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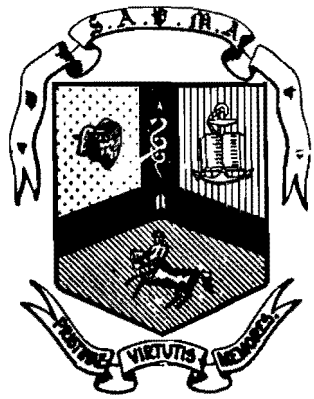

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EDITORIAL

THE 1964 CONGRESS AT CAPE TOWN

Since taking the plunge in 1961, when for the first time in its history, the Association planned and held a Scientific Congress away from Onderstepoort, at Durban, Council has given serious attention to the wisdom of repeating the successful Durban experiment at Cape Town. It had barely instructed the Congress Committee to proceed with the planning of the Cape Town meeting when grave misgivings arose regarding the wisdom of the decision, because of a change in policy by the Department of Agricultural Technical Services.

Only four veterinarians from each of the Divisions of Veterinary Research and Veterinary Field Services were to be permitted to attend Congress and then only if they were contributors. Cape Town was a thousand miles from Onderstepoort and from many places where veterinarians were employed and practised. Would members attend in sufficient numbers at their own expense? Could the Association afford to speculate on what seemed a foregone uncertainty; indeed even perhaps a complete flop?

Fortunately wisdom was not influenced by unrealistic reasoning; cold calculation gave way to warm appreciation of the loyal support and esprit de corps which could be expected from the members of the Association. The Association is thankful for the decision of its Council and proud of the steadfastness of its members.

The Cape Town meeting was a resounding success and if the Association and its members were set back a little financially, there is no doubt that the gain in status and publicity given to the profession will be amply rewarded by this small expenditure.

The papers were of a high standard and the Congress arrangements worked very smoothly. While the President occasionally found himself fettered by the time factor, this is something which inevitably tests the ability of every chairman of a scientific meeting; and in our case we acclaim our President as he stood the test very well indeed.

The social events, organized both for the ladies separately and for Congress-in-community, were most enjoyable and very well taken care of. The bus tour to Darling and its evirons was an unqualified success and Council is advised to give serious consideration to the institution of a regular excursion for one day during Congress, no matter where it is held.

The opening address by Mr. de Villiers Loubser and the Congress Lecture by Professor Kipps will long be remembered by those who had the privilege of listening to these two accomplished speakers. Both are published in this issue and are commended for reading and rereading, as each is a *Congress Classic* in its own field.

The cordial welcome given to us by His Worship the Mayor, at Congress and at his very pleasant Cocktail Party, alone made the visit to Cape Town worthwhile.

The Exhibitors deserve a special word of praise. During the year they agreed to the disbandment of their Medical Exhibitors Association, but in spite of this, turned out in full force for the Cape Town Veterinary Congress. The Association greatly appreciates their loyalty and thanks them sincerely for their continued support.

The President at the conclusion of Congress spent some time recording the thanks and appreciation of the Association to all who assisted in making it the success it was. We would like to endorse this, particularly in respect of the lovely homely farewell party given us by Dr. and Mrs. Faull.

Lastly, but indeed by no means the least, we salute the Chairman, the Secretary, and the executive Committee of the Cape Western Branch and their wives and willing helpers, for all they did for us who had come a thousand miles to learn about their wonderful hospitality and organizing ability.

A.M.D.

ANNUAL SCIENTIFIC CONGRESS AND FIFTY-NINTH ANNUAL GENERAL MEETING HELD AT CAPE TOWN, 28 SEPTEMBER — 2 OCTOBER, 1964

The Annual Scientific Congress and Fifty-Ninth Annual General Meeting of the South African Veterinary Medical Association was held at Arthur's Seat Hotel from 28 September to 2 October, 1964.

THE OPENING CEREMONY

The President Announces the Commencement of Congress

Mr. de Villiers Loubser, Ladies and Gentlemen,

We have arrived I might say. This is only the second meeting to be held away from Onderstepoort in fifty-nine years. Mr. de Villiers Loubser is going to tell you about some very interesting facts and figures. Now before we proceed I will ask His Worship the Mayor to welcome delegates and visitors to Cape Town.

His Worship the Mayor, Councillor W. J. Peters, J.P., welcomes Guests and Delegates.

Dr. Steyn, Mr. de Villiers Loubser, Ladies and Gentlemen,

My duty this morning is a pleasant one and it makes it a little more pleasant when I have somebody on my right who is my size; because I have got to be very careful what I say or he might take me out into the gymnasium. It is my privilege to welcome the delegates and members and guests to this Congress of the South African Veterinary Medical Association. I do so very warmly and trust not only that the deliberations will be rewarding but that the few days spent here will be memorable and enjoyable.

Now Mr. President, I have welcomed guests to Conferences over the past few months and I have never seen so many ladies present at a Conference; whether the ladies are the wives of the delegates or whether they are all veterinary surgeons, I don't know, but if they are the wives I would like to suggest to the husbands that they give them a blank cheque and let them go out and enjoy themselves. Our shops are full of things, and Christmas is not far distant; perhaps they will be able to buy Christmas presents. That is an advert. My welcome to our visitors from overseas is particularly cordial and we hope that this experience will induce them to visit us again when work will not be their paramount interest. We in Cape Town feel honoured that you have come here for your Congress. As you have mentioned, this is only the second time you have left the precincts of Onderstepoort; may I congratulate the convenors of this Congress on their choice of venue, and say that I do not know of any place in the whole country which can compare with Cape Town at this time of the year — *hear hear*!

I am sure that even our friends from the Administrative capital of Pretoria applaud your wisdom.

Dames en here, namens die Raad en Burgers van Kaapstad, is dit vir my 'n voorreg, en besonder aangenaam, om die afgevaardigdes, lede en gaste van die Suid-Afrikaanse Veterinêr-Mediese Vereniging hartelik welkom te heet, en die hoop uit te spreek dat hul verblyf in ons stad genotvol en plesierig sal wees. Ek is ook bly dat ons die geleentheid sal hê om hulle vanaand by die Burgelike skemerparty te ontmoet, en beter te leer ken.

Ladies and Gentlemen, on no account let my enthusiasm for my city run away with my good sense. We appreciate that Onderstepoort is South Africa, to countries connected with veterinary research, and that the standard set by its founder, Sir Arnold Theiler, is maintained to this day. Not so long ago the veterinarian in this country was the man who looked after

The President, Dr. H. P. Steyn, invites His Worship the Mayor, Councillor W. J. Peters, to welcome members and guests to the Congress.

your dog or your cat; today he plays an enormous part in the health of the whole community; humans in particular, dogs and cats as incidentals in our homes, but whose health is important, because of their close contact with us. It is to his skill that we look for the maintainance and development of the highest quality in our meat supplies, dairy products, and of all the products derived from animal sources. This covers an enormous field. I notice from the notes given to me that the veterinary surgeon in this country is a rare species. I have no doubt however, that with the scope open to the profession today, and in the years to come, our young men will be induced to take up this rewarding and important work.

In fact, Mr. President, so much do we owe to the profession, for maintaining a healthy livestock population, that our income from wool, dairy and poultry products, meat etc., is second only to that of the mines. With the marked increase in the numbers of our population and that of the rest of the world, it is becoming even

more important that this country possess a healthy livestock population. There is a great shortage of good protein of animal origin such as meat and milk, which can only be increased by more intensive methods with its attendant dangers of disease. The important task then of the veterinary surgeon is to see that production is increased, and at the same time to prevent the spread of disease.

Now apart from the fact that Cape Town leads the country in natural amenities as I have already told you, it led the country in being the first to enforce that very necessary measure of compulsory pasteurisation of milk, in order to ensure the safe milk supply; and again now in building a very modern abattoir at a cost of over 3 million Rand to ensure a safe and hygienic meat supply.

Dr. Steyn, you and your colleagues, have matters of great importance to us all on your agenda; again let me wish you well in your deliberations.

Dames en here, ek moet nou die veld ruim vir die ander sprekers. Nogmaals wens ek u 'n vrugbare vergadering toe en herhaal my verwelkoming aan u almal. Dankie.

Dr. Albertyn, Chairman of the Cape Western Branch, thanks the Mayor

Mr. President, Mr. Mayor, Mr. Loubser, Ladies and Gentlemen,

It gives me great pleasure to thank our Mayor, Councillor W. J. Peters, J.P., for his presence here today, and for giving us such a warm welcome to the city of Cape Town.

As Capetonians, our popular Mayor requires no introduction. To you who come from further afield may I say that Councillor Peters, or as he is affectionately known to members of the municipal staff, Big Bill Peters, I may say that he has been a prominent business man in Cape Town for many years.

The Mayor of this big thriving city of ours is a busy man and many organizations make demands on his time. However, when we approached him in connection with this Congress, his very willingness to come here imbued us with the sense of confidence regarding the success of this Congress.

We have listened with interest to your remarks, Sir, and may I say that the clarity of your insight into some of the veterinary tasks has been very gratifying to us.

I also wish to thank you for the long and continuous support which the City Council of Cape Town has given to veterinary surgeons in the public health field; I am referring to the fight against numerous diseases transmitted to humans through foodstuffs of animal origin and to the transmission of animal diseases to humans.

May I just refer to two very interesting facts, Mr. President, that Cape Town is the first city in South Africa to introduce the compulsory pasteurisation of milk under veterinary supervision. This happened more than a decade ago. May I also just mention that Cape Town is at present building a great new abattoir, not very far from here, which when it is completed will be the most modern and finest in Africa.

Finally, Mr. Mayor, may I also thank you for inviting us to the very pleasant social occasion, which we are looking forward to, this afternoon in the city hall. Thank you.

Die President stel mnr. J. E. de Villiers Loubser aan die Kongres voor

Vriende ek wil aan u voorstel mnr. de Villiers Loubser, die Voorsitter, eintlik die President, van die Winterreënstreek Boere-Unie.

U mag nie almal weet nie, maar mnr. Loubser staan nou op uit 'n siek bed; hy het 'n ernstige hart kondisie opgedoen 'n paar weke terug, en hy is met die permissie van sy dokter 'n paar uur per dag op sy voete. Omdat hy so belanghebbend is by veeartsenykundige aangeleenthede, het hy hom die moeite getroos om tog hier teenwoordig te wees, om hierdie vergadering vandag te open.

Ek sal mnr. Loubser vra om sy toepsraak te lewer.

Die Kongres word deur mnr. de Villiers Loubser geopen

Mr. J. E. de Villiers Loubser addresses Congress while His Worship the Mayor of Cape Town, Councillor W. J. Peters and the President, Dr. H. P. Steyn, listen attentively.

DIE 1964 JAARKONGRES TE KAAPSTAD WORD DEUR DIE VOORSITTER VAN DIE WINTERREËNSTREEK, MNR. J. E. DE VILLIERS LOUBSER, GEOPEN

MNR. DE VILLIERS LOUBSER aan Kongres deur die PRESIDENT voorgestel

Mr. President, Mr. Mayor, honoured guests, Ladies and learned Gentlemen,

On behalf of organised farming of the winter rainfall area, and I also have authority to speak on behalf of the farmers of the Province, I wish to bid you a most hearty welcome.

We feel honoured to have this Congress of Veterinarians in the deep South. We have done whatever we could and I know to what extent Dr. Albertyn went to have all the preliminaries in order, for a very comfortable and hospitable stay for you in Cape Town. We also feel sure that you will have experienced the hospitality of our mother city, and that you will enjoy this wonderful city of ours, on the shores of the most beautiful Cape in the world.

