kudde-instink, gedrag en ander eieselwige karaktertrekke soos senuweeagtigheid, moed e.d.m.

Die basiese voedingsdeskundige byvoorbeeld, wat voeding al te dikwels afsonder van teling en omgewingsfaktore, is geneig om die dier te beskou as 'n passiewe masjien waarin brandstof in die vorm van voer gegooi word wat dan omgesit word na energie en dierlike produkte met wisselende doeltreffendheid afhangende van die aard van die produk, en die spesies of ras van die dier. Die begrip dat 'n dier op enige tydstip slegs een stadium van 'n ontsettende ingewikkelde proses is, word te dikwels misken.

Die toekoms van die wetenskap en die bedryf wat dit behoort te dien, lê m.i. dus nie in die eenvoudigheid van die ou praktyke of in een of ander spesifieke enkele vertakking van die wetenskap nie, maar ongetwyfeld in die doeltreffende integrasie en koördinasie van die verskillende vertakkinge van die wetenskap wat dit ondersteun, aangevul deur 'n intieme kennis van die praktyk en wat letterlik genoem kan word gesonde verstand. Een van ons vroeëre wetenskaplikes, T. H. Huxley, het tog immers die wetenskap gedefinieer as „organized common sense".

Dit is dus die taak van diegene wat die groot Industrie in een of ander vertakking dien om toe te sien dat deur samewerking, koördinasie en toepassing van die verskillende dissiplines die groot uitdaging wat aan ons gestel is, aanvaar word en dat die wetenskaplike wat in ons diere bevolking belangstel hulle met eer van hulle taak sal kwyt.

Die hoop word uitgespreek dat in die toekoms die samewerking tussen die praktiese boer, die Veeteler en Veearts sal toeneem en bestendig word want alleen dan is sukses moontlik.

H. C. LUITINGH
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PV 2997

EDITORIAL

MORE MEAT BY PARTIAL OR RUSSIAN CASTRATION

Despite our more or less constant food animal population, annual slaughterings increase every year to meet the demands of a growing and more sophisticated total population. Thus 665,346 cattle were slaughtered in 1938/39, and 1.5 million in 1962/63, whilst for the corresponding years the small stock slaughterings amounted to 3.5 and 5.3 million respectively. Improvement in breeds, disease control, animal management and earlier slaughtering are some of the factors which have made this possible.

To meet this increasing and accelerated demand for meat, other methods may also have to be considered, and the 5—10% increase in weight of animals claimed by Russian workers following partial castration merits serious investigation.

Castration of surplus male animals has been practised for many years to control breeding and to render the meat from male animals more acceptable to the consumer.

Despite discrimination of present carcase grading regulations against uncastrated bovine males over six months of age, there is however an increasing interest in the slaughter of young entire males such as is practised in Europe. The consumer's aversion to excess fat and the relatively greater muscular development in entire males accounts for this tendency. Recent comparative work in fat lamb production in South Africa has shown that seven month old rams off the veld had a three lbs. weight advantage at slaughter over castrated controls.

Partial castration claims to improve on the growth characteristics of entire males whilst still achieving breeding control. The method has been the subject of numerous Russian papers, particularly by Kirillov and Baiburtcjan. The Bureau of Animal Breeding and Genetics deserves tribute for translating and reprinting two of these, together with a review article by G. D. Turton on "The effect of partial castration on meat production and quality in cattle, sheep and pigs". These reports have shaken conventional complacency and made available important information to the agricultural and veterinary professions.

The method involves a technique which will remind senior veterinarians of the method of "cutting a horse proud". Despite a still existent controversy over the exact site of male hormone production, Dr. Baiburtcjan's technique ensures retention of remnants of the fibrous tissue stroma of testis and epididymis, tunica albuginea, walls of seminiferous tubules and some Sertoli and interstitial cells. Testicular parenchyma, seminiferous structures and associated tissue is eliminated. The procedure is in fact not true castration but rather sterilization.

After surgical preparations, one testis is fixed by forceps and punctured by a sharp scalpel through the scrotum and into the testis to a depth of 0.5 to 1.0 cm.

The scalpel blade is turned through 90° to 180° on its longitudinal axis to loosen the parenchyma and to facilitate its gentle expression through the puncture by the fingers of both hands. Expression begins near the wound, then from the deeper and middle portions, then the head and finally from the tail portion. It should be gradual since any severe pressure may rupture the scrotal wall and cause hernia of epididymis and testis residue, with infection and undesirable complications. Parenchymal regeneration may result if the head portion is not properly expressed, though it seems unlikely to occur if the tail portion is not fully evacuated. Proper fixation of the testis is stressed so that the openings in scrotum and testis tunic are kept coadapted. The skin around the wounds is treated by antiseptic and fly repellant ointment and healing is complete in a few days and there is little systemic disturbance.

It becomes immediately clear that this operation cannot readily be undertaken by the lay persons who do most of our farm castrations today. Surgical asepsis is demanded and accentuated by the presence of damaged tissue and a small wound. The operation requires several minutes of painful procedure and therefore demands an efficient local anaesthesia. The Animals Protection Act No. 71 of 1962 would certainly be applicable.

A joint investigation into the surgical aspects and the effect on growth, carcase quality and yield etc. under South African conditions is currently being undertaken by the Department of Surgery of the Faculty of Veterinary Science at Onderstepoort and by the Livestock and Meat Industries Control Board. It is hoped to learn in due course whether the method has any merit.

The method undoubtedly requires veterinary assistance or supervision, and partial castration of even a portion of the estimated 4–5 million male food animals slaughtered annually is likely to present the profession with a major problem.

Control of partial castrates will however remain difficult on our extensive farms, and this alone may act as a major deterrent to extensive application of the method.

REFERENCES

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2. Reprint No. 53, *Commonw. Bur. Ani. Breed. Gen.* 1963.
3. *An. Breed. Abstrts.* 1963. **31**: 1–21.
4. *Brit. Vet. J.* (1963) **119**: 565.
5. *Farm. in S.A.* (1964) **40** (1): 14–17.

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PERFORMANCE TESTING OF BEEF CATTLE UNDER RANCHING CONDITIONS

I. G. HORAK* M. L. P. RATTRAY B. CHARLS

"Quest Ranch", Mkuze, Zululand.

Received for Publication May 1964

SUMMARY

A performance testing programme involving 38 bulls, 1,850 cows and 1,834 calves on two ranches in Zululand is described.

Sex, breed of sire, age of dam, time of birth, time of weaning and grazing quality all influenced weaning weight.

Hereford sired calves made the greatest daily weight gains during 1958–59, Brahman sired calves had the heaviest weaning weights in 1959–60.

There was a positive correlation between weaning weight and post-weaning weight, but light weaners made greater post-weaning gains than did heavy weaners.

Mortality rates were influenced by breed and season of the year.

February was the most fertile month on one of the ranches.

Culling for infertility was severe.

INTRODUCTION

The performance of an animal may be measured by its ability to survive, reproduce and produce a marketable product at an early age within a given environment. The performance of beef-producing animals is assessed on fertility, body-weight, conformation and composition and quality of carcass. The validity of comparative studies depends on performance being measured within the same environment.

Performance testing of beef-producing cattle has generally been limited to trials under feedlot conditions, but a number of studies have been carried out under grazing conditions[2,4,8,9]. This paper describes a performance testing programme as applied to beef cattle on two ranches in Zululand during the years 1958 to 1960.

MATERAILS

Ranches

(i) "DUBE RIDGE RANCH" of approximately 8,000 acres in the Ntambanana district of Zululand.

(ii) "QUEST RANCH" of approximately 30,000 acres in the Mkuze district of Northern Zululand.

Cows

(i) Dube Ridge, approximately 450 grade and registered Afrikaner cows.

(ii) Quest Ranch, approximately 1,400 commercial cows of Afrikaner, Drakensberger, Nguni and Friesland type as well as crosses between Afrikaner cows and Hereford, Shorthorn, Drakensberger, Aberdeen Angus and Sussex bulls. Afrikaner and Drakensberger cows were in the majority.

Bulls

(i) Dube Ridge, eihteen registered Afrikaner bulls.

*Present address: Section of Helminthology, Veterinary Research Institute, Onderstepoort.

(ii) Quest Ranch, twenty bulls consisting of Brahmans, Afrikaners, Herefords, Drakensbergers, Sussex, Aberdeen Angus and Shorthorns were used for natural service as well as semen from Braham, Hereford, Drakensberger, Sussex, Afrikaner and Charollais bulls for artificial insemination purposes.

Weighbridge

One Avery cattle weighbridge capable of weighing 2,000 lbs with collecting, holding and crush pens was installed on each ranch.

Methods

Six points of selection were used: (i) the ability to survive in the particular environment, (ii) the ability to reproduce regularly, (iii) the ability to put on the maximum amount of weight in the shortest period, (iv) conformation, (v) carcass evaluation at slaughter, (vi) manageability.

The breeding cows at Quest Ranch were branded with a number between 1 and 1,500 on their right ribs, whereas those at Dube Ridge had an "A" preceeding their numbers. At Quest Ranch the cows were artificially inseminated from approximately October to April, the bulls running with them for the remainder of the year. On Dube Ridge two breeding seasons were employed, one in the spring and the other in summer. The cows on both ranches were regularly examined for pregnancy and when pregnant were removed from the breeding herds to the pregnant herds where they were kept until they calved. Cows failing to calve at least every sixteen months were culled unless they were pregnant at the time. The bulls too were regularly examined for fertility.

When a bull calf was born a blue plastic disk was put into its left ear, a yellow disk was put into a heifer calf's ear. On these plastic disks a letter indicating the year of birth and the calf's number were stamped. An identical letter and number were also tatooed in the calf's left ear. In the calf's and its dam's right ear a plastic disk of a particular colour was clipped to indicate the 37 to 58 day period during which the calf was born. The calf's date of birth, sex, number, breed and dam's number were entered in the calf performance register. If the calf was an artificial insemination calf, its sire's number was also entered in the register. Identical details were entered on the dam's performance card.

As a group of calves born during a particular 37 to 58 day period reached an average age of approximately 225 days the whole group was collected. They were weighed, dewormed, dehorned, branded and inoculated against quarter evil and in the case of the heifers, contagious abortion. By means of a conversion graph each calf's weight was converted to a 205 day weight and the calves were divided into three groups according to their 205 day weights; Group 1 consisting of the heaviest calves and Group 3 of the lightest. All bull calves not falling in the top weight group were castrated. The calves remained with their dams for approximately 50 days before being weighed again and weaned. Cows, the calves from which were in the bottom 10% of the weight group, were culled.

At the start of a new breeding season the heifers were weighed and the replacement heifers were selected on top weaning and eventual weights. These heifers were branded on the right ribs with their numbers, dewormed and innoculated against quarter evil and contagious abortion and taken up into breeding herds.

By the end of 1960 no selection had been done on the basis of carcass evaluation. Any animals which were unmanageable were culled.

The Quest Ranch calf-crop of 1958–59 was the first group of calves in the performance programme and they were not divided into 37 to 58 day groups according to date of birth. On Dube Ridge the calves were divided only into summer and winter born calves. The weighing of the Quest Ranch 1958–59 calf-crop and the Dube Ridge calf-crops was not done at an average age of approximately 225 days, but a little later. Consequently the 205 day weights were not used. The Quest Ranch 1959–60 calf-crop was weighed at an average age of approximately 225 days.

The Quest Ranch 1958–59 heifer calf-crop was weighed again in 1960, so too the entire Dube Ridge summer and winter calf-crop for 1958–59.

For comparative purposes average daily gains in pounds were calculated. These were obtained by subtracting 75 lbs. (estimated average birth weight) from a calf's weight and dividing the remainder by the calf's age.

Results

(a) *Weaning Weights*

The average weaning weights, ages and daily gains of all available calves on both ranches during 1958, 1959 and 1960 are shown in Table 1.

Bull calves were an average of 35.5 lbs heavier than heifer calves. Afrikaner, Drakensberger, Hereford and Sussex sired calves were lighter than the average for the group, whereas Brahman sired calves were heavier.

Calves born to mature cows were 15 lbs heavier than those born to first calf cows. Calves born to Nguni cows were lighter than those from other cows.

(b) *Post-weaning weights*

The post-weaning weights, total ages and daily gains for the Dube Ridge 1958–59 calves and Quest Ranch 1958–59 heifer calves are given in Table 4.

Table 1.—Average Weaning Weights and ages on Dube Ridge and Quest Ranch.

Ranch	Year of Birth	Sex	Number of Calves	Average weight in lbs.	Average Age in Days	*Average Daily gain in lbs.
Dube Ridge	Summer 1958–59	♂	89	380.0	332.0	0.919
		♀	88	352.0	331.8	0.835
Dube Ridge	Winter 1959	♂	42	453.1	292.7	1,292
		♀	45	408.9	301.1	1,109
Dube Ridge	Summer 1959–60	♂	61	375.6	259.0	1,161
		♀	58	338.7	258.7	1,019
Quest Ranch	1958–1959	♂	225	347.1	244.9	1,111
		♀	231	332.5	243.4	1.058
Quest Ranch	1959–1960	♂	271	395.0	232.8	1,375
		♀	281	356.6	234.8	1,199
TOTAL		♂	688	379.2	255.6	1.190
		♀	703	350.0	256.0	1.074

$$* = \frac{\text{(Weaning weight — 75 lbs. (estimated birth weight)}}{\text{Age in days}}$$

The Dube Ridge winter 1959 calf-crop was heavier than the summer 1959 to 1960 calf-crop which, taking age into consideration, was heavier than the summer 1958 to 1959 crop. Calves on Quest Ranch born during 1959 to 1960 were heavier than those born the previous season. Bull calves were heavier than heifer calves.

The breed influence of the sire on the weaning weights of calves is shown in Tables 2 and 3.

Males were heavier than females. Afrikaner and Drakensberger sired calves made smaller daily gains than the average for the group, while Hereford sired calves made greater daily gains.

In Table 3 weight is expressed as a 205 day converted weight. The influence of the dam on the 205 day weight is also given in Table 3.

Daily gains during the post-weaning period were less than pre-weaning daily gains. The Quest Ranch heifer calves had a greater post-weaning daily gain than the Dube Ridge heifer or bull calves.

The influence on subsequent weight gain of grouping the heaviest calves into Group 1 and the lightest into Group 3 at weaning, is shown in Table 5.

Group 2 and 3 calves gained more weight during the post-weaning period than Group 1 calves, and in most instances Group 3 calves gained more than Group 2 calves. In all but one case males gained more weight than females.

TABLE 2.—SIRE COMPARISONS ON WEANING WEIGHTS ON QUEST RANCH DURING 1958 TO 1959.

Sire	Sex	Number of Calves	Average Weight in lbs.	Average Age in Days	*Average Daily gain in lbs.	% Above or Below Average gain for sex group.
Aberdeen Angus**	♂	3	347.3	240.3	1.133	+ 2.0
		9	374.3	293.8	1.019	— 3.7
Afrikaner	♂	9	331.2	259.1	0.989	—11.0
		21	328.3	264.6	0.946	—10.6
Brahman	♂	97	347.5	242.6	1.123	+ 1.1
		105	332.8	244.1	1.056	— 0.2
Drakensberger	♂	40	336.0	246.4	1.059	— 4.7
		41	319.3	235.3	1.038	— 1.9
Hereford	♂	63	356.3	244.3	1.151	+ 3.6
		48	341.0	231.6	1.149	+ 8.6
Sussex**	♂	9	352.6	250.8	1.107	— 0.4
		5	333.4	239.8	1.078	+ 1.9
Nguni**	♂	1	320.0	260.0	0.942	—15.2
		2	269.5	214.5	0.907	—14.3
Unknown	♂	3	329.3	249.7	1.018	— 8.4
Total	♂	225	347.1	244.9	1.111	
		231	332.5	243.4	1.058	

* = $\frac{\text{(Weaning weight—75 lbs (estimated birth weight)}}{\text{Age in days}}$

** Too few calves for comparative purposes.

Table 6 shows the breed influence of the sire on the post-weaning weights of Quest Ranch 1958–59 heifer calves.

Afrikaner sired calves showed a daily gain less than the average for the group, whereas there was no marked difference in the gains of the other heifers.

(c) Environmental influences

Table 7 shows mortality in relation to breed of sire on Quest Ranch during the 1959–60 calving season.

Mortality amongst Brahman and Sussex sired calves was higher than that for the other breeds.

The relationship between month of birth and average daily gain until weaning on Quest Ranch from October 1958 to July 1960 is shown in Graph 1. Graph 1 also shows the number of calves born monthly during the same period. The number of deaths occurring amongst calves born during a particular month from September 1959 to July 1960 is superimposed on Graph 1.

Calves born between July and October 1959 had the greatest average daily rate of gain. Those born during January to March 1959 and February to April 1960 had the lowest rate of gain. The majority of the 1958–59 calf crop was born between December 1958 and May 1959 while the 1959–60 calves were born between October 1959 and March 1960.

Mortality prior to weaning in relation to month of birth amongst the Quest Ranch 1959–60 calves is shown in Table 8.

TABLE 3.—SIRE AND DAM COMPARISON ON 205 DAY WEIGHTS ON QUEST RANCH DURING 1959 AND 1960.

Sire	Sex	Number of Calves	*Average 205 day weight in lbs.	% Above or Below Average weight for sex group.
Aberdeen Angus**	♂	1	407.0	+10.2
	♀	2	298.5	—10.5
Afrikaner	♂	38	346.6	— 6.1
	♀	27	282.6	—15.3
Brahman	♂	177	381.8	+ 3.4
	♀	205	348.9	+ 4.6
Charollais**	♂	2	476.0	+28.9
	♀	1	410.0	+22.9
Drakensberger	♂	34	336.8	— 8.8
	♀	22	300.3	—10.0
Hereford**	♂	7	322.6	—12.6
	♀	5	311.8	— 6.6
Shorthorn**	♂	1	223.0	—39.6
	♀	2	306.5	— 8.2
Sussex	♂	9	356.9	— 3.3
	♀	12	275.3	—17.5
Nguni**	♀	1	240.0	—28.1
Unknown	♂	2	404.5	+ 9.6
	♀	4	320.3	— 4.0
Total	♂	271	369.2	
		281	333.7	
DAM				
Mature cows	♂	218	372.1	
	♀	225	336.7	
First calf cows	♂	53	357.3	
	♀	56	321.7	
Nguni	♂	14	357.6	
	♀	15	301.7	

*All calf weights converted to an average 205 day weight.

**Too few calves for comparative purposes.

TABLE 4.—Post-Weaning Weights and Gains on Dube Ridge and Quest Ranch.

Ranch	Year of Birth	Sex	Number of Calves	Average Weight in lbs.	Average Age in days	*Average Total Daily Gain in lbs.	Average Gain in lbs since Weaning	Days between Weaning and Post-Weaning weights	Average Daily Gain during this period
Dube Ridge	Summer 1958—1959	♂	86	547.0	616.5	0.766	169.1	285.0	0.593
		♀	84	467.8	618.6	0.635	115.6	285.0	0.406
Dube Ridge	Winter 1959	♂	42	476.9	378.7	1.061	23.8	86.0	0.277
		♀	45	424.7	387.1	0.903	15.8	86.0	0.183
Quest Ranch	1958—1959	♀	200	512.3	498.4	0.877	178.9	255.6	0.700

* = $\frac{\text{(Post-weaning weight — 75 lbs (estimated birth weight)}}{\text{Age in days}}$

TABLE 5.—POST-WEANING GAIN IN RELATION TO GROUPING AT WEANING.

Ranch	Year of Birth.	Grouping at Weaning	Sex	Number of Calves	Gain in lbs Between Weaning and Post-Weaning Weighing	Total Present Weight
Dube Ridge.....	Summer 1958-59...	1	♂	26	157.9	591.2
		1	♀	39	106.9	508.8
		2	♂	28	168.2	565.9
		2	♀	27	122.6	459.7
		3	♂	32	178.8	494.6
		3	♀	18	124.1	391.1
Dube Ridge.....	Winter 1959.....	1	♂	6	3.3	562.0
		1	♀	13	7.0	473.0
		2	♂	22	24.6	493.6
		2	♀	15	23.9	444.7
		3	♂	14	31.4	414.2
		3	♀	17	15.3	370.0
Quest Ranch.....	1958–59.....	1	♀	62	162.0	559.6
		2	♀	65	192.5	525.7
		3	♀	73	181.1	460.3

TABLE 6.—SIRE COMPARISON ON POST-WEANING WEIGHTS IN QUEST RANCH 1958 TO 1959 HEIFER CALVES.

Sire	Number of Calves	Average Weight in lbs.	Average Age in Days	*Average Daily Gain in lbs.	%Above or Below Average Gain for Group
Aberdeen Angus**.....	8	568.3	559.3	0.882	+0.6
Afrikaner.....	17	501.9	510.9	0.836	—4.7
Brahman.....	90	530.8	516.1	0.883	+0.7
Drakensberger.....	35	489.9	473.3	0.877	0.0
Hereford.....	44	491.8	471.2	0.885	+0.9
Sussex**.....	4	497.8	482.0	0.877	0.0
Nguni**.....	2	420.0	421.0	0.819	—6.6
Total.....	200	512.3	498.4	0.877	

$$* = \frac{\text{(Post-weaning weight—75 lbs (estimated birth weight)}}{\text{Age in days}}$$

**Too few calves for comparative purposes.

The highest percentage of mortality occurred amongst those calves born between November 1959 and March 1960.

The relationship between month of weaning and daily weight gain during the period immediately prior to weaning is given in Table 9.

Calves weaned from August to October 1960 had the lowest rate of gain during this period, while those weaned during January 1961 had the highest rate of gain.

The monthly rainfall for Quest Ranch from January 1959 to December 1960 and the average

TABLE 7

MORTALITY IN RELATION TO BREED IN QUEST RANCH 1959 TO 1960 CALVES

Sire	*Number of Calves*	*Number of Deaths*	*Percentage Mortality*
Aberdeen Angus*.....	5	0	0.0
Afrikaner.....	81	5	6.2
Brahman.....	442	38	8.6
Charollais*.....	5	2	40.0
Drakensberger.....	151	6	4.0
Hereford.....	18	1	5.6
Shorthorn*.....	4	0	0.0
Sussex.....	31	4	12.9
Nguni*.....	2	0	0.0
Unknown.....	6	1	16.7
Total.....	745	57	7.7

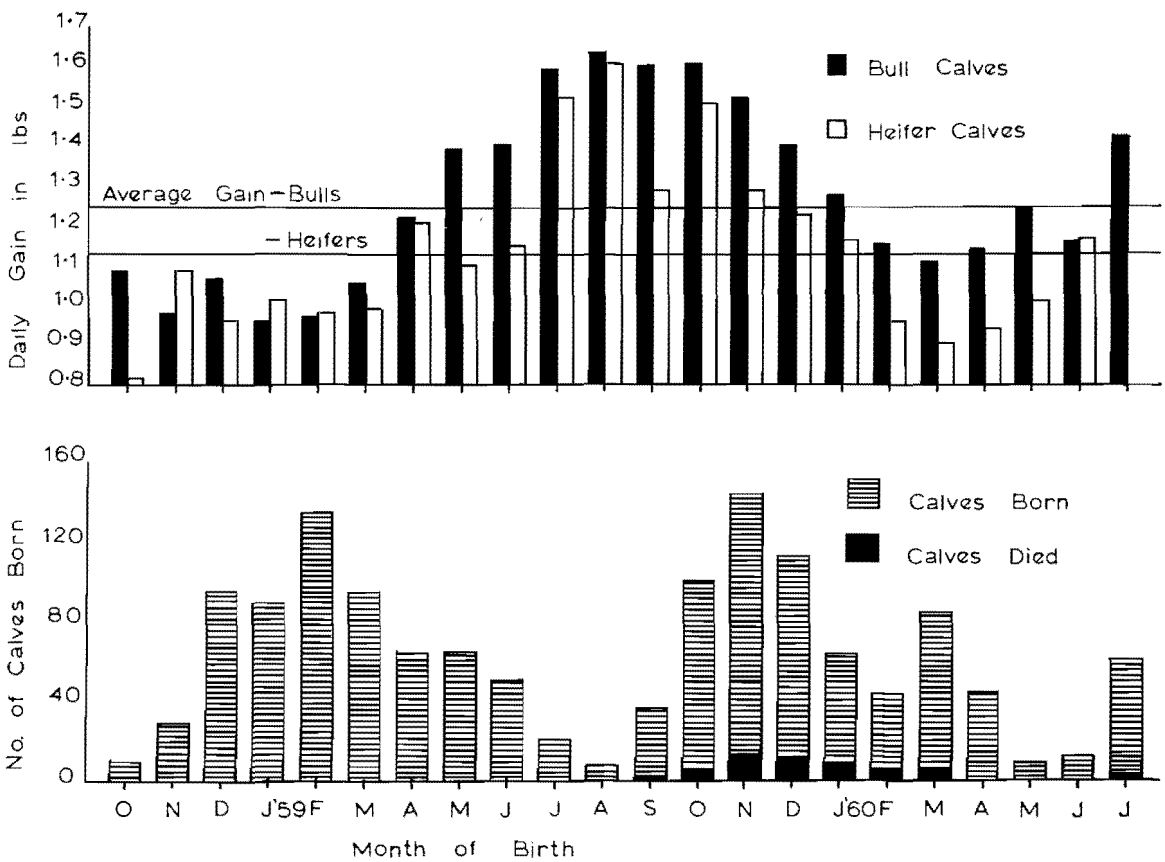
*Too few calves for comparative purposes

TABLE 8

MORTALITY IN RELATION TO MONTH OF BIRTH ON QUEST RANCH DURING 1959 TO 1960.

Month of Birth	*Number of Calves Born*	*Number of Deaths*	*Percentage of Mortality*
Sept. 1959.....	37	2	5.4
Oct.....	101	6	5.9
Nov.....	145	13	9.0
Dec.....	113	12	10.6
Jan. 1960.....	64	9	14.1
Feb.....	44	6	13.6
March.....	85	6	7.1
April.....	45	1	2.2
May.....	9	0	0.0
June.....	13	0	0.0
July.....	61	2	3.3
Aug.....	28	0	0.0

Graph 1.



Average daily weight gains and number of calves born on Quest Ranch.

TABLE 9.—WEIGHT GAIN PRIOR TO WEANING IN RELATION TO MONTH OF WEANING ON QUEST RANCH 1959 TO 1960 CALVES

Month weaned	Sex	Number of Calves	Number of Days prior to weaning	Average gain in lbs. in this period	Average Daily gain in lbs. in this period
July '60	♂	39	55.9	55.5	0.993
	♀	39	55.9	54.9	0.982
August	♂	73	49.4	15.1	0.306
	♀	75	47.5	20.6	0.434
September	♂	4	52.3	37.3	0.713
	♀	8	44.1	13.5	0.306
October	♂	31	58.5	21.8	0.373
	♀	42	57.0	24.5	0.430
November	♂	28	60.2	69.0	1.146
	♀	27	59.6	48.9	0.820
December	♂	49	29.0	45.4	1.566
	♀	55	41.4	46.5	1.123
January 1961	♂	9	42.0	78.7	1.874
	♀	4	49.3	77.8	1.578
Total	♂	233	48.4	38.4	0.792
	♀	250	50.3	36.0	0.716

maximum and minimum monthly temperatures from January to December 1959 are given in Table 10.

born during a particular season and the number of deaths occurring amongst these calves are also shown in Graph 2.

TABLE 10.—RAINFALL AND AVERAGE MONTHLY TEMPERATURES ON QUEST RANCH.

1959 Month	m.m. of Rain	Average Maximum Temperature °C	Average Minimum Temperature °C	1960 Month	m.m. of rain
January	73.1	31.5	20.0	January	(62.5)
February	59.8	32.0	20.0	February	(94.0)
March	6.0	32.1	18.8	March	(54.7)
April	6.5	32.2	17.4	April	(29.6)
May	82.2	28.4	10.8	May	(23.7)
June	0.0	26.6	8.5	June	0.0
July	0.7	26.1	9.3	July	12.5
August	23.1	26.9	9.5	August	12.0
September	30.5	27.9	13.0	September	51.8
October	90.3	28.7	16.9	October	(62.0)
November	65.1	28.3	18.2	November	152.5
December	62.6	31.2	19.7	December	188.3

Figures in brackets indicate average monthly rainfall.

Rainfall is concentrated in the summer months from October to April and the highest average temperatures are recorded during this period.

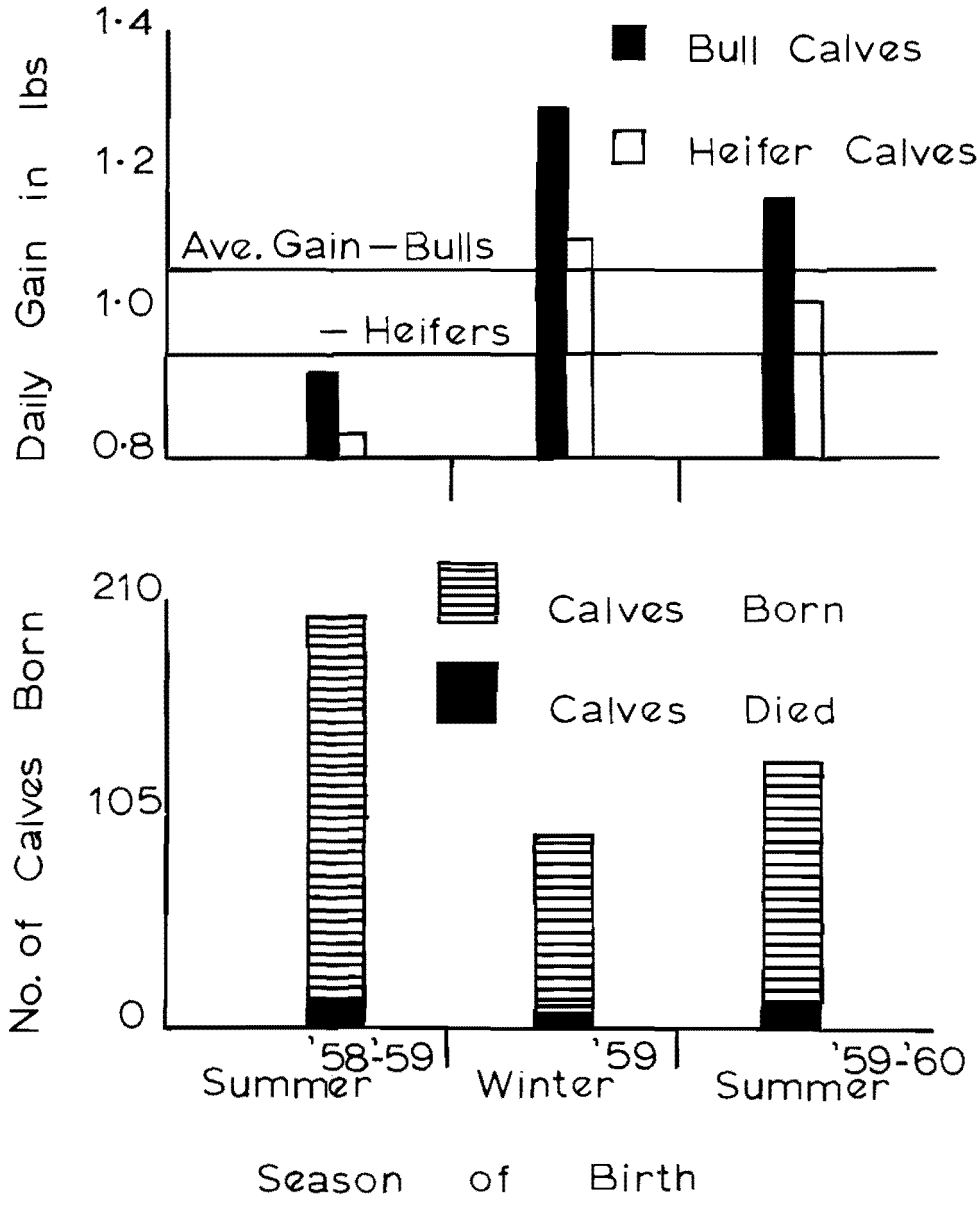
Graph 2 shows the relationship between season of birth and average daily gain until weaning on Dube Ridge. The number of calves

Calves born during the winter months had the greatest daily rate of gain, whereas the greatest number of calves was born during the summer months.

(d) Cow fertility

In a previous paper[6] February was shown to be the most fertile month on Quest Ranch.

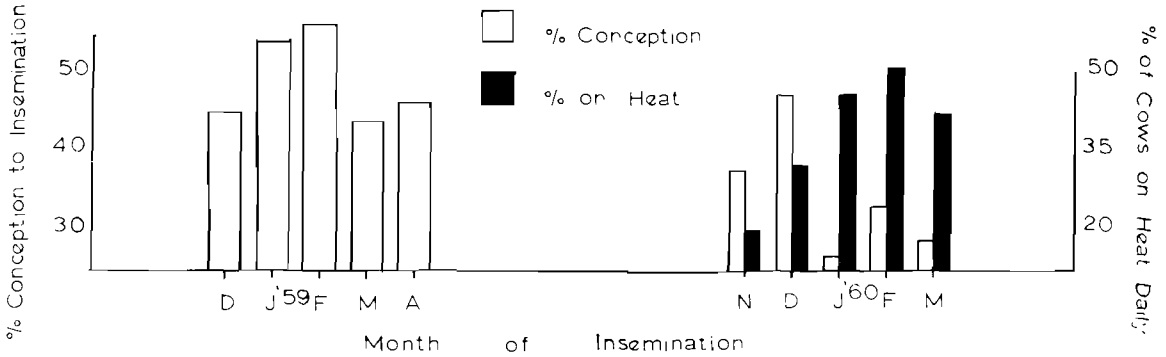
Graph 2.



Average daily weight gains and number of calves born on Dube Ridge.

The findings in that paper are now presented in Graph 3. The percentage of cows conceiving to insemination in relation to the month of the year is shown in Graph 3; so too the number of non-pregnant cows coming on heat daily, expressed as a percentage of the ideal of 1/21st. of the non-pregnant cows which should come on heat daily, during a particular month of the artificial insemination season.

Graph 3.



Percentage of cows conceiving to insemination and coming on heat daily on Quest Ranch.

The greatest percentage of cows conceive to insemination and the greatest percentage of non-pregnant cows come on heat daily, during February.

Discussion

(*a*) *Birth Weight*

No birth weights were taken on either ranch and hence no correlation between birth weight and weaning or post-weaning weights can be drawn. Bennett[1] and Tulloh[9], however, showed that there is no correlation between birth weight and weaning weight and it would appear that the omission of birth weights does not materially affect a performance testing programme.

(*b*) *Weaning Weight*

The greater average weaning weight of bull calves when compared with heifer calves is similar to the sex difference reported by Bennett[1], de Vree[3], Lasley, Day and Comfort[8] and Tulloh[9].

The weaning weights of calves born to first calf cows were 15 lbs lighter than those of calves from older cows. Consequently before any comparisons were made on a weight basis the former calves' weights were loaded by 15 lbs. Other workers have also recorded lower weaning weights for calves from first calf cows when compared with calves from older cows[1,2,3,8,9].

The calves from Nguni cows were so much lighter at weaning than the other calves that apart from a few exceptional cows the remainder of the Nguni herd was culled. This herd had already been heavily culled because of its low calving percentage.

As the animals on the two ranches and during the various calving seasons were not weighed at the same age, comparisons on total weight at weaning were not possible. For this reason average daily gains in pounds were calculated by subtracting 75 lbs. (estimated average birth weight) from the weaning weight and dividing the remainder by the age of the calf.

The smaller daily gains of the Dube Ridge summer 1958–59 calf-crop, when compared with the subsequent summer's calves, were probably due to the fact that the former calves were weighed at a greater age than the latter and had already weaned themselves by the time they were weighed. The majority of the Quest Ranch 1959–60 calves were born during those months favourable to high weight gains thus accounting for their greater weight than the previous season's calves which were born during the poorer months (Graph 1).

On Quest Ranch there were only adequate numbers of Afrikaner, Brahman, Drakensberger, Hereford and Sussex sired calves available for comparative breed studies; calves of the other breeds being too few in number for comparisons to be valid. In 1958–59 Hereford sired calves made the greatest daily gains and Afrikaner and Drakensberger sired calves the least. In 1959–60 Brahman sired calves were the heaviest at weaning while Afrikaner,

Drakensberger and Sussex sired calves were the lightest. The greater weights at weaning of Brahman X Afrikaner calves when compared with Afrikaner calves has also been recorded by Hayward[4].

The lesser weights of Afrikaner and Drakensberger sired calves are probably due to a lack of hybrid vigour as in many cases Afrikaner was mated to Afrikaner and Drakensberger to Drakensberger. The term Drakensberger is loosely used in Zululand and may include any black bovine which itself might be 75% Afrikaner. Any cross between one of these so-called Drakensbergers and an Afrikaner would therefore also be lacking in hybrid vigour.

(*c*) *Post-weaning Weight*

Weight difference, due to sex, increased markedly in favour of males during the post-weaning period. Daily gains of both males and females, however, decreased considerably when compared with pre-weaning daily gains. This can be ascribed to withdrawal of milk at weaning and adverse seasonal influences during the post-weaning period.

Brumby, Walker and Gallagher[2] showed a positive correlation between weaning weights and subsequent weights but no correlation between weaning weight and daily gain post-weaning. The present findings confirm their results but the heavier weaners in these trials actually made smaller gains post-weaning than did the lighter weaners. Nevertheless the total weights of the lighter weaners did not catch up to the total weights of the heavier weaners during the post-weaning period. Hence heavy weaning weights are a valid criterion for performance selection.

As weaning weight depends largely on the milk production of the cow[2,5], it can be assumed that the lighter weaners had had a poor milk supply and hence often weaned themselves before the actual time of weaning. When they were separated from their dams at weaning they had already adapted themselves to veld grazing, whereas the heavier weaners still relied on milk for part of their diet. For this reason the latter calves did not make the same post-weaning gains as the former because they had first to adapt themselves to a diet of veld grass only. Because weaning weight is a function of milk production selection of cows for breeding purposes on their progenies' weaning weight is important[5].

On Quest Ranch the influence of breed on post-weaning weights was in favour of the indigenous stock. Drakensberger sired calves now had overall daily gains equal to the average for the group and Afrikaner calves had improved from 10.6% below the average at weaning to 4.7% below the average. Hereford sired calves dropped from 8.6% above the weaning average to 0.9% above the post-weaning average. Post-weaning weight gains are largely due to the influence of the sire[2], and hence bulls should be selected on the performance of their progeny both at weaning and in the post-weaning period. The improvement of the indigenous breeds during the post-weaning period was probably due to their hardiness under ranching conditions and their better adaptation to the heat, which was often extreme.

(*d*) *Environmental Influences*

Quest Ranch

Lower mortality amongst the indigenous Afrikaner and Drakensberger sired calves partially compensated for their lower weaning weights. The highest mortality occurred in the January and February born calves and the majority of deaths occurred during January and February. The major causes of death in the 1959–60 calf-crop were Sweating sickness (17 deaths from January to April), Heartwater (13 deaths from April to June) and snake bite (10 deaths from October to March)[7]. Although Charollais sired calves had excellent weights at weaning, two of the five calves succumbed to Sweating sickness before reaching weaning age.

The winter and early spring born calves had greater daily weight gains and a lower mortality than the summer born calves. Unfortunately, however, there is practically an inverse ratio between the number of calves born during a particular month and the daily rate of gain for calves born during that month.

The above findings are closely related to monthly rainfall and temperature which are

responsible for grazing quality and the number and activity of the tick and snake populations. Both ranches are situated in a summer rainfall area and the grazing is at its best from November until May. Winter and spring born calves hence have the advantage of lush summer grazing, which also affects their dams' milk production, whereas summer born calves have only a few months of lush grazing while they are very young and are then weaned on to winter veld.

The effect of the grazing on weight gain immediately prior to weaning is strikingly illustrated in Table 9. The calves weaned on to winter grazing during August to October had very poor weight gains whereas those weaned on to summer veld from November to January made progressively larger daily gains.

Dube Ridge

Here too the winter born calves made markedly better daily gains than the summer born calves. Once again there was nearly an inverse ratio between the number of calves born during a particular season and the daily weight gains made by those calves, (Graph 2).

Bennett[1] and Lasley *et al*[8] also found that month or season of birth influence weaning weight and daily gains. For this reason it is necessary to divide a calf-crop into groups according to the time of their birth and only calves born within a particular period should be compared with one another and not with the rest of the calf-crop as a whole.

(*e*) *Cow Fertility*

As previously stated the figures for this section of the work have been borrowed from a previous publication[6]. Cow fertility is markedly influenced by grazing quality, increasing until the height of the summer in February and then decreasing again as the grazing becomes poorer. Fortunately the calves conceived during February are born in November and this group of calves still makes reasonable daily gains until weaning.

The Quest Ranch calf-crop for 1958–59 was abnormally late as the artificial insemination season had only commenced during February and continued until June, whereas the subsequent seasons commenced in October or November and continued until March or April respectively. Hence this particular calf-crop was born during the months when the lowest daily gains were recorded, thus accounting for its lighter average weight than the following season's calf-crop.

The reason for the poor conception rates during January, February and March 1960 was that poor quality semen had been used during these months. However, the percentage of non-pregnant cows coming on heat daily during February 1960 clearly established this month as the nucleus for any breeding season on Quest Ranch.

Both on Quest Ranch and Dube Ridge culling for infertility was high. Approximately 200 cows were culled annually on Quest Ranch because of infertility.

(*f*) *General*

The discrepancy between the number of calves born, the number weighed at weaning and the number weighed post-weaning was due to deaths and to calves not being identified as they had lost their plastic tags or their tatooed numbers had smudged.

Three Aberdeen Angus bulls sired approximately 70 calves in their first year on Quest Ranch, 17 in the second year and five in the third year. These three bulls were culled as they were unable to adapt themselves to the veld grazing and the heat and spent most of the day lying in the Mkuze river unable to keep up with the cow herds. A Hereford, a Simmen Thaler and an Afrikaner bull were culled for infertility when repeated semen examinations failed to show any live spermatozoa. A Hereford bull died from the after effects of Three-day-stiff-sickness and two Afrikaner bulls from snake bite.[7]

The Brahman sired bull calves in the Quest Ranch 1958–59 calf-crop were particularly unmanageable and about 15 of these were culled.

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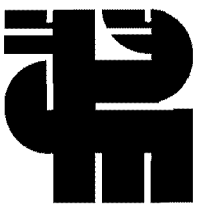

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BIURET AS A SUPPLEMENT TO ROUGHAGE RATIONS FOR SHEEP AND CATTLE

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Received for publication June, 1964

SUMMARY

Feeding trials over two winter periods were conducted to compare the value of crude biuret, urea and mixtures of these two chemicals fed as sources of supplementary nitrogen for sheep and cattle on hay diets.

The addition of non-protein nitrogen to good quality hay did not improve the liveweight gains of cattle and sheep and did not influence the voluntary intake of roughage by sheep. On a low quality hay diet the animals receiving N.P.N., irrespective of source, maintained weight while the controls lost significant amounts of weight. Hay consumption of sheep was increased significantly by the addition of crude biuret and mixtures of crude biuret and urea but not by urea alone. Wool growth and quality were not affected by treatment.

The practical significance of the results is discussed.

INTRODUCTION

Biuret, a pyrolysis product of urea, has been studied over a number of years as a source of non-protein nitrogen (N.P.N.) for ruminants. Published work shows a wide variation in the results obtained but the main factor that always emerges is that biuret is not toxic. When comparing biuret with urea numerous workers have found it to be inferior in promoting cellulose digestion, bacterial growth and general efficiency as a supplier of nitrogen to the ruminant[1,2,3,4,5]. Others have reported that biuret is as efficient as urea and that the nitrogen from biuret is retained by sheep in amounts similar to that of urea[6,7,8].

The discrepancy between the results might be explained by the findings of McLaren *et al*[5,9] who produced evidence to show that the adaptation period for biuret was from 4 to 5 weeks and only after that period were the micro-organisms fully adapted to the use of biuret. Because of this long period of adaptation Hatfield *et al*[10] put forward the hypothesis that a combination of urea and biuret might be superior to pure biuret.

In most of the work published the basal rations have contained at least 50% concentrate feed. There is little information available on the efficiency of biuret as a source of N.P.N. for ruminants where low protein bulk feeds constitute over 80% of the total daily intake. In the summer rainfall areas of Southern Africa the roughage available for winter feed is predominantly of this type, but it has been shown that it can provide maintenance rations when supplemented with urea[11].

Trials were commenced in 1962 to evaluate biuret as a source of N.P.N. for cattle and sheep fed mainly on a roughage diet.

TRIAL 1 — WINTER SEASON 1962

METHODS

Cross-bred German Merino maiden ewes aged 12 months were wintered in two groups (10 ewes per group) on the following two rations per head per day:

Ration A — (Control)

Veld hay (2.9% C.P., 0.07% P_2O_5, 39.0% C.F.)
110g yellow maize meal
10g Pagura* (Sugar content 85%).
10g Salt
10g Dicalcium phosphate

Ration B — (Biuret)

Veld hay (2.9% C.P., 0.07% P_2O_5, 39.0 % C.F.)
110g yellow maize meal
10g Pagura* (Sugar content 85%).
10g Salt
10g Dicalcium phosphate
21g Biuret (92% biuret containing 40.9% nitrogen).

*Pagura — crude sugar product produced by Hulett & Sons containing 85% sugar.

Each group was penned where they had free access to water and hay but the supplements were fed individually and were readily taken by all the sheep. The unconsumed hay was weighed daily.

Results

The difference between the weights lost by the two groups was highly significant ($P = 0.01$). The difference between the fleece weights was not significant.

Table 1.—Winter weights of 12 month old maiden ewes over a feeding period of 9 weeks.

Group	No. of Animals	Av. Initial wt. (lb.)	Av. Final wt. (lb.)	Av. Gain or loss (lb)	Av. gain or loss/day (lb)	Av. Fleece wt. (lb)
A (Control)	10	76.3	65.4	—10.9	—0.17	3.66
B (Biuret)	10	76.0	74.2	—1.8	—0.03	3.89

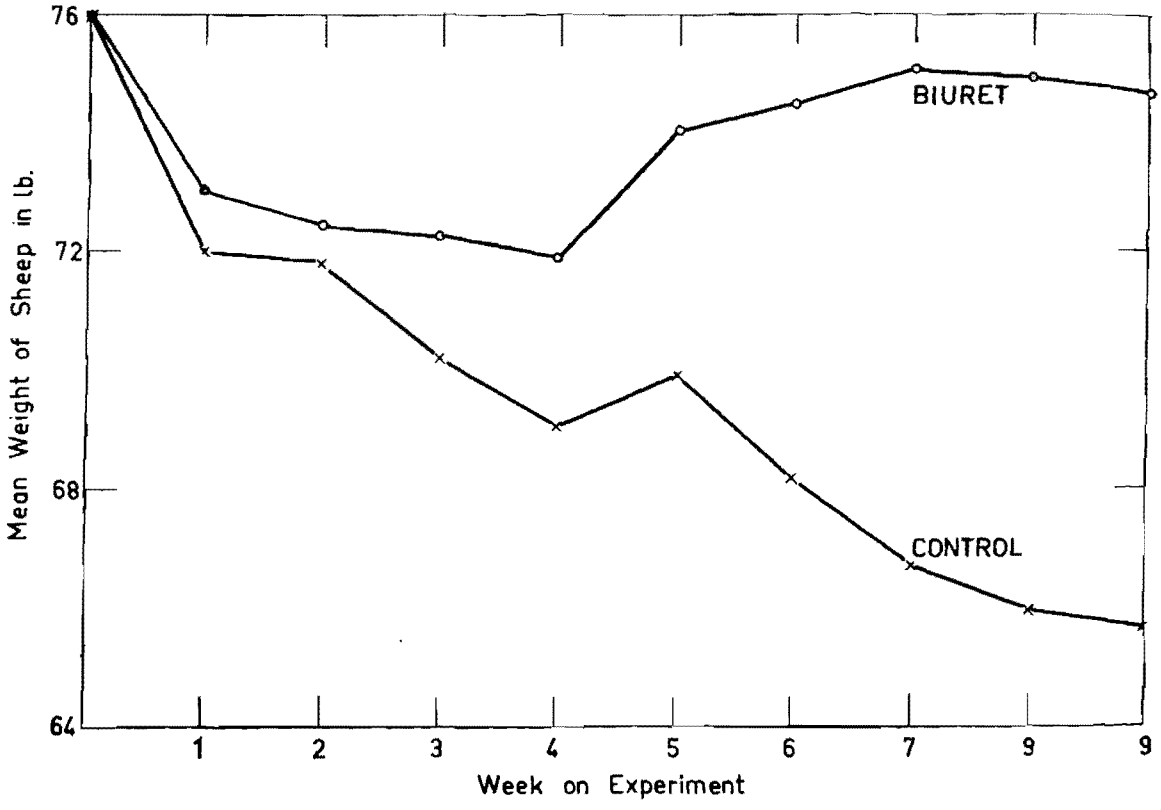

FIG. 1

SHEEP GROWTH CURVES

The quality of the wool from Group B (biuret) indicated that growth was normal while that from the control group showed signs of undernutrition.

The average hay consumed per head per day for the Control Group was 2.0 lb. and for the Biuret Group was 2.27 lb.

Conclusions

The addition of biuret in combination with limited carbohydrate, to a low protein bulk diet produced a maintenance diet for sheep.

The characteristic delay in the liveweight response to biuret feeding is clearly evident from Figure 1.

Trial 2 — Winter Season 1963

Methods

One hundred cross-bred German Merino maiden ewes aged 15 months were penned and fed teff hay for 4 weeks prior to the commencement of the experiment. They were then divided into nine comparable groups of 10 ewes each on the basis of liveweight.

The sheep had free access to the basal ration of hay which was weighed and fed twice daily. The unconsumed hay was weighed and removed before each feed. The supplements were fed once daily. The sheep were fed teff hay for a period of 42 days and then the teff hay was replaced by a poor veld hay for the next 112 days. Individual wool samples from a 9 inch square cut off the shoulder were taken at the conclusion of the experiment for quality evaluation. The sheep were regularly drenched with a vitamin A concentrate and dosed against internal parasites.

Table 2

Composition of hay on a dry matter basis

	C.P.	P_2O_5	*C.F.*
Phase 1 (Teff).....	7.88	0.17	29.29
Phase 2 (Veld grass).....	4.12	0.19	39.07

The crude biuret had an average composition of 64% biuret, 5% urea, 3% cyanuric acid, 3% triuret and 25% moisture and the total nitrogen content was 30% (as fed). The urea contained 46% nitrogen.

The crude biuret, urea and the biuret:urea mixtures supplied 6.4g of additional nitrogen per sheep per day.

The differences between final weights, weight gains and hay consumption were not significant.

The performance of the sheep in Group 2 (Control B) was significantly inferior ($P = 0.05$) to those of groups 3 to 9, but there was no difference between any of the groups receiving supplements (3 to 9). Groups 2 and 4 consumed significantly ($P = 0.01$) less hay than the other groups but the difference between the former two groups was not significant. It should, however, be noted that the average weekly intake of Group 2 fell from 109.0 lb. during the first three weeks of Phase 2 to 89.5 lb. during the final three weeks. Comparable figures for group 4 were 92.2 lb. and 104.8 lb.

The average water consumption of all groups was similar and varied between groups from 0.32 to 0.37 gallons per head per day.

Table 3.—Composition of supplements fed per sheep per day (grams)

Group		Yellow Maize meal	Crude Biuret	Urea	Dicalcium Phosphate Lick	Salt Lick
Control A	1.....	—	—	—	8.6	8.6
Control B	2.....	95.0	—	—	8.6	8.6
Biuret	3.....	95.0	21.5	—	8.6	8.6
Urea	4.....	95.0	—	14.0	8.6	8.6
50:50	5.....	95.0	10.7	7.0	8.6	8.6
60:40	6.....	95.0	12.9	5.6	8.6	8.6
40:60	7.....	95.0	8.6	8.4	8.6	8.6
70:30	8.....	95.0	15,0	4.2	8.6	8.6
30:70	9.....	95.0	6.4	9.8	8.6	8.6

Results

Table 4.—Liveweight gains of ewes over 42 days: basal ration teff hay (Phase 1)

Group	No. of Animals	Average Initial Wt. (lb.)	Average Final Wt. (lb)	Average Gain (lb.)	Average Hay consumption (lb. per head per day)
1.....	10	72.8	74.4	1.6	2.42
2.....	10	73.6	80.7	7.1	2.43
3.....	10	73.4	79.3	5.9	2.47
4.....	10	73.3	77.0	3.7	2.22
5.....	10	72.6	79.0	6.4	2.40
6.....	10	73.5	81.8	8.3	2.46
7.....	10	72.9	78.9	6.0	2.55
8.....	10	72.6	79.3	6.7	2.42
9.....	10	72.5	80.7	8.2	2.62

Table 5.—Liveweight gains and losses over 112 days. Basal ration veld grass hay (Phase 2).

Group	No. of Animals	Average Initial Wt. (lb.)	Average Final Wt. (lb.)	Average Gain or Loss (lb.)	Average Hay consumption (lb. per head per day)
1.....	10	74.4	65.8*	—8.6*	1.44*
2.....	10	80.7	70.6	—10.1	1.38
3.....	10	79.3	81.7	+2.4	1.59
4.....	10	77.0	80.4	+3.4	1.40
5.....	10	79.0	81.6	+2.6	1.62
6.....	10	81.8	81.0	—0.8	1.59
7.....	10	78.9	79.0	+0.1	1.55
8.....	10	79.3	81.7	+2.4	1.51
9.....	10	80.7	80.9	+0.2	1.56

*Note.—Group 1 was withdrawn from the trial after 56 days.

A comprehensive examination of individual wool samples failed to reveal any distinct differences due to treatment.

Discussion

The addition of supplements containing crude biuret, urea and mixtures of these two compounds to a basal ration of good quality teff hay did not exert a significant effect on liveweight or hay consumption. This observation is in agreement with the findings of Williams *et al* and others[12,13,15].

On the basal ration of poor veld grass hay which was low in nitrogen and high in fibre, the sheep maintained weight when supplemented with N.P.N. in addition to maize meal while the control group recorded a significant loss of 10.1 lb. per head.

There were no apparent differences in the value of crude biuret, urea or the mixtures as sources of N.P.N. under the conditions of the experiment. However, it is apparent that the biuret group were conditioned to the use of this compound during Phase 1 when no response to N.P.N. was obtained and that at the commencement of Phase 2, the microflora had attained an adequate level of biuret splitting activity to eliminate any time-lag in liveweight response.

Increased voluntary intakes of low quality roughage by ruminants under the stimulus of added nitrogen have been widely reported[12,13,14,16,17]. All the nitrogen supplemented groups with the exception of group 4 (urea) in this trial conformed to this pattern. It is possible that the relatively rapid consumption of the supplement containing 9.7% urea caused a temporary inhibition of rumination or even temporary ruminal stasis due to an increase in the pH and ammonia concentration of the ruminal fluid in the urea fed group[16].

Liveweight differences were not reflected in wool growth or quality due to the high variability in fleece types within groups.

Trial 3 — Winter Season 1963

Methods

24 Afrikaner Steers and 36 Sussex-Afrikaner steers aged 24 months were penned and fed teff hay for one week. They were then divided into 4 comparable groups according to liveweight and breed.

The basal ration was hay *ad lib.* and a lick containing equal parts (by weight) of dicalcium phosphate and salt *ad lib.* The investigation was divided into two phases by the use of two types of hay.

Table 6

Composition of hay on a dry Matter Basis

	C.P.	P_2O_5	*C.F.*
Phase 1 (Teff).....	7.88	0.17	29.29
Phase 2 (Veld grass).....	4.15	0.27	37.73

The crude biuret and urea were the same products used in Trial 2.

In Phase 1, teff hay was fed for 70 days and in Phase 2 veld hay for 84 days.

Table 7 — Composition of supplements

Group 1 4.6 oz. crude biuret in 1 lb. molasses per head per day

Group 2 3.0 oz. urea in 1 lb. molasses per head per day

Group 3 4.1 oz. of a mixture of crude biuret and urea (70% of the nitrogen supplied by crude biuret and 30% by urea) in 1 lb. molasses per head per day

Group 4 1 lb. molasses per head per day

Supplements 1, 2 and 3 each supplied 39.2 g N per steer per day.

Statistical Analysis

Curves for the growth of each steer were determined from the differences between successive weekly weights. Using the areas under these curves as variates, the Kruskal-Wallis method of analysis of variance was applied and where the test statistic was significant, the Mann-Whitney U-test was used.

The difference in weight gains between the control and the other three groups did not reach significance.

The difference between the weight gained by both the Afrikaner and Sussex-Afrikaner steers in Groups 1, 2 and 3 and the weight lost by their counterparts in Group 4 (Control) was significant ($P = 0.05$). There was no significant difference between the nitrogen supplemented groups.

Conclusions

The results obtained with steers were essentially in agreement with those recorded with sheep, viz. no significant increase in weight gains when N.P.N. supplements were added to a basal hay ration of a high quality (Phase I). When the hay fed was of poor quality significant differences in utilization, as reflected by weight gains were achieved by adding a N.P.N. supplement (Phase 2). All three N.P.N. supplements used produced similar results. Here again it is stressed that the animals in the biuret group were subjected to a period of adaptation to biuret for 10 weeks prior to receiving low quality hay.

General Conclusions

In the trials reported here biuret has been shown to be a suitable substitute for urea.

To date research has been largely confined to investigations in which biuret has been tested under conditions considered suitable for urea

Results

Table 8.—Liveweight gains of steers over 70 days—Basal ration teff hay (Phase 1)

Group		No. of Animals	Average Initial Wt. (lb.)	Average Final Wt. (lb.)	Average gain per steer (lb.)
(Biuret)	1.....	15	754.3	840.7	86.4
(Urea)	2.....	15	757.0	843.3	86.3
(Buiret: Urea)	3.....	15	757.0	839.7	82.7
(Control)	4.....	15	760.0	826.0	66.0

TABLE 9.—LIVEWEIGHT GAINS OF STEERS OVER 84 DAYS: BASAL RATION VELD GRASS HAY (PHASE 2)

Group		No. of Animals	Average Initial Wt. lb.	Average Final Wt. lb.	Average gain per steer lb.
(Biuret)	1.....	15	840.7	853.9	+13.2
(Urea)	2.....	15	843.3	854.6	+11.3
(Biuret: Urea)	3.....	15	839.7	859.8	+20.1
(Control).	4.....	15	826.0	780.9	—45.1

feeding. The two forms of N.P.N. differ in their properties and further studies are required to determine the factors which affect biuret utilization specifically. It is not unlikely that this information could result in biuret playing a major role in raising the level of livestock production economically in those areas where acute protein deficiencies exist and where management practices restrict the use of urea because of possible toxic effects.

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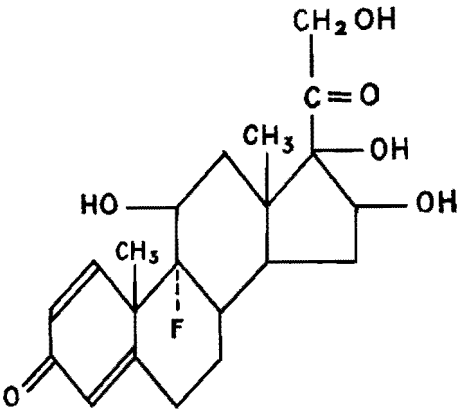

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OBSERVATIONS ON A BIURET-CONTAINING LICK AS A NITROGEN SUPPLEMENT TO LOW QUALITY ROUGHAGE

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Received for publication June, 1964

SUMMARY

A lick containing 3 parts of crude biuret, 2 parts of bonemeal and 1 part of salt was fed *ad lib.* to sheep and cattle. The basal rations were low in nitrogen and high in fibre. Liveweight changes and lick consumption were measured.

The results indicate that the voluntary intake of the lick was sufficient to meet the nitrogen requirements for maintenance of body weight of sheep and cattle on poor quality roughage without the addition of a readily fermentable carbohydrate.

INTRODUCTION

Studies on the use of urea as a supplement for ruminants receiving basal diets which are low in nitrogen and high in fibre, have shown that the presence of limited amounts of readily available carbohydrate in the rumen increases the efficiency of utilization of this compound[1,2,3,4]. The provision of the carbohydrate with urea increases the rate of protein synthesis and thus reduces the loss of nitrogen in the form of ammonia through the rumen wall[1]. The potential wastage of nitrogen, the danger of urea toxicity and the unpalatability of urea in the absence of a carrier, render the feeding of supplements containing high concentrations of urea both dangerous and undesirable.

Biuret is not toxic to ruminants[5,6] presumably due to the slower release of ammonia from it in the rumen and local observations have shown it to be more palatable than urea.

Investigations were initiated to determine the liveweight response of cattle and sheep to licks containing crude biuret fed in combination with low quality roughage and without the addition of readily fermentable carbohydrates.

TRIAL 1

METHODS

Ten maiden ewes were fed a basal ration of veld hay (4.1% C.P. and 39.1% C.F.) plus a lick of salt and dicalcium phosphate for a period of 49 days. Biuret was mixed with bonemeal and salt in the proportions of 3:2:1 by weight and fed in place of the salt: dicalcium phosphate lick for the next 77 days. The biuret was then removed from the lick and only a 2:1 bonemeal, salt lick was fed for a further 42 days. During the three periods the sheep had free access to both hay and lick. Prior to the trial the sheep had never been fed biuret.

TABLE 1.

FEEDING PERIODS AND COMPOSITION OF LICKS BY WEIGHT.

Period	*Duration (days)*	*Composition of lick*
1	49	1 dicalcium phosphate: 1 salt.
2	77	3 crude biuret: 2 bonemeal: 1 salt
3	42	2 bonemeal: 1 salt.

Composition of crude biuret: biuret 64%; urea 5%, cyanuric acid 3%, triuret 3%, moisture 25%, total nitrogen 30%.

Results

Table 2.—Liveweight gains and losses of sheep on a biuret lick trial.

Average weight per sheep	Period 1 (no biuret)	Period 2 (biuret)	Period 3 (no biuret)
Initial weight (lb.)	72.1	65.8	68.9
Final weight (lb.)	65.8	68.9	65.1
Gain or loss (lb.)	—6.3	+3.1	—3.8
Gain or loss per head per day (lb.)	— .128	+ .040	— .090
Lick consumption (g per head per day)	2.8	54.0	41.0
No. of animals	10	10	10

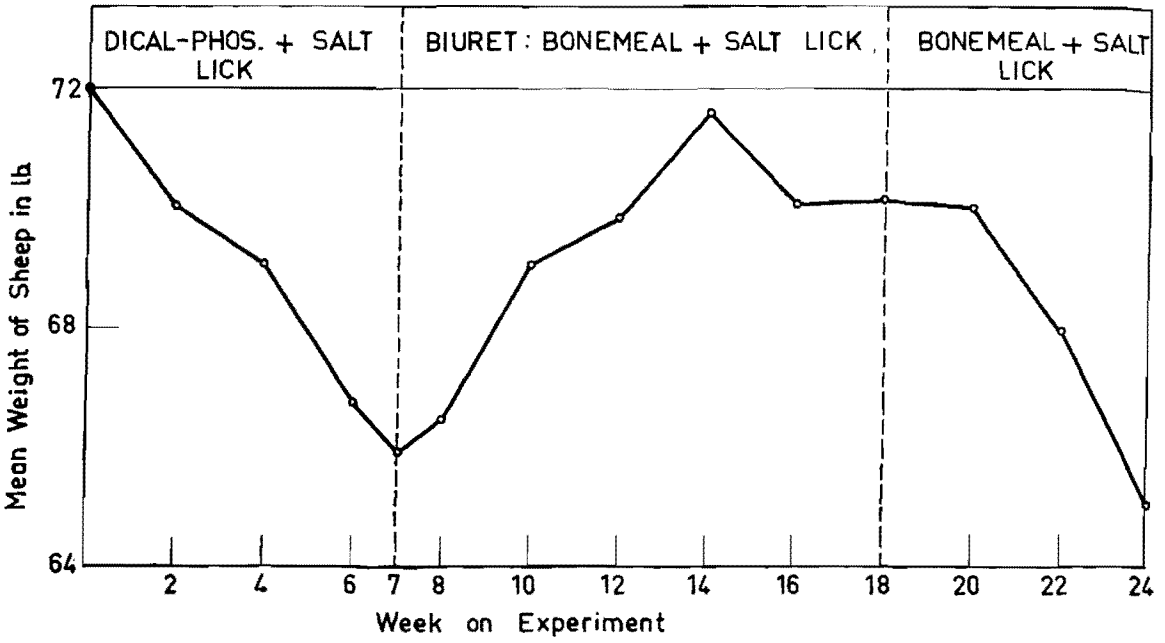

FIG. 1.

SHEEP GROWTH CURVES

Conclusions

The mixture of crude biuret, bonemeal and salt was palatable to sheep.

The addition of crude biuret to the lick containing bonemeal and salt provided a supplement on which sheep receiving poor hay, gained weight. A rapid weight loss occurred when either dicalcium phosphate or bonemeal were fed alone with salt and the hay. It appears that the crude biuret consumed at the rate of 27.0 g (8.1g N) per sheep per day was utilized effectively by the microflora of the rumen with no energy source other than the breakdown products from the basal ration.

The liveweight response of the sheep to crude biuret was immediate (within the first week) and continued with one slight check for 8 weeks. This rapid and sustained response is contrary to the expected performance of unadapted sheep which generally require a conditioning period before responding to biuret feeding [7,8].

TRIAL 2

METHOD

Ten pairs of beef cattle ranging from weaner calves to mature steers were selected according to liveweight, age and sex. One member of each pair was randomly assigned to one of the treatments. The cattle were weighed fortnightly during the first 8 weeks of the trial and weekly thereafter. The animals were unadapted to biuret feeding at the commencement of the trial.

The basal ration was maize cob husks (2.31% C.P. and 42.3% C.F.) *ad lib.*, 20 lb. maize — babala silage per head per day and limited veld grazing. The trial ran for 98 days.

The licks were fed *ad lib.* and the residues weighed weekly. The crude biuret was the same product used in Trial 1. The composition of the licks was as follows:—

Group 1 2 parts bonemeal: 1 part salt (by weight)
Group 2 3 parts crude biuret: 2 parts bonemeal: 1 part salt (by weight)

TABLE 3

LIVEWEIGHT GAINS AND LOSSES OF CATTLE ON A BIURET LICK TRIAL.

Average weight per head	*Group 1 (Control)*	*Group 2 (Biuret)*
Initial Weight (lb)	977.5	979.5
Final Weight (lb)	948.5	984.5
Gain or loss (lb)	— 29.0	+ 5.0
Gain or loss per head per day (lb)	— 0.30	+ 0.05
Lick consumption (g per head per day)	46.5	71.4
No. of animals	10	10

The differences between the weight changes of the two groups was tested by "Students method of paired differences" and was significant ($P = 0.05$).

FIGURE 2

Lick intakes were extremely variable. Group 1 (Control) ranged from 12.7 to 56.5 g. per head per day and group 2 (biuret) consumed 127 g. per head per day during the first week and their subsequent intake varied between 32.9 g. and 95 g. per head per day.

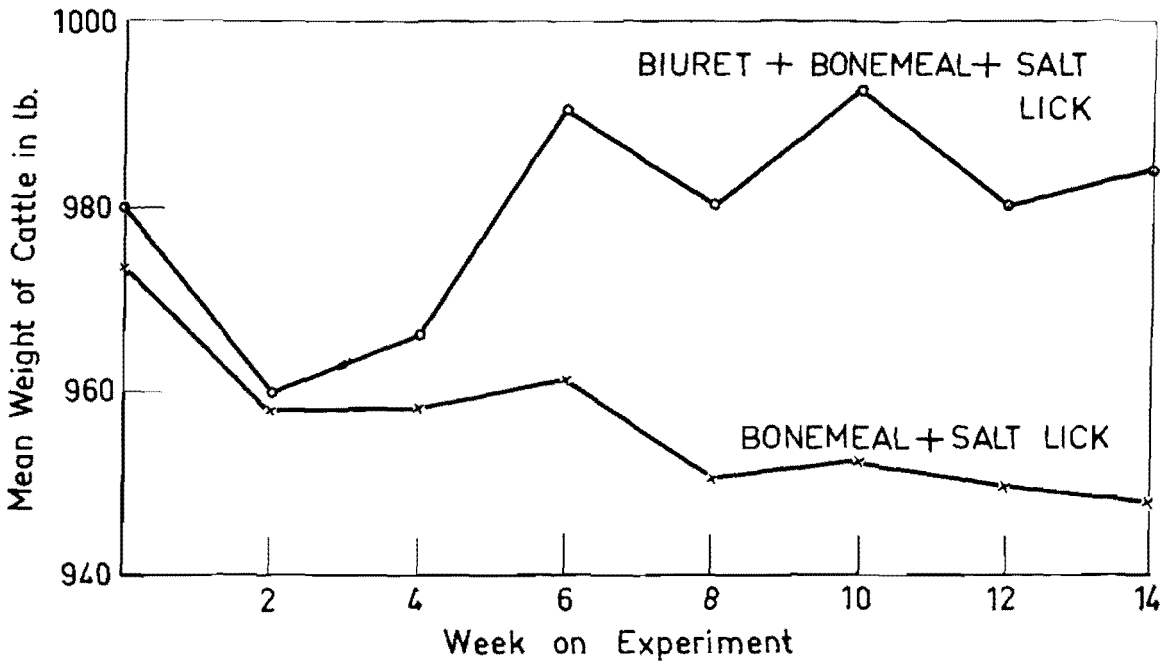

FIG. 2

CATTLE WEIGHT CURVES

Conclusions

The conclusions are generally in agreement with those drawn from Table 1 with sheep and may be summarised as follows:—

The addition of biuret to a bonemeal and salt lick provided a supplement which when fed in combination with low quality dry roughage and silage, supplied a maintenance ration.

The liveweight response of the cattle, unadapted to biuret feeding, was slower than that recorded for sheep, and only became evident 4 weeks after the trial commenced.

The lick was palatable to cattle of varying ages.

The average daily intake of crude biuret was 35.7 g. which provided 10.7 g. nitrogen. This amount is considerably lower than the usual levels of nitrogen supplied by urea in supplements fed to produce maintenance diets from roughage of the type used in this trial[9].

General Conclusions

The observations recorded in this paper suggest that biuret enriched salt:bonemeal licks could play a role in reducing weight losses of cattle and sheep in areas where the animals only have access to low quality bulk feeds during the winter months. It is obvious that further information is required on the application of this method of supplementary feeding to ruminants in different environmental areas before any recommendations can be made.

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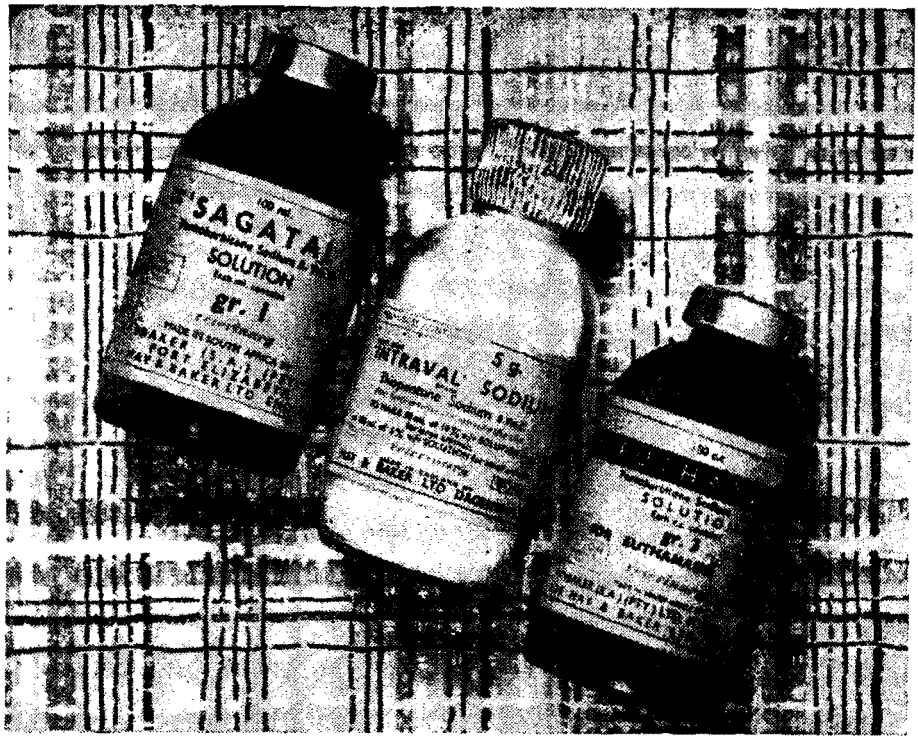

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A DESCRIPTION OF SOME STAGES IN THE LIFE CYCLE OF AN *EIMERIA* SP. FOUND IN THE IMPALA, *AEPYCEROS MELAMPUS* (LICHTENSTEIN, 1812)

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Received for publication July, 1964

SUMMARY

1. An *Eimeria* spp. is described from the impala, *Aepyceros melampus*, of the Republic of South Africa.

2. The oocysts of this *Eimeria* spp. are similar to those of *Eimeria impalae* Prasad and Narayan, 1963. It is therefore suggested that it be regarded as *E. impalae* unless further investigations on the relationship of the two parasites should reveal sustained and significant differences.

3. The morphological features of the oocysts of *E. impalae*, as described by Prasad and Narayan, are therefore extended by those recorded below.

4. The endogenous stages of this parasite were very numerous in histological sections of the small intestine but were rare in the caecum and colon. They were all in the sexual phase of the life cycle, which is apparently the pathogenic phase.

INTRODUCTION

The relevant history, epizootiology and pathology associated with several outbreaks of coccidiosis in impala in the Transvaal Province have been dealt with in another publication[1]. In this article the various stages in the life cycle of the *Eimeria* sp. responsible for the outbreaks are described.

MATERIAL AND METHOD

The following material was received for examination:—

1. Faecal pellets preserved in 2 per cent aqueous potassium dichromate solution from young impala involved in an outbreak of coccidiosis at Loskop Dam Nature Reserve during April and May 1963. These specimens were obtained prior to the first death[1].

2. Specimens consisting of faeces preserved in 2 per cent potassium dichromate solution, smears made from mucosal scrapings of affected areas of the small intestine and sections for microscopical study of the affected portions of the small and large intestines, were obtained at the autopsy of an impala which had died during the 1963 outbreak at Loskop Dam Nature Reserve.

3. Sections for histological study were prepared from portions of small intestine from six other impala that died during the same outbreak. As no faeces specimens were available from these animals, the 10 per cent formalin which had been used for preserving the gross material was collected from each of the six bottles and was centrifuged.

4. Identical specimens as in (3) above were obtained from a single impala that had died on the Hans Merensky Nature Reserve in June 1962[1].

5. Faeces collected at the Loskop Dam Reserve in June 1957 from a group of young impala amongst which deaths from coccidiosis had occurred, was preserved in 2 per cent potassium dichromate solution, and was processed using the techniques described below.

The oocysts in faeces specimens preserved with potassium dichromate solution were concentrated for microscopical study by the glycerine flotation method using standard procedures. The sediment obtained by centrifugation of the formalin used for preserving the gross material was resuspended in water and a sufficient number

of oocysts was obtained for study, following glycerine flotation. Mucosal smears were fixed with methyl alcohol and were stained with Giemsa. Sections of the affected portions of the intestine for histological study were prepared from material fixed in 10 per cent formalin. Sections were cut three microns in thickness and were stained by the haematoxylin-eosin method and the Giemsa method. Faeces preserved in 2 per cent potassium dichromate solution was placed in petri dishes after first removing coarse particles, and was left to stand for two to three weeks with occasional replenishment of water in order to allow the oocysts to sporulate.

Illustrations of the endogenous stages of other *Eimeria* sp. were consulted during this study in order to assist in the identification of structures other than macrogametocytes and oocysts[2,3]. This measure was considered essential because some of the immature microgametocytes could have been mistaken for schizonts.

Results

Large numbers of morphologically identical oocysts were found in all the faeces samples examined during the 1963 outbreak at the Loskop Dam Reserve. This indicated without doubt that a single *Eimeria* species was involved. Oocysts of the same morphological type were demonstrated in the impala that died on the Hans Merensky Nature Reserve in 1962, and, in the 1957 outbreak of coccidiosis at Loskop Dam Reserve. A second type of oocyst was also present in the 1957 outbreak in small numbers, in addition to the coccidium under review. Due to inadequate data this second type of oocyst will not be described further.

The endogenous stages of the parasite were studied in the 1962 and 1963 outbreaks. In both instances they were found to be morphologically identical.

The morphological features of the various stages of the coccidium were as follows:—

Oocysts (*Fig.* 1).

The oocysts were large and relatively robust, and varied in size from 33 to 45 microns in length and from 22.5 to 30.8 microns in width. The average size of 128 oocysts was 38.95 by 26.68 microns. The average length-width ratio was 1.5. In shape, the oocysts were ellipsoidal with the micropylar end slightly flattened and more tapered than the opposite end. The micropyle was formed by a thinning of the oocyst wall. Because this transition was gradual it was difficult to determine the size of the micropyle accurately, and it was found to be about six microns in diameter. No polar cap was observed. With the exception of formalin fixed specimens which were generally lighter in colour, the oocysts were a yellow or yellowish-brown colour. Some oocysts which had been preserved in formalin for long periods were completely colourless, and it is assumed that this phenomenon was caused by a bleaching or leaching effect of the preservative. The oocyst wall was yellowish-brown in colour and was about two microns in thickness.

No attempt was made to determine the exact sporulation time of the oocysts. Sporulation was apparently complete after two weeks. There was no oocystic residuum. Sporulated oocysts contained four sporocysts, which were elongated ovoid structures with inconspicuous Stieda bodies (Fig. 1). Ten sporocysts measured 18 to 21 by 7.5 to 9 microns in size. Sporocystic residual granules were rather loosely scattered, and were probably responsible for obscuring the sporozoites from view, as these could not be distinguished with certainty. Two large, and one or two smaller clear globules could be clearly seen in each sporocyst. The large globules lay at either end of the sporocyst, thus indicating that each one was probably situated in a sporozoite.

Endogenous Stages

The endogenous stages were studied in intestinal sections and smears. The sections were found to be more suitable for morphological studies on the microgametocytes than were the smears.

1. *Schizonts.*

No structures identifiable as schizonts were found in any of the material examined.

2. *Merozoites*

In intestinal smears from one impala small rounded intracellular merozoites with pale blue cytoplasm and pale purple nuclei, and measuring 5 to 6 microns in diameter, could be demonstrated. These were considered to be precursors of male and female gametocytes. No free merozoites were seen. The presence of intracellular merozoites, however, does indicate that schizonts must have been present shortly before death in this case.

3. *Macrogametocytes, macrogametes and oocysts*

Macrogametocytes, macrogametes and oocysts were numerous in all sections. Immature macrogametocytes (Fig. 2) were spherical structures about 9 to 10 microns in diameter with pale purple nuclei and light blue cytoplasm in mucosal smears stained with Giemsa. As they grew in size, eosinophilic granules appeared in the cytoplasm in increasing numbers. In mature macrogametocytes the larger granules lined the periphery of the organism (Figs. 3 & 4). In mature macrogametes, which were ellipsoidal in shape, thus resembling the oocysts, the granules had coalesced to form a thick eosinophilic rim. This rim gave rise to the refractile, usually distorted, wall of the oocysts (Fig. 5). Thirty-one mature macrogametocytes measured an average of 21 by 17 microns in size.

The host cells were virtually obliterated by the parasites, only compressed crescentic nuclei remaining visible in some cases (Fig. 2 & 3). The parasites in the cytoplasm of epithelial cells appeared to be situated distal to the nucleus. The identity of the host cells involved has been discussed elsewhere, but it appears as if both epithelial cells and histiocytes were parasitized[1]. Many of the organisms, including oocysts, in the sections were noticeably shrunken.

4. *Microgametocytes.*

The occurrence in histological sections of scattered clusters of female parasites, all of which were in the same stage of development, was interpreted as indicating a simultaneous infection of a group of host cells with merozoites. Although female organisms predominated in such clusters, a small number of microgametocytes was always present. These microgametocytes were observed to be in a similar stage of development as their female counterparts. Among a group of young oocysts, for example, a small number of mature microgametocytes containing fully developed microgametes could easily be recognised. This phenomenon facilitated the study of the male series of parasites; the male counterparts were sought in the vicinity of these clusters of female parasites which consisted of any one stage of organism from oocysts to immature macrogametocytes.

The youngest microgametocytes seen were approximately 9 microns in diameter. These microgametocytes contained a moderate number of closely packed, more or less cuboidal blocks of basophilic nuclear material, each of which was about 1.5 microns in diameter. As the microgametocytes developed they increased in size and progressively lost their compact structure. The nuclei were reduced in size as they became more numerous. In some microgametocytes the nuclei were arranged along the periphery with one or more whorls of nuclei within the organism (Fig. 6), but in others the entire microgametocyte consisted of whorls of nuclei of varying sizes (Fig. 7). In the next phase the nuclei, which constituted the most clearly resolved portions of the developing microgametes, had become oval black dots lying rather haphazardly around the periphery, with residual cytoplasmic material remaining in the centre of the organism (Fig. 8). At this stage the microgametocytes appeared to lack strength and were inclined to be compressed by the female gamonts surrounding them. Thirty-three microgametocytes measured an average of 37.4 by 28 microns in size with a range of 48 to 30 by 37.5 to 17.5 microns. The microgametes became more elongated as maturation progressed and eventually appeared as crescentic or filamentous bodies about 3 to 4 microns in length (Fig. 9). Flagellae were difficult to distinguish in both sections and smears. In the final stage masses of free-lying microgametes were observed

Discussion

In the cases of coccidiosis in the impala described above it is obvious that, with one exception, a single *Eimeria* species was responsible. Not only were the oocysts, which were

present in very large numbers, identical, but the endogenous stages were also strikingly similar. Proof that the endogenous stages in sections were precursors of oocysts in the faeces was provided by the presence of young oocysts in sections which were similar in shape and size (except for slight shrinkage) to the oocysts found in the faeces.

Very few *Eimeria* spp. have been described from African antelopes, and the description of those that have been reported have been based on oocyst morphology only. *Eimeria impalae* Prasad and Narayan, 1963 has recently been described from impala in East Africa[4]. Ten oocysts were found to vary in size from 30 to 36 microns in length by 20 to 24 microns in width. They were ellipsoidal in shape and the average length-width ratio was 1.47. A micropyle 5 to 8 microns in width was present. The oocysts were yellowish-green in colour "with granulations in the anterior third". Sporocysts were ovoid in shape and measured 8 to 13.6 microns in length by 7.2 to 8.8 microns in width. Their Stieda bodies were inconspicuous. No oocystic or sporocystic residual bodies were seen.

The *Eimeria* sp. from South African impala obviously has a number of features in common with *E. impalae*, most important of which is marked overlapping of the size-range, identical shape and the presence of a micropyle. It differs from *E. impalae* by the absence of "granulations in the anterior third" of the oocyst, the presence of sporocystic residual granules and by having considerably larger sporocysts.