Mr. President, according to our programme, which we have already overlapped to some extent, I am afraid that my message to you will be a little bit longer than scheduled, and for that reason I would ask you to bear with me when I address you in my mother tongue, because then I will be able to be more fluent and perhaps more to the point.

Meneer die President, dit is vir my 'n baie besondere voorreg om as boer, en as boer van Kaapland, genooi te word om hierdie Kongres van geleerde mense, te kom open. Toe die uitnodiging in die eerste instansie na my gekom het, het dit vir my vreemd voorgekom, so vreemd dat dit vir my eintlik snaaks was; maar dokter Albertyn en die President het my oorreed dat daar tog 'n plek is by Veeartsenykunde, en by

The President, Dr. H. P. Steyn, Mr. J. E. de Villiers Loubser, Dr. A. M. Hartshoorn and Dr. P. M. S. Masters.

At the Administrator's Residence.

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Mrs. Malan with Mrs. Greathead, Mrs. Steyn and Mrs. Albertyn.

hierdie Kongres, waar 'n boer sy plek kan inneem langs die veearts. Dit is dan ook die tema van my rede aan u vanmôre — *Die boer en die veearts* as spanmaats. Waar het hulle vandaan gekom, waar gaan hulle geen, hoe lyk hulle pad? En ek wil dan dadelik begin deur vir u te sê dat ek die uitgangspunt as onaanvegbaar beskou. Die boer en die veearts in enige plek ter wêreld, maar te meer in hierdie land van ons, behoort intiem saam te werk, mekaar te verstaan, en in die nouste verhoudinge met mekaar te verkeer. Om daardie ideale verhoudinge te bekom is dit noodsaaklik dat daar ontwikkeling sal plaasvind aan albei kante.

Daar moet ontwikkeling plaasvind aan die kant van die boer tot daardie samewerking.

In die eerste plek sou ek sê dat hy breed en verstandelik moet ontwikkel; maar dit is 'n breë onderwerp en hoort meer tuis by ons landboukongresse. Maar wat hier wel tuishoort, glo ek, is dat die boer elementêr gespesialiseerd moet ontwikkel; die boer moet ontwikkel sodat hy eerstehulp kan toepas op sy vee op die plaas. Die boer moet ontwikkel, meneer die Voorsitter, sodat hy deur sy ontwikkeling sy behoefte kan leer ken. 'n Grondige lewensreël is dit dat die man wat nie ontwikkel is nie ,nie sy behoefte ken nie. Ek wil hier konstateer, dat baie van ons mense op die platteland, nog nie die geleentheid gehad het om sodanig te ontwikkel dat hulle die behoefte aan en omvang van veeartsenydienste, en van die veearts as sy vriend, besef of begryp nie. Sover die boer.

Ons veeartse moet ontwikkel

U moet my hier voor u sien staan as 'n leek op die gebied van u wetenskap. As boer praat ek miskien met u 'n ander taal as dié waarin u met u kollegas die saak bespreek, en daaruit mag moontlik voortvloei dat ons radikale gedagtes kan bekom wat 'n mens kan prikkel op jou pad vorentoe. Ek sê, ons veeartse moet ontwikkel, ontwikkel in die rigting van die mediese, dog daarheen, glo ek, is hulle reeds sterk onderweg; maar waarin ek nie so sterk kan glo nie, is of hulle 'n sterk agtergrond het in die ontwikkeling van die *kennis van die voeding van diere*. Ek glo, meneer die President, dat hier nog heelwat ruimte is waarin ons jong veeartse voorberei, sterk gemaak en bewapen kan word, want op die pad vorentoe is die voeding van diere een van ons allerbelangrikste fasette in ons landbou.

Ons veeartse moet ontwikkel in die rigting van die besef van die waarde van *navorsing* en, ek glo, tot groot hoogte is dit die geval. Maar dan ook moet ons veeartse *ontwikkel in die rigting van voorligting*. en hier twyfel ek of ons manne behoorlik bewapen is, om voorligting te kan gee aan die mense wat met die land se vee werk en om hulle kennis te kan oordra aan die mense wat daardie verantwoordelikheid het.

Op daardie pad van die ontwikkeling van die veearts sien ek 'n paar gevare. Die eerste wat ek hier wil noem, is die gevaar van die moontlike ontstaan van naywer by die veearts, naywer op sy kennis wat hy dra, op sy kennis wat hy versamel, 'n jaloersheid op daardie kennis. Dis moontlik miskien 'n enge benadering in die rigting van persoonlike voordeel, maar, meneer die President, daarin lê vir my die groot gevaar, want sodra daardie gees by 'n veearts aanwesig is, dink hy aan die ekonomiese benadering van sy roeping en sodra hy daardie benadering vooropstel dan verswak hy die groter saak, daardie besondere verantwoordelike taak wat vir hom wag in sy roeping as veearts op die land.

Meneer die President, die besit van daardie kennis moet oorgedra word tot die ontwikkeling van die boer op die plaas, en ek wil u hier net een geval noem wat my getref het en wat ek as boer as verkeerd beskou. U weet dat tot onlangs was daar die geleentheid vir boere, jongboere, om kort kursusse by Onderstepoort te loop, kort kursusse in melkbeeste, vir inseminasie en so meer, waar die jong boer kon leer om die elementêre diens aan sy diere te kon lewer. Die manne wat dit geloop het kan u naspoor en u sal vind dat hulle 'n besondere belangstelling in die welstand van hulle diere ontwikkel het en dat hulle die waarde van hulle diere in so 'n mate agtergekom het dat die mense nou begryp hoe noodsaaklik dit is om in 'n baie noue betrekking met hulle veeartse te leef. Na wat ek verneem, is daardie fasiliteite afgesluit as gevolg van besware wat van veeartse gekom het, asof dit dan die brood van die veearts in gevaar sou stel. Dit is 'n korttermyn benadering; dit is 'n benadering wat veel skade kan doen; want sodra u die kennis, die elementêre kennis, van die boer weerhou, dan bly sy wêreld klein, u hou sy kennis klein en ook sy sin van behoefte, sy insig van behoefte hou u elementêr, en in daardie toestand het hy die veearts nie nodig nie.

Ons veeartse moet waak dat hulle baie noukeurig *dienstariewe* binne redelike perke handhaaf, want as daar gefaal word op daardie pad

The Ladies at the Administrator's Residence. Mrs. Malan tells an amusing story to Mrs. Pols and Mrs. Louw.

lê daar ook gevaar van verwydering tussen veearts en boer en 'n verkleining van die veld.

Daar moet gewaak word teen *tegniese en wetenskaplike oordondering* van die boer; miskien is dit nie 'n algemene kwaal nie — maar, meneer die President, dit is waarneembaar ook in ander beroepe van die wetenskap, dat mense met hulle wetenskaplike kennis, 'n boer só oordonder met hul terme en terminologie dat die moontlikheid van die verstaanbaarheid van die onderwerp onder bespreking, of onder behandeling, vir die boer heeltemal verlore gaan; en die veearts verlaat die plaas van 'n boer wie se belangstelling hy verloor het. Ons jong veeartse moet teen daardie dwaling gewaarsku word.

Ek dink aan die *diensveld* van ons veeartse, en graag noem ek, volgens my mening, 'n lys van waardebepalings. Die eerste en belangrikste diensveld in hierdie land van ons is die manne wat staan tussen die vee, by die boer op die plaas — ons veeartse op die platteland. En glo u my as ek u sê dat ons vandag so min as net ± 85 veeartse op die platteland het! — mense wat hulle lewe in private praktyke by die boer tussen sy vee maak.

Die tweede in belangrikheid vir my kom in die staat se velddienste, met sy diagnostiese sentrums en so meer, voor. Dis 'n baie besondere en belangrike diensveld. Ongelukkig, meneer die President, bly dit Staatsdiens en die ontwikkeling in daardie rigting is baie traag. In hierdie veld staan 81 veeartse.

Die veld probleme wat van die plaas en die boer af moet gaan na die navorsers is so uiters belangrik; dit kom van die veearts op die plaas en van die navorsingsentrum, en die manne wat daardie navorsing moet waarneem vir ons hele ganse land, tel 46 navorsers.

Die gesondheidswaarborg wat ons volk, ons verbruikerspubliek het, het van dié manne, wat in die bresse moet staan en die gesondheid van die diereprodukte moet beheer, en toesien dat gesonde voedsel die publiek bereik — dwarsoor ons land net 17 veeartse — glo my 17! En as u geluister het na ons Burgemeester dan sal u met my saamstem dat as die waarde van daardie diensveld behoorlik geëvalueer word, dan sal 'n plek soos Kaapstad met sy voorstede al 17 daardie manne met groot vrug in diens kan neem. Waar is die res van ons land en waar is die

The Instructional Tour of the Western Province.

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Mr. Hennie Smit, M.P., showing delegates and members over Mr. Duckett's farm and gives details of the Ayrshire herd to the President and Prof. van der Walt

gesondheidswaarborg van ons publiek? Ons dink aan siektes wat na ons stede toe ingedra kan word — miltsiekte, mastitis, tering, masels, ander besmetlike siektes en dit is hierdie manne wat moet keer en toesien dat dit nie binne die stad ingedra word nie.

By maatskappye het ons veeartse in diens soos by die K.I. en by mense wat voer vermeng, as adviseurs. Daar het ons 'n totaal van 20 veeartse; maar van die 20 is alleen 6 manne wat hulle geheel-en-al wy aan die voeding van diere; 6 manne oor ons Vaderland; veeartse wat eintlik die geroepe persone is om voorligting insake voeding van diere aan die boere en ander instansies te doen. Hier in dié veld staan 6 veeartse!!

Dan kom ek by 'n diensveld wat ek nie 'n plek voor aanwys nie. Dit moet daar wees; dit is basies, dit is die dosente aan ons fakulteit; daar het ons 18 veeartse.

En dan kom ons by die grootste groep in die veld, die stedelike veld en dit is ons privaat praktiserende veeartse binne in die stede. U sal my verskoon as ek sê, dat in die groter projek gesien, daardie diensveld miskien nie die belangrikste is nie. Maar tegelykertyd wil ek ook sê ons waardeer dat baie van die manne ook vee besien en bedien wat rondom die groot stede lê. In daardie veld is 'n 100 veeartse. Tel dit almal bymekaar, ook hierdie 100 veeartse wat privaat diens in die stede lewer, dan kom ek by 'n totaal van 373 in die Republiek van Suid-Afrika. Dit is om toe te sien dat al die troeteldiere van die bevolking versorg word, dat al die mense se gesondheid gewaarborg word sover as dit die toevloei van voedsel gaan, en om toe te sien dat plus minus 40 miljoen skape en bokke, 12 miljoen beeste, 2 miljoen varke en al die ander vee waarmee geboer word, nie alleen gesond gehou moet word nie, maar beter versorg moet word. Hulle moet toesien dat die vooruitsigte vir daardie veestapel van die land uitgebrei moet word. Is dit moontlik, meneer die President, ek vra u, is dit fisies moontlik met die klein groepie manne tot ons beskikking?

The Instructional Tour of the Western Province.

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Members and delegates are shown over Mr. de Villiers Loubser's farm.

Wat is ons behoefte?

Ons het groot behoefte aan beter mediese versorging van ons diere; dit is 'n statiese stelling. Ons verloor miljoene aan geldwaarde deur

onvrugbaarheid onder ons veekudde; ons verloor etlike miljoene aan die verlies van die foetus. Foetusverlies is onbepaalbaar; maar ek wonder tog as daar mense benoem word om op die saak in te gaan, of hulle nie vir ons 'n denkbeeld kan gee van wat die verliese is, wat op die groter veld verlore gaan deur erosiesiektes, nie. Ons behoefte aan beter mediese versorging, die geweldige sterfte onder ons jong diere, kalwers, lammers, pasgebore diere; erosiesiekties, totdat die dier gespeen is, is 'n ontsettende verlies. Navorsing kan dit nie bybring nie, ons veeartse in die veld is te min en die boer is nie behoorlik toegelig om die noue verband tussen die veearts se kennis en sy behoefte as voog oor sy vee, te kan uitoefen nie. Dan praat ek nie eers van die parasiete, in- en uitwendig, nie!

Ons het beter voeding vir ons diere nodig. Meneer die President, ons land is bekend vir sy droogte, maar ons het twee soorte van droogtes. Ons het die droogte met die groot „D", ons het die droogte met die klein „d" en waar ek die grootste respek, agting en meegevoel vir dié mense van ons, wat ly onder die smarte van geweldige droogtes, soos ons vandag nog in Noord-Transvaal ondervind, dra, kom die vrees by my op en dit word by my 'n wesenlike, dat ons Suid-Afrikaanse boer gevaar staan om aan 'n droogte-kompleks gewoond te raak. 'n Kleindroogte is ook 'n droogte, en dit het 'n verlammende effek, maar as die boer geroep word om sy vragmotor na die dorp te stuur om voer te gaan haal om die vee kos te gee, dan is dit 'n droogte-kondisie en dan moet daar droogte-maatreëls getref word. Meneer die President, dit is verkeerd, dit is kortsigtig en dit is die groot, beheerende en beperkende faktor wat ons vandag ondervind waarom ons veestapel nie kan groei nie, want baie van ons boere glo nog dat al die groeikrag van die hemel moet kom; ons ken nog nie die waarde van byvoeding in die voeding van ons diere nie. Hier wil ek u die Swartlandgebied kwoteer. U ken die graanstreek hier af, en Woensdag sal u miskien 'n deel daarvan sien. Die Swartlandgebied het vyftien jaar gelede plus minus 'n kwart miljoen skape gedra teen 'n gemiddelde van ongeveer 250 skape per plaas, want sy beperkende faktor was daardie sewe maande droogte van Desember tot Junie van *elke* jaar en die boer kon nie méér vee aanhou as wat hy oor daardie droë droogteperiode van die jaar kon dra nie. Hierdie gebied het 'n permanente, jaarlikse en terugkerende sewe maande droogte uit die twaalf; daarom was die veestapel van hierdie streek onbenullig en van baie weinig waarde op landsvlak gebied.

Meneer die President, wat is die posisie vandag? Met die koms van lupine in die Swartlandstreek, het die boer geleer dat hy sy dier in daardie tyd van die jaar, daardie droogte, kan kos gee, en sodoende kan hy meer diere aanhou, want in die vyf maande gras periode kan hy soveel méér vee op sy plaas dra. En vandag? Die ongeveer 'n kwart miljoen skape van die Swartlandstreek het oor 'n miljoen skape, van 'n baie beter gehalte, wat baie beter en byna dubbel soveel wol, en baie beter en dubbel soveel vleis lewer, geword. My vraag is dit — as dit waar is van die Swartland, wat is die rede waarom dit nie waar kan wees in al die ander dele van die land nie, waarom nie die Karoo nie, waarom nie die Vrystaat of die Oostelike Provinsie of elders in die land nie? Dis 'n begrip wat aanvaar moet word; ons diere moet byvoeding ontvang in die swaar tye van die jaar, of in die swaar jare van jou boerdery, want dit is lonend, en dit is toekomsbeleid.

Maar nou kom ek na die veeartse toe. Is u veeartse bewapen met die kennis van die voeding van diere sodat u in staat is om aan boere voorligting te kan gee en te kan sê: so moet op hierdie plek gevoer word, en so moet hier nie gevoer word nie? Het ons die benadering by ons fakulteit om ons veeartse met daardie kennis, as 'n onderleggende, belangrike, ja, byna allerbelangrikste faset van hulle opleiding, te bewapen?

Dr. van der Wath het op die Wolkwekers Kongres gesê dat hy die moontlikheid daarin sien dat ons 'n 60 miljoen skape in ons land kan dra, teenoor vandag se 38 miljoen. Meneer die President, 60 miljoen lyk nie vir my snaaks nie, want as u ons skaap getalle wil vermenigvuldig oor 'n periode van 10-15 jaar soos in hierdie graanstreek vermenigvuldig is, weet u, dan gaan u na 'n 100 miljoen, sonder dat die veld vertrap sal word. Ons het beter voorligting aan ons boere, om bostaande kennis oor te bring en van toepassing te maak, nodig. Ons het beter gesondheidskontrole oor ons diereprodukte in ons

stede nodig. Ons het uitbreiding op ons diagnostiese sentrums nodig. Ons voel in die hart dankbaar dat die staat hierdie sentrums geskep het, en ons sê ook baie hartlik dankie daarvoor. Dit is 'n progressiewe stap en 'n wonderlike onderneming; maar ek is bevrees dat dit te lank by dié stap en by die onderneming bly, want toe dit jare gelede as stelsel geskep is, is daar vir 36 sulke sodanige sentrums beplan, en weet u dat daar nou nog maar net twee volwaardig gefinaliseer is. Ses is beplan en in wording, maar nog nie klaar nie. Wanneer sal ons kom by die 36? Ek vra u, wanneer sal ons kom by die 36? Grootfontein vra vir 'n diagnostiese sentrum by daardie allerbelangrikste plek binne in die skaap se mekka. En die antwoord wat ons gehad het — nog vyf jaar.

Ons het 'n absolute minimum veeartse in hierdie land. Ons behoefte bestaan sterk in die uitbreiding van navorsing oor al ons veesoorte. Ons haal ons hoed hoog af vir wat die navorsers, daardie 46 manne. vir ons doen; hulle is bekend as van die beste in die wêreld, maar vlees en bloed het 'n perk en ek kan nie vir 'n oomblik glo dat 46 manne die probleme van 'n land soos die Republiek van Suid-Afrika behoorlik met al die bykomende werke wat aan ons navorsers opgedra word nie kan dek nie.

Ons vra vir die skepping van *'n tweede navorsingsentrum;* te min opgeleide manne is daarvoor beskikbaar. U sal u herinner dat by die opening van verlede jaar se kongres, het ons Eerste Minister gesê, en hy het hierdie woorde gebruik: „Daar bestaan 'n relatiewe wanverhouding tussen die getal veeartse en die veebevolking van die land." Dit het vir my soet geklink om dit te lees. Maar ek vra u, meneer die President, Kongres, is daar iemand in hierdie land wat die omvang van daardie wanverhouding ken? Om te sê daar is 'n wanverhouding, is pragtig, en komende van so 'n verantwoordelike persoon, is ons daarvoor dankbaar, maar is hier een persoon in hierdie land van ons wat die omvang van daardie wanverhouding ken. Ek wil die stelling maak dat hier geen mens is wat weet hoe noodlottig die wanverhouding geword het nie. Dit is 'n ware stelling, maar sy omvang is onbekend en swanger aan 'n groot gevaar vir ons vee.

Ek vra uitbreiding van opleidingsfasiliteite vir groter mannekrag, want daar lê ons basiese probleem, dit wil sê die skepping van *'n tweede fakulteit* in hierdie land van ons.

Members and Delegates enjoy the Buffet-Supper at the Arthur's Seat Hotel.

Waarom het ons 'n tweede fakulteit nodig?

Om meer kandidate te kan trek oor 'n wyer veld; om meer intensiewe persoonlike aandag aan kandidate te kan gee. Om meer intensiewe aandag aan kandidate te kan gee, meneer die President, is die teenoorgestelde van die worsmasjienmetode. En nou wil ek vir die pers vra om my nie verkeerd te rapporteer nie, want ek sê nie dat ons land se voorbereiding van veeartse op 'n worsmasjienmetode gedoen word nie, maar ek sê wel dat ek gelees het dat wêreldkenners stateer dat die grootste enkel klas by 'n fakulteit nie hoër as 'n 35 kandidate in 'n jaarklas moet wees nie. Ons getalle word nou al opgestoot tot 45, dis 'n gevaarteken! As dit nie massa produksie is nie, dan is ons op die rand van massaproduksie en nou kom my vraag van die gevaar na vore; indien die staat skielik agterkom wat die omvang van die wanverhouding is, en hy onmiddellik sy getalle van veeartse moet uitbrei, dan sal hy moet gebruik maak van die worsmasjienmetode, en dan sal ons tevrede moet wees met 'n minder goeie opgeleide veearts. Ons het nie plek vir minder goeie opgeleide veeartse in hierdie land van ons nie.

Ons moet gebruik maak van die geografiese, klimatilogiese en die kliniese verskil wat daar in ons land bestaan; ons maak tans nie ten volle daarvan gebruik nie. Ons het 'n tweede fakulteit nodig om *kompetisie tussen fakulteite* in hierdie land te bewerkstellig, ons het nie kompetisie nie, en, meneer die President, laat my toe om te sê dat 'n onderneming wat nie kompetisie het nie, 'n onderneming is wat nie 'n norm het wat vergelykbaar is by iets anders nie; dit is so maklik dat so 'n onderneming in sy eie oë kan groei tot iets wat vir homself baie belangrik skyn en na alles tog miskien heeltemal verwater geraak het op die pad.

Ons het 'n tweede fakulteit nodig om die *kwesbaarheid*, in vele opsigte kwesbaar, van 'n enkel fakulteit en navorsingsentrum uit te skakel. Bedink die moontlike kwade dag wanneer hierdie bestaande fakulteit en navorsingsentrum gekwes moet word. Wat gaan die gevolg wees op hierdie land van ons?

Ons het dit nodig om die gevaar van die ontstaan van 'n *koninkryk* uit te skakel. Ek trap nie op 'n man se tone wanneer ek dit sê nie, ek hoop altans nie so nie, maar, meneer die President, sodra ek baas is op my eie plaas, dan laat ek nie toe dat 'n ander man my kom vertel hoedat ek moet uitbrei of hoe dat ek my veld moontlik mag verdeel nie. Ek vind dit ook meermale by die predikant in die gemeente. Die predikant van 'n sterk gemeente hou nie daarvan dat sy gemeente verdeel word nie, want dan is die status van die gemeente verklein, die inkomste is gehalveer en baie meer probleme kom daarmee saam. En ek vrees net dat in hierdie toestand van 'n enkel fakulteit, 'n koninkryk mag ontwikkel wat later al meer moeilik sal word om te verdeel; 'n verdeelde kroon, en u weet 'n verdeelde kroon het ons reeds op politieke gebied in die verlede al baie probleme besorg.

Waar moet die nuwe fakulteit wees?

Ek glo dit moet wees waar die mees intensiewe veeboerdery beoefen word. U moet self aflei waar dit sal wees. Dit moet wees waar die uitbreidingsmoontlikhede van die veeboerdery die grootste is. Dit moet wees in die provinsie wat die meeste vee dra, en die grootste potensie het vir vee vir die toekoms. Dit moet wees in die provinsie waar die meeste veesiekte en byvoedingsprobleme bestaan. Dit moet wees in die streek wat klimatologies die teenoorgestelde is van dié, waarin die huidige fakulteit gehuisves word. Dit moet wees in die streek wat hom die beste leen tot aangeplante weiding en weidingsnavorsing. Dit moet wees by 'n Universiteit met 'n kragtige landboufakulteit. Dit moet wees in 'n streek waar die groot getalle veeboerseuns rondom daardie fakulteit beskikbaar is as materiaal en kandidate, en laastens dit moet wees in die streek, wat die staat in sy beleid by wyse van afbakening bepaal het, die kern van blanke Suid-Afrika te wees. Maar ek gaan u nie sê waar nie.