In spite of these differences the writer is of opinion that there is not sufficient evidence for the creation of a new *Eimeria* sp. Although the distribution of impala is virtually continuous in these parts of Africa, such geographical isolation as exists may have been responsible for the development of these slight variations in form. However, if further investigations on the morphological features, life cycle and host-parasite relationship should reveal significant differences between *E. impalae* and the parasite above, their toxonomic status will require reconsideration.

Eimeria talboti Prasad and Narayan, 1963[4] differs from *E. impalae* by virtue of its ovoid rather than ellipsoidal oocysts and the absence of a micropyle. The oocysts are 36.16 by 24.75 microns in size. The host is the kongoni, *Alcelaphus cockei*, Günther, 1884.

Two *Eimeria* spp. have been recorded from the eland, *Taurotragus oryx* (Pallas, 1766). *Eimeria canna* Triffit, 1924 has oocysts that are oval in shape and 27.3 by 19.9 microns in size[5]. They differ from *E. impalae* by their smaller size and the conspicuousness of the Stieda body of their sporocysts. *Eimeria triffittae* (Yakimoff, 1934) oocysts are 21.1 by 17.8 microns in size[6]. They have no micropyle and are also different from *E. impalae* in shape and size.

Eimeria macieli Yakimoff and Matschoulsky, 1938, described from the waterbuck, *Kobus ellipsiprymnus* Ogilby, 1833 has oocysts that are 29.7 by 21.2 microns in size[7]. They are therefore smaller than those of *E. impalae*.

Eimeria walleri Prasad, 1960 of the gerenuk, *Litocranius walleri* Kohl, 1886 has oocysts that are 28.5 by 23.5 microns in size and subspherical in shape[8]. They differ from those of *E. impalae* in size and shape.

Oocysts of *Eimeria gorgonis* Prasad, 1960 of the blue wildebeest, *Connochaetes taurinus* (Burchell, 1823) are 22.7 by 16.5 microns in size. They are therefore considerably smaller than those of *E. impalae*, and have no micropyle.

E. impalae differs from the *Eimeria* sp. of sheep and goats by virtue of the large size and ellipsoidal shape of its oocysts, and the absence of a polar cap. With the possible exception of *Eimeria bombayensis* Rao and Hiregaudar, 1954, *E. impalae* oocysts are also dissimilar to those of other bovine coccidia. *E. bombayensis* has apparently only been encountered on one occasion in Bombay[9].

A striking feature of this study was the severe degree of infection of the small intestine with endogenous stages, which had all reached virtually the same stage of development viz. advanced gamogony. No schizonts could be found among the sexual stages in any of the sections. It would, therefore, appear that the sexual phase of the life cycle of *E. impalae* is more pathogenic than the asexual phase, and was responsible for the death of the animals.

ACKNOWLEDGEMENTS

The writer wishes to thank the Chief, Veterinary Research Institute, Onderstepoort, for permission to publish this article. Assistance given by Dr. R. C. Tustin in the preparation of this article is gratefully acknowledged. He is particularly grateful to Drs. T. W. Naude, R. C. Tustin, J. G. Pienaar and S. M. Hirst for supplying material for examination. Thanks are due to Mr. A. M. de Bruyn for the photomicrographs and to Mr. J. H. Schoeman for assistance.

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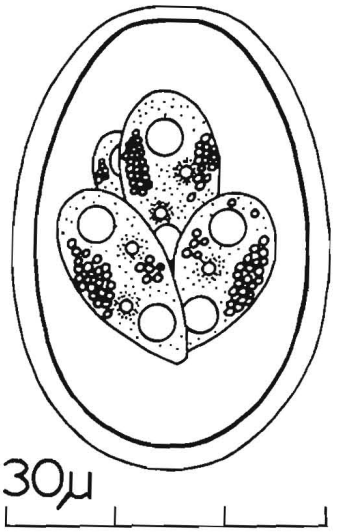


Fig. 1 — Camera lucida drawing of an oocyst.

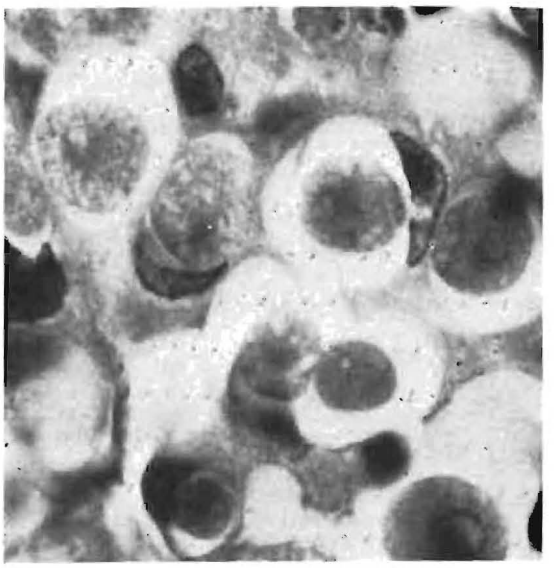
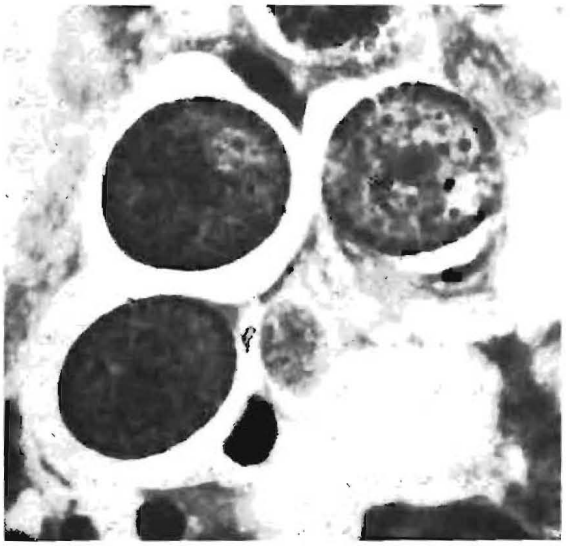
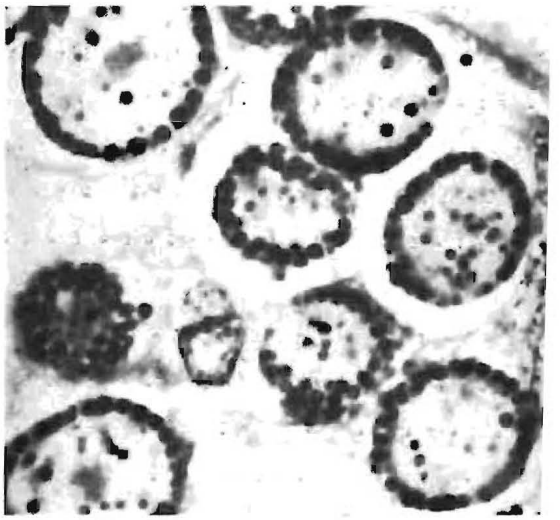


Fig. 2 — Immature macrogametocytes stained haematoxylin and Eosin. X 1,200.





Figs. 3 & 4 — Macrogametocytes with eosinophilic granules staned as before. X 1,200.

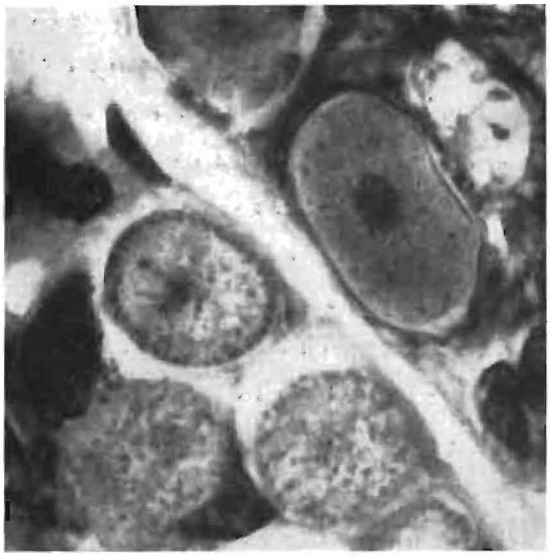


Fig. 5 — Macrogametocytes at left and young oocyst on the right, stained as before. X 1,200.

Fig. 6 — Immature microgametocyte showing peripherally arranged and central whorls of nuclei, stained as before. X 1,200.

Fig. 7 — Two microgametocytes showing numerous whorls of nuclei around residual material, stained as before. X 1,200.

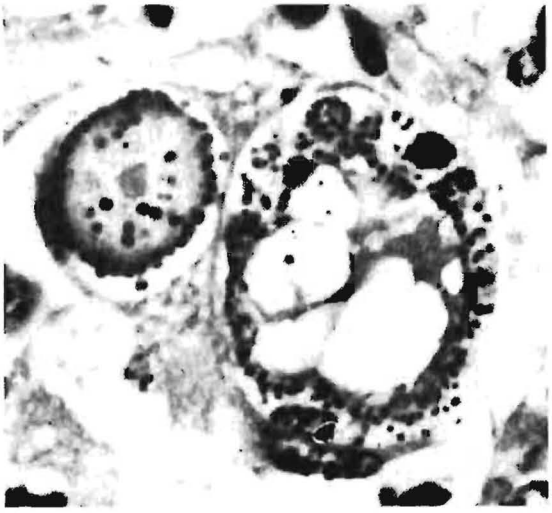


Fig. 8 — More mature microgametocyte on right, stained as before. X 1,200.

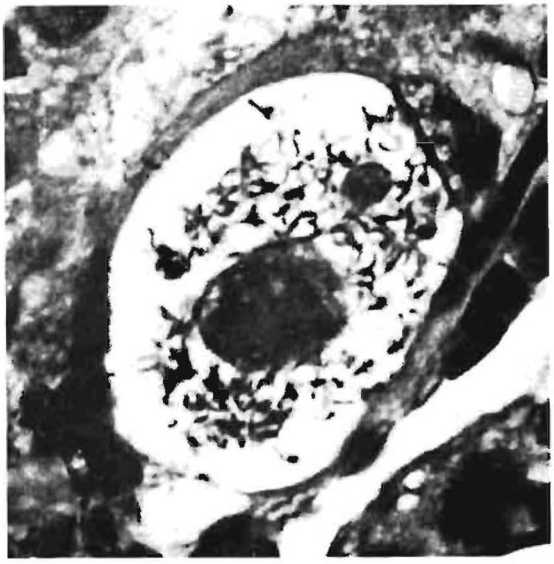


Fig. 9 — Mature microgametocyte containing microgametes around a centrally situated residual body, stained as before. X 1,200.

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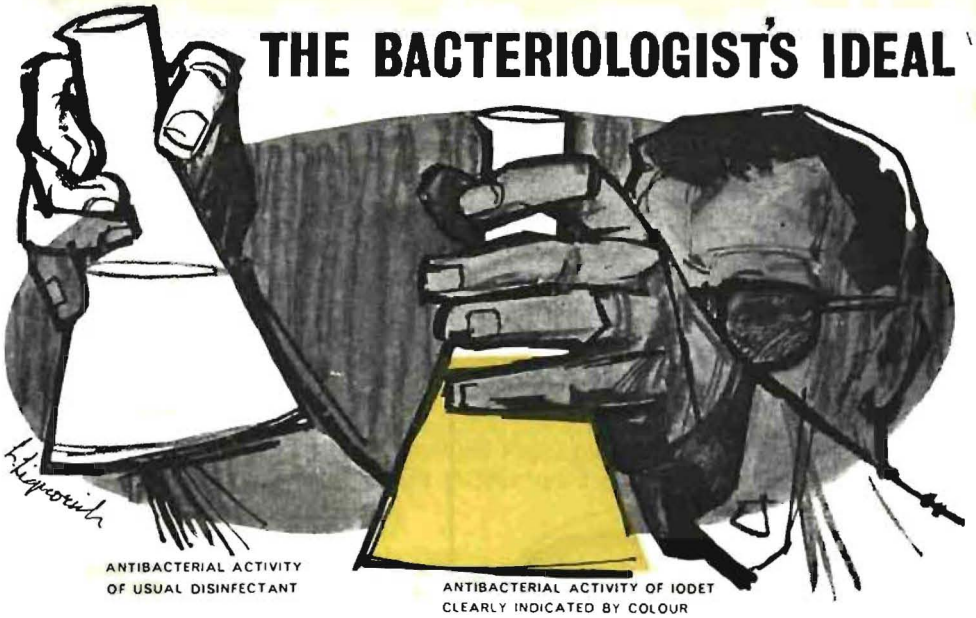

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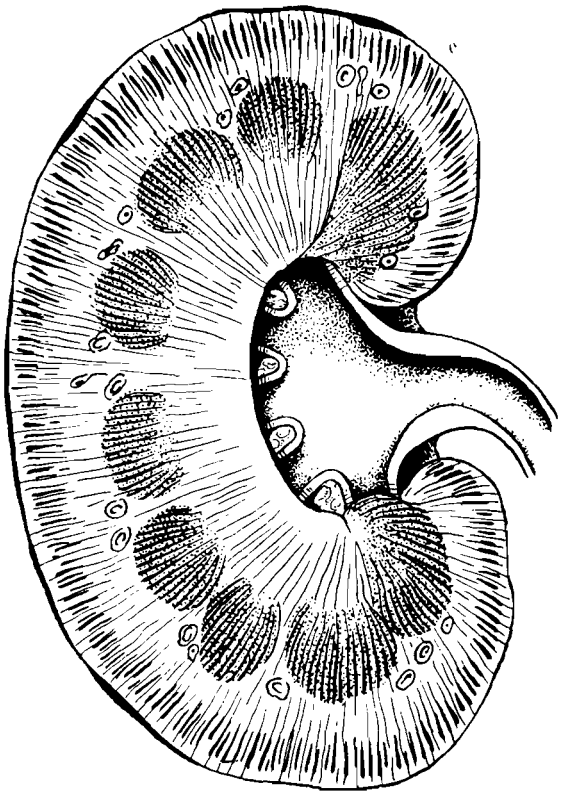

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THE MINERAL METABOLISM OF RUMINANTS IN THE SOUTH WESTERN DISTRICTS OF THE CAPE PROVINCE

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Received for Publication June, 1964

Investigations into the reasons for low wheat yields obtained in the various districts of the South Western Cape, have indicated that a severe copper deficiency exists in the soils of these districts. This deficiency is manifested not only in the low wheat yields; other crops are similarly affected, while symptoms of copper deficiency are frequently observed in the merino sheep flocks in some of these districts. The predominant symptoms observed in sheep are swayback in lambs and 'steely' wool in adults.

Experiments conducted by Roach and Beyers (1959, 1963)[6] have shown that the addition of copper, in the form of copper sulphate crystals, applied either as an aqueous solution sprayed onto the young preflowering stages of the wheat, or as an adjuvant to the normal fertilizers, has led to remarkable increases in the yields obtained; in the majority of cases a ten-fold increase in the yield may be expected.

Since copper in the soil is but slowly leached, forming a very stable combination with the organic material, humus, the question was raised as to the effect on livestock grazing on stubble lands, subsequent to the continued application of copper sulphate and consequent accumulation of quantities of this mineral substance in both the soils and plants. As both copper and molybdenum (the latter applied as a trace element) are toxic substances, the rapid extension of their use by farmers creates a source of potential toxicity to the livestock on these farms. Warnings to this effect have been issued to the farming industry (Roach and Beyers 1959, 1960)[6] but as insufficient data exists on which the assessment of the potentiality of these poisons may be based, the experiment, described below, was planned to determine the effect on sheep when fed plants, which had been grown on soils to which varying quantities of copper sulphate had been added.

Materials and Methods:

The quantities of copper sulphate added to the experimental plots are those which are prescribed for the enhancement of wheat yields, and were not merely arbitrary figures.

(*a*) Three plots of ground at the Outeniqua Experimental Farm, George, were prepared for the cultivation of lupins, lucerne and oats.
Plot No. 1 received copper sulphate at the rate of 7½ lb/morgen.
Plot No. 2 received copper sulphate at the rate of 30 lb/morgen.
Plot No. 3 received copper sulphate at the rate of 60 lb/morgen.

(*b*) Nine sheep of the same breed, age, sex and live bodyweight were divided into 3 groups, placed in pens, and handfed material from

the treated plots. For the control group sheep from the normal farm flock were slaughtered on the prescribed dates.

(*c*) To each of the four groups (including all the normal farm flocks) the following mineral mixture in the form of a lick was supplied *ad lib*:

40 lbs bonemeal.
10 lbs dolomitic lime.
5 lbs flowers of sulphur
40 lbs salt.
10 lbs molasses.
8 oz. copper sulphate.
4 oz. magnesium sulphate.
2 oz. manganese sulphate.
1 oz. cobalt sulphate.

This mineral mixture is fed throughout the year to all the animals on the farm to supplement the copper and other mineral deficiencies which exist in this area. The supplementary feeding of mineral mixtures is advocated as a routine measure to all stock farmers in the coastal regions of the South Western districts of the Cape Province.

(*d*) At the beginning of the experiment on 1.6.62, the animals in the pens were weighed and again weighed just prior to slaughter, when liver specimens were taken for copper analysis.

(*e*) On 7.6.63 the remaining animals were weighed and released from the pens and admitted to the normal farm flock, prior to the slaughter on 12.6.63. The purpose of this exercise was to submit these remaining 3 sheep to the stresses and strains to which sheep are subjected in the course of normal husbandry practices.

(*f*) Livers from the slaughtered sheep were assayed for copper by the methods of Jackson (1958)[4] and the results obtained are expressed as "parts per million"—dry basis.

Results:

A. *Weight gains*:

Group 1 Zero day weights (average of 3 animals)	107
Weights at slaughter (average of 3 animals)	149
Group 2 Zero day weights (average of 3 animals)	107
Weights at slaughter (average of 3 animals)	158
Group 3 Zero day weights (average of 3 animals)	105
Weights at slaughter (average of 3 animals)	162
Group 4 (Controls) Weights at zero day average	100
Weights at slaughter average	111

B. *Consumption of Mineral Supplement.*

(i) 1*st Period*: 1.5.62 to 30.10.62	(183 days)
No. of sheep per group	3
Quantity of supplement consumed per group	80 lbs
Quantity of copper sulphate consumed per group	175 g.
Average daily consumption of $CuSO_4$ per sheep	0.316 g.
(ii) 2*nd Period*: 31.10.62 to 20.3.63	(141 days)
No. of sheep per group	2
Quantity of supplement consumed per group	45 lbs.
Quantity of copper sulphate consumed per group	102 g.
Averange daily consumption of $CuSO_4$ per sheep	0.36 g.
(iii) 3*rd Period*: 21.3.63 to 5.6.63	(78 days)
One sheep per group.	
Quantity of supplement consumed	10lbs.
Quantity of $CuSO_4$ consumed	22 g.
Average daily consumption of $CuSO_4$	0.28 g.

C. *Copper intake through forage—Experimental Groups.*

(i) Average weight of green material fed daily to the 3 groups:

Lupins	65 lbs.
Lucerne	40 lbs.
Oats	42 lbs.

(ii) Average daily intake of copper per sheep fed on fodder taken from the experimental plots:—

7½ lb. $CuSO_4$/Morgen	*30 lbs./Morgen*	*60 lbs./Morgen.*
0.0021 g. Copper	0.0034 g. Cu	0.0040 g. Cu.
o.e. 0.0084 g. Copper sulph.	0.0136 g. $CuSo_2$	0.016 g. $CuSo_2$

D. *Liver Analysis*:

Copper content of livers (parts per million—dry weight).

Date of slaughter	Controls	Experimental Groups- lbs. sulphate per morgen.		
		7½	30	60
19.4.62	(56, 60, 65) 60 (104, 106, 109) 106 (19, 14, 20) 18 Average 61			
1.11.62		(252, 255) 253	(343, 359) 351	(578, 628) 603
26.3.63	(158, 158) 158	(250, 265) 257	(350, 375) 362	(716, 723, 768) 736
12.6.63	(95, 115) 105	(190, 225) 207	(165, 155) 160	(410,420) 415

(Figures in brackets are for separate determinations of different samples from the same livers, followed by average for each liver.)

Discussion:

(i) The figures for the liver copper content of the control sheep stress the severity of the copper deficiency in the soils of the experimental farm. The figure of 250 p.p.m. is generally accepted as the normal copper level. The figure obtained from the Control sheep indicate that:

(*a*) despite the ad lib availability of the lick, there is either an inadequate intake of the lick, or

(*b*) the absorption of copper sulphate is insufficient to meet the accepted normal physiological requirements.

(ii) Despite the fact that the daily copper intake of sheep fed on the 7½ lbs/morgen fodder is in the region of only 0.0021 g., this minimal quantity is apparently sufficient to catalise the absorption of copper sulphate from the lick, so as to bring the liver copper values in this group to within the accepted normal range.

(iii) The addition of 30 lbs. of copper sulphate per morgen raises the liver copper values to the accepted upper range of normal values.

(iv) If the standards as laid down by various authorities (Jones 1959,[5] Blood and Henderson 1960[1], Garner 1961[3], and Tod, et al., 1962[7]) are accepted, then the copper values of the livers of the 60 lb./morgen are well within the toxic range.

(v) Despite the fact that the sheep of all 4 groups had been assimilating copper sulphate at the rate of 6.1 mgm./Kilo. live wt. per day, (range 5.6, 7.2 and 6.2), no clinical symptoms of copper poisoning were observed in any of the animals of the 3 experimental groups. In the control group (normal farm flock) certain animals were evincing clinical signs of a copper deficiency (steely wool etc.)

Conclusions:

(i) The dramatic fall in liver copper values of the final slaughter groups, when compared with those slaughtered on 26.3.63 could be explained by the fact that for a period of 6 days prior to slaughter on 12.6.63 these remaining animals were pastured together with the main flock on untreated soils, although the copper sulphate medicated lick was freely available at all times. The apparent build up of copper values in the liver can be accepted as a form of physiological storage and not as a pathological deposition. As soon as the copper intake through the treated forage ceased, the stored copper was released by the liver and excreted from the body.

(ii) From the liver copper values of the control animals one must conclude that, despite the fact that the mineral lick had an abnormally high copper sulphate content, the copper intake was insufficient for normal requirements. This phenomenon could be explained by one or both of the following suppositions:

(*a*) The control sheep did not eat as much of the lick as the penned animals ate.

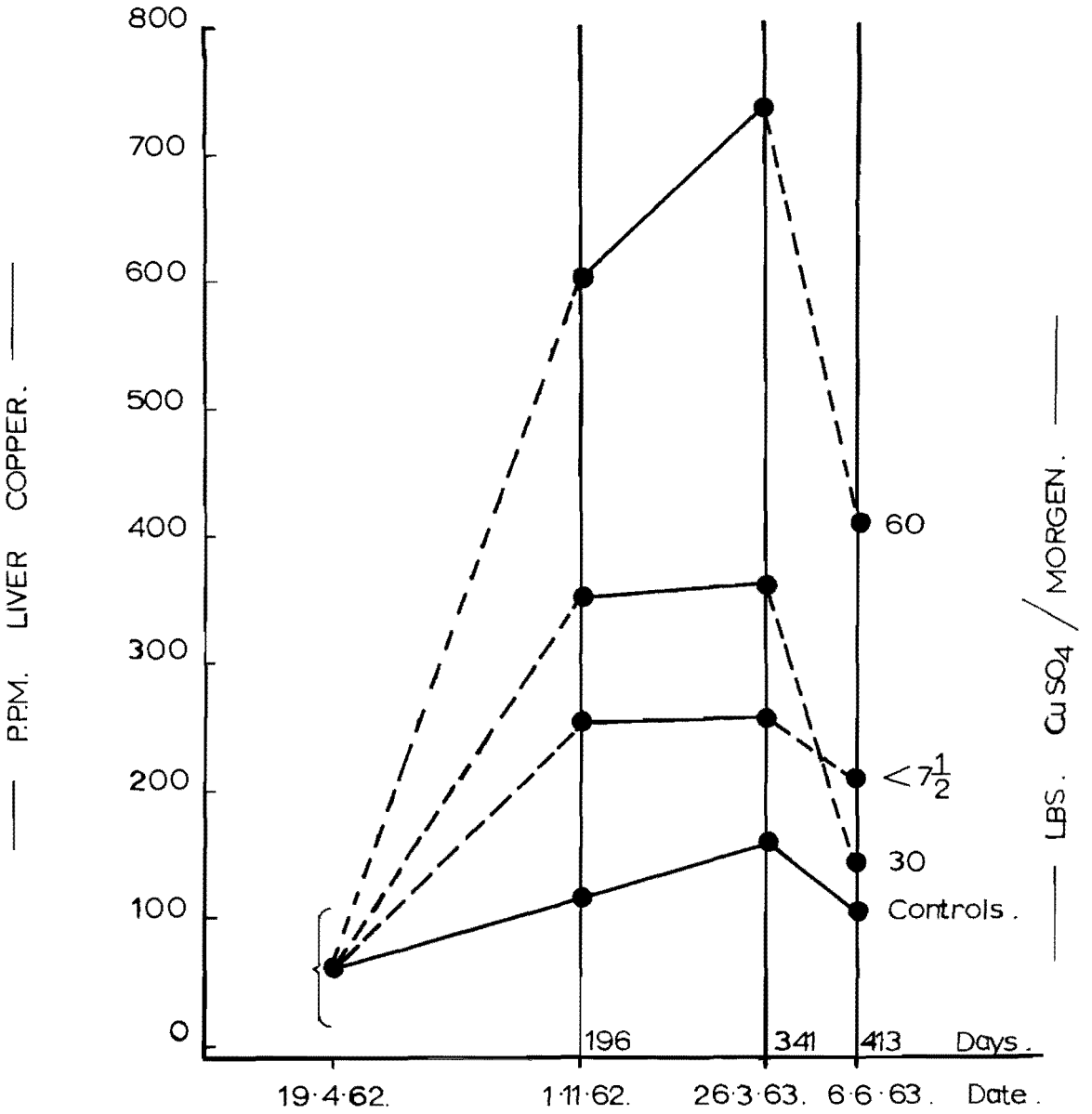

FIG. 1. Graph showing levels of liver copper (dry matter).

(*b*) The copper sulphate in the lick is not so readily absorbed from the gastro-intestinal tract.

It was noted that from sheer boredom, the penned animals ate abnormal quantities of the mineral lick.

(iii) It can be concluded that the addition of 7½ lbs./morgen of copper sulphate together with the *ad lib* intake of mineral supplements, supplies sufficient copper to bring the liver copper values to the accepted normal range.

(iv) Provided that the sheep are not exposed to stress or other abnormal factors for example, sudden deprivation of food as the result of kraaling or penning for the purpose of dosing or inoculations, moving the animals over long distances by road or rail or sub-clinical viraemic infections, such animals with liver copper values as high as 736 p.p.m. will show no clinical signs of copper toxicity. The important part played by sub-clinical virus infections, as a stress factor, cannot be overstressed. (Brown 1964 — personal communication)[2].

(v) Further experiments should be undertaken to determine the actual quantity of copper absorbed from mineral licks containing varying quantities of copper salts.

Summary:

(i) Experiments were uhdertaken to determine the effect on sheep fed with fodder obtained from soils which had been treated with copper sulphate at the rate of $7\frac{1}{2}$, 30 and 60 lbs. per morgen.

(ii) Analysis for liver copper gave values ranging from 61 p.p.m. in the controls, to 736 p.p.m. (dry basis) in the 60 lb./morgen group.

(iii) It is concluded that, in the absence of stress, most of the copper is stored in the liver in a physiological state, and excreted as soon as the sheep were returned to normal, untreated pastures.

ACKNOWLEDGEMENTS

We wish to thank the Director of the Winter Rainfall Region for providing the facilities for conducting this experiment, and for his interest and advice. The Chief, Veterinary Field Services is thanked for permission to publish this work.

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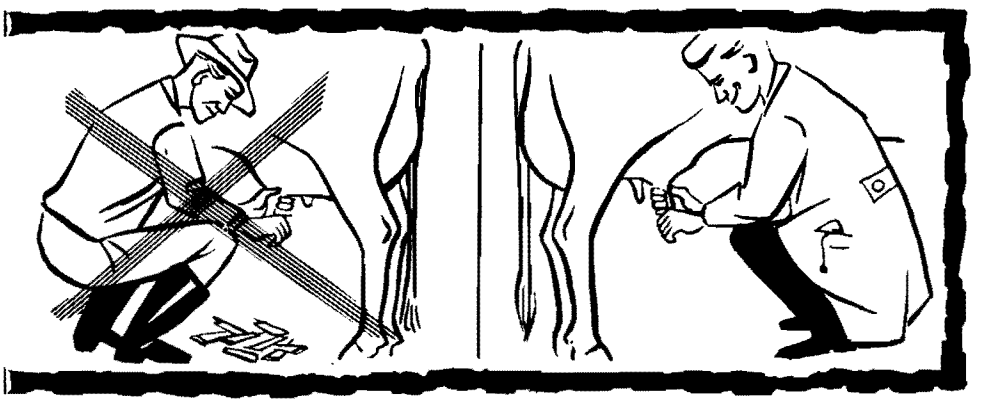

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THE OCCURRENCE OF COCCIDIOSIS IN IMPALA — *AEPYCEROS MELAMPUS* (LICHTENSTEIN, 1812)

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Received for publication July, 1964

SUMMARY

1. Outbreaks of coccidiosis in impala are recorded.

2. The history and epizootiological features of the outbreaks are discussed.

3. A description of the macroscopic and microscopic lesions is given.

4. Various epizootiological aspects regarding the cause of the outbreaks are discussed.

INTRODUCTION

Coccidia are common parasites of domesticated animals, being represented by a large number of *Eimeria* and *Isospora* species. Despite their prevalence they are rarely responsible for disease under conditions of extensive management, where the degree of exposure is limited. If young animals however, are crowded together in a restricted area under climatic conditions allowing a build-up of viable, ripe oocysts in the soil, outbreaks of coccidiosis can be expected to occur. Depending on the pathogenicity of the parasite(s) concerned, severe symptoms and even mortality may be observed.

Very few coccidia have been described from antelopes[1]. Prasad and Narayan[4] in 1963, described a new species of *Eimeria*, which they named *Eimeria impalae*, from the impala. This description was based on the examination of a single sample of faeces which originated from Kenya. No indication of the pathogenicity of this parasite was given. This lack of information does not necessarily mean that coccidia are uncommon in these animals. Their failure to manifest themselves as pathogenic organisms may either be due to the existence of an ideal host-parasite relationship, indicative of good adaptation, or it may be a result of the feeding habits and extensive ecological conditions under which these animals live in their natural habitat. In the latter instance it is likely that any pathogenic coccidia present may cause illness, and thus expose itself, when such antelopes are captured and confined to close quarters under poor hygienic conditions.

In this article we wish to record the course of events leading to outbreaks of coccidiosis in young impala and the pathological changes produced. The single *Eimeria* sp., regarded as similar to *Eimeria impalae* Prasad and Narayan, 1963, responsible for the outbreaks is described in a separate paper.[1]

History

The Department of Nature Conservation of the Transvaal Province annually capture a number of young impala for distribution to farmers who desire to restock their farms. Prior to their release in their new habitat the animals are kept in quarantine for one month on one of the Nature Reserves.

The quarantine camp at Loskop Dam Nature Reserve consisted of a paddock about an acre in extent. A reed shed with a thatched roof and a floor surface area of about 400 square feet, in which the animals were housed every night, was erected in the paddock. Veld grass was used as bedding, and after prolonged use, the soil surface of the shed became rather damp. These facilities have been used for a period of 4 to 12 weeks, annually, since their erection in 1957.

The first outbreak of coccidiosis in young impala at Loskop Dam occurred in June 1957. The diagnosis was made by one of us (T.W.N.) who demonstrated large numbers of oocysts in the faeces of a dead animal. The same quarantine facilities were continued to be used once a year without significant mortality until the autumn of 1962, when 26 animals died from what was considered to be enterotoxaemia.

The most recent mortality occurred in April and May, 1963 when 17 out of 29 young impala from four to six months of age, died. They were all captured on Loskop Dam Nature Reserve and subsequently liberated in the quarantine camp. Concentrates and water were fed in troughs and hay and the branches of indigenous trees were distributed daily on the ground. The condition of the impala, which was rather poor at the time of capture, deteriorated even further. The first death occurred about fourteen days after capture. Some animals died without having shown any definite symptoms. Others appeared to be stiff in the hindquarters, and voided soft faeces, about two days prior to death. Nine out of the sixteen impala captured in March, died during April, and eight of the eleven captured in April, died during May.

Initially enterotoxaemia was again suspected and immunization against enterotoxaemia was instituted. However, on submitting the carcase of an impala that died subsequent to vaccination, to the Veterinary Research Institute, Onderstepoort a diagnosis of coccidiosis was made. Subsequent examination of specimens from further fatal cases revealed infections with coccidia.

At this stage prophylactic and therapeutic measures aimed against coccidiosis were instituted. A new paddock about half the size of the existing one was erected for alternate use with the existing one. Having withheld all drinking water for one day, medicated water containing approximately 0.5% sulphadimidine* was supplied *ad lib* for four consecutive days. After an interval of three days the treatment was repeated. One impala died on the 2nd day of treatment, but thereafter no further cases occurred. The impala were then trasferred to the new paddock and treatment was again repeated for a four-day period.

Coccidiosis was also diagnosed by one of us (R.C.T.) in a single impala that died under virtually identical circumstances on the Hans Merensky Nature Reserve, in the Letaba district of Transvaal, in June 1962. The same *Eimeria* sp. was involved[1].

Material and Method

Specimens of the affected portions of the intestine from the animal examined at Onderstepoort were collected and were fixed in 10% formalin.

Preserved material was also received from six other impala that died at Loskop Dam during the 1963 outbreak and from one animal that died on the Hans Merensky Nature Reserve in 1962. Sections for histopathological study were cut 3μ in thickness from this material and were stained by haematoxylin-eosin method and Giemsa method.

Faecal material and portions of affected gut were taken from several cases for the identification of the coccidium involved[1].

*Sulphamezathine I.C.I.

Pathology

(a) Macroscopic pathology.

The description given below is based on observations made on a single impala presented for autopsy at Onderstepoort.

The skin around the anus was soiled with soft greenish-yellow faeces. Dehydration of the carcase was obvious. The intestinal tract was virtually empty and the mucous membrane, of the posterior third of the jejenum and the ileum was thickened and had a dull greyish-red colour. On close inspection of the affected portion of the intestine it was noticed that numerous greyish foci varying in size from pin-point to pin-head, were scattered diffusely throughout the mucous membrane. Along the whole length of the affected part of the intestine, numerous irregular patchy areas, coloured an intense red, were also present. These varied in size from about 1 cm. in diameter, to areas about 7 cm. in length, which involved the whole width of the gut. No free blood was observed in the faeces or lumen of the gut.

With the exception of a small number of subendo- and subepicardial petechiae, no other lesions were observed in the carcase.

(b) Histopathology

In all the cases examined the histopathological picture was identical. The endogenous stages of the parasite were mainly confined to the epithelial cells lining the crypts of Lieberkühn. In the more severely affected parts of the intestine, which corresponded with the intensely red areas seen macroscopically, almost every crypt and every single epithelial cell of each crypt, was parasitized. Thus a solid mass of organisms, in different stages of development, was seen occupying the area extending from the muscularis mucosae to the orifices of the crypts (Fig. 1).

The infected glands were greatly enlarged and in apposition with each other, obliterating the lamina propria between them. Marked congestion of the blood vessels was present in these heavily infected portions of the intestine. In the less severely affected parts fewer crypts were involved and here single or adjoining groups of crypts, were seen to be packed with organisms, surrounded by unaffected crypts (Fig. 2). An occasional parasite occurred in the epithelial cells covering the villi.

In a few instances small groups of organisms consisting mainly of young oocysts with fewer macrogametocytes and microgametocytes present, were found in the lymph nodules in the submucosa, situated immediately below the muscularis mucosae (Fig. 3 & 4). Some of these parasites appeared to be intracellular; the nucleus of the parasitized cell being compressed to one side. The host cell in which these organisms occurred could not be determined with accuracy. It is assumed that they are macrophages, the organisms probably reaching the lymph nodules via the lymphatics after phagocytosis by macrophages. Many of the parasites did however appear to be extra-cellular. The smaller groups of these organisms were surrounded by cells with large vesicular nuclei resembling macrophages (Fig. 5) whereas these cells were not so evident around the larger groups.

The parasites, observed in the sections of the gut, were in various phases of the life cycle and these have been described in detail, by Bigalke[1]. A striking feature was the presence of numerous crypts or groups of crypts, filled with parasites which were virtually all in the same stage of development, and which resemblde a nest full of eggs (Fig. 6 & 2). The majority of the parasites in these groups or foci of crypts were young oocysts, and these "nests" were particularly conspicious where the adjoining crypts were not infected, or contained immature macrogametocytes. This focal appearance of crypts filled with young oocysts, probably represents the small greyish foci observed macroscopically in the mucous membrane.

There was marked hyperaemia of all the layers of the intestine, but this was most pronounced in the massively affected portions of the gut. The lamina propria and submucosa were slightly oedematous. No definite cellular infiltration was observed although it did appear as if a slight increase in the number of plasma cells and eosinophiles was present in the propria.

In addition to parasites, the lumen of many crypts contained erythrocytes and desquamated cellular debris. There were no signs of regenera-

tion of epithelium. Only a few glands were found to contain organisms in the caecum and colon, of all the cases examined (Fig. 7).

Discussion

The severe degree of infection with coccidiosis, the severity of the lesions encountered in every case investigated and the absence of other lesions justify the conclusion that coccidiosis was the cause of death in the impala described above.

The source of infection poses an interesting epizootiological problem. If the well-known host specificity of *Eimeria* spp. is taken into consideration, it is reasonable to assume, that this parasite has only one host viz. the impala. Impala acting as carriers of oocysts, must have been involved in the original introduction of the organisms onto the premises where these outbreaks of coccidiosis occurred. Two possible epizootiological avenues, leading to the mortality, then present themselves for consideration.

Under ideal environmental conditions it is possible that a build-up of large numbers of viable oocysts, excreted by clinical cases or subclinical "multipliers", may occur[2]. Provided that environmental conditions in the immediate vicinity (microclimate) of the oocysts remain favourable, it is possible that fairly large numbers of oocysts may have remained viable for a year. It is possible that conditions in the shed, mentioned above, fulfilled these requirements. The introduction of highly susceptible young animals from the veld, where the infection rate is probably very low, into such heavily infected quarters, would result in a massive infection, further excretion of large numbers of oocysts, and mortality, if the *Eimeria* sp., concerned is pathogenic.

The second possibility, that should be considered, is that the oocysts may not survive for as long as a year, under favourable environmental conditions, and that in the outbreaks under review, the oocysts were introduced annually by carriers. A rapid build up of parasites, due not only to the high susceptibility of young animals, but also to the artificial conditions under which they were kept, must have taken place. Eventually sufficient numbers of oocysts may have been ingested to cause mortality. This latter theory seems improbable as only 14 days elapsed between capture and the first death.

The significance of reduced resistance, due to the stress of capture, the change of diet in these animals, which are largely browsers, and the fact that they were fed from the ground, must also be considered in the aetiology of these outbreaks.

It is evident that *Eimeria impalae* Prasad and Narayan, 1963[4] is a highly pathogenic parasite of impala. Under natural conditions, the intake of oocysts is probably on such a low level, that it is of no significance, but under conditions of domestication it has to be contended with.

Until the complete life cycle of the parasite has been studied, opinions as to the nature of cells other than the epithial cells of the intestine acting as host cells, must be speculative. Apparently macrophages may also serve as host cells of this parasite. The extracellular parasites seen in the lymph nodules probably originated from degenerated and ruptured macrophages.

Chemotherapeutic studies on avian coccidia have shown that sulphonamides act chiefly against the schizonts[3]. If these results are applicable to all mammalian coccidia, it would seem strange that sulphidimidine appeared to be effective in cases where only sexual stages could be found. Nevertheless, results obtained with sulphadimidine were sufficiently promising to warrant the use of this drug in outbreaks of coccidiosis in impala.

ACKNOWLEDGEMENTS

The authors wish to thank the Chief, Veterinary Research Institute, Onderstepoort, for permission to publish this report. Thanks are also due to Mr. E. J. J. Henrico and Dr. S. M. Hirst of the Department of Nature Conservation of the Provincial Council of Transvaal, for information and specimens supplied by them, to Mr. A. M. de Bruyn for the photomicrographs and to the technical staff of the Section of Pathology, for the preparation of the histological sections.

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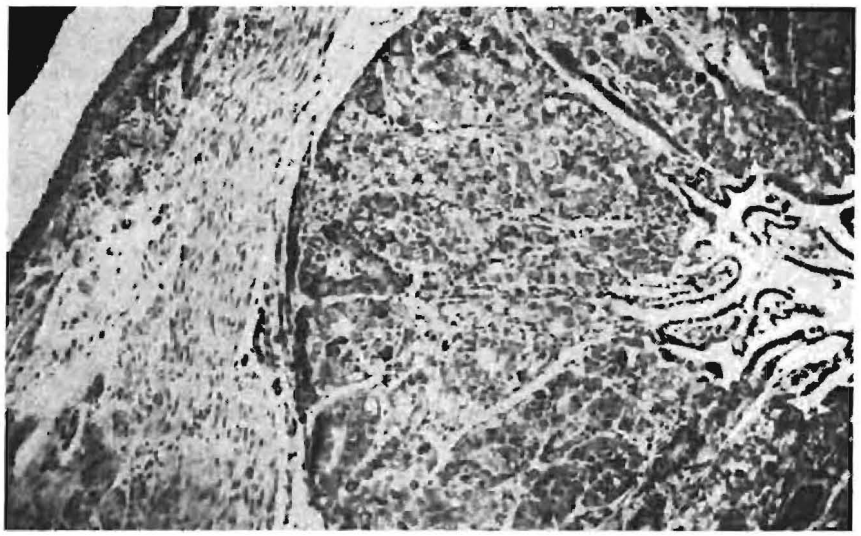

Fig. 1 — Ileum. Severely affected part showing masses of organisms in the crypts of Lieberkühn. No unaffected crypts present. The villi (on the right) contain no parasites. H. & E. stain. X 75.

Fig. 2 — Ileum. Less severely affected portion showing three crypts containing parasites surrounded by unaffected crypts. H. & E. Stain. X 75.

Fig. 3 — Ileum. Focal group of organisms in lymphoid tissue below muscularis mucosae. H. & E. Stain. X 75.

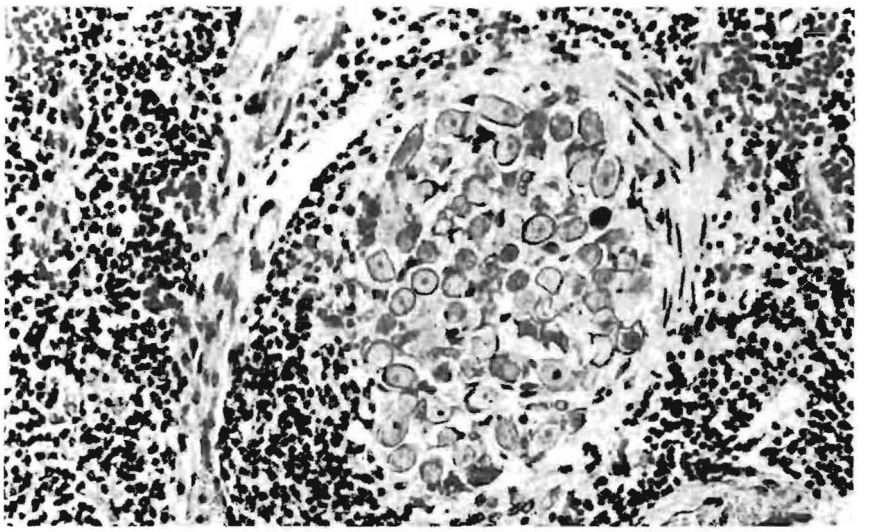

Fig. 4 — Higher magnification of group of organisms shown in Fig. 3. H. & E. Stain. X 190.

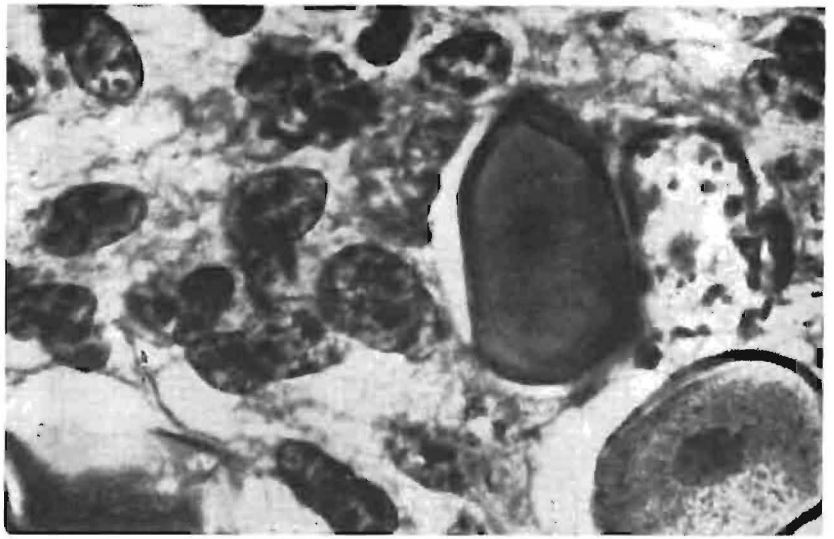

Fig. 5 — A small group of parasites in lymphoid tissue of ileum surrounded by cells with large vesicular nuclei which are probably macrophages. H. & E. Stain. X 1,200.

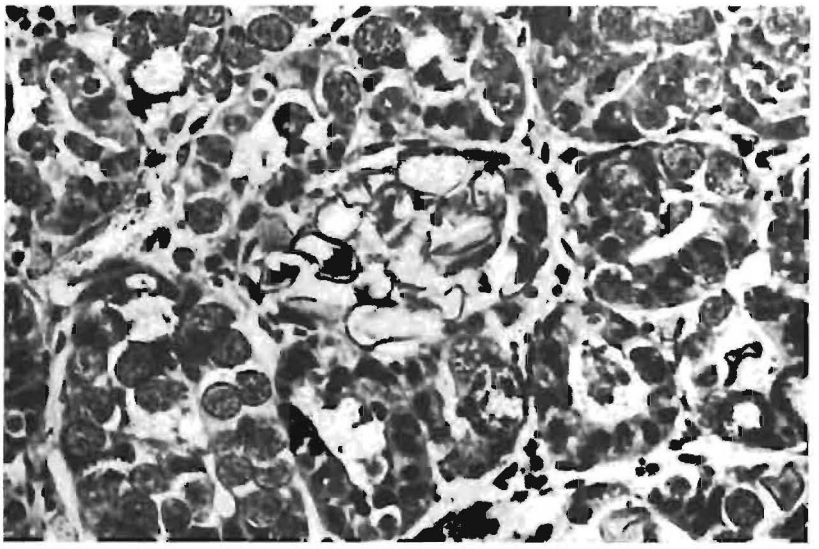

Fig. 6 — Ileum. Single crypt, containing a "nest" of young oocysts, surrounded by unaffected crypts and crypts containing parasites in other phases of life cycle. H. & E. Stain. X 300.

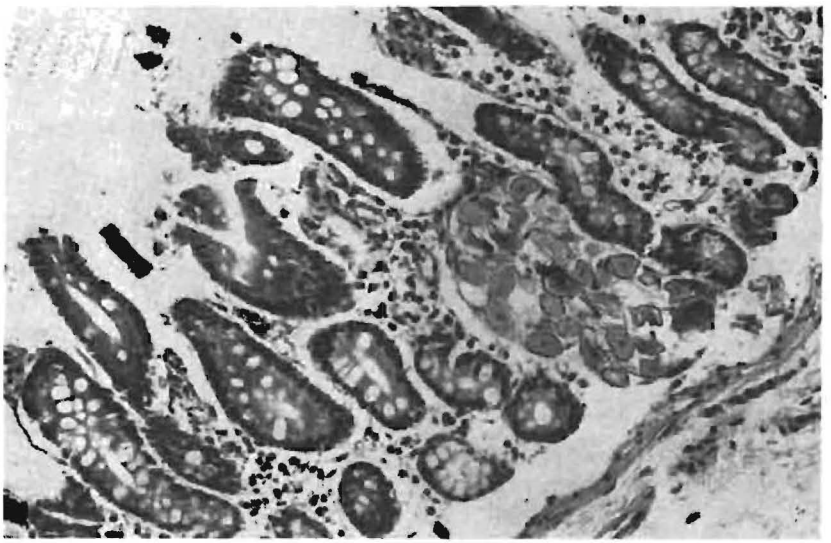

Fig. 7 — Caecum. A single affected gland. H. & E. Stain. X 190.

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OBSERVATIONS ON SNAKE-BITE IN DOMESTIC ANIMALS IN NORTHERN ZULULAND

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Summary

The aetiology, symptoms and treatment of snake-bite in various domestic animals in Northern Zululand are described and discussed.

Introduction

In 1954 human mortality due to snake-bite was estimated at 30,000 to 40,000 deaths per *annum*. These figures exclude mortality in the USSR, Central Europe and China, but nevertheless are considered conservative. Asia alone accounts for 25,000 to 35,000 of these deaths, while the figures for Africa are estimated at 400 to 1,000 deaths annually[11]. No mortality rates for domestic animals are available.

FitzSimons[3] states that despite the fact that most of the experimental work on the action of venoms has been done in domestic animals, very little information is available on the treatment of the latter. In Northern Zululand particularly, the incidence of snake-bite in domestic animals is high[6] and with the exception of two, the cases described here all occurred on one ranch in the Mkuze district of Northern Zululand.

Aetiology

The snake responsible for the bite is often never seen and therefore it is usually impossible to make a definite identification of the species of snake concerned.

Circumstantial evidence, however, such as the sighting or killing of a particular type of snake in the area of strike within a few hours of the bite, or the symptoms of the stricken animal may be an aid to identification. The aetiology in the various domestic animals differs and will therefore be discussed under separate headings.

Cattle

It is common practice on ranches in this area to collect large herds of cattle for dipping, counting, weaning, branding or marketing. These herds are driven through the veld or along roads to the dip, kraal or railway station. Snakes are normally not aggressive[4], but on finding themselves trapped in the midst of a herd, have no other alternative than to protect themselves by striking. This strike is often preceded by a warning hiss, which merely serves to whet the bovine's curiosity so that it sniffs at the place from whence the hiss came thus resulting in the large number of facial or neck bites encountered.

The herd boys usually do not witness these strikes, it is only later when the venoms start taking effect, that cases are seen and reported. These bites are usually inflicted by the Mambas, both Black and Green, (*Dendroaspis polylepis polylepis* and *D. angusticeps*) or the Black-necked Spitting Cobra (*Naja nigricollis nigricollis*) locally known by its Zulu name of 'Mfesi.

The slower moving Puff-adder (*Bitis arietans arietans*) is often the cause of abdomen or flank bites in cattle. The position of these bites suggests that the animals accidentally lie down on the snakes or that the snakes curl up near the reclining animals, and then strike when the animals get up.

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Dogs and cats

In dogs and cats bites are usually on the face or neck as these animals often attack snakes. Any of the above-mentioned snakes might be the cause of such bites. Cases of venom in the eyes of dogs are due to the accurate spitting of the 'Mfesi[3].

Poultry

As the 'Mfesi is often an egg-eater it can be incriminated in most cases of snake-bite occurring in poultry. Muscovy ducks are particularly prone to its attacks as they will protect their nests, whereas the author has seen the domestic hen and guinea-fowl stand to one side while an 'Mfesi rifled their nests.

Venoms

Snake venoms are complex chemical substances consisting amongst others of several proteins capable of producing a variety of effects. Venoms contain neurotoxic, haematotoxic and cytotoxic elements, the venom of any one snake can contain all these toxins and the effects of such a venom depends on which toxin predominates. The venoms from the Mambas and 'Mfesi are mainly neurotoxic with haematotoxic side effects whereas Puff-adder venom consists of haematotoxic andcytotoxic fractions[3,4].

Symptoms and Treatment

The author and various herdboys observed a number of cases of actual strike so that symptomatology and snake species can be linked. The cases in which no definite identification of the snake concerned could be made, will be grouped under certain snake species according to their symptomatology.

Each case is presented as follows; firstly the animal bitten, secondly the symptoms, which include the site of the bite and the approximate length of time that elapsed between the actual bite and the time when the author saw the animal and finally the treatment employed. In all cases the antivenin used was polyvalent.*

A. PUFF-ADDER (*Bitis arietans arietans*)

1. *DRAKENSBERGER COW*

Symptoms: Bite on left side over the ribs. Seen within 24 to 48 hours after strike. Extensive oedematous swelling of whole left side, the neck and limbs were not involved. Crepitation on palpation. The cutis became necrotic with the board-like texture of a dry unflayed hide.

Treatment: Twenty ml of polyvalent antivenin intravenously. 3M IU Penicillin intramuscularly for three days. No improvement. Cow destroyed.

2. *AFRIKANER BULL*

Symptoms: Bite in the region of the preputial fold. Seen within 24 to 48 hours of strike. Extensive oedematous swelling involving the ventral surface of the abdomen, preputial fold and prepuce. Crepitation on palpation. Occlusion of the preputial opening so that even an insemination pipette was passed into the preputial cavity with great difficulty. As the condition progressed there was a purrulent discharge from the prepuce and the cutis became necrotic and hard with the texture of a dry untanned hide. Multiple local abscessation developed in the preputial region.

Treatment: Antivenin injected locally with antibiotic supportive therapy. Bathing and cleaning of the prepuce. No improvement. Bull destroyed.

3. *SEALYHAM X AIREDALE DOG*

Symptoms: Bite on right upper lip. Seen within two minutes of strike. Massive progressive swelling of face and throat so that within a further two minutes the whole face was swollen, the eyes closed and the respiratory passages occluded. The dog collapsed into a coma.

Treatment: Twenty ml. antivenin intravenously and 500,000 I.U. Penicillin intramuscularly for three days. The dog regained consciousness within 15 minutes and the swelling disappeared within a few hours. Complete recovery.

* The South African Institute for Medical Research, Anti-Snakebite Serum.

Identification: A Puff-adder was killed the following day, in the vicinity of the house, where the dog had been bitten.

B. 'MFESI (*Naja nigricollis nigricollis*)

1. *MONGREL DOG*

Symptoms: Site of bite could not be located. Seen within one hour of strike. The dog was extremely hypersensitive to noise or movement, either of which resulted in a reaction similar to that seen in strychnine poisoning. The head and neck were pulled backwards, the fore-limbs thrust stiffly forwards and muscular spasms wracked the whole body resulting in the patient falling over. Salivation was marked and the jaws made champing movements lacerating the tongue. Respiration was extremely rapid and shallow and ceased completely on inspiration when the patient went into a prolonged clonic spasm.

Treatment: Artificial respiration was applied by means of alternate pressure and release over the thorax until voluntary respiration started again. Muscular spasms and jaw champing continued. Twenty ml. of antivenin was given intravenously. Muscular spasms stopped within a short while. The dog remained prone but calm for a further six hours and then recovered completely.

Identification: An 'Mfesi was seen within half an hour of the time of strike in the area where the strike had occurred.

2. *GREY KITTEN*

Symptoms: Site of bite could not be located. Seen within one hour of strike. Hypersensitivity and salivation.

Treatment: Five ml of antivenin intravenously and five ml subcutaneously. Complete recovery by the following day.

Identification: The 'Mfesi was destroyed immediately after treating the cat.

3. *BANTAM ROOSTER*

Symptoms: Site of bite could not be located Seen within four hours of strike. Marked hypersensitivity and muscular spasms. The head and neck were pulled over backwards at the slightest noise or movement and the fowl collapsed on the ground.

Treatment: Ten ml of antivenin intraperitoneally. The rooster died a few hours later.

4. *TWO MUSCOVY DUCKS*

Both ducks were found dead on their nests. They had been bitten on the breast and the bite was surrounded by haemorrhage and congestion in the sub-cutis.

C. BLACK MAMBA (*Dendroaspis polylepis polylepis*)

1. *DRAKENSBERGER HEIFER CALF*

Symptoms: This heifer had been collected with the rest of the herd for artificial insemination purposes[5]. She was seen at the time of strike which occurred on the face or neck. Torticollis set in immediately with the neck bent towards the left hand side. Salivation and slight champing of the jaw were present. Severe bloat developed within 15 minutes.

Treatment: Ten ml antivenin intravenously within ten minutes. Ruminal puncture to releave bloat. Ten ml. antivenin intravenously approximately one hour later. Antibiotic supportive therapy. The heifer died two days later showing torticollis and a complete disinterest in her surroundings.

Identification: Black Mamba seen at the time of strike.

2. *SUSSEX BULL*

Symptoms: The bull was collected with the rest of the herd and driven along the road to the dip. The bite occurred during this herding. Site of bite could not be located. Seen within minutes of strike. The bull collapsed at the dip, breathing was laboured, the eyes rolled wildly and the limbs projected stiffly, death was imminent.

Treatment: One hundred ml of antivenin intravenously. As the last 10 ml vial of antivenin was administered the bull got up and charged the

author and his assistants. It then charged and battered every shrub and bush in the vicinity. Recovery was immediate and complete.

Identification: The herdboys identified the snake as a Black Mamba soon after the strike had taken place.

3. *OLD ENGLISH GAME HEN*

This hen was nesting in a shed. A Black Mamba, which was being chased by the garden boy, fell from the shed roof on to the hen and bit it on the head. Death was instantaneous. The snake was destroyed.

D. 'MFESI OR MAMBA

The following cases are placed in this group on symptomatology alone as no identification of the snakes concerned was possible.

1. *ONE SEALHYAM X AIREDALE DOG, ONE POINTER DOG AND A MONGREL BITCH*

Symptoms: The three dogs were bitten at the same time. The sites of these bites could not be located. Seen within an hour of strike. Marked hypersensitivity, muscular spasms, heads pulled backwards, jaws champing, salivation, limbs rigid, shallow rapid respiration followed by complete cessation of breathing.

Treatment: The Sealyham was given 30 ml of antivenin intravenously which resulted in some relief but the dog was still hypersensitive. Sagatal* was administered intravenously until the dog was anaesthetised. Heart beat was extremely irregular, a beat was missed every five to eight beats. The dog remained anaesthetised for practically 24 hours and then made a complete recovery.

The Pointer was given a tranqilliser (Diquel) intravenously, no alleviation of symptoms was noticed. This dog and the bitch were destroyed *in extremis*.

2. *FOX TERRIER BITCH*

Symptoms: Site of bite could not be located. The owner brought the bitch in within eight hours of strike. The bitch was hypersensitive but extremely weak and near collapse. Salivation and diarrhoea were marked.

Treatment: Twenty ml of antivenin intravenously. The bitch made a complete recovery within the next few days.

3. *FOX TERRIER DOG*

Seen within 16 hours of strike. Symptoms and treatment were identical to those just described above but the dog died the following day.

4. *BRAHMAN COW*

Symptoms: A diagnosis of snake-bite was made on the symptoms and on their response to antivenin treatment. Site of bite could not be located. The cow was seen within a few hours of the commencement of symptoms. Hypersensitivity, on occasions the head was pulled backwards and the cow walked with the forelimbs extended stiffly. No temperature, bloodsmear negative.

Treatment: Twenty ml of antivenin intravenously. Complete recovery.

E. VENOM IN THE EYES

1. *SEALYHAM X AIREDALE DOG*

Aetiology: An 'Mfesi attacked by three dogs, spat venom into the eyes of one.

Symptoms: Marked lachrimation, hyperaemia of the conjunctiva.

Treatment: Ten ml of antivenin intravenously Both eyes were bathed with milk. Complete recovery.

Discussion

The symptoms described for Puff-adder, 'Mfesi and Mamba bites are similar to those listed by FitzSimons[3] but in addition strychnine-like symptoms were encountered with Mamba and 'Mfesi bites. Wall[12] in a detailed account of the effects of viper and cobra venom on dogs and

* Maybaker brand of Pentobarbitone Sodium.

fowls states that salivation is a constant feature of cobra- and does not occur with viper-venom. He also demonstrated the marked effect cobra venom has on respiration. Both these observations were confirmed in the present cases.

FitzSimons[4] states than when aroused a Mamba will bite and release its hold immediately afterwards to be ready for a further strike and quotes cases in which a number of dogs have been killed by one Mamba. This appears to have occurred in the above case report of the two dogs and a bitch bitten at the same time. Further evidence incriminating a Mamba in this attack is the fact that Mamba venom causes a wild and rapid pumping of the heart[4] and one of these dogs exhibited cardiac irregularity.

Schöttler[9] demonstrated the importance of early antivenin treatment in snake-bite. He injected mice subcutaneously with the venom of either *Bothrops jararaca*, (Pit viper) which causes circulatory collapse and local necrosis or *Crotalus terrificus* (Rattle snake) which causes respiratory collapse by peripheral paralysis. After various time intervals he administered specific antivenins by either the subcutaneous or intravenous routes. In the case of *Bothrops* venom antivenin had to be administered within four minutes subcutaneously or 215 minutes intravenously to ensure survival of 50% of the mice, in the case of *Crotalus* venom these time intervals were 116 and 319 minutes respectively.

These findings are confirmed by the present observations as in only one case (the fox terrier bitch) did the animal survive when antivenin was not administered immediately. Intravenous administration of antivenin being the route of choice[7].

The death of the Drakensberger heifer, despite immediate administration of antivenin can be ascribed to the fact that the polyvalent antivenin used is only partially effective against the venom of the Black Mamba.[1] The Sussex bull survived a Black Mamba bite because of the large amount of antivenin administered and the short time interval between strike and administration.

Supportive therapy in the form of artificial respiration in cases of respiratory collapse, general anaesthetics in cases of extreme hypersensitivity and antibiotics to prevent secondary bacterial infection of a bite, is of great importance. In all cases animals should be kept quiet after treatment as cardiotoxic elements in the venom may cause cardiac collapse several days later[3].

Several workers have tried various drugs as non-specific treatments for snake-bite, few with much success. Schöttler[10] used antihistamine, ACTH, cortisone, hydrocortisone and local anaesthetics in mice and guinea pigs experimentally injected with venom. In his experiments there was no statistical evidence in favour of the application of any of these drugs. Parrish, Scatterday and Moore[8] used the antihistamine Phenergan in dogs injected with venom. This drug appeared to potentiate the toxicity of the venom as the treated dogs died more quickly than the untreated controls. Christensen and de Waal[2] tested the efficacy of a 10% magnesium sulphate solution and a 5% watery solution of carbolic soap injected locally in guinea pigs experimentally injected with venom. The magnesium sulphate solution afforded no protection while the carbolic soap was effective against Cape Cobra and Ringhals venom but not Puff-adder venom.

To combat the high mortality from snake-bite in the ranch's cattle[6], the herd boys were paid a bonus for each poisonous snake they killed. Puff-adders and 'Mfesis were worth one rand each while Mambas were worth four rand each. Some herd boys brought in as many as four Black Mambas and three 'Mfesis in one calendar month. Contrary to the observations of Mason[7] the author found the Zulu herd boys particularly accurate in identifying snakes as it was seldom that a non-poisonous snake was presented as a claim for a bonus. Chiefly because of this system cattle mortality on the ranch due to snake-bite decreased from above 40 head in 1958 to 20 head in 1960, the number of poisonous snakes killed decreased correspondingly.

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FOCAL SYMMETRICAL ENCEPHALOMALACIA IN SHEEP IN SOUTH AFRICA

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SUMMARY

1. Focal symmetrical encephalomalacia (F.S.E.) in sheep is reported for the first time in the Republic of South Africa.

2. The literature on this condition is briefly reviewed.

3. A description of the history, clinical symptoms, macroscopic and microscopic pathology is given.

4. The disease condition, as seen in South Africa, is compared to cases described from New Zealand and the possible association of the toxin of *Cl. welchii* type D to the malacic lesions in the brain, is discussed.

INTRODUCTION

Hartley (1956)[1], described the occurrence of a specific brain abnormality in lambs in New Zealand, characterised by lesions of a focal and symmetrical encephalomalacia. On at least ten properties where this condition was diagnosed there were also simultaneous losses due to enteroxaemia. In the majority of the outbreaks reported by Hartley, in addition to the typical cases of enterotoxaemia, a certain number of lambs showed a wide variety of neurologic symptoms and lived 2 to 7 days before dying. On post mortem examination some of these cases showed lesions frequently associated with proven cases of enterotoxaemia. Focal symmetrical lesions in the brain, varying from haemorrhages and/or gelatinous softenings to lesions consisting of yellowish-grey, gelatinous material, according to the age of the lesion, were observed. Lesions were present in the region of the internal capsule, adjacent to the basal ganglia, lateral to the iter beneath the posterior corpora quadrigemina and in the cerebellar peduncles, in these cases.

In other cases there were no visceral lesions associated with enterotoxaemia present and lesions were restricted to the central nervous system. Almost all animals showed a similar symmetrical distribution of lesions in the same neuro-anatomic areas. No inclusion bodies were found and no significant organisms were cultured from affected brains. Attempts to transmit the disease by intracerebral inoculations of affected brain material into healthy lambs, failed.

The copper content of the livers of some of the lambs was examined and was found to be within the normal range. From some of the lambs *Cl. welchii* type D toxin was demonstrated from the ileal contents. The suggestion was made that the disease might be due to *Cl. welchii* type D toxin in animals surviving the acute disease.

According to Innes and Saunders (1962)[2], Jubb (1957) observed the same condition in sheep in Ontario, Canada. Barlow[4], (1958) and Robertson and Wilson, (1958)[5] described a similar condition in sheep in the British Isles. Hadlow, (1956)[3] in Montana, observed a lamb with symmetrical haemorrhagic lesions in the brain identical to those seen in New Zealand, and a

possible connection with enterotoxaemia was also considered.

In 1961, Griner[1c] described natural and experimental, and acute and subacute forms of *Cl. welchii* type D intoxication in sheep. Microscopic foci of softening or liquefaction necrosis were found in the basal ganglia, thalamus, internal capsule, substantia nigra, subcortical white matter and cerebellum of most cases with acute experimental type *D* intoxication. The anatomical sites of lesions in acute intoxication compared favourably with those observed grossly in subacute enterotoxaemia. He also studied the chronclogic pathogenesis of lesions in mouse brains caused experimentally by *Cl. welchii* type D toxin.

In this paper we wish to record the occurrence of a similar disease syndrome in sheep, in the Republic of South Africa.

History

The following is a short account of the outbreaks of mortalities amongst sheep, in which cases of focal symmetrical encephalomalacia were diagnosed:—

(*a*) During March 1962, 37 deaths occurred in a flock of 650 lambs running on green oats, on a farm in the Graaff-Reinet district. These animals were immunized against enterotoxaemia for the first time only after the initial deaths took place, which appeared suspicious for this disease. Six animals seen 3 days after vaccination showed nervous symptoms and the brains of two of these cases, one which died naturally and one which was killed *in extremis*, were collected for histopathological examination.

(*b*) In May 1963, mortalities occurred in a group of 6 to 7 months old lambs, running on lands containing green oats and green lupines, on another property in the same district. No deaths took place in older sheep that grazed on the same lands. The lambs had been vaccinated once against enterotoxaemia at the age of 2 to 3 months. The older sheep were immunized against this disease the previous October, and annually, before that. One of the lambs that exhibited nervous symptoms was forwarded alive to the Veterinary Research Institute, Onderstepoort, for examination.

(*c*) The brain of a 10 months old wether which had shown inappetance, stupor and aimless wandering for three days before it died, was received from the Graaff-Reinet district during May, 1963. Deaths occurred in the flock from which this animal originated that closely resembled enterotoxaemia. These sheep were grazing on Karoo veld and were vaccinated against enterotoxaemia 4 days before the first animals died. The sheep from which the brain was received, died 9 days after vaccination.

(*d*) During July, 1963 56 out of 400 sheep grazing on veld, suplemented by Japanese Radish, died over a period of 3 weeks, on a farm in the Standerton district, Transvaal. Mortalities in all age groups occurred shortly after feeding with crushed maize was started. The flock was vaccinated against enterotoxaemia during 1961, but not during 1962 or 1963, previous to the occurrence of deaths. A diagnosis of enterotoxaemia was made on the post mortem appearance of two sheep submitted to Onderstepoort, for autopsy. One eight months old wether exhibiting nervous symptoms was also obtained alive, for clinical examination and autopsy, from the same flock.

Mortalities ceased in all the flocks in which the abovementioned outbreaks took place, subsequent to the development of immunity, after vaccination with enterotoxaemia vaccine.

Symptoms

Many of the cases seen in the first outbreak in the Graaff-Reinet area during 1962, were found in a position of lateral recumbency and showed opisthotonus, intermitted galloping movements of all four legs, twitching of the face muscles, nystagmus, grinding of the teeth and hyperaesthesia. The sheep forwarded alive from the Graaff-Reinet district during May, 1963 exhibited marked mental dullness and dejection. It wandered aimlessly for hours on end with its head lowered, taking no notice of its surroundings. Occasional head pressing and circling was also manifested by this animal. Similar symptoms were observed in the case from which the

brain only was received, also from the Graaff-Reinet district, and in the animal obtained alive from the Standerton district. The latter animal also showed torticollis and impairment of vision. No temperature was present in any of the cases examined clinically. Animals showed symptoms for 3 to 14 days before dying.

Material and Method

Three brains of sheep showing encephalomalacic lesions were obtained in 10% formalin for histopathological study. Two cases were examined clinically before being autopsied. The brains and spinal cords of these animals were also fixed in 10% formalin.

Coronal sections were cut from the fixed organs and blocks were selected for histopathological study. Microscopic sections were cut at 3μ thickness and stained with haematoxylin-eosin, various myelin stains and special stains for fungi and bacteria.

Antitoxin values against *Cl. welchii* type D toxin of sera from sheep from the flock in the Standerton district were determined in terms of Welcome units.

Pathology

(*a*) Macroscopic Pathology

In none of the cases were macroscopic lesions present in any organs other than the brain. The brain lesions were present in the region of the internal capsule lateral to the basal ganglia, in all cases. In some, lesions were also found in the cerebellar peduncles and thalamus, and in one case, in the white substance of the frontal gyri. The lesions were of a focal nature, bilaterally symmetrical in distribution and were seen as irregularly outlined yellowish-grey gelatinous foci of malacia, varying in size from 1.5 cm. in diameter to foci which were only detected on microscopic examination. Some of the larger lesions showed irregular cavitation. No lesions were present macroscopically in the spinal cord in three cases in which this organ was available for examination.

In all the outbreaks in which cases of F.S.E. were encountered, other animals in the flocks, dying of the acute disease showed the gross pathological features usually associated with enterotoxaemia[7].

(*b*) Histopathology

Histopathologically the lesions were approximately at the same stage of development in all the cases. Compared to the description of the lesions observed by Hartley[1], the cases under review corresponded to the more advanced stages of the evolution of the brain lesions. None of the cases examined by us showed the early stages, consisting of focal areas of non-inflammatory parenchymal necrosis and vascular changes, as was seen by Hartley[1].

The lesions consisted of irregularly outlined focal areas of malacia, containing numerous granular corpuscles (Gitter cells) in every instance. Proliferation of capillaries was present in and adjacent to these malacic foci. Marked vacuolation of the nervous tissue immediately surrounding these foci was seen in the haematoxylin-eosin stained sections. Staining with myelin sheath stains showed depletion of myelin in these vacuolated areas. At the periphery of the lesions, numerous, oval to round, with fewer elongated or irregularly shaped, eosinophilic structures were present. These eosinophilic bodies showed a finely granular structure and apparently represented swollen degenerated axis cylinders.

In the two cases showing the most extensive lesions, peri-vascular infiltration of some of the bloodvessels, in and adjacent to the malacic foci, with varying numbers of round cells were observed. The infiltrating cells were mostly plasma cells, while fewer cells exhibiting small hyperchromatic nuclei, similar to small lymphocytes, were also noticed. These infiltrations were restricted to some of the larger lesions and then were only seen around some vessels. They were absent in the majority of lesions, and were not observed in other parts of the brain or meninges.

No inclusion bodies were observed in any of the sections examined and no bacteria of fungi could be demonstrated in sections stained with various staining methods to show up these organisms. Microscopic examination of sec-

tions of the spinal cords of three cases did not reveal any lesions. The most extensive microscopic lesions were observed in the two cases from outbreak (*a*), which also showed a shorter course than the rest of the cases. The lesions observed in the white substance of the frontal gyri in one case were also bilaterally symmetrical and histologically consisted of small focal areas of vacuolation which proved to be areas of early demyelination with the Marchi staining method.

The malacic lesions in the brain were restricted to the white matter. None were observed in the grey matter.

Immunity against Enterotoxaemia

No antitoxin could be demonstrated in the sera of two sheep bled before vaccination against this disease from the flock in the Standerton district. In ten healthy sheep, from the same flock, which were selected at random and bled 3 weeks after vaccination, antitoxin levels from 0.3 to 30 units/m.l. were found.

Copper content of livers

Liver material from four cases was examined chemically for copper content. All four sheep were found to have copper figures within the normal limits.

Discussion

Taking the epizootiology and the distribution, macroscopic and histological appearance of the lesions seen by us, into consideration, it appears that we are dealing with the same condition as observed in New Zealand. The age of the lambs affected in New Zealand as described in the first report on this condition varied from a few days to ten weeks[1]. The cases observed in this country occurred in sheep of older age and the early haemorrhagic lesions as described by Hartley were not seen. According to this author the early lesions, consisting of non-inflammatory necrosis and haemorrhages showed a transitory neutrophilic invasion, followed on the second and third days by the appearance of faintly granular eosinophilic strutures at the periphery of the lesion which were thought to be swollen axis cylinders. The foci of malacia were invaded by granular compound corpuscles from the third day and by the tenth day they were occupied by these cells. Compared to the abovementioned, the lesions described in this resport, were thus all of an advanced nature.

Based on the demonstration of *Cl. welchii* type D toxin in the ileal contents of one lamb in a group of four lambs that died without showing any symptoms and in which lesions of encephalomalacia were found in three, together with lesions found at post mortem simulating enterotoxaemia, Hartley[1] states that these findings tend to incriminate the toxin of *Cl. welchii* type D toxin as the cause of the brain lesions.

Innes and Saunders (1962)[2], suggested that the toxin of *Cl. welchii* type D may be the cause of the brain lesions observed by Hartley[1], but only if the animals survived, in contrast to the sudden collapse and death seen in the acute cases of enterotoxaemia. They also pointed out that Morrison and Zamecnick (1950)[8], produced experimental demyelination *in vitro* with the alpha toxin of *Cl. welchii*.

Jubb and Kennedy (1963)[6], remarked apparently from personal experience, that it appears, from epidemiological and serological evidence, that sub-acute cases of enterotoxaemia may occur in sheep and be followed by recovery. According to them, there is some, but little, doubt that brain lesions occur in some lambs with enterotoxaemia of this type, which is not immediately fatal. They further stated that in these cases the brain lesions are of two patterns and that the lesions in each, are quite symmetrical bilaterally. The commonest pattern being haemorrhage and softening in the basal ganglia, internal capsule, dorsolateral thalamus and substantia nigra. There are apparently some minor variations in the distribution of lesions in this pattern. The second pattern is that of lysis and liquefaction of the white matter of the frontal gyri, sparing only the communicating U fibres.

Our observations are in accordance with those of the abovementioned authors. All the cases examined by us were associated with outbreaks

of mortality highly suspicious for enterotoxaemia. Further support is also lent to the hypothesis that the lesions of focal symmetrical encephalomalacia in sheep is associated with the toxin of *Cl welchii* type D by the finding that sera from sheep in the flock from the Standerton district did not contain protective values of antitoxin against the toxin of *Cl. welchii* type D. This finding is no proof that mortality in this flock was due to enterotoxaemia. But when considered with the following facts; that mortality ceased after vaccination against enterotoxaemia, that no further cases of F.S.E. occurred in this flock as well as in the other flocks in which cases of this condition were found after development of immunity against enterotoxaemia, that deaths occurred in these flocks under conditions highly suitable for the occurrence of enterotoxaemia and that the acute deaths accompanying the cases of F.S.E. showed gross pathological changes usually associated with enterotoxaemia, points to the possible association of F.S.E. with the toxin of *Cl. welchii* type D.

Griner (1961)[10], examined the brains of 31 lambs killed experimentally by one or more intraveneous dose of trypsin-activated *Cl. welchii* type D toxin at a dosage rate of 3.7 to 9.3 µ.g. per pound of body weight. He also described the lesions observed in the brains of 19 field cases of natural enterotoxaemia. These experimental and natural cases he classified as acute or sub-acute on the basis of clinical symptoms, macroscopic and histopathological lesions. In 42 acute cases no macroscopic lesions were discernible in the brain. The microscopic lesions present in these acute cases were characterised by vascular congestion: degeneration of endothelium and walls of the vessels, pronounced perivascular oedema and varying degrees of intercellular oedema. The distribution of these lesions in the brains of the acute cases was mentioned in the introduction of this article. Histopathologically the lesions in the group of 8 sub-acute cases were of the same general character as those of lambs with the acute form, but were more severe and further advanced. In three of these sub-acute cases multiple bilateral symmetrical foci of malacia were seen in the same neuro-anatomic areas in which the microscopic lesions appeared in the acute cases.

The time interval between completion of toxin injection and onset of symptoms in the acute cases varied from none to four hours, while the period from onset of symptoms to death varied from 30 minutes to 4 hours. In the subacute cases the duration of the symptoms was longer, one experimental case showing signs of intoxications for 11 hours, and two natural cases for 36 to 48 hours before death.

The chronologic pathogenesis of the brain lesions caused by intravenous administration of *Cl welchii* type D toxin in mice as studied by Griner[10] appeared to be related to an initial increase in vascular permeability followed by oedema, softening, liquefaction necrosis and healing by glial scarring. At no stage of the development of the neurologic lesions in mice were compound granular corpuscles recognised. These cells were also absent in the lesions of experimental and natural cases in sheep examined by Griner[10]. According to Griner the anatomical distribution and character of the lesions observed by Hartley, resembled those observed in his experimental cases, with the exception that no haemorrhages or granular corpuscles were found in the experimental cases and that oedema was not mentioned in F.S.E. He further states that although the evidence presented by Hartley indicates that F.S.E. may result from the toxin of *Cl. welchii* type D it is not conclusive since there are some variations in the nature of the lesions observed by Hartley as compared to that of experimental type D intoxication and remarks that if malacic lesions are present in the cerebral cortex of lambs with F.S.E., this condition might more closely resemble polio-encephalomalacia as described by Jensen *et al*[11].

It should however be kept in mind that the lesions in most of Griner's cases were artificial produced lesions, with the animals surviving a relative short period after administration of the toxin as compared to the longer periods of survival observed in Hartley's cases and in all the cases seen by us. One would expect that in the more advanced or chronic cases, should the animal survive for a number of days, that the vascular changes will no longer be as evident as in the acute phases of the lesion, and that compound granular corpuscles will make their appearance in necrotic areas. Griner did not state what number of the 19 field cases examined by

him, he considered as acute or sub-acute. Neither did he give an indication as to the duration of illness of these cases before death.

Focal symmetrical encephalomalacia has not been reported before in sheep in the Republic of South Africa. Smit (1964)[9], observed cases in sheep showing nervous symptoms, associated with outbreaks of enterotoxaemia and following a protracted course of a few days to three weeks before, dying, in the past. It was suspected by him that such cases were due to the effect of the toxin of *Cl. welchii* type D on the brain. The brains of such animals were not examined for lesions. Sporadic cases of F.S.E., associated with outbreaks of enterotoxaemia are probably more widespread, in this country than is realised.

ACKNOWLEDGEMENTS

The Chief, Veterinary Research Institute, Onderstepoort, is thanked for permission to publish this report. Thanks are also due to Mr. F. Visser, Department of Bacteriology, for the determination of antitoxin levels of sera, Mr. S. J. Myburgh, Department of Biochemistry, for the determination of the copper content of livers, Mr. A. M. du Bruyn for the photomicrographs, Dr. R. C. Tustin for assistance in preparing the manuscript and the technical staff of the Department of Pathology for the preparation of microscopic sections.

FOOTNOTE:

Since the completion of this article, small bilaterally symmetrical lesions of encephalomalacia, identical to those observed in natural cases of F.S.E., were observed in the internal capsule of the brain in one experimental sheep, by the senior author. This animal which was one of a series of priliminary experimental cases of *Cl. welchii* type D, intoxication, received sublethal doses of trypsin-activated toxin intravenously over a period of two days. On the third day it showed nervous symptoms and slowly recovered and appeared normal on the sixth day after the administration of the initial toxin dose. The serum of this animal showed an antitoxin level of 0.7 units/m.l. before administration of toxin. Six days after the beginning of toxin administration the antitoxin value was 2.0 units/m.l. and further large doses of toxin failed to provoke any symptoms. The animal was destroyed seven days after symptoms were observed, for autopsy.

This single case does not permit the drawing of definite conclusions, but points to the possibility that F.S.E. in sheep may be associated with a very low level of antitoxin value, insufficient to protect the animal fully but sufficient to prevent the acute deaths usually seen in enterotoxaemia.

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Fig. 1 — Focal malacic lesions in internal capsule.

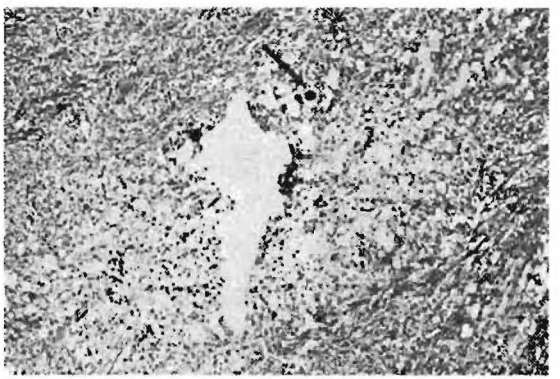

Fig. 2 — Cerebellar Peduncle. Small focal lesion. Note eosinophilic bodies at periphery of lesion (arrow). X. 70 H. & E.

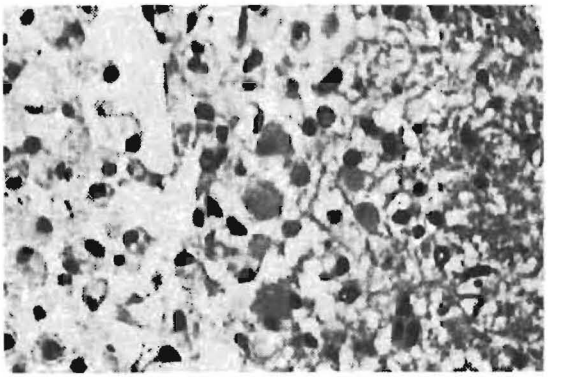

Fig. 3 — Internal capsule. Periphery of malacic lesion with compound granular corpuscles on the left. adjacent vacuolation area with eosinophilic bodies (middle) and unaffected nervous tissue on right. X. 440. H. & E.