En die prosedure, meneer die President? Die prosedure wat ons moet volg glo ek is dat die Staat onmiddellik 'n inter-departementele kommissie van ondersoek aanstel om die hele aangeleentheid van veeartsenydienste *in sy volle omvang* te ondersoek, met spesiale klem op die skepping van *'n tweede fakulteit en tweede navorsingssentrum* en om 'n verslag uit te bring wat as basis kan dien vir 'n deeglike, uitvoerbare, versiende en so absoluut noodsaaklike beplanning.

Tweedens, dat die staat ernstige oorweging sal skenk aan die wenslikheid van die aanstelling van distriksveeartse op dieselfde grondslag as distriksgeneeshere, om sodoende die gebrekkige kontak tussen die boer en die veearts noodsaaklikerwys te kan aanvul.

Ten slotte, indien ons nie onmiddellik met sodanige ondersoek en beplanning 'n begin maak nie, sien ek dat daar vir die boer, die verbruiker en vir die Staat 'n tydvak voorlê waarin probleme, wat hande uitruk, en verwyte ons volk se pragtige toekoms op landbougebied gaan benewel en bederf. Die mediese professie beplan die vermeerdering van die menslike bevolking en hulle slaag „onrusbarend goed" daarin. Dit is die plig van die veearts om toe te sien dat daar voldoende gesonde voedsel en kleding vir die groeiende miljoene mense verskaf word. U het 'n edel roeping en 'n verantwoordelike taak en ek wil u die krag en volharding vir sukses op hierdie wonderlike strewe van u paar mense, die Gideonsbende, toebid.

Mr. President, it now is my very great privilege and pleasure to declare this Congress as formally opened.

Mnr. die President, dit is vir my 'n groot voorreg en 'n eer om hierdie kongres met die beste wense as behoorlik geopen te verklaar.— Dankie.

PRESIDENTSREDE

In 'n onlangse toespraak het dr. J. G. van der Wath, voorsitter van die Wolraad, gesê dat die behoefte aan skaapwol in die Republiek so groot is dat ons skaapbevolking behoort op te stoot na 50-60 miljoen.

Ons skaapbevolking bly nou vir die afgelope 30 jaar min of meer konstant op 37 en 38 miljoen, maar gedurende 1929–30 was dit 48 miljoen. Toe kom die groot droogte van 1933 en daarna het die skaapboere nooit weer gewaag om so veel skape aan te hou nie.

Dr. van der Wath is hiervan goed bewus, daarom sê hy ook dat om baie groter as die huidige getalle aan te hou meer intensiewe boerdery metodes toegepas moet word. Dit sou beteken byvoeding, kunsweiding ens. want ons veld kan nie meer vee dra nie.

Wat vleis produksie betref word iets soortgelyk ondervind. Hier vind ons 'n beesbevolking van ongeveer 12 miljoen, waarvan slegs 7½ miljoen aan blankes behoort, en die getal het oor die afgelope 20 jaar nie veel toegeneem nie. Daar is egter 'n toename aan die getal beesslagtings, alhoewel die beeste wat geslag word blykbaar ligter is as vroeër.

Intussen vind daar vinnige vermeerdering aan die mensebevolking van die land plaas en dit skyn dat die lewensstandaard moontlik vinniger styg as die aanwas aan getalle.

Suiwelprodukte word op die oomblik op groot skaal ingevoer.

Die algemene prent is dus een van 'n vinnig toekomende aanvraag na diereprodukte. Daar is egter geen teken van 'n verhoogde produksiepeil oor die afgelope 20–30 jaar nie. Dit kom dus hierop neer dat ons alleen deur veranderde boerdery-metodes 'n verhoogde produksie kan verwag.

Onder die huidige omstandighede kan daar langs twee weë 'n verhoogde produksie verkry word. Die eerste is 'n verhoogde aanwas, dus groter kalwer- en lammeroeste, gepaard met 'n verlaagde sterftesyfer onder ons vee. Die gemiddelde jaarlikse verlies aan beeste het ons 'n paar jaar gelede beraam as sowat 7 na 8 persent. Verder word daar beswaarlik een kalf van drie volwasse koeie per jaar in die Republiek groot gemaak.

Die tweede moontlike weg na verhoogde produksie is deur die toepassing van meer intensiewe boerdery metodes.

Laasgenoemde sal moet gepaard gaan met 'n verhoogde pryspyl vir diereprodukte.

Die ou wet van voorsiening en aanvraag gaan natuurlik pryse bepaal en vleispryse styg alreeds, en sal waarskynlik aanhou styg.

Die algemene neiging blyk dus te wees dat daar 'n vinnig stygende aanvraag na diereprodukte is met geen noemenswaardige styging wat voorsiening betref nie.

Heelwat word deur die Landbou Departement gedoen om veeboere behulpsaam te wees, maar word voldoende veeartsenykundige beplanning vir toekomstige uitbreiding gereël? Sover ons kan bepaal word enige veeartsenykundige uitbreiding wat beplan word beperk tot een inrigting en streeks diagnostiese laboratoriums. Is dit voldoende in 'n land waar die inkomste uit veeboerdery jaarliks sowat R380,000,000 na R400,000,000 beloop?.

Ons erken dat daar gedurende die afgelope jare groot verbeterings aan staats veeartsenydienste aangebring is, maar ons het nog nie gehoor van 'n beplande uitbreidingsprogram wat betref navorsings- en opleidingsentra vir die toekoms nie.

Ons sou dink dat 'n geweldige groot bedryf soos die van skaap en wol geregtig sou wees op 'n veeartsenykundige navorsingssentrum in een van die voorneme skaapproduserende gebiede van die land.

Afgesien van die bekende onopgeloste veesiekte probleme wat in die land heers en wat bekend staan as die sogenoemde erosie-siektes,

is dit alombekend dat intensiewe boerdery nuwe probleme skep. Dat intensiewe veeboerdery reeds plaasvind en met die verloop van tyd 'n versnellende tempo gaan aanneem is nie te betwyfel nie, en alhoewel daar reeds meer veeartse opgelei word as ooit tevore, bestaan die vraag nog of voldoende geriewe beplan word vir die voorsiening van genoeg veeartse in die toekoms. Ek persoonlik betwyfel dit.

Ons het geen rede om twyfel uit te spreek omtrent die gehalte van die werk, wat wel gelewer word. Daar kan slegs met trots gepraat word omtrent wat gedaan is, maar ons het ernstige bedenkings omtrent toekomstige beplanning. Die bedenkings word veroorsaak deur die geskiedenis van die verlede. Dit het ons beweeg om te vra na 'n deeglike ondersoek na alle veeartseny aangeleenthede. Die versoek is voorlopig geweier en daarom het die Raad van die S.A.V.M.V. besluit om self so 'n ondersoek in te stel. 'n Onderkomitee is aangestel om die ondersoek te probeer uitvoer. Daar was ongelukkig 'n tydelike onderbreking met die werk van die komitee wat veroorsaak was deur sekere huishoudelike moeilikhede. Ek, tesame met ons Sekretaris, het voorverlede week die Sekretaris van Landbou-Tegniese Dienste gaan spreek in verband met die saak, en die moeilikhede uit die weg geruim. Ons sal dus hopelik binnekort kan voort gaan met die onderneming.

U sal dadelik begryp dat hier 'n werk van geweldige omvang ons voorlê en soms is ons onseker omtrent die deurvoerbaarheid van so 'n onderneming met die beperkte geldelike en ander geriewe tot ons beskikking. Ons vertrou dat georganiseerde Landbou en ander instansies ons gaan bystaan deur inligting in te samel en aan ons beskikbaar te stel.

Maar selfs met al die moontlike hulp wat tot ons beskikking mag wees is dit heel duidelik dat die werk geweldige persoonlike en geldelike opoffering van sekere lede van die komitee gaan verg, en dit is ook duidelik dat dit nie van staatsamptenare verwag kan word nie.

Ons is egter bereid om tot die uiterste van ons vermoeë deur te druk met die onderneming en om sodoende voldoende inligting in te samel en te formuleer, sodat 'n meer helder begrip van die benodighede van die veeteelt bedryf verkry kan word. Ook is ons begaan oor sekere verskynsels in die veeartsenypraktyk wat opheldering vereis. Ons dink bv. aan moontlike weë vir die daarstelling van veeartsenydienste aan veeboere op die platteland teen 'n, vir die boer, ekonomiese peil.

The Instructional Tour of the Western Province.

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The Braaivleis-lunch on Mr. F. Basson's farm.

The Instructional Tour of the Western Province.

- - -

The Braaivleis-lunch on Mr. F. Bason's Farm.

Dit spreek vanself dat ekonomiese oorwegings dit dikwels vir die praktisyn onmoontlik maak om op die platteland te bestaan, en op die oomblik daar 'n onoorbrugbare gaping tussen wat die boer kan bekostig om te betaal en wat die veearts moet verdien om sy belegging aan sy opvoeding en om 'n ordentlike lewensstandaard te regverdig. Tog voel ons dat deur die behoorlike koordinering van die baie bronne van diensverskaffing tot die beskikking van veeartse baie meer praktisyns op die platteland gevestig kan word.

Die voorgestelde ondersoek sou onder andere die moontlikhede probeer nagaan en die nodige aanstelling verkry.

Dit is vanselfsprekend dat verwikkelinge van die aard nie oornag kan gebeur nie maar alhoewel hulle miskien met die verloop van tyd onwillekeurig tot stand mag kom is dit ons oortuiging dat daar doelbewus na gestreef moet word indien ons nie ons plig teenoor die veeboere wil versuim nie, soos wat ons oortuig is die geval in die verlede was.

Die vooruitsig is dat ons vir baie jare nog aan 'n tekort aan veeartse gaan ly. Ons besef dat daar 'n tekort aan allerlei opgeleides is maar dit is ons oortuiging dat die tekort aan veeartse moontlik een van die dringendste is; veral in agnemend die waarde van ons veeteelt-bedryf. Die tekort veroorsaak geweldige geldelike verliese aan veeboere. Die werklike omvang van die verliese is moeilik beraambaar, waarskynlik kan hulle glad nie bereken word nie. Opnames word blykbaar nou geneem om te probeer bepaal watter probleme daar bestaan en hoe hulle aangepak moet word.

Voltooiing van die opnames gaan baie lank neem en die vraag is of daar nou gewag moet word tot die opnames voltooi is voor doeltreffende stappe geneem word om hulle op te los? Dit is beleidsvrae wat nie sonder versigtige oorweging beantwoord kan word nie. En wie gaan hulle op 'n realistiese en betroubare wyse antwoord?

Daar was die ongelukkige ondervinding van die verlede dat op veeartsenykundige gebied voortuitbeplanning vir die behoeftes van die land baie ontoereikend was. Leiding word nou aan ander manne toevertrou, maar is dit reg teenoor hoofde van departemente wat hulle hande meer as vol het met administratiewe en ander verpligtinge om van hulle ook te verwag dat hulle verantwoordelikheid vir toekomstige beplanning moet aanvaar?

Daar is nou 'n nuwe Landbou Administratiewe organisasie wat maar 'n paar jaar in werking is. Die persone daarop is baie bekwaam, maar niemand weet wat daar beplan is vir die uitbreiding van veeartsenykundige geriewe nie. Dit word as huishoudelik beskou. Dit is egter nie huishoudelike sake nie maar sake van nasionale belang en daarom behoort die beplanning bekend gestel te word.