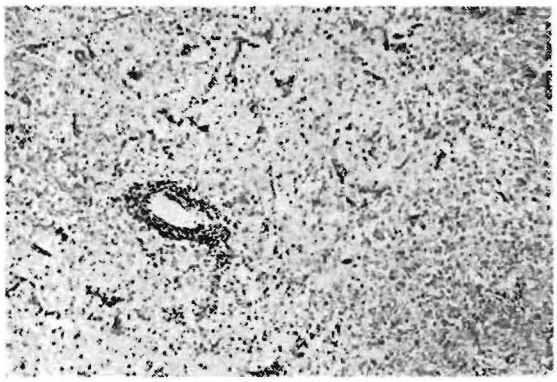

Fig. 4 — Cerebellar Peduncle. Malacic lesion showing numerous compound granular corpuscles and infiltration of round cells in perivascular space of vessel towards centre of lesion. X 70. H. & E.

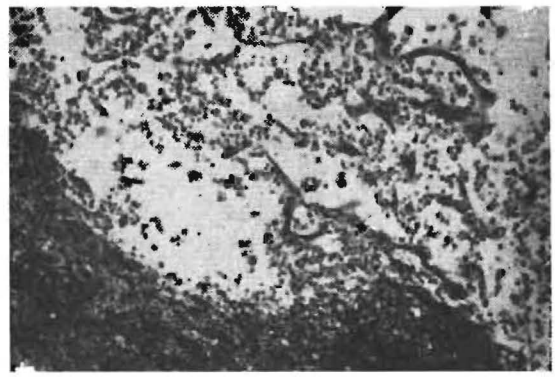

Fig. 5 — Internal capsule. Compound granular corpuscles and proliferation of blood-vessels in malacic lesion. X. 70. Luxol Fast Blue Stain.

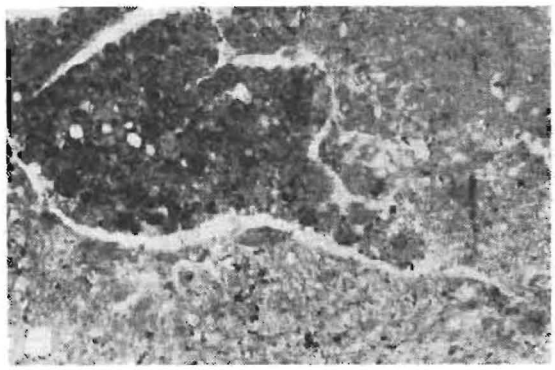

Fig. 6 — Malacic lesion in internal capsule stained Marchi's method. X. 175.

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RECENT RESEARCH ON BIOCHEMICAL POLYMORPHISM IN LIVESTOCK*

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Polymorphism in general is the occurrence together of two or more varieties in the same population at the same time in such proportions that the rarest of them cannot be maintained by mutation alone. Biochemical polymorphism is the occurrence of varieties attributed to biochemical differences which are under genetic control. There is a growing conviction among research workers that a better understanding of biochemical differences and their genetic bases may lead to selection procedures which will be important supplements to conventional selection for improvement of livestock. Aside from its possible application to animal improvement, knowledge concerning genetic control of biochemical polymorphism will increase our understanding of how genes act and how heterozygosity is maintained in livestock populations.

The gene-controlled diversity of chemical structure hitherto revealed has provoked much discussion as to its origin maintainance and significance. The geneticist, microbiologist and the biochemist are interested in these very basic problems and are aware of the fact that the differences observed demand explanations which can be given only by a fusion of knowledge of all disciplines involved.

This paper excludes red cell antigens not because they are outside the field of gene-controlled biochemical polymorphism but because they comprise a large and distinct class, too comprehensive for the present discussion. Recent developments in the field of blood group studies in infrahuman species have been published by the New York Academy of Sciences[39].

Very recently new biochemical techniques have appeared which allow us to distinguish very small differences between protein molecules. These techniques are extensions of the well-known techniques of paper chromatography and depend on the migration of the protein molecules under electric gradient in specially treated blocks of starch gel[48]. The distance which a particular protein molecule will migrate under a given set of electrophoretic conditions is a function of the size of the molecule and the charge which it possesses. The sorting effect provided by the starch gel which hinders the migration of the larger protein molecules more than that of the smaller proteins is largely responsible for the improved separations over those obtained in other electrophoretic systems.

A complete standardization of all steps of the method is necessary, i.e. the preparation of buffers, through mixing of gels, actual electrophoresis, slicing and staining of gels, removal of the excess stain is absolutely imperative if a high degree of repeatability is to be achieved.

The observation of biochemical polymorphism follows a basic pattern. Firstly the observation of clearly distinguishable variants in a specific character is made. Then, the transmission of these variants is followed in related animals and their genetic basis and mode of inhertance is established. Using this information, appropriate gene frequencies are calculated from a random sample of the species and the observed frequencies of the various phenotypes are tested for agreement with those expected on the genetic hypothesis. Finally an attempt is made to establish correlations between blood polymorphism and fitness factors and furthermore, find possible clues as to the nature of the selective pressures involved in maintaining the observed balance of types.

* Lecture presented at a meeting of the South African Genetic Society on 23 June 1964.

Haemoglobins

The first protein analysed was human haemoglobin. A large number of types has been established and some of the variants are known at the level of molecular structure, e.g. Hb-A normal and Hb-S sickle cell are known to differ merely in the substitution of one amino acid—Glutamic acid—by another—Valine—at one of the two pairs of protein chains which constitute this molecule. The many different types of haemoglobins in human are mainly abnormalities with pathological changes which originated by mutations. The literature on this subject is reviewed by Allison (1958)[2].

Table 1

Number of different haemoglobin types in livestock (Schmid, 1962a)[46]

Species	*No. of alleles*
Cattle	2
Horse	(2)
Donkey	1
Mule	2
Pig	1
Sheep	2
Goat	2
Dog	1
Cat	1
Chicken	3

In domesticated animals, however, a true polymorphism could be established. Besides the foetal haemoglobin which is present only in the foetal and early life after birth two alleles are known which control the haemoglobin type in most species of domesticated animals.

It is interesting to note that in *horses* always two types are present which are not true allelomorphs. (Bangham et al., 1958)[16].

In our laboratory we recently started a new investigation on haemoglobins in equidae, since mules, donkeys and of course zebras have never been investigated in starch gel medium. Apparently we deal with different types of haemoglobins in these species indentified by different mobility in the starch block.

The haemoglobin polymorphism in *cattle* provides a number of problems due to the fact that some breeds show a clear polymorphism with two alleles and three possible phenotypes (A/A, A/B and B/B), while others present only one type (A/A). No satisfactory explanation can be given at this stage for this striking difference between breeds, which can be seen in Table 2.

The differentiation of the Jersey, Guernsey, South Devon, Brown Swiss and the African breeds from the others is most puzzling. The cattle haemoglobins illustrate a future possible use to which this type of study can be put, namely the slightly speculative study of the genealogy of particular breeds. Bangham and Blumberg (1958)[15] showed that the presence of the Bovine Hb^B-gene in Southern Europe and African

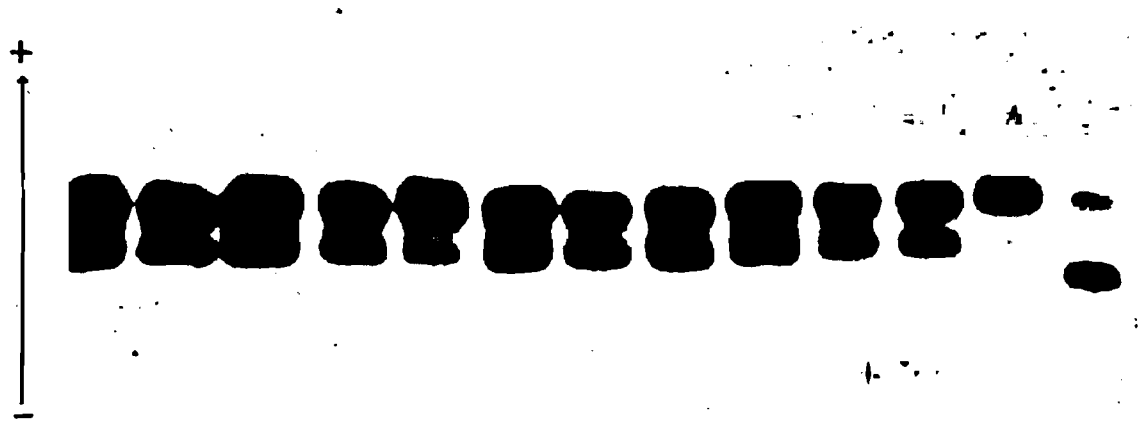

Fig. 1 — Haemoglobin types of 12 different horses together with one cattle sample of haemoglobin type AB.

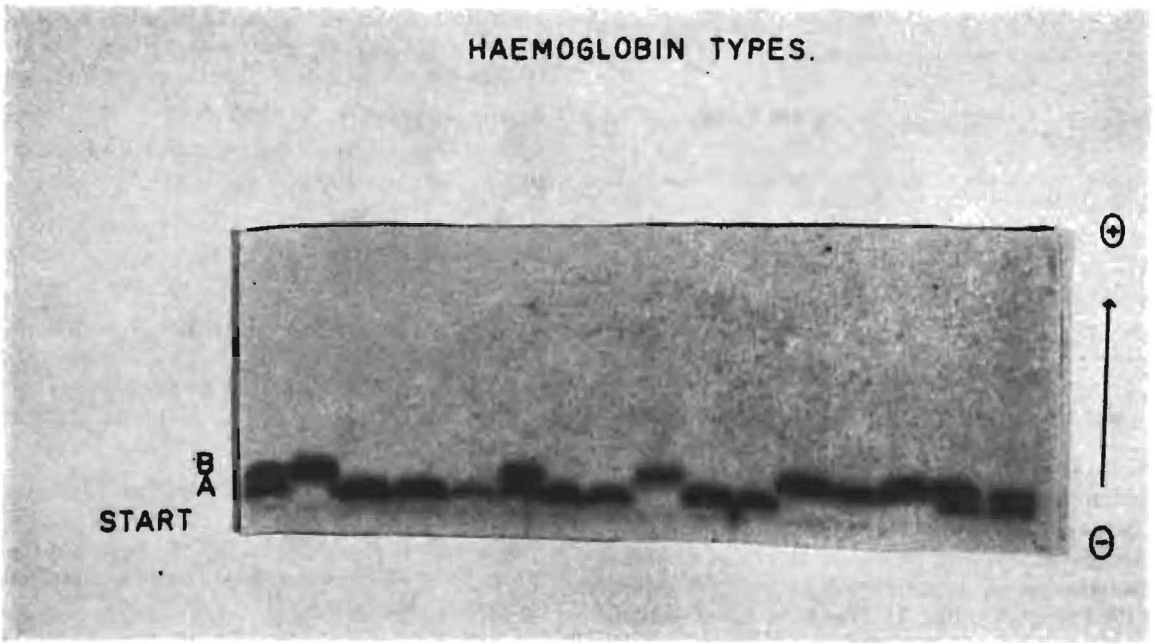

Fig. 2 — Haemoglobin differentiation in cattle, showing the thre possible phenotypes A/A. A/B and B/B.

TABLE 2

HAEMOGLOBIN TYPES IN SOUTH AFRICAN CATTLE BREEDS.

Breed	*Hb—A*	*Hb—B*	*Hb—AB*
Afrikaner	+	+	+
Friesian	-		
Jersey	+	+	+
Dairy Shorthorn	+		
Red Poll	+		
Brown Swiss	+	+	+
Beef Shorthorn	+		
Aberdeen Angus	+		
Ayrshire	+		
Hereford	+		
Drakensberger	+	+	+
South Devon	+	+	+
Devon		not tested	
Sussex	+		
Guernsey	+	+	+
Ankole*	+	+	+
Boran*	+	+	+

* Breeds from Kenya.

breeds of cattle was consistent with one of the suggested ancestral lines of the Jersey breed. It was postulated that the Jersey are descended from cattle of the ancient Indus valley civilization.

In the South African Jersey the frequency for gene Hb^A was established to be 0.55, thus the frequency for Hb^B is 0.45. In comparison herewith 140 Ankole samples in a Hb^A-frequency of 0.72 which is in contradiction to the original assumption that a high frequency of Hb^B and also an excess of heterozygotes would favour the animals in regard to trypanosomiasis and possibly also East Coast fever. Unfortunately, we are now unable to obtain more blood samples from Kenya, but our South African breeds alone offer interesting material for the study of these correlations.

It should be mentioned that in several breeds a further differentiation of Hb-A in three subtypes A_1, A_2 and A_3 was recently achieved in our laboratory (Osterhoff et al., 1964a)[43]. Studies of the inheritance of these three types are under way.

The practical use of the haemoglobin types in parentage determinations can be demonstrated by a single case where the sire was blood typed in California, but the haemoglobin type was not known. By testing mother-daughter combinations it could be proved that the bull had the haemoglobin type Hb^A/Hb^B.

TABLE 3

HAEMOGLOBIN TYPE OF BULL BRAMPTON SIXTH GENERATION.

Possible Matings Genotype			Obtained Offspring Genotype		
Sire		*Dam*	Hb^A/Hb^A	Hb^A/Hb^B	Hb^B/Hb^B
?	×	HbA/HbA...	6	1	0
?	×	HbA/HbB..	1	3	2
?	×	HbB/HbB...	0	1	0

In general we can say, that by haemoglobin determinations 3 to 4 per cent more parentage cases can be solved than by blood grouping alone. This applies, however, only to breeds with polymorphism in haemoglobin types.

In *sheep* the fastest moving haemoglobin is the haemoglobin A, in contradiction to all other species. The most complete survey of different breeds of sheep in regard to their haemoglobin polymorphism was recently given by Meyer (1963b)[38]. He listed all breeds so far investigated—53 in all—and demonstrated the frequency of the Hb^A-gene varying from 0.00 in the English Dartmoor breed to 0.97 in the German Heidschnucke. Here again, a great deal of assumption and speculation can be recognized in the discussions of different authors. Falconer (1961)[25] believes that the reason for the establishment of a polymorphism in a population is a selection advantage of the heterozygotes over the homozygotes. Huisman et al (1958)[32] were able to show a higher oxygen affinity of the red cells of Hb^A-type sheep, which is in agreement with the results of Evans et al., (1958a, b)[23,24] who stated that sheep in greater altitudes have a higher frequency of the Hb^A-gene.

From Meyers' results (1963b)[38] it can be learned that other breeds which are usually regarded as lowland breeds have a very high frequency of Hb^A which is in contradiction to the high-altitude-theory. But all breeds discussed in the three last mentioned papers have several characteristics in common, all of them pointing in the direction of good adaptability to unfavourable climatic and nutritional conditions.

An example of our own results show how difficult it is to give a suitable explanation for the polymorphism obtained.

These results show that we deal with complex influences which may act on the genetic differences between breeds.

The *pig* appears to have only a single haemoglobin (Rodnan et al, 1957)[45]. This is in contrast to the complexity of the pig serum protein. No explanations for this contrast have been given as yet.

In *fowl* the haemoglobins are heterogenous, but this heterogeneity is common to all samples examined. Rodnan et al. (1957)[45] suggested that synthesis of fowl haemoglobins may be controlled by at least three genes.

No further species shall be discussed here, only the foetal haemoglobin should be mentioned which is found in most species in the prenatal and early life after birth.

From Fig. 3 it can be seen how the foetal haemoglobin changes to adult haemoglobin and how the foetal haemoglobin can interfere with the identification of different types. In dizygous cattle twins, we were able to show that males are slower in exhibiting their proper haemoglobin type than females, the difference being at least four weeks. This difference has never been reported before.

TRANSFERRINS.

The method of differentiation of globulins according to the molecular size of proteins with starch gel as supporting medium has been widely used for the fractionation of human and

TABLE 4.—HAEMOGLOBIN POLYMORPHISM IN THREE SOUTH AFRICAN SHEEP BREEDS

Breed	Total tested	Phenotypes			Gene Frequency	
		AA	AB	BB	A	B
Karakul & crosses	670	12	200	458	.17	.83
Merino Grootfontein	267	80	121	66	.53	.47
Letelle	361	12	113	236	.19	.81

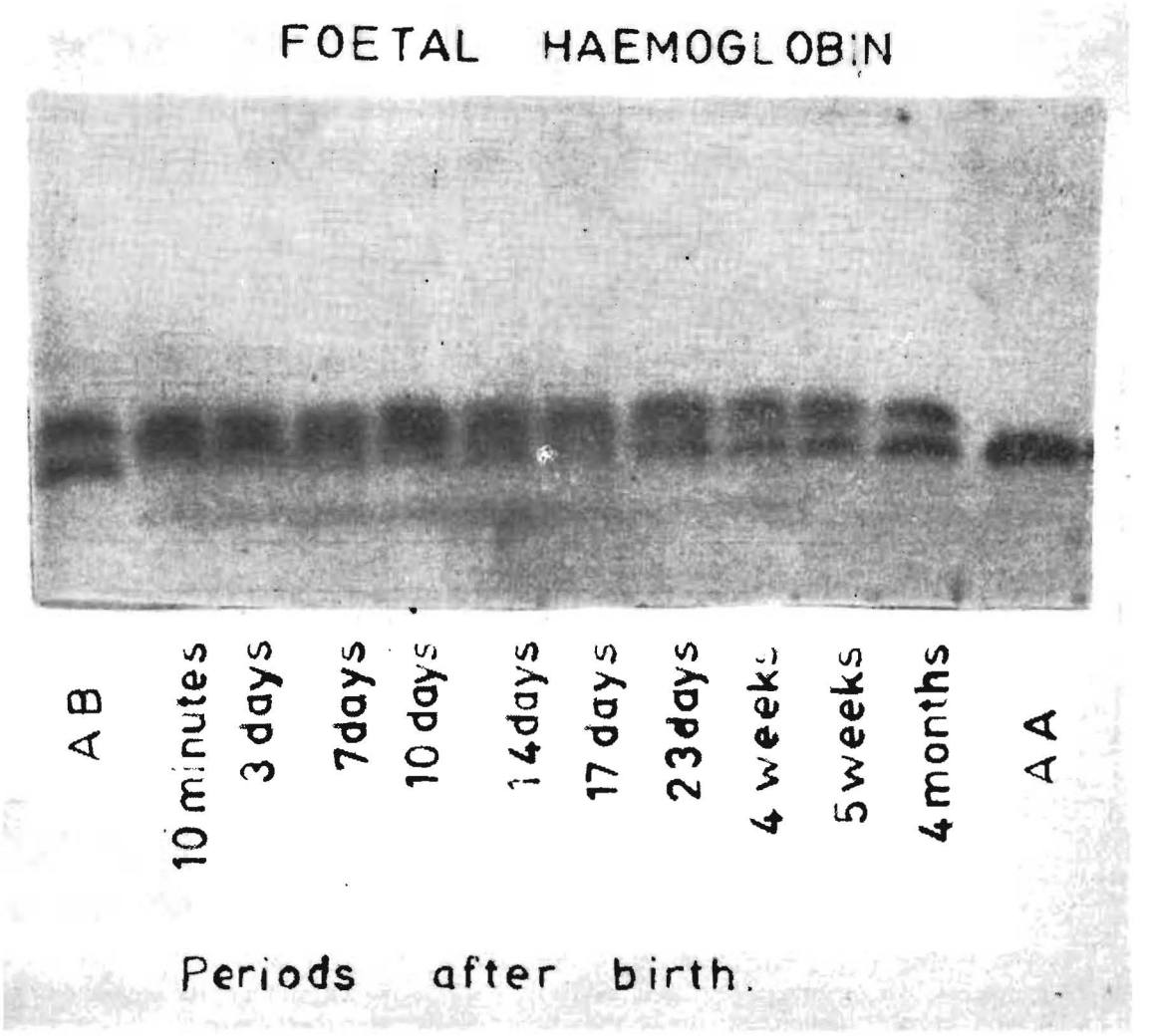

Fig. 3 — The canges from haemoglobin to adult haemoglobin.

animal serum proteins, especially the beta-globulins.

Genetical investigations carried out by Smithies et al., (1958)[49] and Ashton (1958a)[4] proved that the beta-globulin types were inherited according to Mendel's laws and the possible genetic mechanism could be established for some of our domesticated animal species. Giblett et al., (1959)[29] were able to prove that these genetically variable beta-globulins are the specific iron binding proteins, called transferrins. The experiments were performed with Fe^{59} isotopes added to the serum before electrophoresis and an autoradiograph of the gel was taken after the completion of the electrophoresis.

In horses the identification of the genetic background of the transferrins provided difficulties which were overcome by Braend et al., (1964)[18], only recently.

Sixteen transferrin phenotypes were observed in horses and attributed to the action of six codominant, autosomal alleles. Aware of the fact that a denomination of alleles must leave

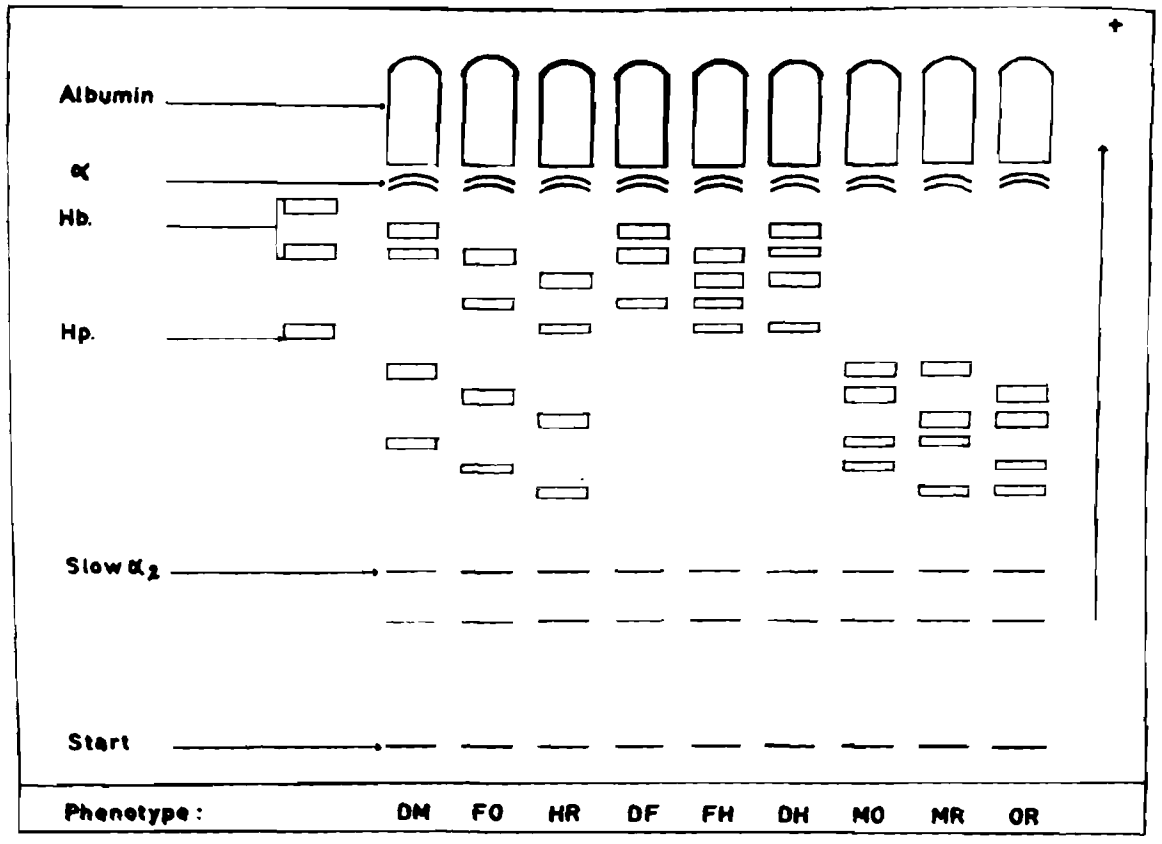

Fig. 4 — Diagram showing nine different transferrin types in hirses.

letters or numbers out for other alleles detected later, Braend et al.[18] designated the horse alleles Tf^D, Tf^F, Tf^H, Tf^M, Tf^O and Tf^R. The hypothesis of six alleles was tested by means of family data. No breed differences have been reported until now; our own data, which include the transferrins of mules, are also still incomplete.

Genetically controlled polymorphism in sera from normal cattle has been demonstrated by different workers (Hickmann et al., 1957[30]; Ashton, 1957[3], 1958a[4], 1959[7], 1960b[9], 1961[10]; Ashton et al., 1958[14]; Gahne et al., 1960[28]; Ogden, 1960[40]; Hojgaard et al., 1960[31]; Gahne, 1961[26]; Brummerstedt-Hansen et al. 1962[19]; Ashton et al., 1962b[13]; Schmid, 1962b[47]; Bushmann, 1963[20]; Datta et al., 1963[21]).

Fig. 5 shows a diagram of the protein differentiation after starch gel electrophoresis representing the six possible types of transferrins found in all breeds of bos taurus origin. These six phenotypes are attributed to three transferrin alleles designated Tf^A, Tf^D and Tf^E.

Here some of our own results will be reported. Serum samples from 3146 pure bred animals of 16 different breeds in South Africa and from the Boran breed in Kenya were collected. The breeds were classified according to their purpose into beef, dairy and dual purpose breeds, and according to their origin into imported indigenous and crossed breeds.

In the four dairy breeds included in our study interesting differences could be established as it

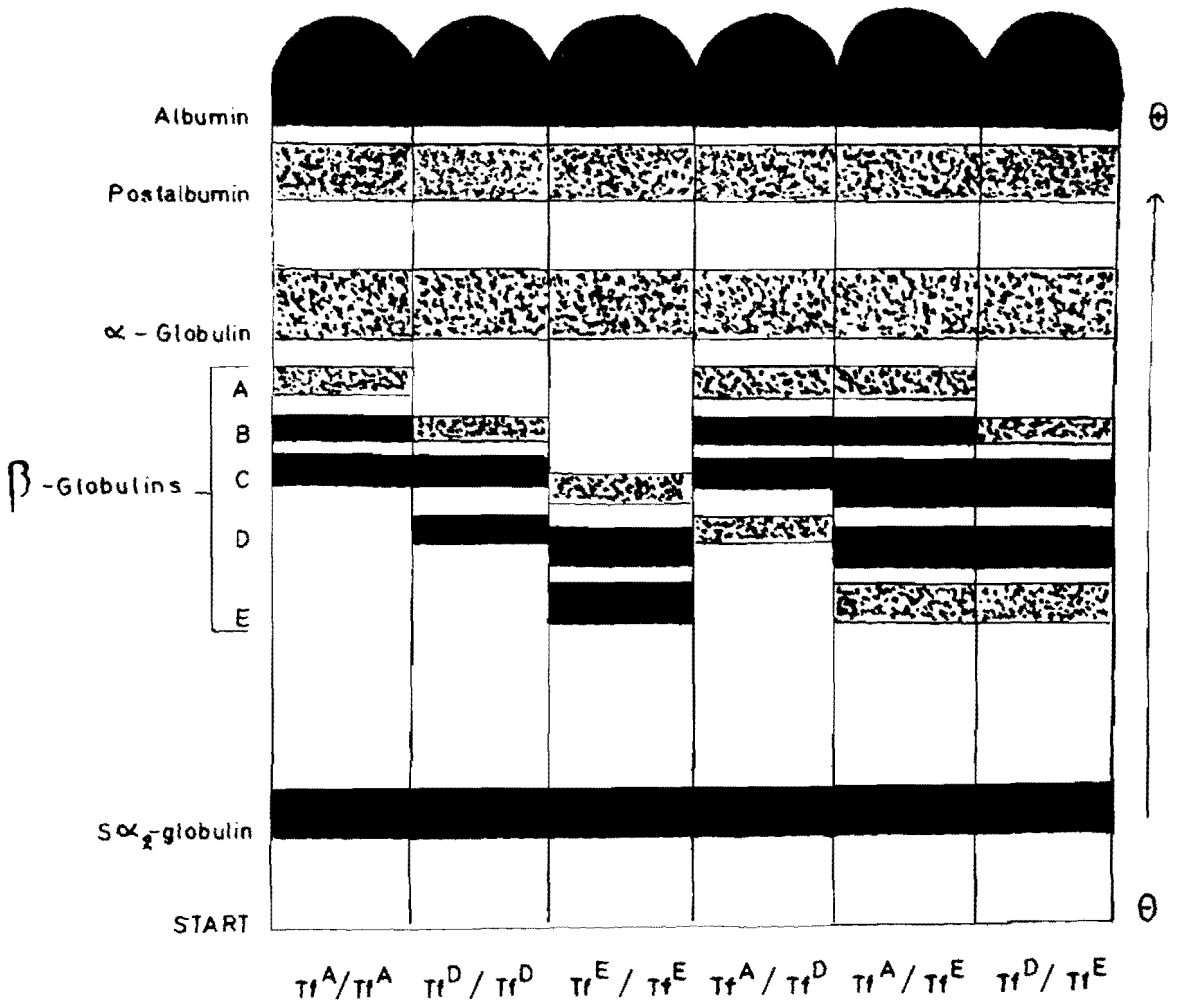

Fig. 5 — Diagram showing the six different transferrin types in cattle of Bos taurus origin.

can be seen in Table 5 where our results have been compared with those of other workers.

The results obtained with South African Friesians are much in agreement with those of Friesians in other countries. The Jersey and Guernsey results compare fairly well with those in other countries, but the Ayrshire in South Africa shows great differences with the most striking deviation in the frequency of Tf^D from that in other countries. This is by far the largest number of Ayrshires investigated so far and no reasons for the discrepancy can be given at this stage.

The situation in beef breeds is more complicated due to the fact that breeds from bos indicus origin are included. Ashton (1959)[7] described for the first time two new beta-globulin alleles in Zebu cattle. The two alleles were called Tf^B and Tf^E, the first one showing a mobility in starch gel between the zones of Tf^A and Tf^D, and the other having a relative mobility between the zones of Tf^D and Tf^E.

Fifteen transferrin types are thus possible:

AA
AB BB
AD BD DD
AE BE DE EE
AF BF DF EF FF

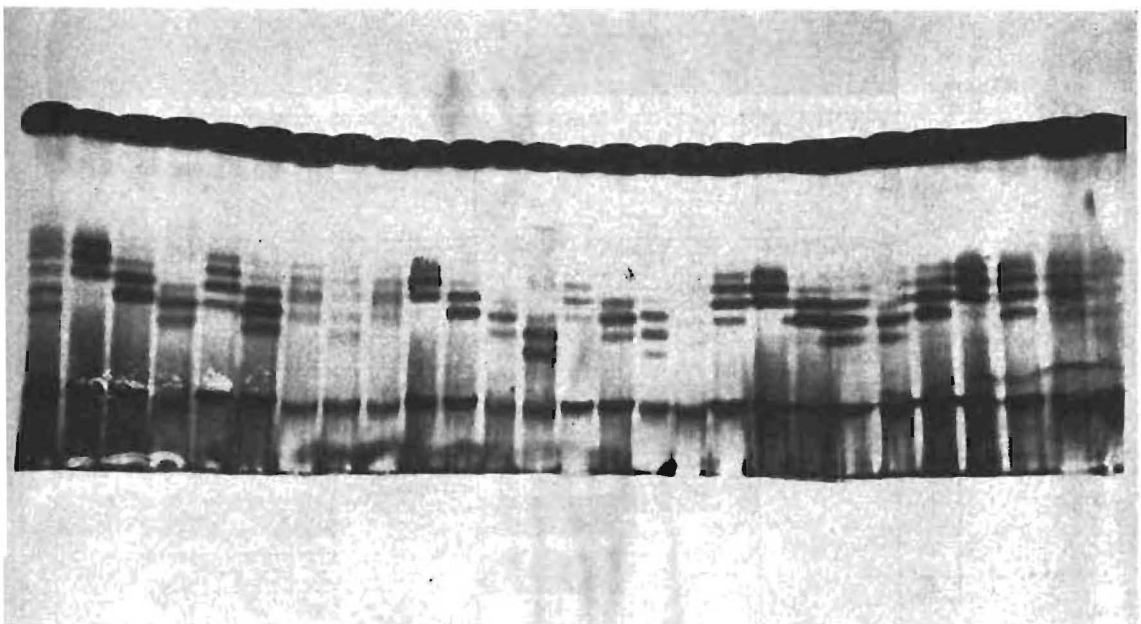

Fig. 6 — Actual protein differentiation after starch gel electrophoresis.

TABLE 5.—THE TRANSFERRIN GENE FREQUENCY IN SOUTH AFRICAN DAIRY BREEDS COMPARED WITH RESULTS OBTAINED IN OTHER COUNTRIES.

Breed	Country	Gene frequency			Total animals tested
		A	D	E	
Ayrshire	South Africa	.054	.805	.141	244
	Australia	.241	.583	.176	139
	Britain	.272	.641	.088	124
	Canada	.297	.453	.250	42
Friesian	South Africa	.435	.530	.035	116
	Britain	.463	.474	.062	261
	Canada	.563	.374	.063	102
	Denmark	.518	.459	.023	238
	England	.522	.447	.031	179
	Australia	.362	.638	.010	203
	Sweden	.485	.500	.015	204
	U.S.A.	.460	.530	.010	473
Guernsey	South Africa	.318	.682	—	99
	Britain	.494	.507	—	62
Jersey	South Africa	.678	.322	—	276
	Britain	.737	.264	—	49
	Australia	.534	.457	—	375
	Denmark	.706	.291	.003	194

However, the new alleles are extremely rare and several types have not been found in the material under discussion. Of all South African beef breeds only the Aberdeen Angus, Beef Shorthorn and Hereford have been investigated before—in England by Ashton, (1958a)[4]. In

the following table Ashton's results are included for comparison.

TABLE 6.—SOUTH AFRICAN BEEF CATTLE TRANSFERRIN RESULTS COMPARED WITH THOSE OF CATTLE IN OTHER COUNTRIES

Breed	Country	Gene frequency					Total animals tested
		A	D	E	F	B	
Aberdeen Angus	South Africa	.610	.379	.011	—	—	232
	Britain	.628	.260	.113	—	—	52
Afrikaner	South Africa	.397	.332	.267	.004	—	223
Beef Shorthorn	South Africa	.675	.325	—	—	—	100
	Britain	.629	.358	.014	—	—	141
Bonsmara	South Africa	.448	.359	.193	—	—	96
Boran	Kenya	.559	.020	.225	.010	.020	102
Drakensberger	South Africa	.308	.494	.198	—	—	185
Hereford	South Africa	.192	.739	.061	.008	—	123
	Britain	.387	.606	.008	—	—	77
Red Poll	South Africa	.545	.171	.273	—	—	283
Sussex	South Africa	.439	.557	.004	—	—	271

The results show great differences between the breeds with the Hereford differing the most, also from the British comparison results. The low frequency of Tf^A together with the high Tf^D is strange, but the fact that two animals were found with the F-gene is even more strange, because so far, the Tf^F- and also the Tf^B-gene has been discovered only in Zebu-type cattle. (In the meantime the owner said, that there could be a possibility that these two animals were not purebred).

Ashton (1959)[7] suggested the relative high frequency of Tf^E in Zebu-type cattle as an indication for the wellknown climatic and ecological tolerance of these cattle. This seems to be true for the Afrikaner, Bonsmara, Boran and Drakensberger, but the high frequency of Tf^E in the Red Poll would be difficult to explain along the same lines.

The transferrin types of the dual purpose breeds investigated differed very much as can be seen in Table 7. No foreign gene frequency was available.

The frequency of Tf^A and Tf^D in the Brown Swiss and the Dairy Shorthorn is almost the opposite, while a difference between Dairy Shorthorn and Beef Shorthorn is almost nonexistent in regard to their transferrin make-up. Also in the Nguni the relatively high frequency of Tf^E is found, and three of the other phenotypes which are apparently bound to the Zebu-type of cattle breeds could also be traced.

The search for a possible association of transferrin types with production traits and fitness factors started immediately after the inheritance was known. Datta et al., (1963)[21] were able to show that cows of Tf^D/Tf^D genotype had a slightly higher average milk yield than cows of Tf^A/Tf^A and Tf^A/Tf^D genotypes. Ashton, (1960b)[9] was able to present similar results. In his material the Tf^D/Tf^D animals produced 46 gallons of milk more than the Tf^A/Tf^A cows. If this is true, then dairy farmers should, together with their selection for higher milk production, automatically have increased the frequency of Tf^D. In fact, in the present material the frequency of Tf^D of all dairy breeds together is 0.564, in comparison with the frequency of

TABLE 7.—THE GENE FREQUENCY OF TRANSFERRINS IN DUAL PURPOSE BREEDS.

Breeds	Gene frequency					Total No. tested
	A	D	E	F	B	
Brown Swiss	.170	.830	—	—	—	271
Dairy Shorthorn	.703	.297	—	—	—	101
Nguni	.385	.265	.330	.001	.001	100
South Devon	.359	.592	.048	—	—	324

0.414 in all beef breeds. In the dual purpose breeds the frequency of Tf^D was 0.595 for all breeds given in the above table.

Ashton et al., (1962b)[13] reported that serum transferrin polymorphism affects fertility in dairy cattle, matings between homozygotes being significantly more fertile than matings involving heterozygotes. These workers were able to show that an excess of heterozygotes is born.

The distribution of returns 25 days or longer after artificial insemination, used as an index of embryonic death, as well as the distribution of genotypes from known matings, support the conclusion that homozygotes are less viable than heterozygotes in utero. The distribution of returns 0 to 24 days after insemination, however, showed that matings between homozygous parents of like genotype have a greater chance of achieving fertilization than matings between homozygous parents of unlike genotype. In other words, the transferrin locus in cattle affects fertility in two ways, both at fertilization and in utero.

Studies, also in our laboratory, have been initiated to prove these interesting findings. Unfortunately, the rats from Onderstepoort, which we attempted to use in breeding experiments were 100% homozygous at the transferrin locus, most probably due to the high inbreeding which has been practised during the past 25 years.

The study of the ratio homozygotes to heterozygotes could possibly throw some light on the problems mentioned above. One should expect, under normal conditions, a 50:50 distribution between homo- and heterozygous calves at birth. If the distribution at a later stage of life differs widely from this ratio, one must accept

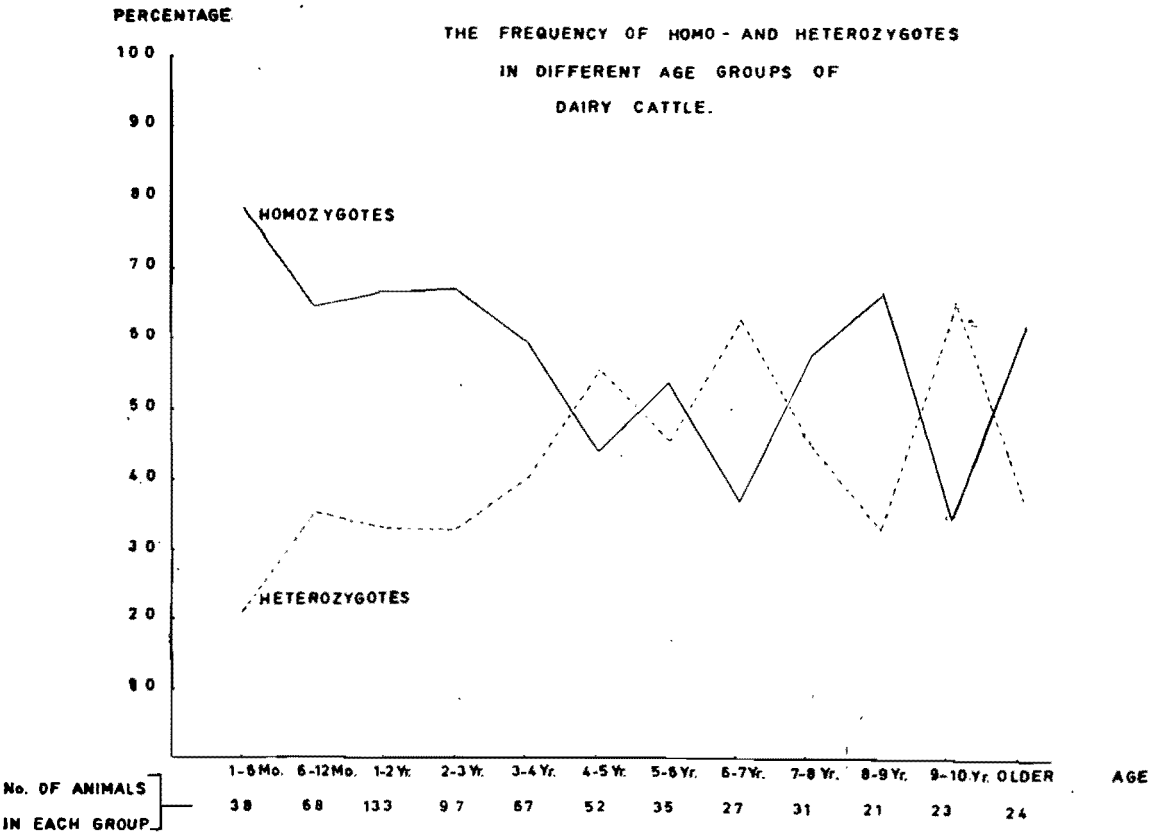

Fig. 7 — The frequency of homo- and heterozygotes in different age groups of dairy cattle.

that with the normal selection taking place in well managed herds the ratio automatically also changed.

TABLE 8

COMPARISON OF THE HOMOZYGOUS AND HETEROZYGOUS TRANSFERRIN TYPES IN DIFFERENT BREED GROUPS.

	Homozygous	*Heterozygous*
Dairy breeds.....	455	280
Beef breeds.....	766	849
Dual purpose breeds...	443	353
Total.....	1,664	1,482

The differences between dairy and beef breeds and also between beef breeds and "Total" are highly significant ($P<0.001$). We have a very great excess of heterozygotes especially in the Afrikaner, Drakensberger and Sussex, which needs an explanation.