Groot uitbreiding aan die enigste navorsingsinrigting van die Republiek is reeds in aanbou, maar is dit voldoende om alle navorsing van landswye belang op een plek te sentraliseer?

As daar op ander landbougebiede die noodsaaklikheid bestaan om 'n aantal navorsings- en opleidingssentra te hê moet dit tog tot 'n mate geld vir veeartsenykunde. Dit is dikwels onmoontlik om biologiese probleme wat in nou verband staan met die omgewings invloede wat hulle verwek oor te plant na 'n sentrale navorsingsinrigting.

Die toekoms van die biologiese wetenskappe, waaronder veeartsenykunde sorteer, is onpeilbaar en die vooruitsig is dat vordering in die rigting geweldig gaan wees in toekomstige jare. Dit sou dus gesonde beleid wees om ons daarvoor voor te berei.

Prof. Luitingh het onlangs in 'n toespraak wat in ons jongste tydskrif verskyn 'n pleidooi gelewer vir nouer samewerking tussen die boer, die veearts en die veeteler. As ons ons regmatige deel aan die samewerking wil bydra moet ons daarvoor sorg dat daar genoeg veeartse is en dat hulle verspreiding oor die land doeltreffend is.

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OPENING OF THE TRADE EXHIBITS BY THE VICE PRESIDENT

PROFESSOR R. CLARK

Prof. R. Clark opened the Trade Exhibits with these words:

"It is not necessary to introduce the Exhibitors — their contribution has been going on for many years and is a very valuable contribution as it helps us to keep up to date — especially those of our members who do not live near scientific centres. They give us information regarding the latest instruments, remedies and books.

The Exhibition this year is quite up to the usual standard and has been organized through the good offices of Mr. Stabler of Johannesburg and Mr. Veitch of Cape Town, both members of a wellknown firm dealing in instruments and appliances. We hope you will take advantage of the Exhibits and visit them as often as you can.

I might add that these Exhibitors contribute very materially to our Benevolent Fund and that is a Fund which deserves every bit of support we can get. The Exhibit is up on the 4th Floor.

I now declare the Trade Exhibit open and would ask both His Worship the Mayor and Mr. de Villiers Loubser to visit it with us and see the instruments. I would like them to see these instruments and appliances. We have got beyond the stage where our equipment consisted of a pen knife, a bottle of linseed oil and a corkscrew.

The corkscrew figures on the Onderstepoort's Students Badge. The history of this badge goes back to the days of Sir Arnold Theiler. He said veterinary students only needed a couple of things amongst which was a corkscrew. One student asked "why a corkscrew Sir Arnold?" to which he replied 'to open the medicine bottles'."

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SOME MECHANISMS OF MAMMALIAN IMMUNITY

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Guest lecture delivered at the Annual Scientific Congress of the South African Veterinary Medical Association

INTRODUCTION

It is a great honour to have been asked to address you this evening during the Annual Congress of the South African Veterinary Medical Association, and I would like to take this opportunity of expressing to you my sincere appreciation of your kind gesture in electing me last year, an Honorary Member of your Association. I have paid many visits to Onderstepoort to exchange ideas with colleagues working in fields allied to my own. I regard it as one of my most treasured privileges to be welcome there. They, both old and young, have taught me much.

The subject of my talk this evening — "Some Mechanisms of Mammalian Immunity" — is one of great importance to all of us. There is hardly a single field in human or veterinary medicine that has remained uninfluenced by the new concepts and the theoretical developments of the last 20 years in immunology.

The phenomena of immunity and immunological reactivity are apparently confined to vertebrates, and the earliest studies were concerned with the nature of antibody and with the specificity of the antigen-antibody reactions. Soon it became apparent that the phenomena of hypersensitivity were basically similar to those that were regarded as imparting immunity. For a long while these constituted the foundation of the subject.

But last year Gell and Coombs (1963)[16] wrote 'At the beginning of the century Immunology, then an infant barely 10 years old, promised to be the wonder child of Medicine. During the next 20 years it grew up and was put out to work; and if it did not quite live up to the promise of its golden infancy, it did a very valuable job of practical therapeutics in human and veterinary medicine. But signs developed that it was being sent out into the world too young, without the benefits of a University education — without, that is, having had time to think and to make up its mind about its real place in the world. Hence it is not unfair to say that by the Second World War a certain lethargy had fallen on the subject.'

Dramatic conceptual advances have followed the realization that immunological reactions are involved in a wide variety of phenomena and not confined only to consideration of infectious disease and the classical manifestations of hypersensitivity.

In this presentation I shall, naturally, call heavily, but not only, upon human examples to illustrate important mammalian responses.

IMMUNOLOGICAL TOLERANCE AND TRANSPLANTATION OF TISSUES

The first major development followed the discovery of immunological tolerance.

It had, for a long time, been taken for granted that an animal would not normally react immunologically to its own antigens and tissues either by antibody production or in any other way. In considering this, Burnet and Fenner (1949)[9] postulated that the exposure of foetal or embryonic animals to an antigen during this period of immunological immaturity, would

cause the animal to accept this substance as 'self' rather than 'foreign' or 'not-self'. Burnet (1959)[8] later suggested that this acceptance was due to the supression or destruction of the cell line or clone of cells genetically endowed with the potentiality to react to the presence of this antigen.

The earliest experiments to induce such a state of specific unresponsiveness to a particular antigen failed, but Medawar and his colleagues (Billingham *et al* 1953)[5] succeeded in making mice unresponsive to skin grafts in post-natal life by the administration of donor cells into the foetal mice. This induced state they called acquired immunological tolerance.

Some years previously Owen (1945)[36] had demonstrated that dizygotic (non-identical) cattle twins, sharing the same placental vessels, were born with cells of two genotypes consisting of those of its own and those transfused into it from its twin. Each was immunologically tolerant of the antigens of the other and this was confirmed by the fact that grafts from one to the other were successful, whereas grafts from other calves were rejected.

Surgical transfer of tissues or organs between individuals of the same species, i.e. homografts, almost invariably end in the destruction of the graft by a process which is clearly immunological.

This was confirmed in mice. The first graft would appear to 'take' but in about 11–12 days it would be rejected. A second graft from another donor of the same inbred line as the first, resulted in an accelerated reaction, with destruction of the graft in about 6 days, and this was termed the second set phenomenon.

If, however, living cells from the spleen or bone marrow of the donor-line of mice were injected intravenously into the foetus, or into the newborn mice soon after birth, then when these mice grew up they were found to be incapable of rejecting a donor-line graft which settled down and took up permanent residence in the skin of the recipient mouse. This response was specific since a graft from a third party would be rejected in the usual fashion.

It is now known that immunological tolerance is a general phenomenon and has been demonstrated in a variety of animals such as the mouse, monkey, rabbit and the fowl.

It was soon observed, however, that tolerance could be induced more readily in certain host-donor combinations (Billingham and Brent 1957,[4] Martinez *et al* 1958)[30] than in others, pointing to a genetic basis for the success or otherwise of this type of experiment. The most successful results are achieved with tissues which are antigenically not too distantly related to those of the host.

The immunological responses of the homograft reaction are concerned with a number of T antigens (as opposed to the ABO and Rh antigens) present only in nucleated tissue cells. They are determined by so-called histocompatibility genes of which there are several (in the rabbit at least 7 and in man at least 20) on the chromosome. The genes at this histocompatibility-locus on the chromosome are numbered and the H-2 locus is the strongest one concerning histocompatibility in the mouse. That is to say that transplantation experiments between mice of the same H-2 genotype are more likely to be successful than between mice of different H-2 genotype.

Tolerance is not necessarily permanent. The duration of the tolerance is dependent upon the nature and the amount of the antigen originally introduced. The tolerance induced by a single injection of an inert antigen may be measured in weeks or months but it may be prolonged by further injections of antigen during the tolerant period. Longer lasting tolerance may be expected when the antigen is in the form of live cells, which persist in the recipient.

Homografts may be successful when the recipient is either naturally, or artificially made, immunologically unresponsive. There are many such circumstances. Such is the case with:—

(1) autografts;

(2) identical twins who accept isografts;

(3) cattle or human twins (chimeras) of dizygotic origin by exchanging cells in utero, become completely tolerant of each other;

(4) mice, made immunologically tolerant by injection of spleen cells of the donor

line of animals into the foetal or neonatal animals;

(5) recipient animals in which a reduction or elimination of the immunologically competent cells is brought about by X-irradiation, by the use of cytotoxic drugs or by the use of drugs such as 6-mercaptopurine which interferes with RNA synthesis. Such animals may accept not only homografts, but even heterografts because they no longer possess the cellular components with which to reject them.

Graft — Versus — Host Reactions

It would be misleading to give the impression that all of these are equally as satisfactory for the transplantation of tissues. There are a number of other considerations.

Skin grafts normally contain no immunologically competent cells, but when grafts of spleen, lymphglands, bone marrow or kidney are attempted, the immunologically competent cells which these tissues contain may react against the antigenically different cells of the recipient. This graft-versus-host reaction is almost invariable resulting in wasting, impairment of growth and eventually death of the host said to be suffering from runt disease.

If the unresponsiveness is induced by X-irradiation or other means the graft-versus-host reaction is often called homologous disease, or secondary disease. The risk of infection in this state is very high and for the duration of the experiments the lethally irradiated mice are given suitable antibiotics to ensure their survival, but for the complete success of the experiments it is necessary to restore the bone marrow, by a transfusion of bone marrow cells from the donor. These cells too are immunologically competent and will show a graft-versus-host reaction, but they will not damage the graft in situ.

Bone marrow cells, collected before the experiment started, from the host, and used for this transfusion will reject the graft. Foetal or embryonic bone marrow, which is immunologically immature, would in theory be most suitable for this experiment since the only way it could react either to the graft or to the host cells would be by becoming tolerant to both of them.

Before considering the nature of the reaction in the tissues which is responsible for the transplantation reaction it is of interest to mention a controversial matter of homograft immunity in pregnancy.

Homograft Immunity in Pregnancy

The most successful homograft is the normal mammalian foetus and its placenta in the normal pregnant animal.

It is still not clear why the mother does not reject the antigenically distinct foetus as she would a skin graft from the father.

The foetus, as we have already indicated, is immunologically non-reactive, so it is unlikely to react to the maternal antigens in any other way than by becoming tolerant to them. There is ample evidence, however, that the mother may become sensitized to the antigens of the foetus for this is the very basis of Rh disease or acute haemolytic disease of the new-born.

It was suggested that the onset of labour might result from such a graft rejection, but there are objections to this proposal. (Lanman *et al* 1964)[26]:—

(1) animals inbred to the point of histocompatibility would then be incapable of delivering their young. There is, in fact, no difference in the gestation period of highly inbred and outbred strains of mice;

(2) studies in man and animals have failed to reveal maternal homograft sensitization against the foetus. If the mother is sensitized with grafts from the father before pregnancy, a graft of the foetus to the mother will show an accelerated reaction, but the pregnancy is normal;

(3) rabbits sensitized by skin grafts to both members of a breeding pair were simultaneously implanted with eggs from the sensitizing pair, and with eggs from a second breeding pair against

which no sensitization had been produced. The survival rates of the two types of eggs and the number of young from each kind of egg indicated that the maternal sensitization had not harmed either.

This shows that the foetus is protected. The protection appears to lie in the physical separation of the mother and the foetus by the intact placenta, and the specialized properties of the trophoblast. Perhaps the placenta is devoid of T antigens. Possibly the maternal response is somewhat suppressed by the changes in corticosteroid metabolism.