In our material Ashton's findings (1962b)[13] could not be realized. Young calves showed a higher frequency of homozygous types which decreased to the age of four years. After that no further clear picture could be obtained.

In Fig. 7 the changes between homozygotes and heterozygotes are shown only for dairy breeds. A similar picture was obtained in all beef breeds.

The results of Datta et al., (1963)[21] and Ashton, (1960b)[9], however, could be confirmed. A clear picture of the influence of the transferrin allele Tf^D on milk production was obtained in an indirect way. It can be accepted that selection for higher milk yield is a continuous process with the result that the older cows usually are the best producers. In Fig. 8 the increase of the transferrin allele Tf^D can be recognized which manifests itself in animals of higher age.

Even if the material from the Irene performance tests is very limited it was hoped to get an indication in regard to a possible association of transferrin types and daily gain or any other

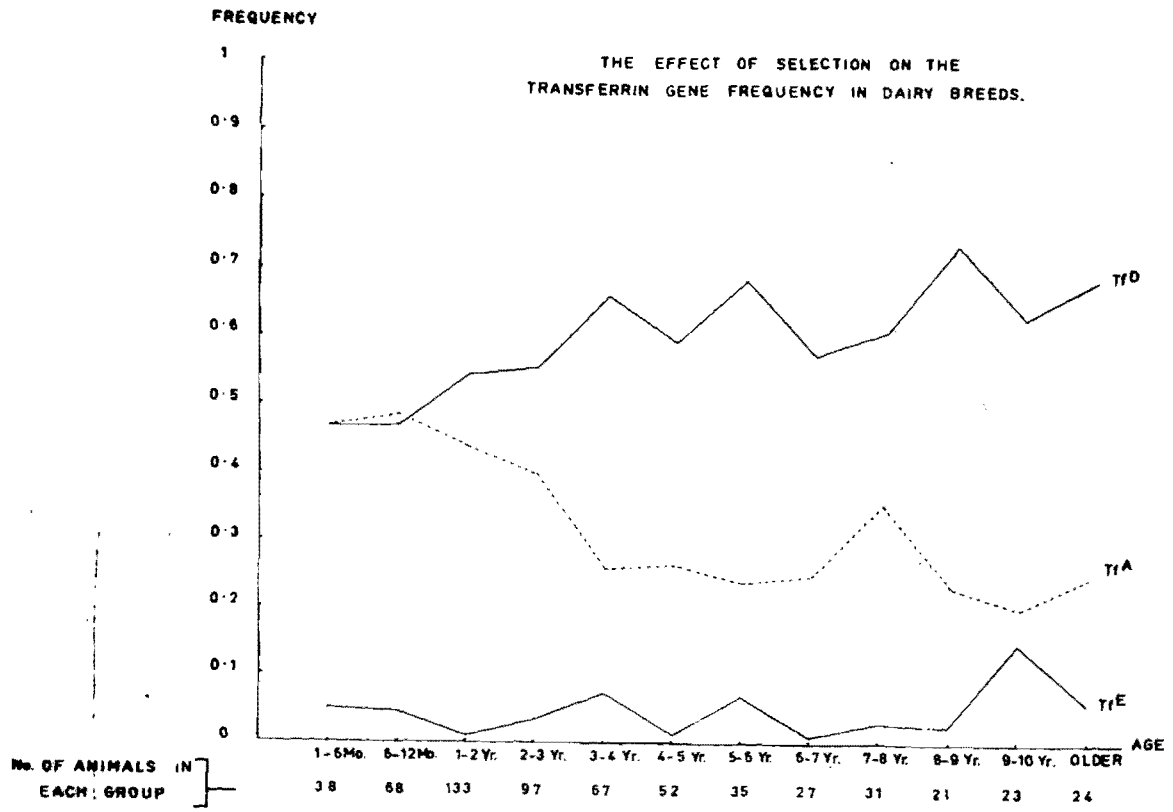

Fig. 8 — The effect of selection in the transferrin gene frequency in dairy breeds.

performance test measurements. Only 83 bulls could be used for this first study. The results obtained are very promising, because it seems that bulls of Tf^A/Tf^A genotype have a higher daily gain than bulls of Tf^D/Tf^D genotype, and these again a higher gain than bulls of Tf^E/Tf^E genotype; the heterozygote types falling in between on their respective order. Further work is necessary to provide more evidence for this association.

In conclusion to the discussion of transferrin types in cattle it should only be mentioned that recently a new type of transferrin has been discovered in our laboratory (Osterhoff et al., 1964b)[44]. It is the Tf^G-allele which expresses itself in a very slow moving triple band, slower than all the others. Both the homozygote Tf^G/Tf^G and several Tf^G heterozygotes could be recognized. Nothing can be said about the significance of this new allele at this stage.

Sheep exhibit the most extreme diversity; their transferrins have a tendency to travel unusually close to the albumin and there are a number of features which make precise typing more difficult in this species. Originally Ashton, (1958b)[5] identified fourteen distinct sheep phenotypes. These were regarded as representing all but one of the 15 possible types from a five allele system (A, B, C, D, E, in decreasing order of electrophoretic mobility). Work in our laboratory indicated that not all types were included in this original classification and recently Ashton et al., (1962a) published a new classification which included 78 possible types in a 12 allele system.

It will immediately be clear to everyone that careful cross matching of reference sera will be essential if desirable maps of world breed distribution are to be established. Also for comparison studies of biochemical polymorphism and fitness factors in sheep an international cooperation is absolutely necessary in regard to exchange and control of reference sera.

A transferrin polymorphism has also been described in the *goat* (Ashton, et al., 1958)[14], where one pair of alleles gives rise to three phenotypes.

Studies of serum polymorphism in the *pig* have been reported by Ashton (1960a)[6] and

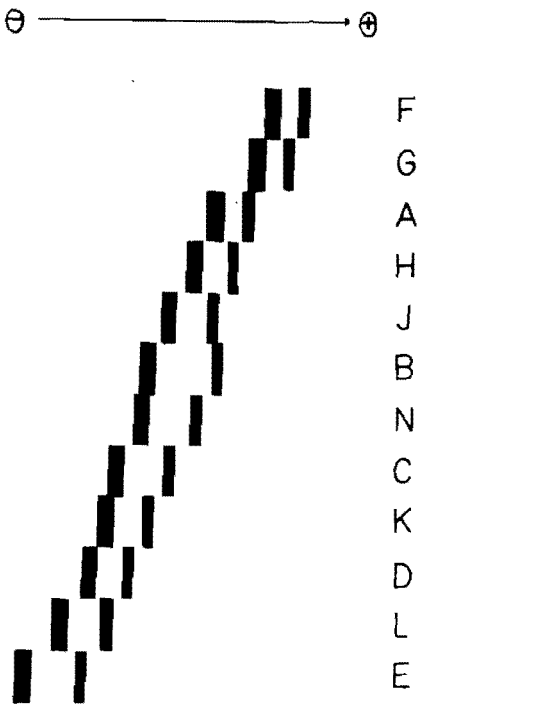

Fig. 9 — Diagram of the mobility of sheep transferrins in starch gel.

Kristjansson (1960a & b)[34,35]. It is clear that considerable polymorphism exists, which became more complicated after the development of a high voltage starch gel electrophoresis system. Originally three alleles Tf^A, Tf^B and Tf^C were described, all of them being now resolved into three fractions. Interesting results will be obtained in the next future also in regard to the question if a single allele may control multiple protein zones in several species.

Probable genetic variants in the serum proteins of domestic *poultry* have been reported by Ogden (1962)[42] but apparently age plays an important role in the proper establishment of the transferrin types.

Table 9 summarizes the relationship between biochemical polymorphism and fitness factors so far established.

TABLE 9.—BIOCHEMICAL POLYMORPHISM AND FITNESS FACTORS IN LIVESTOCK (ASHTON, 1963)[11]

	Transferrins	Haemoglobins
MILK YIELD (Dairy cattle)	*Ashton* (1960, 1963) D/D 46 gall. better than A/A. *Datta and Stone* (1963) D D better than A/A	*Bangham* (1963) No difference
BUTTERFAT PERCENTAGE (Dairy cattle)	*Ashton* (1960, 1963) No effect	*Bangham* (1963) No difference
FERTILITY (Cattle)	*Ashton and Fallon* (1961, 1962). Matings between homozygotes more fertile than matings involving heterozygotes. *Ogden and Woolf* (1963) Confirmed findings of Ashton and Fallon *Hickman and Dunn* (1961) Suggested E type bulls more fertile.	Not reported
FERTILITY (Sheep)	*Ashton, Evans and Turner* (1963) Matings between homozygotes more fertile than matings involving heterozygotes	*Ashton, Evans and Turner* (1963) Hb A ewes poorer lambing percentage than A/B or B.
CLIMATIC ADAPTATION	*Ashton* (1958) Cattle of Tf^E better adapted in unfavourable climates.	*Huisman et al.* (1958) Oxygen combining powers of two Hb types of sheep. *Dawson and Evans* (1962) Oxygen combining ability in sheep
DISEASE SUSCEPTIBILITY	Not reported	*Huisman et al.* (1958) Hb type and anaemia in sheep *Bangham and Blumberg* (1958) Hb type and trypanosomiasis in cattle *Evans, Blunt and Southcott* (1963) Hb A more resistant to *H. contortus* infection in sheep

HAPTOGLOBINS

The haptoglobins are those proteins which have the ability to bind haemoglobins. They are detected on starch gel by adding a small amount of haemoglobin to the serum before electrophoresis and staining the gel after electrophoresis with benzidine reagent. The benzidine reagent produces a deep blue colour reaction at all locations where there is peroxidase activity and therefore at all locations where haemoglobin is situated.

In *horses* no individual variation could be found in haptoglobin types.

In *cattle* the haptoglobins can be demonstrated only with great difficulty.

Three haptoglobin alleles (Hp^1, Hp^2, Hp^3) in *pigs* have been reported in Canada by Kristjansson (1961). Later, three more alleles Hp^0, Hp^4 and Hp^5 have been described in Denmark (Brummerstedt-Hansen et al., (1962). Apparently, more fractions are present, but the genetic background of these new types has not yet been established.

The haptoglobins observed in *chickens* have not been well resolved and further technique studies will be required in order to obtain clear, reproducible resolution.

SLOW ALPHA$_2$-GLOBULIN.

The Sα_2-globulin is a glycoprotein of high molecular weight and is also called 19 S alpha$_2$-glycoprotein or alpha$_2$-macroglobulin. The physiological function of Sα_2-globulin is not

		POSSIBLE GENES FROM EWE											
		F	G	A	H	J	B	N	C	K	D	L	E
POSSIBLE GENES FROM RAM	F	FF											
	G	FG	GG										
	A	FA	GA	AA									
	H	FH	GH	AH	HH								
	J	FJ	GJ	AJ	HJ	JJ							
	B	FB	GB	AB	HB	JB	BB						
	N	FN	GN	AN	HN	JN	BN	NN					
	C	FC	GC	AC	HC	JC	BC	NC	CC				
	K	FK	GK	AK	HK	JK	BK	NK	CK	KK			
	D	FD	GD	AD	HD	JD	BD	ND	CD	KD	DD		
	L	FL	GL	AL	HL	JL	BL	NL	CL	KL	DL	LL	
	E	FE	GE	AE	HE	JE	BE	NE	CE	KE	DE	LE	EE

78 POSSIBLE TRANSFERRIN COMBINATIONS IN SHEEP.

Fig. 10 — The possible transferrin combinations in sheep.

known, but a protein zone named Sa_2-globulin has been found in all mammalian sera so far examined.

Ashton (1958c)[6] described two Guernsey cattle whose sera lacked the slow-α_2-glosbulin zone in starch gel electrophoresis. Later, Gahne (1962)[27] found three animals without this zone. In our investigations four animals were found without the slow-α_2-zone. No sign of abnormality could be established in these animals.

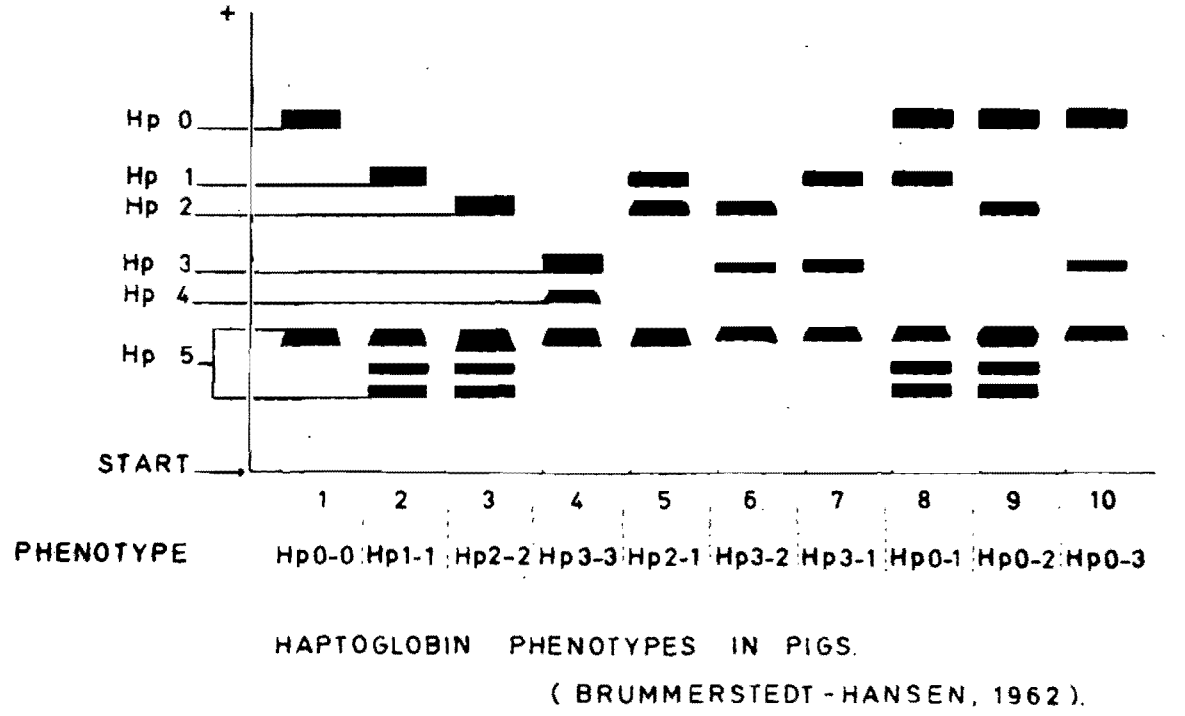

Fig. 11 — Diagram of Haptoglobin phenotypes in pigs.

Post-Albumin.

Smithies (1959)[48] got evidence suggesting that genetic factors are involved in the variation of the serum proteins which migrate immediately behind albumin, the so-called post-albumins. This original work was performed on human serum. Gahne (1961)[26] was able to demonstrate the genetic variation in cattle post-albumins.

In our laboratory Gahne's involved method was simplified and reproducible resolution in three phenotypes AA, AB and BB could be obtained. The post-albumin phenotype AA is characterized by a protein zone migrating a little faster than the corresponding zone of the type BB. In young animals a classification is not possible because the post-albumin pattern is overshadowed by the abundant amount of fetuin.

Albumin

Braend (1964)[17] reported on a polymorphism in the horse albumin types. One should not be surprised if by alterations of techniques a polymorphism is detected in other livestock species.

Acid Phosphatase

Allison et al., (1957)[1] following a chance observation, demonstrated a tendency towards a bimodal distribution in the erythrocyte catalase activity of dogs. Individuals were consistent in their level of activity on repeat testing. On the basis of tests on 70 offsprings from 19 matings they suggested control by a single pair of alleles, C-c, and that in the presence of C more catalase is produced than when c is present. From their evidence they concluded that the heterozygote's activity is intermediate.

Gahne (1962)[27] was able to demonstrate a variation of the cattle acid phosphatase by using the so-called zymogram staining method after the normal starch gel electrophoresis procedure. He suggested a genetic system which determines the cattle acid phosphatase, which was sym-

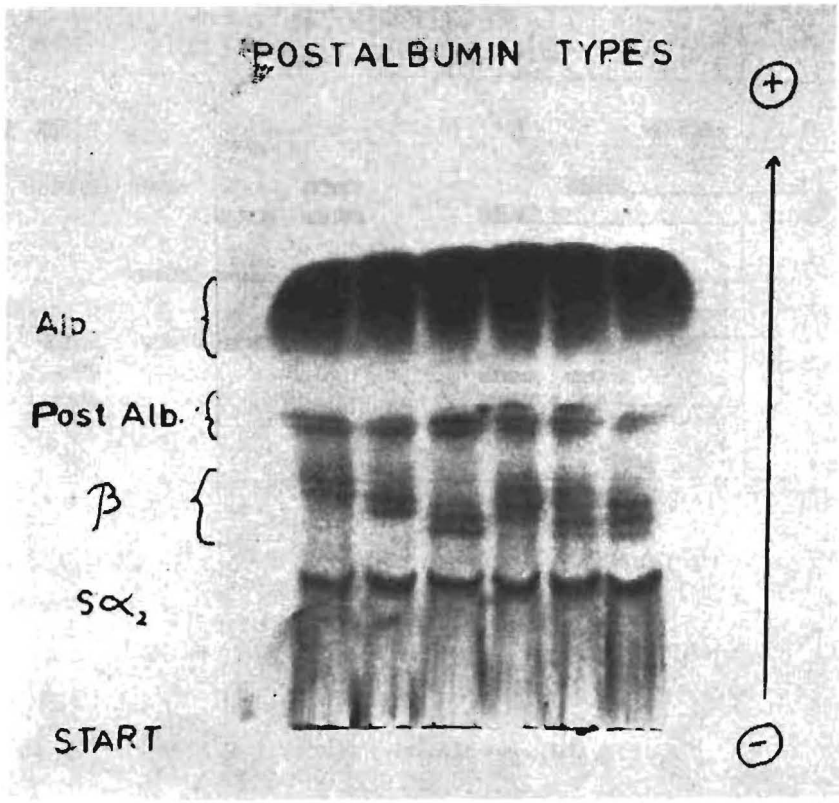

Fig. 12 — Post-albumin differentiation in starch gel.

bolized F. One allele called F^A determines the presence of the enzyme Zone A and other alleles give rise to the other kinds of zymogram. Those other alleles are taken together and designated by F^O. So far, only a phenotypic classification in two groups A and O can be obtained.

Milk Proteins

Besides genetically determined differences in seminal fluid and egg albumen proteins the beta-lactoglobulins in cow's milk must be mentioned. Protein zones corresponding in band pattern and number, and in migration rate with cattle serum transferrins have been detected in milk, notably colostral milk (Gahne et al., 1960)[28]. With the aid of immunoelectrophoresis the serological relationship with the serum transferrins could be established (Gahne, 1961)[26].

Ogden (1961)[41] reported on a number of papers dealing with the existence of genetically controlled variants of the milk proteins of different cattle breeds. After the establishment of gene frequencies of two beta-lactoglobulins which are controlled by two alleles (Lg^A and Lg^B) by different workers, Kiddy et al., (1964)[33] and Thompson et al., (1964)[50] described the genetic control of both alpha$s$- and beta-casein variations. More interesting results in regard to the inherited milk proteins, also in other species, can be expected.

Blood potassium content

Evans (1954)[22] has shown that sheep fall into one of two groups on the basis of the concentrations of sodium and potassium in their red blood cells. Sodium concentration varies inversely with potassium concentration and these two main

types were therefore originally designated simply "high potassium" (HK) and "low potassium" (LK). The genetics of this dimoprhism was explained by one pair of alleles, with that responsible for the HK phenotype (K^h) behaving as a recessive. On this basis LK phenotypes are, therefore, of two genotypes, those homozygous and those heterozygous for the LK allele (K^l).

The most complete survey of different breeds of sheep in regard to their blood potassium types was recently published by Meyer (1963a)[37]. He listed all breeds so far investigated—56 in all—and demonstrated the frequency of the K^h-gene varying from 0.06 in the English Leicester to 0.98 in the Islandic Sheep. The author assumes that the distribution of the K-alleles is directed by ecological factors together with other factors such as genetic drift, origin, crossbreeding and artificial selection.

Merinos investigated in our laboratory showed a very low frequency of the K^h-gene, which is in agreement with the Australian results.

Conclusions.

It is clear that there is a gene-controlled biochemical diversity in farm livestock.

The value of such studies in respect of problems of gene action and protein synthesis is unquestionably high.

These fields are amongst the most intriguing in contemporary biology.

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A NOTE ON THE TOXICITY OF *PAPAVER NUDICAULE L.* (ICELAND POPPY)

M. TERBLANCHE AND T. F. ADELAAR

Section Toxicology, Veterinary Research Institute, Onderstepoort.

SUMMARY

Nervous symptoms and death occurred in some cattle after the ingestion of a large amount of Iceland Poppys. The symptoms and post mortem appearances seen and treatment applied, are described.

Two sheep were dosed in the laboratory at different dosage rates and one died. All evidence at our disposal indicates that this plant is possibly toxic.

The plant contained an insignificant amount of nitrate and was negative for prussic acid.

INTRODUCTION

In 1929 McLennan[1,2] encountered two natural cases of poisoning in sheep which he ascribed to Iceland Poppy poisoning. Since then no further reference to its toxicity could be found in the literature. In South Africa no cases have been described or brought to our attention. We, therefore, wish to bring to the notice of our colleagues this possible source of poisoning.

ANAMNESIS

The owner's Iceland Poppies were not fetching economical prices on the market; he decided to feed them to his cattle. A group of 25 calves which received about 15 kg of the fresh plant in the full flowering stage was not affected. Another group, consisting of 6 cows and 2 heifers received about 80 kg of the plant; one heifer died peracutely, four cows and one heifer showed severe symptoms while the remaining two bovines were not affected.

Symptoms:

The animals appeared to be anxious and had a nervous expression on their faces. They ran around continuously, went down and stood up again. One developed a stiff gait. There was a complete loss of appetite in the beginning. The dead animal was severely bloated. At post mortem there was, however, no submucosal haemorrhages in the anterior portion of the trachea as is usually seen when animals die of bloat.

Treatment:

The animals were prevented from drinking water for 3 days, during which time they were given Prostigmine intramuscularly. Some purging took place and they all recovered.

LABORATORY INVESTIGATION.

The actual green fodder they received consisted of *Papaver nudicaule L.* and a small amount of *Vicia varia Host* and *Vicia sativa Host*. The three plants, specimens of the decomposed carcass as well as fresh blood samples of the remaining animals, gave negative results when tested for hydrocyanic acid.

The *Papaver nudicaule* L. was found to contain an insignificant amount of nitrate.

The two animals that showed the most severe symptoms on the previous day was bled for a chemico-pathological analysis:—

In the one animal a slight increase in serum transaminases (SGOT = 200 King units and SGPT = 118 King units) a slight hyperglycaemia (90 mgm %) and a mild hyperproteinaemia (= 10.9 Gm %) were found.

The other one showed normal transaminases (SGOT = 146 and SGPT = 42) also a slight hyperglycaemia (71 mgm %) and a hyperproteinaemia (11.4 Gm %). The fresh green plant was then dosed to a 6 tooth 44 kg wether at the following rate, per rumen fistula.

Days	1	2	3	4	5	6	7	8	9
Dosage in g/kg.....	22.7	45	45	0	45	45	0	0	68

The animal was found dead and in an advanced state of decomposition on the morning of the 10th day.

CHEMICO-PATHOLOGICAL ANALYSES ON THE BLOOD:

A significant increase in serum glutamic-oxaloacetic transaminase was seen:—

Days	1	2	5	6	7	8
SGOT.....	194	125	247	233	342	342

King units.

Daily clinical observations revealed no symptoms at any stage. Another sheep weighing 36.3 kg was dosed by stomach tube at the following rate:—

6 g/kg on the first day and 12 g/kg/day from the second to the 10th day. The green material, which had been kept in a refrigerator, became too decomposed for further dosing.

The only symptoms seen were a slight listlessness on the 4th day and a mucoid nasal discharge on the 5th day. Chemico-pathological analyses on the blood over 15 days in this case, only showed a slight anaemia developing towards the end.

Unfortunately the experiments could not be continued due to lack of material.

DISCUSSION

McLennan[1,2] was not able to pursuade the sheep in his laboratory to eat the plant. However, in two "natural" cases, where wheelbarrow loads of Iceland Poppys were disposed of in places where the sheep had access to them, the symptoms were strychnine-like and he described more severe nervous symptoms than we had seen. These were polypnoea, convulsive movements of the libms, flexing of the head on the neck, frothing from the mouth and nostrils.

Although very little experimental evidence is available the circumstantial evidence is such that this plant can be regarded as toxic.

It is to be concluded from this evidence that the plant is not highly toxic and a large amount of the plant is necessary to cause any effect. The possibility of the cause of death being merely bloat due to overeating can not be entirely excluded.

However the specific nervous symptoms seen in this case and by McLennan[1,2]; the absence of submucosal haemorrhages in the anterior trachea; no bloat in McLennan's rabbit which started eating the plant after 4 days and then died on the 6th day; and the absence of bloat in our two experimental sheep, indicates the presence of an inherent toxic factor in the plant.

ACKNOWLEDGEMENT

The Chief of the Onderstepoort Veterinary Research Institute is thanked for permission to publish this report.

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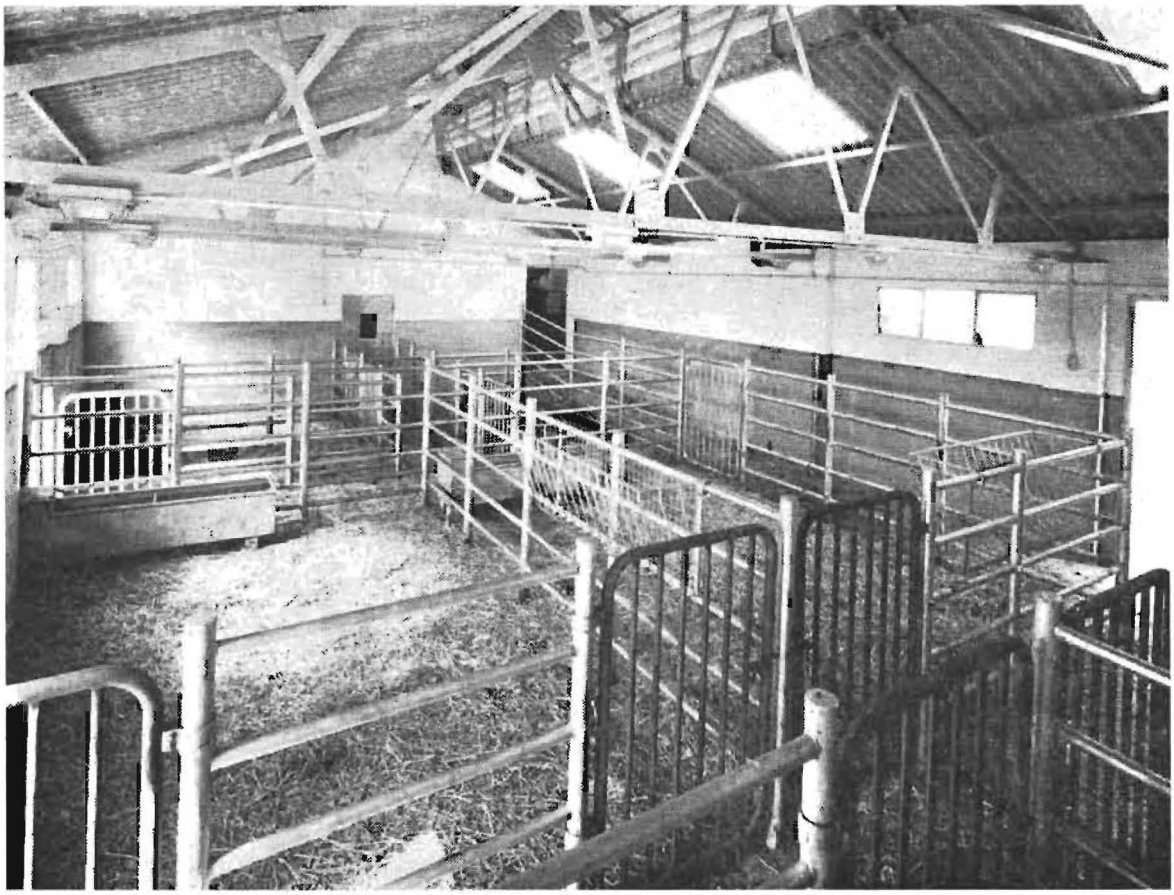

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VETERINÊRE AANGELEENTHEDE IN DIE LANDBANK ORGANISASIE

P. J. J. FOURIE

Land- en Landboubank van Suid-Afrika, Paul Krugerstraat, Pretoria.

Die verskaffing van krediet fasiliteite vir die boeregemeenskap was 'n dringende noodsaaklikheid na die uiterste nood en armoede onder die plattelandse boere as gevolg van die verwoesting en vernietiging van die plase en die vee-stapel gedurende die Boereoorlog.

Die Transvaalse Verantwoordelike regering het die voortou geneem en het alreeds in 1907 die Transvaal Land- en Landboubank gestig met 'n kapitaal fonds van twee miljoen pond wat bewillig is deur die Transvaalse regering. Toe volg Natal ook in 1907 met die stigting van Land- en Landbou-leningsfonds (£500,000), die O.V.S. in 1907 die Land- en Landbouleningsfonds (£500,000). Die Kaap Kolonie het ook in 1907 'n wet passeer vir die stigting van „De Landbouw Kredietbank", maar dit was nooit in werking gestel nie.

Op 1 Oktober 1912 is die Land- en Landboubank van Suid-Afrika gestig om krediet fasiliteite vir lang- en kort-termyn-doeleindes aan die boere in al vier die provinsies te verskaf.

Die bank staan lenings toe op eerste verband om grond te koop, of om bestaande laste af te los of verbeterings aan te bring op grond wat alreeds in besit is; vir die bevordering van Landbou bedrywe (tabak, suiwel, sitrus ens.); die oprigting van omheinings; die voorsiening van water; die oprigting van dipbakke en voerkuile en voorskotte aan koöperasies en beheerrade.

Die tydperk waarvoor die lenings betaalbaar is varieer van een jaar (Kaskredietvoorskotte) tot 35 jaar (verbandlenings).

Veterinêre aangeleenthede het geen noemenswaardige rol gespeel in die werking van die lenings nie.

Op 1 Oktober 1959 het die Bank as gevolg van 'n wysiging van die Landbankwet, lenings toegeken op hipoteek van roerende goed. Die doel van die skema is om middeltermynkrediet aan voltydse boere te verskaf om lewende hawe en ander boerderybenodighede (Implemente—soos trekkers ens.) te koop, wat noodsaaklik is om 'n doeltreffende en gebalanseerde boerderystelsel te verseker. Die sekuriteit in die geval is die roerende goed, waarvan die lener die gebruiks- en beskikkingsreg besit. Dit sluit ook in die aanteel van die lewende hawe en die opbrengs van die verhipotekeerde goed.

Waar diere vir wol, vleis of melkproduksie gekoop word, is dit vanselfsprekend dat die diere moet voldoen aan die vereistes waarvoor hulle aangeskaf word. Die boer moet nie alleen in die geleentheid gestel word om die regte tipe dier te koop nie, maar wat ewe of nog meer belangrik is, die diere moet normaal gesond wees. Vir die rede het die Landbank in sy kontrak met die skuldenaars hierdie gesondheidsvereistes gestel:

(i) Alle diere wat gekoop word moet sigbaar vry wees van enige siektes en gebreke en in goeie gesondheid verkeer. Aanteel diere moet veral vry wees van geslagsiektes en uiersiektes.

(ii) Waar aanteel diere van meer as R80 stuk gekoop word, moet 'n veterinêre sertifikaat gelwer word dat 'n algemene kliniese inspeksie van die aangekoopte aanteelbeeste sowel as die applikant se eie aanteelbeeste gedoen is en dat die diere sigbaar vry is van geslag- en uiersiektes en andersins in goeie gesondheidstoestand verkeer.

(iii) Waar bulle of ramme gekoop word moet 'n veterinêre sertifikaat gelewer word dat die diere klinies ondersoek is en dat die diere sigbaar vry is van geslagsiektes en andersins in goeie gesondheid verkeer.

Waar applikante in hartwatergebiede boer, moet hulle beeste wat alreeds geakklimatiseer of geïmmuniseer is, koop.

Om die boere sover as moontlik behulpsaam te wees en om sy eie sekuriteit so sterk as moontlik te maak, het die Landbank vee-inspekteurs by al hulle takke aangestel. Voordat die koop van die lewende hawe voltrek word, moet die inspekteur elke dier ondersoek om seker te maak dat die dier se ouderdom geskik is, dat dit die regte tipe dier is en dat die dier sigbaar klinies gesond is.

Dit is op die oomblik die beleid van die Bank om enige inspekteur wat aangestel word die geleentheid te gee om saam met 'n veearts, wat toevallig op die oomblik in diens van die Bank is, op 'n inspeksie reis te gaan, sodat hy onderrig kan word in 'n roetien kliniese ondersoek om sover as wat dit prakties moontlik is in die omstandighede, te verseker dat die diere wat gekoop gaan word in klinies normale gesondheid verkeer. Die doel van die onderrig is om die inspekteur te help om enige kliniese abnormaliteite te kan waarneem, sonder om 'n diagnose te maak en daar is nie die minste gevaar dat sodanige inspekteurs hulle self later as 'n soort veearts sal beskou nie. Waar inspekteurs met hul roetien ondersoek van Landbank verhipotekeerde diere verdagte simptome teëkom, mag hulle nie die boer oor behandeling inlig nie, maar moet hulle die boer aanraai om meteens in aanraking te tree met 'n privaat of staatsveearts.

Om inspekteurs te help met die roetien kliniese ondersoek van die diere, is 'n beskrywing gegee onder andere, van belangrike simptome waarvoor hulle op die uitkyk moet wees; dit is ingelyf in die handleiding van die Bank en tot die beskikking van alle Landbankbeamptes. Hierin word sekere siektes behandel wat belangrik is om die boer te beskerm en te probeer verseker dat die Bank se sekuriteit so goed as moontlik sal wees.

Dit word van die inspekteurs verwag om in goeie samewerking met staatsveeartse te verkeer. Dit is veral belangrik in die bosluisgebiede. Dit gebeur ongelukkig soms dat skuldenaars nie die mas opkom nie en die Bank is dan genoodsaak om die diere in beslag te neem en te verkoop, om sodoende die skuld te probeer dek.

Die inspekteurs is dan verantwoordelik vir die versorging van die diere en as hulle vervoer word en uiteindelik verkoop moet word, moet die inspekteurs op hoogte wees met veterinêre vereistes in verband met behandeling van die diere vir bosluise met middels wat aanneemlik moet wees vir die staatsveeartse.

Waar sodanige diere soms tydelik onder die sorg van 'n ander boer geplaas word, moet redelike voorsorg geneem word dat hully vry is van infeksie siektes en selfs 'n besmetting soos lewerslak, sodat die toestande nie oorgedra word op die diere van die boer waar hulle tydelik versorg word nie.

CASE REPORTS

1.—FRACTURE OF THE SYMPHYSIS MANDIBULAE IN A JERSEY BULL

A. E. Fair

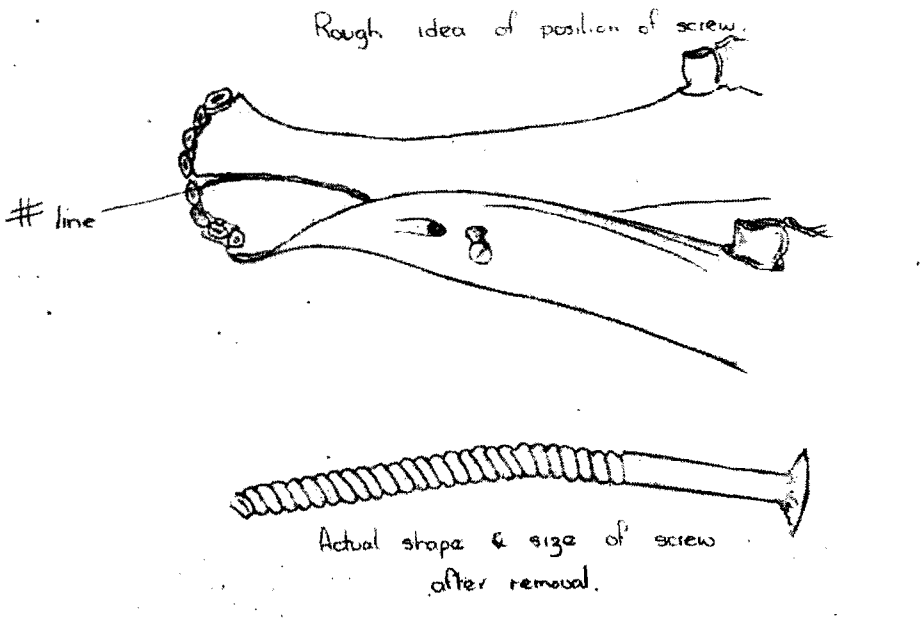
*State Veterinarian, Ladysmith, Natal.

The following case may be of interest to other colleagues.

On the 23rd. March 1964 I was called in to examine a 5 year old Jersey bull with a fracture of the symphysis mandibulae. The owner was positive that two days prior to my visit the bull was sound and presumed that he had fractured his jaw in a fight with another Jersey bull.

On examination there was no external evidence as to the nature of the damage; no swelling, no coagulated blood etc; but there was obvious pain. The bull was taking a small amount of grain and water but refused to graze. Closer examination showed a complete fracture of the jaw, the rami being freely moveable. A small amount of pus exuded from the break when the jaw was manually clamped closed. The soft tissue was broken only on the lingual aspect of the gum but not between the lip and incisors.

Atropine (25 mgm) was administered s.c. half an hour before sedating the bull i.v. with approximately 700 cc of 40% chloral hydrate.



*Now veterinary practitioner Box 400 Rustenburg, Transvaal.

The bull was cast on the right side; 5% local anaesthetic with adrenaline was infiltrated into an area already shaved and disinfected.

A 2 cm skin incision was made, parallel to the jawbone, just behind and above the mental faramen, and the periosteum scraped with the blade.

A small hand-brace, $\frac{3}{16}''$ steel bit and screw-driver were suitably prepared beforehand. A hole was then drilled through the bone and soft tissue below the tongue to the opposite ramus of the mandibulae. Using a sterilized 4" wood screw the mandibles were then screwed together; the screw was inserted till it emerged from the opposite ramus but without breaking the skin; the jaw visibly pulled together. A 4 day course of oxytetracycline was prescribed and contact maintained with the owner. The next day the bull was grazing; a slight swelling had developed at the site.

On April 17 the bull was examined. He was well, but held his head low as if pain was present. Granulation tissue had obliterated the screw head but there was a clear fluid discharge. Examination of the floor of the anterior buccal cavity revealed soft tissue repair and no swelling.

On May 28 (i.e. 66 days later) the screw, which was loose, was removed with the fingers after local anaesthesia, and an incision through granular tissue over the screw head. There was a slight pus discharge around the screw head. Granulation tissue about the size of a golf-ball had developed. The screw cavity was irrigated with a penicillin solution. The healing of the fracture appeared complete. There was a slight gap between the central incisors. On removal the screw showed a 20 bend and had become black in colour, but no corrosion was evident.

2.—AN UNUSUAL CASE OF BOVINE TUBERCULOSIS IN MAN

R. W. WORTHINGTON

Department of Bacteriology, Onderstepoort.

SUMMARY

An unusual case of tuberculosis in man, caused by *M. bovis*, is described. This form of tuberculosis could be regarded as a professional hazard to veterinary surgeons.

INTRODUCTION

It is often said, that *Mycobacterium bovis* is of minor importance as a cause of tuberculosis in humans in South Africa. Reviewing the available literature, Dormer[1] reported that 18 cases of tuberculosis caused by *M. bovis*, had been recorded up until 1957; only one of these isolates was from a case of pulmonary tuberculosis[2]. In 1961 the finding of seven children, all receiving un-pasteurized milk from the same herd, who were suffering from tuberculosis led to an investigation of the herd. The herd proved to be 60% infected[3]. A further strain isolated by Dr. Martinaglia at the King George V hospital from a Bantu, has been identified in this laboratory, as *M. bovis*.

The position in other countries before the eradication of tuberculosis, is in marked contrast to that reported in South Africa. In a review of the world literature, Goertler and Weber[4] reported that of 93,000 strains of tubercle bacilli isolated from humans, up to 1952, 10.2% were *M. bovis*. With the eradica-

tion of bovine tuberculosis from many countries, the position is now changed and M. bovis infection of man is becoming increasingly rare. At present, the incidence of bovine tuberculosis in our commercial dairy herds, is still high[5] and it is surprising that more cases are not reported from humans. The case reported below has been recorded as it may be of particular interest to Veterinary Surgeons.

History and Clinical Findings

In January, 1959 the author was asked to perform autopsies on two cattle which had died suddenly. While removing the skin, he accidently cut three fingers of the right hand over the flexor tendons. The cut on the small finger penetrated the skin. After washing and bandaging the finger, with a piece of rag, the post mortem was continued. One of the cows was found to be suffering from a tuberculous peritonitis.

The wound healed quickly, but complete flexion of the small finger was impossible. Within one month the hand became painful and stiff, four months later, what appeared to be a slight contraction of the tendon, was evident, which interfered with the extension of the wrist joint. Six months after the accident, a tendovaginitis of the flexor sheath developed which persisted for the next four years.

Diagnosis

Approximately four years later, an orthopaedic surgeon made a diagnosis of suspected tuberculous synovitis of the tendon sheath. A granuloma of the synovial sheath was removed surgically, and the diagnosis confirmed histologically and bacteriologically.