The Nature of the Graft Reaction

It is necessary to attempt an analysis of this immunological response in graft rejection.

Histological examination of the graft during the process of rejection shows a diminution of the vascularity and an increasing infiltration of the tissue with lymphoid cells and histocytes, but without a corresponding infiltration of plasma cells. On the occasion of the accelerated response of the second set phenomenon the cellular infiltration occurs with dramatic rapidity.

While it is true that antibodies to the graft cells may be demonstrated in the serum of the recipient, graft immunity cannot be passively transferred by serum alone. Transfer is successful only by means of cells either from lymph glands or spleen from immunized mice to a normal mouse or to a tolerant mouse with a graft in situ, which then becomes rejected.

These observations make the mechanism of graft rejection remarkably similar to that responsible for the delayed hypersensitivity response to certain bacteria, viruses and other microorganisms. Guinea pigs rendered immune by skin grafting, react with a typical delayed hypersensitivity skin reaction to tissue extracts from the donor of the graft, whereas the same extract causes only a minor non-specific reaction in the skin of a normal animal.

I shall return to a consideration of the participation of delayed hypersensitivity in resistance, after a brief presentation of a problem in human disease which has occupied our minds recently.

Herpes Simplex Infections in Man

Herpes simplex infection in young children provides an example of a problem in human immune responses.

Herpes simplex infections in man are so common that it is very surprising that they are so poorly understood. The very common primary infection — the primary herpetic stomatitis — occurring as a rule in young children between the ages of 2 months and 5 years, no longer stimulates much interest. Some observers benignly believe that there is little more we need to know about them. But it would be nearer the truth to say that all we really know about them is:

(1) that they commonly occur in children no longer protected by the passive immunity of maternal antibodies;

(2) that the mortality is insignificant under most circumstances and that the vast majority of the world's children recover completely from this mild disease;

(3) that a very high proportion of the infections are so mild as to be entirely subclinical and missed;

(4) that recovery is associated with solid immunity, associated with neutralizing antibodies in the serum and presumably with persistence of virus in the tissues, since

(5) some of the immunes are likely to be plagued in later life with recurrent fever sores about the lips (or rarely elsewhere) after a common cold, too much sun-bathing or something of that sort.

In Cape Town, starting in 1957, children were encountered dying from this otherwise rather trivial infection. Although this infection is sometimes fatal in the newborn, children between the ages of 2 months to 2½ years should not succumb. This initiated a programme of research which has included virological, immunological and pathological investigations and I

can now report that we have seen 33 such cases.

There was a time when we thought the disease was confined to Cape Town. We have since ascertained, however, that it does occur elsewhere and that typical cases of "Non-neonatal Fatal Disseminated Herpes Simplex Infections" have been seen in other centres in the Republic. We have seen the post mortem material of 50 typical cases in Durban in Professor Wainwright's Department and we have heard of 3 or 4 cases in Pretoria and Johannesburg.

After we had described what we had seen (McKenzie *et al* 1959,[31] Becker *et al* 1963)[3] we received a visit from Professor Armengaud of Dakar, Senegal. He had seen the same disease. In his area he had been impressed by the frequent association of herpes simplex infections with the convalescent stage of measles, and of these thirteen had died with the disease as we see it in Cape Town.

In Cape Town 69 per cent of the cases were in children with kwashiorkor. In Durban 63 per cent of the cases had kwashiorkor or were severely malnourished and a further 21 per cent were convalescent from measles.

The failure of recovery was associated with a generalization of the infection and with multiple foci of necrosis in the liver and the adrenal. There was practically no cellular reaction to these necrotic cells, and the surviving cells about them showed typical intranuclear virus inclusions.

It is generally known that there is no gross deficiency of gammaglobulin in the sera of patients with kwashiorkor, and electrophoretic analysis of many of the sera of our fatal herpes cases showed clearly that they were not hypogammaglobulinaemic.

Our interest soon turned to the apparent failure of recovery from this usually insignificant virus infection. Since we knew nothing about unduly virulent hepatotropic or adrenotropic strains of herpes simplex virus, it appeared to us that this strange syndrome might result from a failure of a basic mechanism of immune response. Was it a specific failure of response to the herpes simplex virus or was it a more general failure of response to any infection?

Previous experience with kwashiorkor led us to believe that there was some basic immunological deficiency in clearing the blood stream of bacteria of the type that may have entered the blood from the intestinal tract or from the lung (Smythe 1959)[45]. The most common causes of this septicaemia were Gram negative bacilli of the intestinal tract such as salmonella, pseudomonas, escherichia and klebsiella. This state was accompanied by high mortality despite the administration of antibiotics to which the invading bacterium was sensitive.

The other immunological defect encountered in kwashiorkor and in the post-measles convalescent case is a temporary supression of the mechanisms responsible for the delayed hypersensitivity reaction. We found that in children with measles who were known to have received BCG vaccine, 80–90 per cent have negative tuberculin tests, but within 6 months 66 per cent of these had re-converted back to a positive reaction. In a comparable group of children with measles who were not known to have received BCG vaccine, a similar proportion were negative to the tuberculin test but only 20 per cent had become positive within the following 6-month period.

We were encouraged, therefore, to see whether there was any similar immunological defect in the patients who fail to recover from other relatively insignificant primary virus infections.

Recovery from Primary Virus Infections

The second major development followed the recognition of the clinical condition of agammaglobulinaemia which influenced dramatically our concept of the mechanism of recovery from a primary infection caused by a virus.

Until recently it was more or less generally believed that, within a few days of infection neutralizing antibodies appeared in the body fluids bringing about the inactivation and opsonization of the virus particles which were then taken up by phagocytic cells wherein they were disrupted and destroyed.

There are now, however, many reasons to doubt the major role of neutralizing antibodies in this first recovery, without at the same time

questioning their important role in preventing the establishment of second or subsequent infections.

1. It is well established that most agammaglobulinaemic or severely hypogammaglobulinaemic children, though tormented throughout life with recurrent bacterial infections of certain types, (due to streptococci, staphylococci, meningococci and pneumococci) react to and recover from certain other types of bacterial infection such as salmonellosis and tuberculosis, as well as from primary virus infections as effectively and as efficiently as normal children. (Good and Zak 1956)[18], and that the immunity which follows is both specific and long lasting. This is so for infections such as measles, varicella and vaccinia.

Solid immunity to poliomyelitis virus has been demonstrated in persons with no demonstrable antibody in the serum (Sabin 1959)[42] in feeding experiments with live virus.

Although the absence of gammaglobulin is seldom complete and very low levels of antibody may be demonstrable by specially sensitive techniques, the deficiency is such that it would be unlikely to provide effective protection, but it should be noted that recovery from these infections is associated with the development of delayed hypersensitivity to the viral antigens.

2. The administration of large amounts of pooled gammaglobulin or even hyper-immune gammaglobulin fails to initiate or to hasten the recovery from common primary virus infections in children. (Mitus *et al* 1960,[34] Kempe 1960,[25] Connolly *et al* 1962)[10].

3. Neutralizing antibodies in the serum in high concentration may fail to prevent the evolution of the lesions, the progress of the disease and a fatal outcome in smallpox (Downie 1951)[12] or in measles (Mitus *et al* 1960)[34].

4. Recovery from viral infections in experimental animals does not require the participation of antibody (Friedman *et al* 1961,[13] Friedman *et al* 1962[14]) and it has been suggested that recovery from primary virus infections is well on its way before antibody makes its first appearance in the body fluids (Hitchcock and Isaacs 1960[22], Baron and Isaacs 1962[2]).

5. The spread of infection from one cell to another in tissue culture infected with herpes simplex (Stoker 1958)[46], vaccinia or varicella viruses occurs whether neutralizing antibody is present in the medium or not.

Carriers of Immunological Configuration

The host immune mechanism has four major carriers of the configuration complementary to the antigen which stimulated it (Smith 1960)[44]. A lymphoid cell, a high molecular weight globulin, a low molecular weight globulin and a globulin with a marked affinity for tissue cells in cases of atopic allergy.

The low molecular weight globulins have a sedimentation constant of about 7 S and constitute 90 per cent of the adult gammaglobulins. They are the antibodies of the conventional type, produced by plasma cells. They pass readily across the placenta to give the newborn child antibody levels equal to that of the mother.

The high molecular weight globulins have a sedimentation constant of 19 S but they represent only 3–10 per cent of the adult gammaglobulins. They do not readily cross the placental barrier so the newborn child commences life with a temporary macroglobulin deficiency (Gitlin *et al* 1963).[17] Antibodies composed of 19 S macroglobulins are the saline anti A, anti B and Rh antibodies; the antibodies to the somatic lipopolysaccharide of the coliform organisms etc.

Since recovery from primary virus infections in the agammaglobulinaemic child may occur in the face of gross antibody deficiency, it is important to consider the immunologically competent cells which become sensitized by contact with antigen, participate in the secondary response, account for the delayed hypersensitivity reactions in man and animals and, therefore, also for the rejection of homografts. These cells may transfer resistance which cannot be transferred by serum alone.

The Thymus Gland and Immunity

The third major development was the discovery that the thymus gland plays a very im-

portant role in the development of immunological competence since its removal at birth is likely to result in a gross immunological deficiency in later life.

In 1961 Miller (1961,[32] 1962[33]) showed that mice thymectomized at birth ahd fewer lymphocytes and a higher mortality than normal controls. At 6 weeks they showed prolonged survival of skin grafts but 70 per cent of them died before the 4th month from 'wasting disease' associated with lymphopenia and marked lymphoid involution. Grafts of thymus tissue, however, restored the homograft immunity.

The thymus is thought to be the source of immunologically competent precursor cells during the late embryonic and early neonatal life so that the thymus is fully active at a time when the lymphoid structures throughout the body are underdeveloped.

Thymectomy at birth prevents the seeding of thymic cells into the spleen, bone marrow and lymphnodes in guinea pigs, mice and rabbits. It decreases their ability to produce antibody, permits homografts to be accepted, inhibits the development of delayed hypersensitivity and lowers their resistance to infection. The spleen cells from such mice are unable to elicit a graft-versus-host reaction, i.e. they are immunologically inactive (Hilgard *et al* 1962)[11].

There is increasing evidence to suggest that the cells of the thymus are not essential to seed the lymphoid tissues (Levey *et al* 1963,[28] Levey 1964[27]) but that a thymic hormone may initiate the maturation of cells in the spleen and lymphglands, into immunological competence.

Cellular Immunity

If it is logical to suggest that neutralizing antibodies do not play a major role in recovery from primary virus infections, then it is almost inevitable that we should believe that cells do.

It seems that there are three important ways in which these cells may do this:

(1) by the enhancement of phagocytic activity of reticulo-endothelial cells;
(2) by the production of interferon;
(3) by the development of delayed hypersensitivity.

Enhancement of the Reticulo-endothelial Activity

The phenomenon of 'cellular immunity' has recently been re-investigated by a number of workers from very different points of view. In order to account for the transfer of delayed hypersensitivity and transplantation immunity by means of cells, it was common to refer to 'cell-bound' antibodies at a time when there was no experimental evidence to justify this inference. In fact the continued failure to demonstrate such cell fixed antibodies led Boyden (1963)[6] to propose that delayed hypersensitivity was due to a tissue response different from antibody production, and that antibodies were not involved in the tuberculin reaction, cell-bound or otherwise. Other workers (Furness and Ferreira 1959,[15] Saito *et al* 1962[43]) also regarded these reactions as dependent upon phagocytic cells and without the participation of antibody.

Very recently there has appeared a series of papers by Rowley and his colleagues in Australia (Jenkin *et al* 1964,[24] Turner *et al* 1964,[47] Rowley *et al* 1964a[41]) pointing out that living attenuated *S. typhimurium* in mice gave rise to solid immunity which was characterized by early production of 19 S gammaglobulin, for the first 2–3 weeks following infection. Appearing a little later but persisting for much longer there were the prominent, conventional 7 S gammaglobulin antibodies. The early macroglobulins were readily adsorbed to phagocytic cells, and these antibody-coated cells were strikingly correlated with the enhanced opsonic and bactericidal properties of the immunized mouse.

Their experiments showed convincingly that the 19 S macroglobulins were attached to mouse peritoneal macrophages which were effective in transferring immunity, and that these cells, destroyed by freezing and thawing, were almost as effective. The macroglobulins could be eluted from the cell debris with 2 M urea and this eluate may impart immunity. The transfer of this type of immunity may also be transferred be serum alone provided the serum was rich in 19 S macroglobulin.

In another paper (Rowley and Turner 1964)[40] it was shown that injections of lipopolysaccha-

ride from Gram-negative bacilli produced profound changes in the immune state of mice and pigs. Within a few days there was a remarkable elevation of the macroglobulin level in the serum lasting 8–15 days. This change was not specific since it could be produced by a variety of lipopolysaccharides, and the sudden rise of macroglobulin suggested a release rather than a synthesis of immune antibodies.

There are many examples of this type of non-specific enhancement of resistance. Similarities between Gram negative bacterial endotoxins and their lipopolysaccharides and crude extracts of tubercle bacilli, in their ability to enhance resistance to homologous and heterologous infectious agents, were drawn by Weiss *et al* 1964,[48] and their experiments show that a non-toxic residue from phenol killed acetone washed, methanol extracted tubercle bacilli of the BCG strain conferred a high degree of resistance on mice to otherwise lethal infections of *Klebsiella pneumoniae* and with a number of other organisms. This heightened resistance lasted from 24 hours to at least 10 days. Halpern (1964)[20] speaks of a similar effect from his own extract of tubercle bacilli which enhanced the immunity of mice to *S. typhimurium* and other bacteria despite the fact that his extract is almost completely incapable of inducing the production of antibodies. Old *et al* 1961[35] showed that BCG enhanced resistance of mice to Mengo virus.

These and similar findings may indicate that the effect of these large molecular substances on the reticulo-endothelial system is one of non-specific stimulation.

Boyden (1964),[7] however, has described an elegant technique for demonstrating the presence of cytophilic antobidies adsorbed to cells of the guinea pig and the rabbit, but these antibodies appear to have the properties of 7 S gammaglobulins. The experiments showed an important association between the development of delayed hypersensitivity and the ability to produce cytophilic antibodies although it has not been proved that the cytophilic antibody is responsible for the hypersensitivity.

So cytophilic antibody appears to account for the immunity transferred by 'immune cells' and current concepts emphasise the participation of adequate numbers of phagocytic cells and adequate amounts of the right kind of antibody, for its full expression.

Interferon

Interferon production, first described by Isaacs and Lindemann (1957)[23] is now recognized as a more or less general response of cells in birds, animals and man to the introduction of a foreign mucleic acid into the cells. It has the property of rendering normal cells incapable of supporting the multiplication of a variety of viruses, but not all to the same extent.

The observations of Hitchcock and Isaacs, 1960[22] and of Baron and Isaacs, 1961[2] indicated that interferon plays an important role in recovery from many virus infections. It has been shown to be effective in tissue culture and in vivo. Because it appears earlier than demonstrable antibody and because peak concentrations of interferon appear to coincide more closely with onset of recovery, it has been suggested that it may be the prime factor in recovery from some types of infection. This finds support in the recovery of the embryo or the foetus which are thought to be immunologically unreactive, and in the experiments of Friedman *et al* (1962)[14] who showed that guinea pigs treated with X-irradiation to block antibody production and methotrexate to inhibit delayed hypersensitivity responses, could still recover from vaccinia infections as rapidly as normal animals do. Their capacity to produce interferon was left intact by these procedures.

However, the effect of interferon on cells is short lived and at present is not thought to explain the long lasting immunity following recovery.

Delayed Hypersensitivity

There is increasing interest in the part played by delayed hypersensitivity in the various mechanisms of antibacterial and antiviral immunity.

There are several ways of presenting some of these developing concepts.

The development of delayed hypersensitivity to antigens of an infectious agent has been demonstrated in a multitude of infections and probably occurs as part of the recovery process in all types of infectious disease. It has been shown to occur in vaccinia, psittacosis, trachoma, herpes, mumps, measles, lumpy skin disease, mouse pox and others.

It has been shown (Pincus and Flick 1963)[37] that after primary vaccination in man against smallpox, hypersensitivity develops early (within four days) and in the rabbit that this hypersensitivity is responsible for the local papular-vesicular lesion (Pincus, Flick and Ingalls 1963).[38] The cellular mecrosis which occurs at the peak of the reaction at 8–11 days is not due to the cytopathic effect of the virus but to the damaging effect of immunologically competent lymphoid cells already hypersensitive, on the cells containing the viral antigen.

These immunologically competent lymphoid cells account for the accelerated reaction of re-vaccination, and impart to the host the long lasting immunity. It appears that the allergic reaction is also an immunizing event since it is on this that the agammaglobulinaemic child has to rely for recovery from virus infections.

The exanthematous rashes, for instance the rash of measles, may be regarded as multiple disseminated cutaneous delayed hypersensitivity events.

Although agammaglobulinaemic children lack the conventional antibody mechanisms they develop typical tuberculin skin reactions after BCG vaccination. Their lymphoid cells are capable of transferring delayed hypersensitivity to normal persons, and they accept transfer of delayed hypersensitivity from others. Good *et al* 1964[19] have shown that there may be varying degrees of deficiency in delayed hypersensitivity manifest by slow or very slow rejection of skin grafts.

The extreme immunologic defect in human children is exemplified by agammaglobulinaemia with an absence of lymphocytes from the spleen or lymphnodes, an incapacity to develop delayed hypersensitivity and an inability to reject a graft (Rosen *et al* 1962).[39] This is the naturally occurring human equivalent of the neonatally thymectomized mouse.

There are many clinical states in human medicine which are associated with unexpected mortality from virus infections of the type usually regarded as mild and insignificant. These states include most of the gross disturbances of the lympho-reticular system of cells such as acute leukaemia, lymphatic leukaemia, Hodgkins disease, aplastic anaemia, etc., also patients receiving whole body irradition, or antimetabolites and those on prolonged courses of steroid therapy.

All these situations are the very ones in which infections by viruses such as measles, vaccinia or chickenpox are likely to run to a fatal termination. Might we add to the list, from our experience with fatal herpes simplex infections, the conditions of kwashiorkor and the convalescent stage of measles?

The speculations of Mackaness (1964)[29] are interesting in this context. His experiments with three bacterial pathogens in mice indicate the invariable accompaniment of acquired resistance by delayed hypersensitivity, so that he raises the possibility that the antibody which confers increased cellular resistance may be identical with that which imparts delayed hypersensitivity.

The proposition that the mediator of delayed hypersensitivity provides the immunological reactivity on which acquired cellular resistance depends is not in conflict with the generally held views on the separability of the two phenomena, but it emphasises the currently increasing belief to which we subscribe, that resistance depends for its full expression on the presence of delayed hypersensitivity.

REFERENCES

1. ARMENGUAD, Professor M. Personal Communication.
2. BARON, S. and ISAACS (1961). Interferon and Natural Recovery from Virus Infection. *New Scientist*, **11**, 69.
3. BECKER, W., NAUDE, W. du T., KIPPS, A. and McKENZIE, D. (1963). Virus Studies in Disseminated Herpes Simplex Infections. *S.Afr. J. Med.*, **37**, 74.
4. BILLINGHAM, R. E. and BRENT, L. (1957). Acquired Tolerance in Newborn Mice. *Ann.N.Y.Acad.Sci.* **69**, 678.
5. BILLINGHAM, R. E., BRENT, L. and MEDAWAR, P. B. (1953). Actively Acquired Tolerance of Foreign Cells. *Nature* (Lond.), **172**, 603.
6. BOYDEN, S. V. (1963). Use of Radioactive Proteins for Detection of Cell-fixed Antibodies. In Immunological Methods, Blackwell Scientific Publications, Oxford.
7. BOYDEN, S. V. (1964). Cytophilic Antibody in Guinea Pigs with Delayed Hypersensitivity. *Immunol.*, **7**, 474.
8. BURNET, F. M. (1959). In The Clonal Selection Theory of Acquired Immunity, Cambridge University Press, Cambridge.
9. BURNET, F. M. and FENNER, F. (1949). In Production of Antibodies, 2nd Edit., Macmillan, Melbourne.
10. CONNOLLY, J. H., DICK, G. W. A. and FIELD, C. M. B. (1962). A Fatal Case of Progressive Vaccinia. *Brit. Med. J.*, **ii**, 1315.
11. DALMASSO, A. P., MARTINEZ, C. and GOOD, R. A., (1962). Further Studies on the Supression of Homograft Reaction by Thymectomy in Mice. *Proc. Soc. Exp. Med., N.Y.*, **111**, 143.
12. DOWNIE, A. W. (1951). Infection and Immunity in Smallpox. *Lancet*, **i**, 419.
13. FRIEDMAN, R. and BARON, S. (1961). The Role of Antibody in Recovery from Infection with Vaccinia Virus. *J. Immunol.*, **87**, 379.
14. FRIEDMAN, R., BARON, S., BUCKLER, C. E. and STEINMULLER, R. I. (1962). The Role of Antibody, Delayed Hypersensitivity and Interferon Production in Recovery of Guinea Pigs from Primary Infection with Vaccinia Virus. *J. Exp. Med.*, **116**, 347.
15. FURNESS, G. and FERREIRA, I. (1959). The Role of Macrophages in Natural Immunity to Salmonella *J. Infect. Dis.*, **104**, 203.
16. GELL, P. G. H. and COOMBS, R. R. A. (1963). In Clinical Aspects of Immunology, Blackwell Scientific Publications, Oxford.
17. GITLIN, D., ROSEN, F. S. and MICHAEL, J. G. (1963). 19 S. Gammaglobulin Deficiency in the Newborn Infant. *Ped.* **31**, 197.
18. GOOD, R. A. and ZAK, S. J. (1956). Disturbances of Gammaglobulin Synthesis as Experiments of Nature. *Ped.*, **18**, 109.
19. GOOD, R. A., MARTINEZ, C., PAPERMASTER, B. W., CONDIE, R. M. and GABRIELSON, E. (1964), Studies on Transplantation Biology, in Injury, Inflammation and Immunity, Williams and Wilkins, Baltimore.
20. HALPERN, B. (1964). The Reticulo-Endothelial System and Immunity. *Triangle*, VI, 174.
21. HILGARD, H. R., YUNIS, E. J., SJODIN, K., MARTINEZ, C. and GOOD, R. A. (1964). Reversal of Wasting in Thymectomized Mice by Injection of Syngeneic Spleen or Thymus Cell Suspensions. *Nature* (Lond.), **202**, 668.
22. HITCHCOCK, G. and ISAACS, A. (1960). The Role of Interferon in Recovery from Virus Infection. *Lancet* **ii**, 69.
23. ISAACS, A. and LINDEMANN, J. (1957). Virus Interference. The Interferon. *Proc. Roy. Soc. B.*, **147**, 258.
24. JENKIN, C. R., ROWLEY, D. and ANZINS, A. (1964). The Basis of Immunity to Mouse Typhoid. *Aust. J. Exp. Biol. and Med. Sci.*, **XLII**, 215.
25. KEMPE, C. H. (1960). Studies on Smallpox and Complications of Smallpox Vaccination. *Ped.*, **26**, 176,
26. LANMAN, J. T., HEROLD, L. and FIKRIG, S. (1964). Homograft Immunity in Pregnancy. *J. Exp. Med* **119**, 781.
27. LEVEY, R. H. (1964). The Thymus Hormone. *Scientific American*, **211**, 66.
28. LEVEY, R. H., TRAININ, N. and LAW, L. W. (1964). Evidence for the Function of Thymic Tissue in Diffusion Chambers Implanted in Neonatally Thymectomized Mice. *J. Nat. Cancer Inst.*, **31**, 199.
29. MACKANESS, G. B. (1964). The Immunological Basis of Acquired Cellular Resistance. *J. Exp. Med.*, **120**, 105.
30. MARTINEZ, C., SMITH, J. M., AUST, J. B. and GOOD, R. A. (1958). Acquired Tolerance to Skin Homografts in Mice of Different Strains. *Proc. Soc. Exp. Biol. and Med.*, **97**, 736.
31. McKENZIE, D., HANSEN, J. D. L. and BECKER, W. (1959). Herpes Simplex Virus Infection. *Arch. Dis. Childh.*, **34**, 250.
32. MILLER, J. F. A. P. (1961). Immunological Function of the Thymus. *Lancet*, **ii**, 748.
33. MILLER, J. F. A. P. (1962). The Effect of Neonatal Thymectomy on the Immunological Responsiveness of the Mouse. *Proc. Roy. Soc. B.*, **156**, 415.
34. MITUS, A., LEUCHTENBERGER, C., LEUCHTENBERGER, R. and ENDERS, J. F. (1960). Further Studies on Giant Cell Pneumonia. *Am. J. Dis. Child*, **100**, 615.
35. OLD, L. J., CLARKE, D. A., STOCKERT, E., PORTER, C. and ORENSKI, S. W. (1961). Protection against Mengo Virus by Agents Affecting the RES. *Fed. Proc.*, **20**, 265.
36. OWEN, R. D. (1945). Immunogenetic Consequences of Vascular Anastomoses Between Bovine Twins. *Science*, **102**, 400.
37. PINCUS, W. B. and FLICK, J. A. (1963). Hypersensitivity in the Pathogenesis of Vaccinia Virus Infections in Humans. *J. Pediat.*, **62**, 57.
38. PINCUS, W. B., FLICK, J. A. and INGALLS, T. H. (1963). The Response of Inbred Rabbits to Vaccinia Virus Infection. *J. Immunol.*, **91**, 58.