Bacteriology

A few colonies of acid fast bacilli were isolated from the biopsy material on Lowenstein Jensen medium. The colonies were extremely slow growing, small, dysgonic, pale and round. Subcultures grew well on medium containing no glycerine, but not at all on medium with 0.75% glycerine added. The strain proved to be highly pathogenic for rabbits and guinea-pigs, urease positive and niacin negative. The strain was found to be sensitive to isoniazid, P.A.S. and streptomycin. There can therefore be no doubt that this strain is *M. bovis*.

Treatment

After surgical removal of the granuloma, chemotherapeutic treatment consisted of six weeks treatment with Isoxyl* and Isoniazid, followed by a further five months of isoniazid treatment. The surgical wound healed without complications and full movement of the wrist and finger, returned within two months.

Conclusion

It is suggested that tuberculosis of this form may constitute a professional hazard to veterinary surgeons, slaughtermen and other people who come into contact with tuberculous carcasses. Should accidents similar to the one described above, occur, then a course of prophylactic treatment with Isoniazid, should be instituted immediately.

ACKNOWLEDGEMENTS

The author wishes to thank Prof. Smit who confirmed the diagnosis histologically and Dr. H. H. Kleeberg for isolating the strain, also the Chief, Veterinary Research Institute, Onderstepoort, for permission to publish this report.

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* 4—4 Di-iso-amy-thio-carbanilide

3. MISPLACEMENT OF THROAT BOTS (*PHARYNGOBOLUS AFRICANUS BRAUER*) IN THE AFRICAN ELEPHANT

H. H. ROTH

Veterinary Research Laboratory, Salisbury, Southern Rhodesia.

The larvae of the oestrid fly *Pharyngobolus africanus* Brauer develop normally in the pharyngeal region of the African elephant.

Already RODHAIN and BEQUAERT[2] have found that in the Congo every elephant examined harboured some larvae of *P. africanus* in the throat, and ZUMPT[3] has given a full account of our present morphological and biological knowledge of this elephant parasite. The Elephant Throat Bot had been recorded so far from East Africa (MARNO 1868)[1], the Congo (RODHAIN) and BEQUAERT 1919)[2] Uganda, and Northern Rhodesia (ZUMPT 1964)[3], but parasitological examinations of elephants in Southern Rhodesia have shown that the distribution of *P. africanus* extends at least as far south as the southern drainage of the Zambezi.

Two elephants shot at random in a parasitological survey of this species in the Zambezi Valley were both parasitized with throat bots. This indicated that the parasite is very probably quite common in elephant in this area. The individual animal is however far less heavily infested compared to the infestation with larvae of the gastrophilid fly *Platycobboldia loxodontis* (Brauer) which are found by the hundreds in the stomach of almost every African elephant in Southern Rhodesia. The elephants examined harboured each only three larvae in the pharyngeal region. No bots were present in the nasal and frontal cavities, nor in the oesophagus. In another young elephant a careful parasitological postmortem examination produced, however, a live 3rd instar larva of *P. africanus* from the lung. This maggot was found jammed in the most distal end of the left main bronchus. In fact it was so tightly enclosed by the fibrous walls of the bronchus that it could only be recovered after careful cutting and preparation. It was quite obvious that this larva had already been dislodged from the pharynx into the lungs long before the death of the host. Such misplacement is known from the closely related *Cephenomyia spp.* in European species of deer.

The odd larvae attach themselves to the larynx in the course of their development and may be accidentally inhaled. Being more or less immobile in their last stage they are incapable of leaving the air tube and are sucked into the lungs.

The elephant calf in which this displaced throat bot was found, was in a very poor general state of health, suffering from purulent arthritis and being heavily infested with various types of helminths. In some other twelve elephants, including four young calves, of which the lungs were carefully examined for aprasites, there were no bots in the bronchi, and therefore the misplacement of *Pharyngobolus africanus* is probably rare. Whereas *Cephenomyia spp.* in deer quite cause considerable suffering and ill-health, sometimes even suffocation, there is no evidence so far that *Pharyngobolus* larvae cause much irritation to elephants. In the examined elephants there were no pathological lesions seen, neither in the pharynx nor in the lung. The mature larvae leave the elephant most probably through the trunk from which ZUMPT (1964)[3] has received fully grown maggots. In the Zoological Gardens of Basel it was observed that the host may discharge them actively by sneezing and throwing his trunk.

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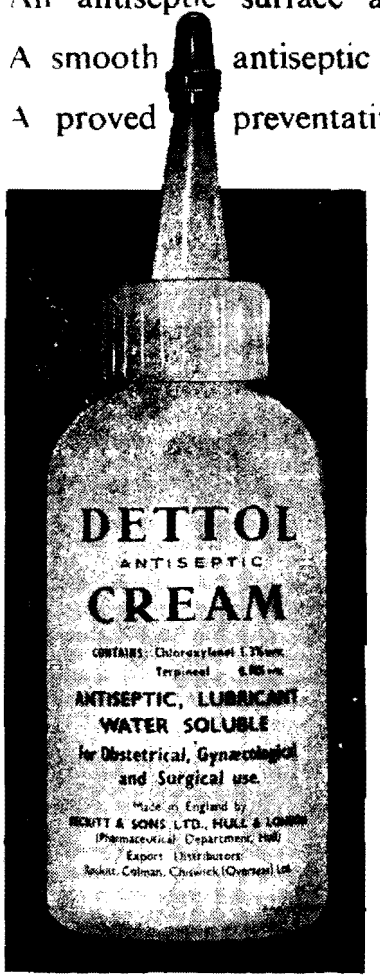

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DIE PLATTELANDSE VEEARTS

D. J. Louw

Parfittlaan 79, Bloemfontein.

Die plattelandse praktiserende veearts wat ongeveer twaalf jaar gelede sy verskyning op die toneel gemaak het, en baie daartoe bygedra het om die boerderygemeenskap veearts-bewus te maak, is haas weer besig om te verdwyn tot nadeel van die veestapel, die boere en ook die professie.

Hierdie veeartse het 'n spesiale leemte aangevul omdat hulle kliniese werk verrig het wat normaalweg buite die bestek van die staatsveearts val, en selfs dié van die stedelike grootdiere-praktisyn, wat meestal met streng kommersiële sake te doen het. Die staatsveearts is grotendeels belemmer in sy kliniese aktiwiteit deur roetine en klerikale werk, terwyl die stedelike praktisyn dikwels, weens tekort aan tyd, sy aandag moet bepaal tot 'n spesifieke pasiënt, en nie die geleentheid het om algemene kontak met die boer en sy boerdery te maak nie.

Die plattelandse veearts het 'n besondere rol in die veeartsenykunde gespeel. Hy het die geleentheid gehad om die boer en sy familie op 'n baie meer intieme sosiale gebied te leer ken, sonder die reserwe wat dikwels voorkom wanneer 'n „amptenaar" betrokke is. Hy was in nouer voeling met die gemeenskap waar hy praktiseer het as die predikant, onderwyser, magistraat en bankbestuurder, waar 'n mate van formaliteit en sosiale reserwe gewoonlik te bespeur is.

Die geaardheid van sy werk het hom die geleentheid gegee om die verhouding tussen veeartsenykunde in die teorie en boerdery in die werklikheid beter te korreleer, en op die wyse praktiese oplossings vir probleme te vind, veral waar boerdery betreklik extensief is, teenoor die intensiewe boerdery waarmee die stedelike praktisyn te doene het. Nie alleen het hy 'n besondere tipe diens gelewer in sy omgewing nie, maar hy het self 'n besonder tipe persoonlikheid ontwikkel. Omdat hy nie 'n amptenaar was nie moes hy hom by die boere aanpas en nie die boere by hom nie. Oorhaastig of burokraties kon hy nie wees nie, want dit duld die boer nie, maar hy het wel ook die boer takvol daarvan begin bewus maak dat tyd waardevol is, en samewerking tot beide se voordeel is. Die boer is gewoonlik ook baie meer openlik teenoor hom as teenoor die amptenaar.

Ons moet erken dat van hierdie kollegas wat op hierdie wyse „skool gegaan" het en later om een of ander rede by die geledere van die navorsing- veld- en opleidingspersoneel aangesluit het, 'n besondere veelsydige persoonlikheid van menslikheid en verdraagsaamheid aan die dag gelê het.

Nou is hierdie groepie besig om te verdwyn, en ongelukkig, ook nie stadig nie. Die veeartsontvolking van die platteland oor die afgelope vyf jaar is verontrustend, nie alleen vir die platteland, maar ook vir die professie. Streekslaboratoria is beplan en ontwikkel maar hulle en hul bemanning kan nie die „G.P." vervang nie, want daar kan nooit dieselfde noue voeling ontstaan wat daar tussen praktisyn en kliënt is nie. Die praktisyn leer sy gemeenskap en omgewing intiem ken — dit kan die streekslaboratorium nie doen nie. Die „plaas-skole" vir ons professie is besig om te sluit, en dit gaan tot nadeel van die professie wees as stappe nie gedoen word om hulle oop te hou nie.

Die redes vir die uittog uit die platteland is hoofsaaklik ekonomies. Tariewe vir dienste is noodsaaklik laag weens die betreklike lae inkomste wat die boer uit sy lewende hawe maak. Die ekonome maak die stelling dat as 'n

afdeling van die boerdery nie betalend is nie moet dit afgeskaf word. In die praktyk is dit egter nie so nie, want die boer moet sekere gedeeltes van sy plaas benuttig al is dit nie ekonomies nie. Skaf hy sy lewendehawe af word ander aspekte van sy boerdery ook onekonomies.

Meeste van ons plattelandse Veeartse bedien die „gemengde boer" by wie kontantoeste die grootste gedeelte van die inkomste uitmaak en die veestapel tot 'n mate subsidiêr is, om vir die boer 'n maandelikse inkomste te voorsien. Die inkomste uit hierdie bron is nie genoeg om alle lopende rekenings te dek nie, en dikwels moet die veearts ook wag tot die oes voordat sy rekening betaal word — misluk die oes, dan moet hy langer wag.

Baie boere is intelligent genoeg, (of nie genoeg nie), om die veeartse se diagnostiese metodes en sy middels self naderhand te gebruik en ook by sy bure toe te pas, tot nadeel van die veearts se inkomste. Daarby moet ook gereken word dat daar seisonale wisselings in siekteuitbreke in sommige distrikte is waar die praktisyn nie kan byhou met werk nie, en ander tye weer wanneer hy feitlik geen werk het nie. Om hierdie rede is dit dan ook selde dat die plattelandse veearts dit kan waag om 'n vennoot in te neem, terwyl 'n locum tenens, wat nog nie vertroue by die boere kon wek nie, vir hom nie van veel waarde is as hy met vakansie wil gaan nie.

'n Ander groot ekonomiese faktor by plattelandse praktyk is vervoerkoste en die tyd wat in beslag geneem word van een geval na een op 'n ander plaas. Dikwels wil die boer nie verstaan dat 'n besoek van 40 myl nie net twee gellings petrol kos nie, maar ook die professionele tyd, beroeps-risiko op die pad, verskeie assuransies, depresiasie op die motor ens. Dan ook is daar die feit dat hoe verder die geval van die kantoor is hoe groter die gevaar is dat 'n ander waardevolle pasiënt en klient verlore kan raak soos b.v. in 'n geval van akute melkkoors. Die plattelandse praktisyn kan nie een van sy kollegas kry om in noodsgevalle sy klient uit te help nie.

Die plattelandse praktisyn het ook meer te kampe met die middelsmouse wat by elke boer aanry met 'n bondel pamflette, 'n kas vol medisyne en klaargemaakte diagnose.

Dit is duidelik dat die plattelandse praktisyn, om dit so te stel, bars om 'n bestaan te maak. Goed, volgens die ekonome moet hy dan maar padgee of ondergaan, maar nog die boerdery nog die professie kan dit bekostig, want altwee word beroof van 'n onontbeerlike lid van die samelewing en wetenskap.

Wat is nou die oplossing? In die verlede is daar verskeie geleenthede geskep vir veeartse om hulle op die platteland te vestig deur Kooperatiewe verenigings, Boere-verenigings, deur waarborge van minimum inkomste ens. ens. maar feitlik al hierdie pogings het skipbreuk gely. Die groot rede daarvoor was feitlik in elke geval dat die professionele man onder verpligtings was teenoor individue of groepe wat teenstrydig was met sy professionele pligte en individualiteit. Dit is altyd die direkteur of een van die borge wat te veel vereis of hom inmeng in sake tussen die veearts en ander kliënte, en as professionele man moes hy gehoor gee aan leke wat hul inmeng met sy professionele aktiwiteit. Die gevolg was, en is, dat afgesien van gebrek aan finansiële sekuriteit, elke veearts wat sy sout werd is onder sulke omstandighede dan na 'n paar jaar die omgewing verlaat en die distrik is dan weer sonder hulp. Sal die staat deur opleiding en wyer verspreiding van veeartse die platteland beter kan bedien? Dit is twyfelagtig en onwaarskynlik aangesien die staatsveearts vereistes moet nakom wat hom nie daartoe in staat stel om die werk van 'n algemene praktisyn te verrig nie. Tydelike massaverskuiwings veral van jong staatsveeartse tydens uitbreke van Bek- en Klouseer kan so 'n plattelandse distrik vir weke of maande hulpeloos laat lê, terwyl promosies ook gedurige onderbrekings veroorsaak.

Dan is ook daar distrikte waar die dierebevolking betreklik hoog is maar die veeprobleme nie van so 'n geaardheid is dat dit nie die aanstelling van 'n voltydse staatsveearts regverdig nie, tog is daar soms staatsprobleme wat vereis dat 'n staatsveearts van elders blote roetine werk daar moet kom doen.

Myns insiens is die enigste oplossing van die plattelandse probleem, die aanstelling van Distriksveeartse op dieselfde basis as die van Distriksgeneeshere. Deur noukeurige ondersoek kan baie van die haakplekke en probleme

wat by Distriksgeneesheerdienste ondervind is uitgestryk word en so 'n diens aan die gemeenskap verstrek word. Deur die jong veeartse 'n redelike gewaarborgde inkomste te verseker vir staatswerk, aangevul deur gewone kliniese praktyk, kan hierdie streke bedien word tot voordeel van die gemeenskap deur die groter verspreiding van veeartsdienste; staatsveeartse sal minder in hul normale werk verhinder word, en die „plaasskool" vir veeartse sal weer 'n bron word van veelsydige kollegas in die professie, wat later moontlik tot groter voordeel van die land hul plek kan inneem in die verskillende vertakkings van ons professie.

BESLUITE VAN DIE VEEARTSRAAD

Met sy vergadering gehou te Onderstepoort op 8 Mei 1964 het die Veeartsraad die volgende besluite, vir die inligting van geregistreerde veeartse, vrygestel.

1. WETSPOSISIE IN VERBAND MET PUBLIKASIE VAN GRADE WAT IN WESE NIE VEEARTSENYKUNDIGE GRADE IS NIE.

Volgens die Wetsadviseer van die Raad is die Veeartswet na verwant aan die Wet op Geneeshere, Tandartse en Aptekers en dat daarin nie voorsiening gemaak is vir sodanige grade nie. In die Mediese-deel is daar egter 'n lys gepubliseer van sodanige grade wat wel gebruik kan word, dog almal is in wese mediese grade.

Aangesien in opdrag van die lede van die S.A.V.M.V. opgetree was toe destyds besluit was dat vir aantekeninge in die Veeartsregister alleen grade of diplomas erken word wat in wese veeartsenykundige praktisynsgrade of diplomas is, sal dit nodig wees dat eers weer tot die lede teruggegaan word indien 'n verandering beoog word.

2. TOEPASSING VAN HERSIENE WET OP VEEARTSE IN DIENS VAN DIERE-WELSYNSORGANISASIES.

Die Registrateur van Veeartse is versoek om die inhoud van die Veearts-wysigingswet onder die aandag van Diere-Welsynsorganisasies te bring en hulle aandag spesiaal te vestig op die wysiging van Artikel 17 van die Veeartswet waarin bepaal word dat die uitvoering van take wat spesifiek tot die beroep van 'n veearts behoort alleen toegelaat word om deur 'n geregistreerde veearts verrig te word. Die Registrateur sal die organisasies ook aanraai om hulleself op hoogte te bring met die voorwaardes waaronder veeartse deur nie-professionele liggame in diens geneem mag word en waarsku dat enige oortreding in 'n ernstige lig beskou sal word.

3. DIE OPSTELLING VAN 'N REGULASIE WAT VOORSIENING SAL MAAK VIR VEEARTSENYKUNDIGE ASSISTENTE, GENIET TANS DIE AANDAG VAN DIE VEEARTSRAAD.

4. HERSIENING VAN DIE GEDRAGSKODE

Die Veeartsraad is besig om aandag te skenk aan die hersiening van die „Handleiding vir Professionele Gedrag" en 'n komitee om die nodige aanvoorwerk te doen is alreeds benoem.

5. REGISTRASIE VAN VEEARTSE

Die volgende persone is onlangs as Veeartse in die Republiek geregistreer:—

Austin, J. C.	Russel, W. D.
Bilbrough, R. B.	Sutherland, R. J.
Brightman, M. P.	Sykes, R. D.
Brown, P. H.	Taylor, M. J. O.
Crewe, O.	Thomson, M.
Deacon, C.	van der Vywer, J. W.
Downes, S. J. T.	van der Merwe, C. P.
du Preez, J. H.	van Jaarsveld, A. S.
Faul, A.	van Schalkwyk, J. H.
Freeman, F. M.	van Wyk, C. J.
Harte, C. P.	Veary, C. M.
Immelman, A.	Weaver, D. B.
Jordaan, A. J.	Wilson, R. A.
Lombard, S. S.	Young, E.
MacNab, E.	Zumpt, J. F.

6. REGISTRASIE VAN BESOEKENDE VEEARTSE:

Die Rhodesiese Veeartsenykundige Owerhede het die sienswyse van die Veeartsraad gevra oor die kwessie van registrasie van besoekende gekwalifiseerde en registreerbare veeartse wat in die verteenwoordiging van hulle onderskeie firmas produkte verkoop, veeartsenykundige advies verskaf en ook vir konsultasie beskikbaar is.

Die sienswyse van die Raad is as volg:

(i) Dat in terme van die Veeartswet geen persoon in die Republiek toegelaat word om te praktiseer nie tensy hy as veearts geregistreer is en dat daar geen rede is waarom dieselfde houding nie ook in Rhodesië aangeneem word nie;

(ii) Die feit dat 'n veearts in die Republiek geregistreer is beteken nie dat hy ook in Rhodesië geregistreer is nie en vice versa. Dit sal dus nodig wees dat 'n besoekende gekwalifiseerde veearts eers geregistreer moet word alvorens hy kan praktiseer, hetsy in die Republiek of in Rhodesië;

(iii) Daarbenewens word dit in die Republiek ook nog van geregistreerde veeartse verwag, of hy in die handel is al dan nie, om aan die vereistes van die gedragskode soos uiteengesit in die „Handleiding vir Professionele Gedrag" te voldoen.

DECISIONS OF THE VETERINARY BOARD

At its meeting held at Onderstepoort on 8 May 1964 the Veterinary Board released the following decisions for the information of registered Veterinarians:—

1. LEGAL POSITION REGARDING THE PUBLICATION OF DEGREES NOT CONSIDERED TO BE VETERINARY DEGREES.

According to the Board's legal advisor the provisions of the Veterinary Act are closely related to those of the Medical, Dental and Pharmacy Act in so far as no provision is made for specific degrees. A list has however been published under the Medical, Dental and Pharmacy Act of degrees which may be used and which are in fact medical degrees.

The decision to recognise for registration purposes, only those degrees and diplomas which are in fact practising Veterinary degrees, was taken as the result of a resolution by members of the S.A.V.M.A. and any contemplated charge would have to be referred back to the S.A.V.M.A.

2. APPLICATION OF THE AMENDMENT ACT TO VETERINARIANS IN THE SERVICE OF ANIMAL WELFARE ORGANIZATIONS.

The Registrar of Veterinarians was requested to draw the attention of Animal Welfare Organizations to the context of the Veterinary Amendment Act and draw their attention particularly to the amendment of Section 17 of the Veterinary Act which requires that only registered veterinarians are allowed to undertake duties specifically assigned to veterinarians.

The Registrar shall also suggest to these organizations that they become *au fait* with the conditions under which veterinarians can serve non-veterinary bodies and warn them that any transgression will be regarded in a serious light.

3. THE PROMULGATION OF A REGULATION TO MAKE PROVISION FOR VETERINARY ASSISTANTS IS PRESENTLY RECEIVING THE ATTENTION OF THE VETERINARY BOARD.

4. REVISION OF THE CODE OF ETHICS

The Veterinary Board is engaged in the revision of the Guide to Professional Conduct and a committee has been appointed to undertake the preliminary work.

5. REGISTRATION OF VETERINARIANS

The following persons have recently been registered in the Republic as Veterinarians:

Austin, J. C.	Russel, W. D.
Bilbrough, R. B.	Sutherland, R. J.
Brightman, M. P.	Sykes, R. D.
Brown, P. H.	Taylor, M. J. O.
Crewe, O.	Thomson, M.
Deacon, C.	van der Vywer, J. W.
Downes, S. J. T.	van der Merwe, C. P.
du Preez, J. H.	van Jaarsveld, A. S.
Faul, A.	van Schalkwyk, J. H.
Freeman, F. M.	van Wyk, C. I.
Harte, C. P.	Veary, C. M.
Immelman, A.	Weaver, D. B.
Jordaan, A. J.	Wilson, R. A.
Lombard, S. S.	Young, E.
MacNab, E.	Zumpt, I. F.

6. Registration of Visiting Veterinarians

The Veterinary authorities of Rhodesia have approached the Veterinary Board for its opinion on the registration of visiting qualified and registerable veterinarians, who sell, consult, and advise on, the products of their firms.

The attitude of the Board is:

(i) That in terms of the Veterinary Act no person can practice in the Republic unless he is registered as a veterinarian and that there is no reason why the same attitude should not be adopted in Rhodesia.

(ii) The fact that a veterinarian is registered in the Republic does not mean that he is registered in Rhodesia and vice versa. It will thus be necessary for a visiting qualified veterinarian to be registered before he can practice, whether in the Republic or in Rhodesia.

(iii) In addition a veterinarian registered in the Republic is expected to comply with the provisions of the Guide to Professional Conduct, whether he is in commerce or not.

INTIMATION FROM THE CHIEF, VETERINARY FIELD SERVICES

RESTRICTED MOVEMENTS OF DOGS/CATS AND RABIES VACCINATIONS

From time to time reports come in that dog or cat owners are sometimes considerably inconvenienced when moving pets to or from declared rabies infected areas, or even to or from neighbouring territories. The difficulties generally arise through lack of knowledge of rabies vaccination and permit requirements.

Attempts have been made to obviate this by radio talks whereby owners were inter alia advised to contact their nearest State Veterinarian for full details before transporting their pets. Unfortunately the results have been somewhat disappointing and it is felt that an appeal to members of the Association, to advise their clients whenever the occasion arises, might be more fruitful.

Visitors to neighbouring territories must firstly comply with all the conditions imposed in the import permit issued by the veterinary authority of the country it is intended to visit but, if the pets are to be brought back to the Republic, an import permit must be timeously obtained from this office. It must also be kept in mind that with the exception of Basutoland, no dogs or cats will be permitted quarantine-free re-entry, unless they were vaccinated against rabies at least 30 days before leaving the Republic or 180 days after vaccination in the neighbouring territory. The duration of immunity is officially recognised as three years for dogs and one year for cats.

The whole of the Natal province, the districts of Matatiele, Mount Currie and Umzimkulu in East Griqualand as well as the following districts in Transvaal; Waterberg, Warmbad, Potgietersrus, Pietersburg, Soutpansberg, Messina, Sibasa, Letaba, Pilgrims Rest, Nelspruit, White River, Barberton and Piet Retief have been declared rabies infected areas in terms of the Animal Diseases and Parasites Act. No dogs or cats may be moved into these areas unless they are accompanied by an official removal permit. Before such permits are issued it is a general requirement that the dogs or cats are vaccinated against rabies. For movements into and out of the areas the vaccination condition is similar to that applying to neighbouring territories. Under certain circumstances, where there is not such a direct threat of infection, some relaxations are permissible particularly in regard to the movement of pups. As these dispensations vary according to the rabies position in the area, owners are best advised to obtain such information through the nearest State Veterinary Office.

It will be much appreciated if you could possibly draw the attention of all our colleagues to the above requirements. Any assistance given will be advantageous to their clients as well as to the country.

M. C. LAMBRECHTS,
Chief: Division of Veterinary
Field Services.

THE LEGAL ASPECT OF PROVIDING VETERINARY HOSPITALS IN THE AREAS OF LOCAL AUTHORITIES IN SOUTH AFRICA

In the previous issue of the Journal of the South African Veterinary Medical Association, (35(2)—**249**) reference was made to a judgement given in the Magistrate's Court of Pretoria, on May 6th., 1964, in a case heard on 9th. April 1964, in which Dr. C. L. Craig was charged for contravening the provisions of the Municipal Bye-law, which made it an offence to maintain dog Kennels on any premise in a built-up area, as required by Regulation 22 of the City Council's regulations, proclaimed in Administrators Notice No. 1058 of 30th November 1955.

Council for the defence attacked the Authority of the Bye-law and in a well reasoned judgement of over 2,000 words, the Magistrate in giving the verdict in favour of Dr. Craig, considered Regulation 22 as *ultra vires* to Section 80 of the Ordinance.

Much research will be necessary to ascertain the provisions of the appropriate Bye-laws of each local authority, and the extent to which they may be *intra vires.*

By and large, the town planning schemes of the various City and Town Councils, govern the requirements concerning the erection and maintainance of Veterinary Hospitals in the areas of local authorities.

Private practitioners should satisfy themselves that they are acting within the provisions of the Bye-laws of the local Authority serving their areas, more particularly when they buy existing practices or set up new ones.

EDITOR

'N MEMOIRE — PHILIP RUDOLPH VILJOEN

Philip Rudolph Viljoen M.R.C.V.S. Dr. Met. Vet. een van die sterk manne in die Veterinêre Professie is nie meer by ons nie. As seun van 12 jaar het hy alreeds sy durf bewys deur by die Boere-kommando aan te sluit gedurende die Boereoorlog. Hy was een van die eerste Afrikaner seuns wat deur Generaal Louis Botha, die destydse Eerste Minister en Minister van Landbou van die Transvaalse Verantwoordelike regering oorsee gestuur is om veeartsenykunde te studeer aan die Royal Veterinary College— Londen.

Hy was 'n entoesiastiese rugby speler en het vir die Royal Veterinary College, London en Old Charltonians, Kent, se spanne gespeel.

As Ere Vise-President van die Suid-Afrikaanse Veterinêr-Mediese Vereniging het hy selfs in die latere jare nog aktief deel geneem in professionele aangeleenthede.

Na werwing van die M.R.C.V.S. was hy op Onderstepoort werksaam. Kort daarna het die eerste wêreld oorlog uitgebreek en Viljoen was een van die veeartse wat die Unie-burgermagte vergesel het met die inval in Duits-Suidwes Afrika. Vir uitstaande diens gedurende daardie tyd het hy die Militêre onderskeiding M.C. ontvang. Ek wil hier net 'n paar uitstaande bakens noem in die kleurryke lewe van die uitstaande persoonlikheid.

1. As sleutel figuur in die oplossing van die lamsiekte probleem.

Na Militêre ontslag is hy terug na Onderstepoort en word verplaas as verantwoordelikebeampte op Armoedsvlakte.

Duncan Hutcheon het alreeds jare tevore beweer dat stywesiekte 'n afosforose is (Rachitis) en dat lamsiekte ook 'n fosfor tekort is, met 'n onbekende bykomende faktor. Die faktor was volgens die ondervinding van die boerebevolking orals, die vrot bene wat die beeste in sekere streke so lief voor is. Theiler en van sy medewerkers het dit probeer uittoets en uiteindelik tot die gevolgtrekking gekom dat die pica of osteophagia niks met lamsiekte te doen het nie en het 'n ingewikkelde plant intoksikasie teorie as oorsaak beskrywe.

Toe Viljoen op Armoedsvlakte kom en orals van die boere moes hoor dat die beenkouery gou gevolg word deur gevalle van lamsiekte, het dit so 'n indruk op hom gemaak dat hy daar en dan besluit om sekerheid hieroor te kry en het toe baie gou eksperimenteel bewys dat vrotvleis- en bene in daardie geweste wel lamsiekte veroorsaak.

As gevolg van hierdie werk van Viljoen kon Theiler en sy medewerkers toe bewys dat die bykomende faktor van Duncan Hutcheon niks anders as botulisme is nie.

2. SY AANDEEL IN DIE STIGTING VAN DIE FAKULTEIT VAN VEEARTSENYKUNDE.

Na sy terugkeer van Armoedsvlakte word hy benoem as Professor in Veeartsenykunde in die Fakulteit Landbou, Transvaal Universiteits Kollege.

In die Senaat van die T.U.K. het hy en Prof. Malherbe (Prof. van Chemie T.U.K.) sterk vertoë gemaak vir die stigting van 'n Fakulteit van Veeartsenykunde en ek glo ek is reg as ek sê dat as gevolg van die vertoë gemaak deur 'n komitee van die Transvaal Universiteits Kollege, het die regering uiteindelik besluit om 'n kommissie (waarvan Theiler lid was) te benoem wat aanbeveel het dat 'n Fakulteit van Veeartsenykunde gestig moet word.

3. DIE VEEARTSWET

Die Veterinêre Vereniging het sy fooie verhoog met die doel om 'n fonds op te bou vir die indiening van 'n lywige Veeartswet by die Parlement, min of meer soos die van die Mediese Professie.

Toe kom Viljoen met die voorstel om 'n eenvoudige wet op te stel, wat deur die goedgunstige medewerking van die destydse Minister van Landbou Generaal Kemp aanvaar is as 'n regeringsmaatreel, wat sonder enige of min onkoste aan die kant van die Veterinêre Vereniging, deur die Parlement geloods is. Onder die wet is die Veeartsraad 'n statutêre liggaam, die Voorsitter waarvan deur die Minister van Landbou benoem word en die Registrateur waarvan 'n publieke diens-amptenaar is en deur die Staat betaal word.

4. DIE BEMARKINGSWET

Hierdie Wet is onder die inisiatief en leiding van Dr. P. R. Viljoen as Sekretaris van Landbou opgestel en is 'n mylpaal in die geskiedenis van die boerebevolking van ons land en 'n kragtige instrument vir die bemarking en stabilisasie van pryse van Landbou produkte.

Hy was later ons gesant in Kanada en Australië, wonderlik bygestaan deur sy vrou, wat dwarsdeur sy lewe soveel vir hom beteken het.

Hy het 'n wonderlike pligsgevoel gehad. Die werk het by hom altyd eerste gekom. As hy 'n fout gehad het dan was dit dat hy geneig was om self teveel te doen.

Die veterinêre professie word steeds armer deur die verlies van die vorige geslag veeartse, waarvan Dr. P. R. Viljoen 'n uitstaande voorbeeld is en wat so 'n belangrike rol gespeel het, dat die prestasie van die veearts in die wetenskap, onder ons boerebevolking en die publiek so hoog aangeskrewe staan.

In hom het die veterinêre professie 'n kollega van internasionale formaat en getroue vriend verloor en sy vrou en kinders 'n voortreflike eggenoot en waardige vader.

In dankbare herinnering betuig ons ons innige meegevoel met die familie.

P. J. J. FOURIE

OBITUARY

LEONARD RIPPON MORFORD

Dr. Morford's many friends will have learnt with deep regret of his sudden and unexpected death on the 26th June.

Leonard was born in London 58 years ago and, before entering the Royal Veterinary College, London, was engaged in farming for five years. He qualified in 1933, spent one year in the R.A.V.C. and about 18 months in practice, first as assistant to Mr. G. N. Gould of Southampton and later to Mr. P. Gautley of London.

In 1936, he joined Mr. S. T. A. Amos of Durban and in 1937 married Mr. Amos' daughter, Vera, whom he had met when they were students together in the London College. In 1946, they bought the practice from Mr. Amos and, together with Dr. D. G. Clow who joined them in partnership later, have carried it on ever since. He was commissioned in the S.A.V.C. during World War II and retired, on the cessation of hostilities, with the rank of Captain.

Leonard had a zest for life, liked to see things, visit places and experience the unusual. He was keen on outdoor life and a frequent visitor

to Game Reserves where he could watch and photograph wild life, particularly bird life. He was a member of the Bird Club, the Wild Life Association, the committee of the Oceanographic Research Institute and of the Royal Society of South Africa. He was an enthuasistic Rotarian, a one-time member of the Point Yacht Club and a keen yachtsman.

His charm, cheerfulness and kindness endeared him to all. He was frienldy to everybody, a delightful companion and, in the words of a former assistant, a chief in a thousand. He found it difficult to bear malice and preferred to shrug off a slight or unkindness rather than to harbour a grievance.

Because he was a thoroughly efficient practitioner, he had the respect of his clients, mostly owners of small animals, and of his racing friends of the Greyville and Clairwood courses to both of which he was veterinary surgeon. He took his work seriously and regarded it somewhat in the nature of a challenge. He was genuinely upset over a mistaken diagnosis but almost boyishly thrilled when he correctly pinpointed the cause of an obscure illness. Whatever the outcome of his advice and treatment, the client was always left with the feeling that all was done that could have been done.

His passing is truly regretted by his veterinary colleagues, his fellow Rotarians, his racing friends and the many owners of small animals in and around Durban.

To his widow and four children, the profession tenders its sincere sympathy.

J. H. M.

SOCIAL AND PUBLIC RELATIONS SERVICE

Dr. S. W. van Rensburg has been awarded the Antonio Perocchi Prize for the best paper submitted on fundamental research on any aspect of animal reproduction. He submitted a paper on "Possible Endochrine Mechanisms in Gestational Failure". This contribution will be published in the Proceedings of the Fifth International Congress on Reproduction and A. I. in Animals, which is to be held at Trento, Italy from 5th. to 13th. September 1964.

Our hearty congratulations go to Dr. Schalkie van Rensburg on this very fine achievement.

Dr. S. W. J. van Rensburg has been awarded a Medal, by the Lazarro Institute of Artificial Insemination in recognition of his contributions to the advancement of knowledge on Infertility and A.I. in animals.

Our special congratulations go to Schalk for this well earned recognition.

We cannot recall an occasion in South Africa where father and son, as veterinarians, are honoured at the same International Congress.

Dr. C. T. McDonald of the Transvaal A.I. Co-op is also to attend the Fifth International Congress on Reproduction and A.I. in Animals at Trento, Italy.

Dr. Coskuner, a Veterinarian from Turkey is a visitor at Onderstepoort, and spending some time with Dr. W. O. Neitz in the section of Protozoology of the Veterinary Research Institute.

Dr. Gerda von Philipsborn from Western Germany has been appointed to the staff of the Pathology Section of the Veterinary Research Institute, Onderstepoort.

Mr. W. H. Gerneke M.Sc. *Cum Laude*, Senior lecturer in histology, of the Faculty of Veterinary Science Onderstepoort, and Dr. F. M. C. Gilchrist of the Section of Digestion and Metabolism of Ruminants, have unanimously been proposed by the Council, to be Honorary Associate members of the S.A.V.M.A.

Dr. J. F. Figueroa, one of the Vice-Presidents of the World Veterinary Association, will be visiting the Republic at the time of the Congress and hopes to give a short address on topical aspects of the W.V.A.

A manuscript copy of the Report of the Commission of Enquiry into Abattoir and Allied Facilities is now available in limited supply. It is a formidable report and occupies 376 pages.

18th WORLD VETERINARY CONGRESS

The 18th W.V.C. will be held at Paris from Monday 17th to Saturday 22nd July 1967. Thursday 20th July will be reserved for the commemoration of the bicentenary of the foundation of the Ecole Nationale Vétérinaire at Alfort (near Paris). Details will be published later.

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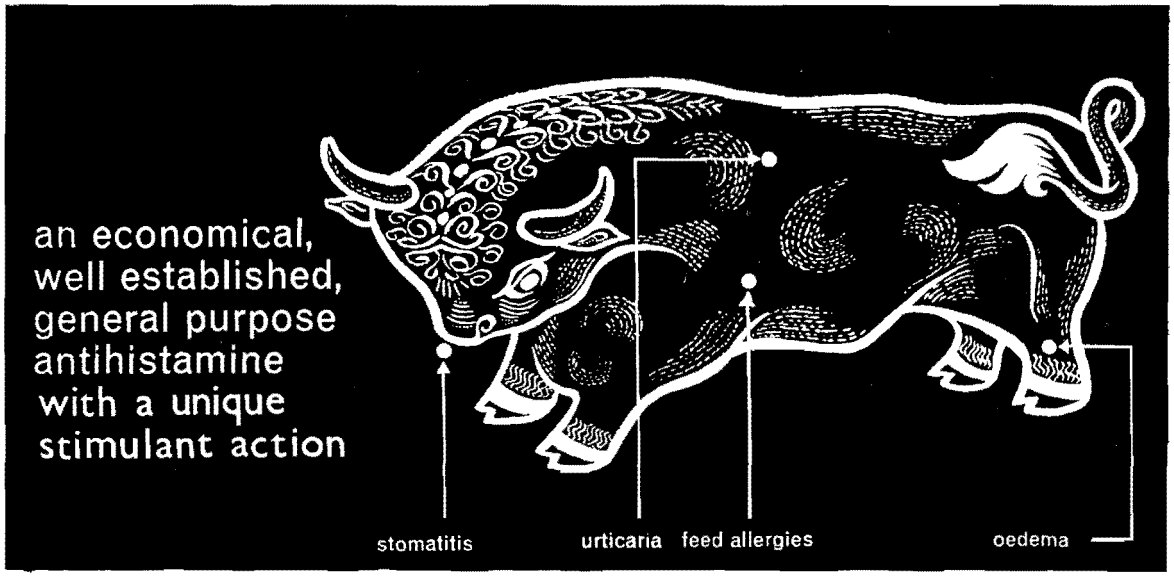

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OUT OF THE PAST

PROF. DR. ROBERT KOCH

Prof. Robert Koch was born on December 11th. 1843. He died on 28th May 1910.

He received the Nobel Prize in 1905. His contributions to the control of Rinderpest and East Coast Fever in South Africa are very well known.

This photograph was probably taken in Southern Rhodesia.

J. A. EDMUNDS
Glen Larne Salisbury,
On 1890 Pioneer.

WORLD SCIENCE DELEGATES AT COOPER CONFERENCE

Scientific delegates from associated companies and branches of Cooper, McDougall & Robertson Ltd. all over the world gathered at Berkhamsted, Hertfordshire, during the first fortnight in May for a conference on the development of new products from synthesis to marketing.

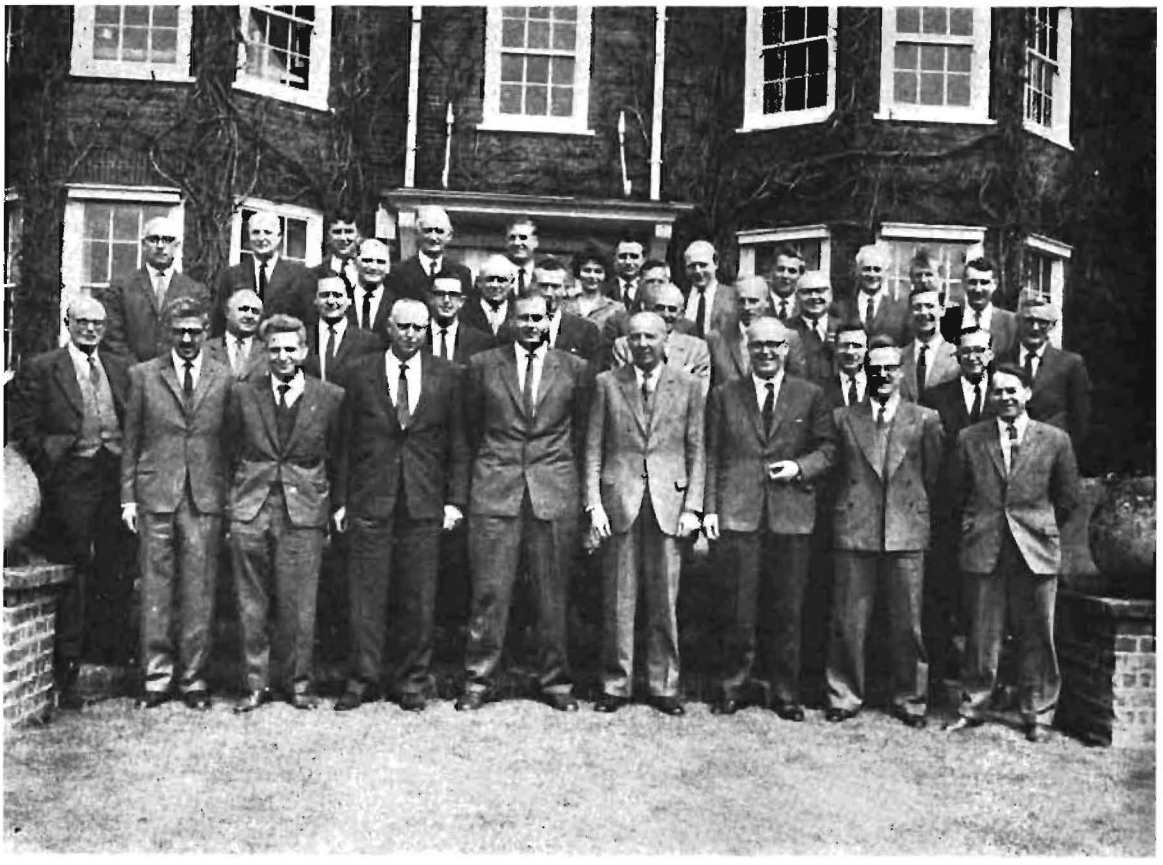
Delegates came from the United States, Argentine, Uruguay, Colombia, East Africa, Central Africa, South Africa, Australia, New Zealand, Spain and Eire.

Subjects for study and discussion included screening methods; analytical techniques; the development of anthelmintics; the control of cattle tick, sheep scab and other parasitic diseases; the development of insecticides for household and industrial use; toxicity and residue studies and formulation.

Delegates conferred with their scientific colleagues from the Cooper Technical Bureau in the reception room of the company's veterinary research station at Berkhamsted Hill. In the chair at the conference was Mr. H. E. Harbour, Technical Director, Cooper, McDougall & Robertson Ltd. Previous world-wide conferences were held at Berkhamsted in 1951 and 1959.

The following is a full list of delegates.

Dr. C. N. Ault	Dr. in Vet. Med. (La Plata)	Technical Director	Productos Veterinarios Cooper S.A. Argentine.
Dr. B. D. Booth	B. Sc., Ph.D. A.R.I.C. A.R.A.C.I.	Research Manager	Wm. Cooper & Nephews (Aust.) Pty. Ltd. Australia.
Dr. J. A. Thorburn	B. V. Sc. (S. Africa)	Senior Veterinary Advisor & Director	Cooper, McDougall & Robertson (Central Africa) (Pvt) Ltd. Central Africa.
Mr. W. S. Allan	M. Agr. Sc.	Technical Advisor	Cooper, McDougall & Robertson (New Zealand) Ltd. New Zealand.
Dr. W. M. McHardy.	B. V. Sc. (S. Africa)	Senior Technical Advisor	Cooper & Nephews (South Africa) (Pty) Ltd. South Africa.
Mr. H. G. Wallace	M.R.C.V.S.	Technical Advisor & Director	Cooper, McDougall & Robertson (East Africa) Ltd. East Africa.
Dr. D. Abaracon	Med. Vet. (Montevideo)	Chief of Vaccine Department	Cooper, McDougall & Robertson Ltd. Uruguay.
Dr. P. Moraleda	Med. Vet. (Madrid)	Technical Advisor	Cooper, McDougall & Robertson Ltd. Colombia.
Mr. W. V. Miller	B.Sc., M.S. (Ento.)	Head of Research & Product Dept.	Wm. Cooper & Nephews Inc. Chicago.
Dr. N. J. O'Connor	M.Sc., Ph.D.	Technical Advisor	Cooper, McDougall & Robertson (Ireland) Ltd. Dublin
Dr Campos Onetti		Technical Director	Cooper-Zeltia S.A. Spain.



Back Row:

Mr. R. W. SEAR. Mr. R. McINTYRE. Mr. R. D. SHAR. Capt. R. OLIVER-BELLASIS. Mr. J. WICKHAM. Mr. R. H. LEE. Mr. P. R. M. BROWN.

Third Row:

Mr. R. M. NUMFORD. Mr. W. S. MANSON. Miss M. WOOD. (Secretary) HIDDEN. Mr. S. ALLEN. (N.Z.) Mr. E. P. POLLARD. Mr. L. C. STONES. Mr. N. C. BROWN.

Second Row:

Mr. D. M. SIMPSON. Dr. S. O'CONNOR. (EIRE) Mr. B. D. BOOTH. (AUST.) Mr. W. MILLER. (U.S.A.) Mr. K. GOODWIN-BAILLY. Mr. W. H. BEAUMONT. Dr. R. F. PHIPERS.

Front Row:

Mr. W. DOWNING. Rr. D. ABARACON. (URUGUAY) Dr. P. MORALEDA. (COLOMBIA) Dr. J. A. THORBURN. (C. AF.) Dr. W. M. McHARDY. (S.A.F.) Dr. C. N. AULT. (ARGETINE Mr. H. E. HARBOUR. Mr. R. G. WALLACE. (E. AF.) Mr. J. C. WOOD.

BOOK REVIEW

CHIRURGIA VETERINARIA

VERLAG PAUL PAREY DM26 FOR EACH EDITION.

A quarterly Journal (commencing July 1st. 1964) providing a periodical and exhaustive review in the field of veterinary surgery, including the field of anaesthesiology, ophthalmology and roentgenology.

The abstracts will be published in German, English and French.

Subscriber must agree to accept complete volume.

Chirurgia Veterinaria provides a periodical and exhaustive review of the particular results of works in the field of the international veterinary surgery which has otherwise not been possible up to now. When published, Chirurgia Veterinaria will provide a remarkable intensification and support of the international collaboration in the field of veterinary surgery. For the first time, Chirurgia Veterinaria makes it possible to observe thoroughly the whole bibliography of veterinary surgery including neighbouring fields of medicine.

In order to support the international collaboration between the veterinary surgeons in all the world as well as between veterinary surgeons and physicians, veterinary surgeons joined in the European and World Association for Veterinary Surgery decided to found a particular organ of reports of the international veterinary surgery.

Abstracts of every scientific or professional paper referring to the field of veterinary surgery, including the field of anaesthesiology, ophthalmology and roentgenology — wherever it might be published — , will be published at once in the Chirurgia Veterinaria. This Journal will publish abstracts of important original papers from other journals, reviews, dissertations, theses for habilitation, monographs, lectures given at congresses and conferences which appear in print, as well as books and special publications; every abstract will be completed with information about dosages, chemical and patent names of preparations, and with a detailed bibliography. Publications on the subject in the field of medicine — as far as they are of importance to the veterinary science — will also be taken into consideration.

The abstracts will be published in German, English, and French, irrespective of the language of the original paper and they will be provided with a special system of English key notes which represents a new system of classification. This system is very useful for a standardized classification of the abstracts by the aid of the normal terminology. Thus you will recognize at once the principal theme and the most important statements of a paper. This list of key notes will furthermore form the basis for classifying the abstracts in a card-index. An electronic data collecting system where all abstracts will be gathered, will be of great advantage for the extensive and systematic work of this international journal. Thus all abstracts are collected by the aid of the new key note system in order to supply efficiently and quickly the index at the end of every year. Besides this method makes it possible to establish quickly at any time — even after years — bibliographies referring to special themes in the field of veterinary surgery.

LEXICON OF PARASITES AND DISEASES IN LIVESTOCK

by Manuel Merino-Rodríguez.

Elsevier Publishing Co. 1964. pp. 125. Price 40s.

This useful little book is one of a series of lexica published by the Elsevier Publishing Company. It is divided into three main sec-

tions:— Parasitology, Bacteriology and Virology, and Other Diseases. The Latin name of the parasite or causal organism is given first and then its English, French, Spanish, Italian and German name; this is followed by the common name of the disease. It is unavoidable that the two parts are not always complete, for as the author points out, some organisms or the disturbance caused by them, may not have a specific name in all languages. The value of this book is enhanced in that synonyms are also given.

The reviewer, a helminthologist, is not in a position to review any but the helminthological section in a critical manner. In so far as the helminthological side, with the exception of a few ommissions, is well covered, it may be assumed that the other sections are on a par with it.

In the helminthological section the common name of *Ascaris* and the sexual state of *T. solium* are ommitted. The term "cestodiasis" for infestations of tapeworms is only mentioned once, and then as "cestodose". The disease complex of *Paramphistomum microbothriodes* is given as "paramphistomiasis" but this term is ommitted for infestations of *P. cervi.* Infestations of the bladderworm of *T. solium* and *T. saginata* (cysticercosis) are listed, but not the term "taeniasis". The names of infestations by *Dictyocaulus filaria*, *Dictyophyme renale* and *Dirofilaria immitis*, viz. dictyocaulusiasis, dictyophymiasis and dirofilariasis respectively, are also ommitted.

This book will be of particular value to biologists and veterinarians, who are not *au fait* with foreign languages as the second part consists of alphabetical indexes to the six languages.

A. Verster

VETERINARY ORTHOPAEDICS

by

John Hickman

John Hickman—Veterinary Orthopaedics.

Oliver & Boyd., Edinburgh & London 1964
p.p. 479, Illustrations 296
Price

This is the first book published, that is devoted entirly to veterinary orthopaedics in domestic animals. It is a comprehensive, simple and yet detailed work, especially written for veterinary students but should also be of great value to the private practitioner.

This book is extremely well illustrated, with 73 photographs, 123 Radiographs and 100 line drawings.

The work is subdivided under the following chapters:—

Chapter I.—Deals with the diagnosis and treatment of lameness.

Chapter II.—Deals with tendons, sheaths, bursae and muscles.

Chapters III, IV, V, VI, which cover 264 pages, are devoted to diseases and injuries of bones and joints.

Chapter VII.—Covers injuries and diseases of the hoof and claw, as well as the dog's foot.

Chapter VIII.—Deals with the spine and peripheral nerves and also includes a section on neurectomy.

The last chapter is especially devoted to amputations of limbs. There is also a section on materials used in bone and joint surgery and an appendix on orthopaedic instruments and appliances.

Only an authority on veterinary orthopaedics can produce work of such high quality. e.g. The Chapter on navicular disease.

It is difficult to critisize a book of this nature because it has to cover such a wide field; but I do feel that seeing the book is essentially written for students, more techniques should have been described for some operations, e.g. a very detailed description is given for the amputation of the front leg of the dog, through the mid shaft of the humerus, whereas a very popular operation through the shoulder joint is just mentioned, but not described.

It is also noted that the author makes no mention of corticosteroid therapy in his chapter on the treatment of lameness.

I think the author should be complimented on work put into the preparation of this book, and the high quality achieved.

As this is the first book of its kind, it is a welcome addition to that part of our veterinary literature which has been sadly neglected in the past.

C. H. BASSON.

MEETINGS OF BRANCHES

MINUTES OF THE ANNUAL GENERAL MEETING, NATAL BRANCH, S.A.V.M.A., HELD AT ROYAL HOTEL, PIETERMARITZBURG at 9 am. ON June 16, 1964

PRESENT:

Dr's F. Ducasse, H. Strydom, A. Tarr, F. Cavanagh, A. M. Diesel, R. Bezuidenhout, A. Fair, R. Sykes, M. Brightman, A Downes, R. Nixon, G. Bishop, A. Thiel, R. Solomon, I. Canham, M. Brown, B. Paine, J. du Preez, G. Bisschop, P. Bisschop, D. B. Weaver, J. Chandler, G. Martinaglia, C. Nilsen, L. Blomefield, H. Holtz, J. Fick, P. J. Anderson, P. Collier, A. Gillespie, D. Clow, L. Morford, V. Morford, P. Wacher, D. Coles, P. Snyman, B. Jansen, M. Lambrechts, R. Every, D. le Roux, B. Baker, A. J. Louw, J. Doré, S. Turner, D. Osborn, I. Banks and J. O'Grady.

VISITORS:

Dr's. B. C. Jansen, M. C. Lambrechts and M. Brown.

The Chairman, Dr. Doré welcomed the visitors as well as Dr. A. Diesel an Hon. Life member of the Natal Branch.

APOLOGIES:

Dr's. H. P. Steyn, M. Shires, W. Hobbs, W. Barnard, K. Dalzell, W. Viljoen and D. McMillan.

Dr. A. M. Diesel then addressed the meeting. He conveyed the greetings of the President, Dr. H. P. Steyn. During his address Dr. Diesel mentioned that for the first time in the history of the Association the Journal had paid for itself. He also expressed his confidence in the young members of the profession and said that in his opinion the profession was going from strength to strength.

MINUTES OF PREVIOUS A.G.M.:

Read and confirmed.

MATTERS ARISING:

At the request of Dr. Coles, the chairman made a statement on the resolutions passed at the previous A.G.M. i.e. A scroll had been presented to the Minister of Agricultural Technical Services. Dr. Diesel said that it might be some time before the possibility of a second Faculty became a reality.

CHAIRMAN'S ADDRESS:

Ladies and Gentlemen,

It is my duty to present to you an account of the stewardship of your committee for the past year.

Only one committee meeting was held during my term of office, and that was mainly to make arrangements for the Annual General Meeting of this branch. It has been our experience that much of the work previously delegated to the branches is now being efficiently dealt with by the Association's Secretary, Dr. Diesel; and that all we had to do was to reply to a few letters from headquarters. This was propmtly carried out by our branch secretary, Dr. O'Grady. I would therefore, like to express my appreciation and thanks to Dr. Diesel and Dr. O'Grady for making my term of office so easy and pleasant. Also I am sure that members who attended the two informal meetings held during the year would like me to thank Dr. O'Grady for his efforts of organising them. For the record and for those who were unable to attend them, the first meeting was held at Dr. Tarr's residence where Dr. le Roux gave us a short talk on "Snakes in South Africa", and Dr. Pudifin related to us some recent advances in chromosome studies. Then followed films on "Foul in the foot of the Dairy Cow", and "Muscle Relaxants in the Dog".

The second meeting was held at the Caister Hotel in Durban. Dr. Shires brought us up to date with a short talk of Aflotoxicosis. The films that were shown were "The Epol Story", "The Gentle Doctor", "The Atrial Septal defect in Humans", and "New Techniques of Anaesthesia of the Horse". I am grateful to the members who attended these meetings. It makes us feel that our efforts were not in vain.

It is also incumbent upon me as chairman to draw your attention to current affairs of the profession in general and Natal in particular. Glancing around us, we note a general rise in prosperity in the country. There is talk of the development of the Tugela Basin, and we must ask ourselves whether the profession is ready to march side by side with these new developments. If there is no preparation for these advances, obviously the profession must fall into the background. I would like to pay tribute to the pioneer veterinary surgeons in private practice in the rural areas. They have battled against the many odds, but chiefly the distances they have to travel has been the greatest bugbear. Nevertheless they have established a good name for the profession, and it is a feather in the cap to them that there are many farmers willing to pay high mileage fees to obtain their services. The only way of overcoming this distance problem is to have more veterinarians scattered at intervals throughout the country so that the farmer need not have to pay such high mileage fees when he requires the service of a veterinary surgeon. In fairness to the farmer and to the veterinarian, our profession should take steps to encourage practices in the smaller rural towns by finding part-time state and municipal employment to keep these practices going. Only in this way will we be able to regain our control over certain drugs which the 'do-it-yourself' farmer should never be allowed to handle. But first we need more veterinarians.

It can be construed that from newspaper reports, veterinarians will be called upon to play a greater role in abattoir supervision, construction and administration. In the past, especially in the rural areas, the standard of hygiene, and the standard of milk and meat inspection, have been shockingly low. Sometimes even cruel acts of slaughter have been perpetrated. Only the veterinarian is qualified to rectify these matters. But first we need more veterinarians.

Another problem mounting in gravity is the millions of Bantu stock at present standing in the liability columns of the country's books. This liability is calculated in terms of the ravages caused by overstocking, overgrazing, soil erosion, tick and worm infestation, and losses of stock from poverty and diseases with little or no salvageable by-products. The only tool that can convert this liability into an asset is enlightenment and knowledge and only the veterinarians are qualified to wield this tool.

If we are to assume these rightful responsibilities then we shall have to increase our numbers considerably. Veterinary education must be the building stone of our profession's expansion. The standard of theoretical knowledge of graduating students from Onderstepoort is good, and we congratulate the professors and lecturers for their efforts. The faculty has no control over the amount of clinical material availale for demonstration and giving the students experience; therefore any lack of clinical experience is not the direct fault of the faculty. But to increase the number of students at one university will only mean that each student will have less clinical experience than ever. This will be a great pity and the young veterinarian may find himself gravely handicapped when faced with a demanding public.

I reiterate therefore, the plea made by the Association's President for a second faculty of Veterinary Science. I have taken into consideration the burden of responsibilities we shall be called upon to shoulder. I also recognise the fact that the country is proud of its veterinary profession and all its accomplishments. The standard of our veterinary education is such that our graduates are in demand overseas. These are the laurels we have gathered; let us not rest upon them now, but let us muster our strength to increase our numbers, keeping the same high standard that we have produced in our past, so that when the order is given to advance, we can say with confidence; "We are ready".

ELECTION OF NEW MEMBERS

Dr. D. B. Weaver — prop. Dr. L. Blomefield who also proposed Drs. M. M. Brightman & A. Gillespie.

Dr. R. Sykes —prop. by Dr. I. Canham.
Dr. P. J. Anderson — prop. by Dr. D. le Roux.
Drs. P. & G. Bisschop—prop. by Dr. A. Fair.
Dr. A. Downes — prop. by Dr. J. Doré.
Dr. C. Nilson — prop. by Dr. L. Blomefield.
Dr. P. Brown — prop. by Dr. S. Turner.
Dr. I. Banks — prop. by Dr. R. Nixon.

ELECTION OF OFFICE BEARERS:

(*a*) Chairman — Dr. J. L. Doré.
(*b*) Vice-chairman — Dr. F. B. Ducasse.
(*c*) Secr./Treasurer — Dr. J. M. O'Grady.
(*d*) Members of Committee: Drs. A. Tarr, W. Hobbs, A. Thiel, R. Solomon, L. Blomefield.

FINANCE.:

Credit Balance of R112.56 Honorarium of R10 for Secretary proposed by Dr. I. Canham and seconded by Dr. P. Bisschop.

CORRESPONDENCE.:

Three letters from Dr. A. M. Diesel dealing respectively with Schedule of fees for sheep, appeal for material for S.A.V.M.A. Journal and a letter from the President of the R.C.V.S. were read to members.

GENERAL

Dr. R. A. Solomon enquired of the meeting whether it was ethical for a Cedara Agricultural student to see practice with a veterinarian.

Dr. B. Jansen replied that he did not think this could be regarded as unethical as we were protected by the Veterinary Act.

Dr. B. Paine mentioned that at a previous enquiry the President of the Association had advised against this practise. Dr. Paine expressed the opinion that he regarded this as a dangerous practise.

Dr. G. Bisschop enquired what control existed over stock inspectors and assistant stock inspectors.

Dr. M. C. Lambrechts replied that 3 weeks training at Ermelo in the use of syringes, performance of post mortems etc was the practise. The intention was to intensify this training. Any complaints against staff were severely dealt with.

Dr. J. Fick suggested better education of farmers to make intelligent use of veterinarians.

Dr. I Canham enquired about the courses given to inseminators.

Dr. M. C. Lambrechts explained that there were two courses of instruction — one for inseminators and one for farmers. A very selective method of enrolment was employed. At Potchefstroom 8 courses per year were provided but the demand was still great. In his opinion these courses were essential for the dissemination of knowledge. The Act provided adequate protection to veterinarians.

Dr. M. Brown addressed the members after tea — his paper "Metabolic Disturbances and Trace Element Deficiencies in Domestic Animals" was very well received and some discussion followed.

Prof. B. C. Jansen brought the members up to date with his paper "Recent Advances in Veterinary Research."

After a very well-attended lunch Dr. A. M. Diesel addressed the members on "The Association of Branches of the S.A.V.M.A. to the Parent Body".

Out of this arose the fact that the majority of members present were in favour of an annual Congress in preference to a bi-annual one.

Dr. M. C. Lambrechts capped a most successful day with his talk on the "Aims and Functions of the State Service".

A very enjoyable and well-attended Cocktail Party followed at the Royal Hotel.

A feature of the day's proceedings was the virtual 90% attendance of members which was most gratifying.

VERSLAG AANGAANDE DIE JAARVERGADERING VAN DIE O.V.S.-TAK: S.A.V.M.V. GEHOU TE BLOEMFONTEIN OP 13 JUNIE 1964.

Vyftien lede van die plaaslike tak sowel as die President Dr. H. P. Steyn en Dr. B. J. Erasmus as gasspreker, het die vergadering bygewoon.

Die besigheidsvergadering is die voormiddag afgehandel waarvan die verloop as volg saamgevat kan word:

Voortvloeiend uit die notule van die vorige jaarvergadering word 'n besluit deur die vergadering geneem dat die tak voortaan as die O.V.S. Tak bekend sal staan.

In die Voorsittersrede meld die voorsitter eerstens dat die takvergadering deur min Staatsveeartse bygewoon word as gevolg van 'n hersonering van die gebiede, met die gevolg dat nabygeleë Staatsveeartse nie die vergadering bywoon omdat hulle nie in die Vrystaat streek val nie, en dus op eie onkoste moet kom om die vergadering by te woon.

Die voorsitter meld verder dat hy hoofsaaklik iets wil sê omtrent die verdwyning van die Plattelandse Veeartse wat sulke waardevolle dienste aan boere in die verlede gelewer het. Die redes vir die verdwyning is hoofsaaklik ekonomies. Die plattelandse veearts moet hom gewoonlik aanpas by omstandighede en dikwels maande en selfs jare wag vir betaling van fooie as gevolg van mislukte oeste en ander omstandighede van die boere, waarmee die veearts vertroud is. Vervoerkoste en tyd deur ritte in beslag geneem, speel ook 'n groot rol in die praktyke.

Die boere is gewoonlik ook intelligent genoeg om die veearts se diagnostiese metodes self, en by bure, toe te pas en met die maklike beskikbaarheid van alle middels, deur allerhande verkoopsorganisasies verskaf, die behandeling ook toe te pas, met nadelige gevolge vir die veearts.

Die plattelandse veearts vind dit uiters moeilik onder die omstandighede en gee maar eerder pad om 'n beter lewe elders te maak. Gevolglik vind verliese vir die professie sowel as die boere wat weer aan hulle lot oorgelaat word, plaas deurdat diere vrek as gevolg van swak veeartsenydienste.

Die oplossing vir die probleem is die aanstelling van Distriksveeartse op dieselfde basis as Distriksgeneeshere. Die veearts word dan van 'n redelike gewaarborgde inkomste van staatsweë voorsien wat aangevul word deur kliniese werk in 'n privaat praktyk. Tot voordeel van die gemeenskap word die stelsel toegepas deur groter en intensiewer verspreiding van veeartsenydienste. Boere sal in die gevalle ook meer veeartsbewus word en oneindige werksgeleenthede sal op die manier aan die professie verskaf word.

Dr. Steyn word vervolgens aan die woord gestel deur die voorsitter wat meld dat dit 'n uitstekende reeling is dat die President of Sekretaris van die moederliggaam, takvergaderings bywoon. Dit bewerk baie noue kontak tussen die moederliggaam en die takke.

Eerstens wens Dr. Steyn die voorsitter geluk met sy voorsittersrede en meld dat daarin baie stof tot nadenke is.

Verder meld die President dat dit ontstellend is om te sien dat Staatsveeartse die takvergadering so swak bywoon, aangesien die vereniging, moederliggaam sowel as die takke, baie in die verlede vir die Staatsveearts gedoen het bv. verbeterde salarisskale.

Wat betref die deeltydse indiensneming van Privaat Praktisyns om staatswerk te verrig, meld Dr. Steyn dat daar ondersoek ingestel is in die verband, maar dat daar besware geopper is van sekere dele van die professie wat nie die nodigheid van so 'n stelsel insien nie.

Vervolgens deel hy die vergadering mee dat daar op die oomblik 'n aantal kommissies van ondersoek is, aangaande verskillende aspekte van die professie:

(i) 'n Kommissie van ondersoek betreffende die hele professie is by die Minister aangevra, maar die vereniging is versoek om 'n memorandum aan die Minister voor te lê, wat die samestelling van genoemde kommissie regverdig. 'n Kommissie is saamgestel om in alle vertakkings van die professie vraelyste te sirkuleer om sodoende die verlangde memorandum op te stel en aan die Minister voor te lê;

(ii) 'n Kommissie van ondersoek betreffende ko-operasies is reeds voltooi en 'n memorandum reeds voorgelê;

(iii) 'n Kommissie wat ondersoek ingestel het na gevaarlike middels, het reeds rapporteer.

Wetgewing word tans geloods in verband met aptekers en die wetgewing sal die professie ook raak.

Verder meld Dr. Steyn dat die Fakulteit van Veeartsenykunde bekommerd is oor die verhouding van veeartsstudente jeens militêre opleiding. Geen Veterinêre korps bestaan meer in Suid-Afrika nie. Die veearts speel absoluut 'n vitale rol in moderne oorlogsvoering in soverre dit die voeding van troepe met gesonde voedsel asook die behoud en versorging van die veestapel aanbetref.

Die President is deur die Voorsitter bedank. Melding word gemaak dat dit nie alleenlik koöperasies is wat 'n rol speel in verspreiding van gevaarlike middels nie, maar ook wolmakelaars, romerye, kaasfabrieke en die farmaseutiese firmas self. 'n Ander sondebok wat in die latere tyd al hoe meer 'n rol speel is die entstof verspreiders wat nie alleen entstowwe versprei nie, maar alle middels vir veeartsenykundige gebruik verkoop, en selfs behandelings word vryelik vir siektes deur ongekwalifiseerde persone voorgeskryf.

Die President meld dat die vraag gestel moet word of die professie voldoen het aan die vereistes wat die publiek stel. Hierop is die antwoord: „Nee". 'n Geweldige tekort aan veeartse bestaan en daarom het die publiek geleer om hulle diere self te behandel met behulp van middels verkry van genoemde instansies.

'n Lid noem ook dat Tegniese Assistente van die Streekslaboratoria uitgestuur word na vrektes van vee om monsters te neem. Indien dieselfde kondisie 'n aantal kere teëgekom word maak die tegniese assistent later die diagnose self en skryf die behandeling en voorkoming daarvan voor.

Wat korrespondensie aanbetref, is briewe ontvang, aan die vergadering voorgelê:

(i) Betreffende vleis en melk inspeksie dienste word verneem dat geen deeltydse werk deur lede verrig word nie;

(ii) Sover verneem is, is daar geen beperkings aangaande die aantal honde in stede en dorpe, wat op een perseel aangehou word in die streek nie;

(iii) Kennis word geneem van 'n brief ontvang van die „Royal College of Veterinary Surgeons" betreffende veeartse wat later praktiseer in gebiede waar hulle vroeër in diens was;

(iv) 'n Navraag ontvang betreffende die jongste lys van fooie vir die tak is voorgelees. Hierna volg 'n bespreking van 'n konsep-fooiskedule vir die tak. 'n Onderkomitee word benoem om die fooie op te stel, aan lede vir kommentaar te sirkuleer en daarna 'n eenvormige fooiskedule op te trek vir die tak.

Die volgende komitee is verkies vir die volgende jaar:

Voorsitter: Dr. D. J. Louw.
Onder-Voorsitter: Dr. N. Barrie.
Ere-Sekr./Penningmeester: Dr. H. G. J. Coetzee.

Addisionele Lede:
Drs. W. J. Ryksen, P. de la Harpe en T. A. Theron.

Die namiddag is die volgende wetenskaplike voordragte gelewer:

(i) Virusse as oorsaak van asemhalingstoornisse by perde.
Dr. B. J. Erasmus.

(ii) Gevalsverslae—
(*a*) Neguvon vergiftiging.
(*b*) Dieldrin vergiftiging.
Dr. W. J. Ryksen.

(iii) Iets omtrent die uitroeiing van meerkatte.
Dr. H. N. v.d. Made.

(iv) Aansteeklike oogontsteking by beeste en skape.
Dr. D. J. Louw.

(v) Gevalsverslag: Verlamming in die aap.
Dr. A. J. D. Williamson.

Die dag se verrigtinge is afgesluit met 'n skemerkelk by die Bloemfontein Klub.

MINUTES OF THE ANNUAL GENERAL MEETING OF THE WITWATERSRAND BRANCH HELD AT THE WANDERER'S CLUB, JOHANNESBURG, ON 5th MAY, 1964.

Present:

Members:Drs. Hempstead (Chairman). Mason. H. Steyn. Shone. Purchase. de Jager. Leibenguth. Kempster. Anema. Greathead. Poole. Ehret. Theron. Kunnen. Cotton. Frost. Breytenbach. Robson. P. Keep. (Sec.).

Apologies: Drs: Craig, Erasmus, Langlands, Boswell, M. Keep, Warnes, Liebmann, Davies, Von Bachström, Kruger, Vogelnest, Snyders, McHardy, Lubbe.

Resignations: Drs. P. Bisschop, G. Thompson.

Visitors: Drs. J. Hofmeyr, van Zyl, Veary & Beverley.

New Members:

Dr. van Zyl proposed by Dr. Breytenbach, seconded by Dr. Frost.
Dr. Vearey proposed by Dr. Leibenguth, seconded by Dr. Cotton.
Dr. Beverley proposed by Dr. Frost, seconded by Dr. Kunnen.
Dr. Hofmeyr proposed by Dr. Theron, seconded by Dr. Poole.

The latter two only provisionally as they are not yet members of the parent body.

Minutes of the 92nd Meeting. These were approved and confirmed.

Correspondence. 1) Licensing of spayed bitches. Agreed that we suggest the parent body try to get uniformity throughout the country.

Chairman's Annual Report. The Chairman mentioned the branch's activities during the past year. He thanked the sub-committee of Drs. Kempster, Azzie, Dickson & Louw for the way in which they had handled the Anti-Cruelty League matters. The present membership was 67. The annual Clinical Medal of the branch had been awarded to Dr. S. Lombard. He thanked Dr. H. Steyn and his wife for acting as hosts at our very successful and enjoyable Annual Social Function. He also thanked the Secretary and Treasurer for her help in the past year.

Treasurer's Report. The Treasurer apologised for not producing a Balance Sheet in time, but the Bank had made an error in our statement and it had taken some time to straighten-out the position. She first asked the approval of the meeting to take R4.25½ out of funds to balance the finances of the Annual Social Function and this was readily granted. She then reported that our balance in the bank at the beginning of the year had been R177.71 and now stood at (approx.) R294.58; R187.85 having been from subscriptions. We also had in hand several Clinical Medals for future years.

Election of new office bearers. It was proposed by Dr. Steyn that the Chairman, Dr. Hempstead, the Vice-chairman, Dr. Warnes; the Secretary and Treasurer, Dr. Pearl Keep, be re-elected en bloc and this was unanimously agreed to by the meeting.

Talk by Dr. H. Purchase. "A method of feeding Monosodium phosphate through water without a dispensing machine", this was given by Dr. Purchase with the aid of printed notes and graphs to illustrate a simple way of feeding soluble phosphorus to stock through piped drinking water from a reservoir without a metering device. Drs. Steyn, Poole & Shone all questioned Dr. Purchase with regard to evaporation, the heavy irons, the human element, the supply of water from the catchment reservoir

and the water levels. It was pointed out that brackish water forms calcuim phosphate as a deposit but only in small quantities and this could be used as a lick when the reservoir was cleaned out. It was essential to use a mixing reservoir on a hill and have the water piped to troughs. It was much more expensive than feeding degelatinised bone meal, but kept the Ca/P ratio correct and ensured a regular supply.

General. 1) Next meeting to be on Tuesday 4th August at the Wanderer's Club, speaker to be decided upon later.

2. Dr. Steyn mentioned that the East Rand wished to start their own branch and raise their fees. The Cape Western Branch also wished to start a small Animal Practioner's Branch, this was on the agenda for the A.G.M. of the parent body.

3. Dr. Breytenbach mentioned that inseminators were treating animals at the same time, the Co-ops supplying Penicillin, Streptomycin, Agavin, etc., he agreed to obtain further information on this matter.

The chairman mentioned that deep-frozen Ayrshire semen was being flown up from Natal and that dairymen could inseminate their own animals.

Dr. Frost queried the issuing of certificates and Dr. Steyn replied that no certificates could be given by an unqualified inseminator nor could be registered pedigree animals. If the client was not a member of the co-op then there was no need for a certificate.

There being no further business, the Chairman adjourned the meeting at 9.55 p.m.

NORTH EASTERN TRANSVAAL BRANCH ANNUAL MEETING HELD AT TZANEEN

6th JUNE, 1964

PRESENT:

Dr. M. J. N. Meeser (Chairman), Drs. S. Solomon, P. Davies, H. P. Steyn, M. C. Lambrechts, A. M. Diesel, J. W. van Niekerk, J. Krige, J. Coetzee, A. D. Thomas, J. H. Schoeman and Mr. Deeks of Messrs. Hall and Sons.

APOLOGIES FOR ABSENCE:

Dr. M. de Lange, Osborne family, Dr. J. Schuss and his Swaziland contingent.

Dr. Meeser welcomed the guests and members and expressed the hope that the meeting would be pleasant and instructive. He regretted that Drs. M. de Lange, K. E. Weiss of Onderstepoort were not able to be present. He was pleased that the President of the Association, Dr. H. P. Steyn was able to attend. He welcomed Dr. and Mrs. Lambrechts and Dr. Diesel from Pretoria, and Mr. Deeks from Hall and Sons.

He then called on Dr. H. P. Steyn to open the Conference.

Dr. H. P. Steyn in thanking the chairman for his remarks, drew attention to the steady progress made by the South African Veterinary Medical Association but warned against complacency.

The Minister of Agricultural Technical Services, Mr. P. M. K. le Roux, had, about three years ago, approved the principle of part-time State employment of veterinary private practitioners.

A Sub-Committee of Council had recently considered, and was dealing with, the question of encouraging veterinarians to rural practice. Dr. Lambrechts was a member of this Subcommittee and would soon submit his recommendations in this respect.

As regards veterinary education, the President gave a brief review of the situation as he saw it; he felt that the development of another Veterinary Faculty would be beneficial and would create a wider horizon from which student's could be drawn. As regards the availability of staff for a second faculty, this would have to be drawn from existing and developing teaching personnel.

The next Annual Congress was to be held at Cape Town. It would occupy 5 days: the programmes would soon be sent out. Some viewed this Congress with a certain amount of misgiving. He was glad that Dr. Harthoorn had agreed to present a paper: he was optimistic that the Congress would be a success. A day had been set aside for an educational tour of the Western Cape. Mr. Smit M.P. of Stellenbosch would arrange and conduct this tour in collaboration with Dr. G. L. Muller.

He hoped as many Veterinarians would attend as possible, as the State had only authorised four from the Research and four from the Field Services, to attend officially.

It was beneficial for the Association to hold occasional Congresses at different centres in the Republic; this gave publicity to the profession and enlightenment to the public.

With these words the President wished the meeting every success with its deliberations and declared the Third Annual Meeting open.

The Chairman welcomed Dr. Solomon and Dr. Harthoorn who had just arrived. He called on Dr. Lambrechts to address the conference on "*Some Aspects of State Veterinary Field Policy*".

Dr Lambrechts briefly reviewed the services rendered by the Veterinary Field Services in the past and sketched its future activities, with particular reference to the role which the diagnostic centres would play to encourage the detection and control of disease and assist in the establishment of veterinary private practice in rural areas.

Dr. Davies felt that the establishment of rural practitions should be encouraged as a stimulus to the development of the Regional Diagnostic Laboratories.

Dr Lambrechts referred to the position in some other countries.

Dr Steyn referred to the provisions of the Veterinary Amendment Act 1963 regarding the entry and registration of veterinarians from other countries.

Paper by Dr. K. E. Weiss

The paper by Dr. Weiss on "New developments in Virus Research", was read by Dr. Lambrechts.

The Chairman paid compliments to the lucid manner in which Dr. Weiss had presented his paper and his logical approach to the transmission of snotsiekte from wildebees to domestic stock.

Dr. Weiss could unfortunately not agree to the publication of the paper as he had referred to unpublished work.

Many members contributed to the discussion of this most interesting paper.

Dr. Meeser thanked Dr. Lambrechts for presenting his own and Dr. Weiss' contribution.

The Chairman then requested a change in the order of the programme and called on Dr. Harthoorn to present his paper on "Physiological points of interest in the Tranquilization of Game". Dr. Harthoorn is a physiologist from the Royal College Nairobi. Dr. Harthoorn presented a most interesting paper on this subject, for which he received a great ovation. He discussed the value of M 99 as a tranquilizer in game and domestic animals, from the physiological aspect, and the manner in which it was administerde.

The Chairman thanked Dr. Harthoorn for his outstanding contribution.

The Third A.G.M.

The Chairman asked for confirmation of the minutes of the previous meeting which had been published in the Journal of the S.A.V.M.A. He was authorised to sign them as correct.

He then called on the Hon. Secretary/Treasurer to present the financial statement.

The Secretary informed the meeting that the Branch had the amount of R34.15 to its credit at the Bank.

The Chairman then called for nominations for Chairman, Hon. Secretary/Treasurer and additional member of Committee. The following were unanimously elected:—

CHAIRMAN — Dr. M. J. N. Meeser.

HON. SECRETARY/TREASURER — Dr. Sue Solomon.

ADDITIONAL MEMBER ON COMMITTEE — Dr. P. V. A. Davies.

Dr. Solomon explained that she may be leaving and was elected on this understanding.

VENUE OF THE NEXT MEETING.

The meeting agreed that the next meeting should be held at Skukuza. If the Executive Committee was not able to arrange this to their satisfaction it had the authority to arrange another venue. The National Parks Board set aside the month of May for Conferences in the K.N.P.

The Chairman advised the meeting that Drs. Steyn, Lambrechts and Diesel had been proposed as honorary members of the N.E. Transvaal Branch.

The Chairman then welcomed these honarary members to the Branch and hoped they would attend many meetings in future.

CONCLUDING PAPER ON THE AGENDA

Dr. P. Davies then presented his contribution viz. "Primary Pulmonary Emphysema in Bovines", being the history of individual clinical cases in which this diagnosis had been made.

An interesting discussion followed.

The Chairman thanked Dr. Davies for his very interesting contribution.

Mr. Deeks thanked the Chairman for inviting him to attend the meeting.

The Chairman thanked all who had contributed to the success of the meeting, particularly those who had been responsible for the refreshments and the luncheon.

A vote of thanks to the Chair was adopted unanimously.

FILMS

The meeting concluded with the projection of the following films.

1. *Cattle-tick Control*—prepared by Messrs Cooper & Nephews.
2. *Wetenskap Oorwin*—Deur die Departement van Landbou-Tegniese Dienste voorberei.
3. *African Horsesickness*—prepared by the Section of infectious Diseases, U.S. Department of Agriculture.

INTIMATION FROM THE ASSISTANT CHIEF VETERINARIAN PIETERMARITZBURG

RABIES

I wish to inform you that many Veterinarians are under the impression that the marking of dogs inoculated against Rabies need not be enforced.

This is entirely wrong as under Section 1 (ii) of GN 407 of 18.8.61 as amended, such marking is ordered by the Minister.

As only the Minister or a higher authority can countermand an order of the former and as such a countermand order has not been given, all dogs inoculated against Rabies in Natal must also be marked in the ear with the last digit of the year of inoculation by means of a tattoo punch.

In the case of small dogs whose ears are too small for marking, the person inoculating may refrain from doing so.

L. C. BLOMEFIELD
Asst. Chief. Vet. (Natal).

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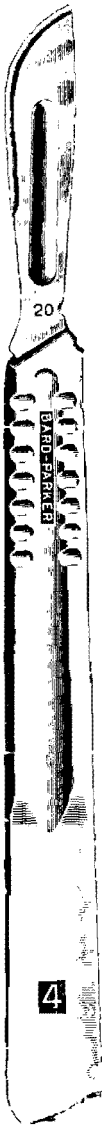

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LETTERS TO THE EDITOR

The Editor,

J.S. Afr. vet. med. Association.

Dear Editor.

PENICILLIN ALLERGY OR ANAPHYLAXIS

In the March, 1964 Edition of the Journal of the Association, Dr. T. C. Wessels introduced a very interesting condition e.g. allergic reactions following the administration of certain drugs.

I also had the unfortunate privilege of experiencing a few cases of allergy from drug reactions.

Allergic sensitivity is nearly always the result of previous administration of the drug. It was interesting to note that in Dr. Wessel's case no antibiotic was administered previously. Time elapsed since last administration of the drug and route of administration seem also to play a role.

A large proportion of reactions to penicillin follow the use of procaine preparations but these also form a large proportion of the total penicillin used. It would nevertheless seem that the sensitivity reactions are produced in the sensitized patient regardless of the form of penicillin given.

Procaine penicillin will produce side effects if an injection is accidentally made into a vein. Some Penicillin preparations are not solutions but suspensions and can cause a pulmonary embolism. One, however, feels that to obviate such reactions, elementary precatuions should be taken against intravenous injection of procaine penicillin by simple traction on the plunger or detaching the syringe to see that no blood flows from the needle.

Symptoms noticed were:

1. Shock, facial oedema, sometimes laryngeal oedema and urticaria.

2. Vertigo—visual and auditory disturbances and restlessness. In one case there was loss of consciousness.

In conclusion it would seem that one has to know the allergic potentialities of the drug to be given and the patient's history with respect to allergies. It is always advisable to keep the necessary emergency medicaments handy e.g. adrenaline, dextrole 40% solution, and a corticosteroid.

One would like to know whether Dr. Wessel's case ever was treated for mastitis and what proprietary preparation was he using?

The Chief: Veterinary Field Services is thanked for permission to publish this letter.

W. P. VAN AARDT,
Senior State Veterinarian
Grahamstown.

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Hierdie diere was vernietig gedurende 'n uitdunningsprogram en geen verdere toetse kon uitgevoer word nie. Dit blyk dus, dat wild van die Kruger-wildtuin met *Brucella*-besmetting in aanraking kom.

Die Navorsingsinstituut wil graag saamwerk om verdere toetse op alle species van wild, uit te voer.

Monsters van volwasse diere van die vroulike geslag word verkies, *alleen van streke vry van Bek- en Klouseer en geen varkmonsters van varkkoorsstreke.*

bv.:— (*a*) Serum vir Serologiese toetse.

(*b*) Waar moontlik, ook limfkliere, milt en gedeeltes van die geslagsorgane vir die afsondering van die organisme.

L.W.—*GEEN MONSTERS VAN BEK- EN KLOUSEERSTREKE EN GEEN VARKMONSTERS VAN VARKKOORSSTREKE KAN HIERVOLGENS NA ONDERSTEPOORT GESTUUR WORD NIE.*

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