39. ROSEN, F. S., GITLIN, D. and JANEWAY, C. A. (1962). Alymphocytosis, Agammaglobulinaemia, Homografts and Delayed Hypersensitivity. *Lancet*, **ii**, 380.
40. ROWLEY, D. and TURNER, K. J. (1964). Increase in Macroglobulin Antibodies in Mouse and Pig Following Injections of Bacterial Lipopolysaccharides. *Immunology*, **7**, 394.
41. ROWLEY, D., TURNER, K. J. and JENKIN, C. R. (1964). The Basis of Immunity in Mouse Typhoid. *Aust. J. Exp. Biol. Med. Sci.*, **XLII**, 237.
42. SABIN, A. B. (1959). In Immunity and Virus Infections, ed. by V. A. Najjar. Chapman and Hall, London.
43. SAITO, K., NAKANO, M. and AKIYAMA, T. (1962). Passive Transfer of Immunity to Typhoid by Macrophages. *J. Bact.*, **84**, 500.
44. SMITH, R. T. (1960). Immunity in Infancy. *Ped. Clin. N. Amer.*, **7**, 269.
45. SMYTHE, P. M. (1959). The Significance of Bacteraemia of Kwashiorkor. *S. Afr. J. Med.*, **33**, 777.
46. STOKER, M. (1958). Mode of Intercellular Transfer of Herpes Virus. *Nature*, **182**, 1525.
47. TURNER, K. J., JENKIN, C. R. and ROWLEY, D. (1964). The Basis of Immunity to Mouse Typhoid. *Aust J. Exp. Biol. Med. Sci.*, **XLII**, 229.
48. WEISS, D. W., BONHAG, R. S. and PARKS, J. A. (1964). Studies in the Heterologous Immunogenicity of a Methanol-Insoluble Fraction of Attenuated Tubercle Bacilli. *J. Exp. Med.*, **119**, 53.

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THE BUSINESS MEETING

MINUTES OF THE FIFTY-NINTH ANNUAL GENERAL MEETING HELD AT ARTHUR'S SEAT HOTEL, SEA POINT, CAPE TOWN ON THURSDAY, 1ST OCTOBER, 1964

PRESENT

Dr. H. P. Steyn—President—(in the chair).
Prof. R. Clark, Vice President, the Secretary.
Dr. A. M. Diesel and the following members:

Abrams, L., Adelaar, T. F., Bischop, J. H. R., Brown, J. M. M., Brownlie, J. F., Buchalter, R., Coetzee, J. D., Coetzee, H. G. J., Colly, L. P., De la Rey, R., De Villiers, O. T., De Wet, G. J., Doré, J. L., Du Toit, I. F., Gilchrist, F. M. C., Goosen, P. J., Hempstead, F. D. J., Hofmeyr, C. F. B., Howell, C. J., Jansen, B. C., Jensen, C. A. S., Krog, F., Louw, D. J., Loveday, R. K., Malherbe, W. D., Marnewick, J. J., Masters, P. M. S., Meara, P. J., Philip, J. R., Pols, J. W., Scholtz, H. E., Schulz, K. C. A., Shone, D. K., Snijders, A. J., Solomon, R. A., Stephan, S. A. R., Sutton, G. D., Thorold, P., Trichard, C. V. J., van Heerden, W. W., van Maltitz, L., van Rensburg, G. F., van Wyk, J. A., van der Merwe, G. F., van der Walt, K., Wessels, C. C., Williamson, J. D.

APOLOGIES FOR ABSENCE

Alexander, R. A., Barnard, W. G., Coles, J. D., du Toit, P. J., de Boom, H. P., de Lange, M., de Villiers, W. J. B., Flight, C. H., Falconer, J., Jenkins, W. L., Irvine-Smith, J., Lambrechts, M. C., Mason, J. H., Matthew, A., Paine, R., Robinson, E., Tarr, A. F., van Rensburg, S. W. J.

IN MEMORIUM

The President, before opening the Congress requested members to stand in silence as a mark of respect and honour to the following members who had passed on during the year:

Major D. D. Morton.
Dr. P. R. Viljoen.
Dr. L. R. Morford.
Dr. R. J. Ortlepp.

and to the late Mr. J. Adams who had been a staunch friend of the Association and of the profession.

THE PRESIDENT WELCOMES MEMBERS

The President in welcoming members to the *Fifty-Ninth* Annual General Meeting requested them to repeat the customary procedure and practice in order to be able to conclude the proceedings dutifully and timeously.

He declared the meeting properly constituted and called for attention to the agenda. He reminded members that roneod copies had been posted to all members.

1. CONFIRMATION OF THE MINUTES OF THE FIFTY-EIGHTH ANNUAL GENERAL MEETING.

The President reminded members that the minutes of the 58th Annual General Meeting had been published in the December, 1963, issue of the Journal of the Association.

The adoption of the minutes was proposed and seconded and the President authorised to sign them as correct.

2. MATTERS ARISING FROM THE MINUTES OF THE 58TH ANNUAL GENERAL MEETING

(a) *Resolutions from the 57th Annual General Meeting*

The President explained that Resolution No. 3 of the 57th A.G.M., arranged for a standing Sub-Committee consisting of Dr. Tarr, Prof. Jansen and Dr. Lambrechts to inaugurate the

Diagnostic Laboratories and maintain them without seriously prejudicing the livelihood of the practitioner in the area.

(*b*) *Resolutions from the 58th Annual General Meeting*

A Committee comprising the President, Dr. J. D. Coles, Dr. M. C. Lambrechts, Dr. L. W. van den Heever, had been appointed to consider this resolution which requested Council to take steps to promote rural private practice.

The matter was considered in two parts viz:—

(i) Part-time service by the Department of Agricultural Technical Services.

(ii) Part-time service by the local authorities in rural areas for the veterinary inspection of meat and milk supplies.

The matter was still receiving the attention of Council. The Department of Agricultural Technical Services was due to be approached in the near future.

(*c*) *The Veterinary Amendment Act* (Act No. 49 of 1963)

Prof. Jansen, Voorsitter van die Veeartsraad gee inligting aan Kongres aangaande die verrigtinge wat gedurende die afgelope jaar deur die Veeartsraad onderneem is.

Geen ernstige gevalle van oortreding van die Wet of die Gedragskode het gedurende die jaar plaasgevind nie. Die voorsienings van Artikel 89 van die Wet op Geneeshere, Tandartse en Aptekers, wat betref die goedkeuring van geregistreerde veeartse word nou deur die Veeartsraad vir lede onderneem.

Handleiding van Professionele Gedrag word tans in hersiening geneem en lede behoort die nodige voorstelle te maak wanneer hulle daarvoor gevra word.

3. Presidents Report

In presenting his Annual Report on the activities of Council and of the Association, the President referred to the work of the following Sub-committees appointed by Council.

(*a*) *The State of all Veterinary Services in the Republic*

This Committee comprised Drs. J. D. Coles, J. H. R. Bisschop, L. W. van den Heever, L. R. Mansvelt, M. de Lange, S. W. J. van Rensburg, the President and the Secretary. The work of the Sub-committee had been interrupted due to a decision by the Department of Agricultural Technical Services not to permit its employees to take part.

This objection has now been withdrawn in the light of further disclosures by the President. The investigation will proceed after Congress matters have been attended to. The investigation constituted a considerable undertaking — the intention was firstly to break down every field of veterinary endeavour into as many self contained fields as possible and investigate each separately as regards needs, personnel etc. Questionnaires would be drawn up and after the initial investigation had been undertaken the Department of Agricultural Technical Services would be approached for any specific information required by the Association.

(*b*) *Sub-Committee on Co-operative Affairs*

A Commission of Enquiry into Co-operative Affairs in the Republic was now sitting. A memorandum had been submitted and verbal evidence would be presented on 28th October, 1964. The object of the Association was to draw attention to the apparent interference of Co-operative Societies in the activities of veterinary private practitioners.

(*c*) *Sub-committee on Veterinary Ethical Drugs*

This Sub-committee's work had virtually been taken over by that concerned with Co-operative affairs.

A Veterinary Foundation

The President then referred to the matter of a *Veterinary Foundation* and indicated that the Witwatersrand Branch had proposed a *nidus* to start the Fund. The object was to create a fund chiefly for *Veterinary Education*. A Sub-committee would be necessary to investigate the matter. Council would be the Trustees. This matter was still in the initial stages of analysis.

The Case of Dr. Craig

The President then referred to the case of Dr. Craig of Pretoria who had been charged by the Municipality for contravening a by-law which prohibited the keeping of more than 3 dogs in a residential area. Dr. Craig had won his case, a Sub-committee was going further into the matter and would report back to Council.

Activities of the President

The President referred to the Annual Meetings of Branches which he and the Secretary had attended during the year. He had also attended a meeting on Medical Education at Durban.

Congress Fees

The President enlarged on the fees usually required to defray Congress expenses. Most Associations took advantage of such occasions to improve their financial status and thereby develop their objects and endeavours. At the Durban Congress on Medical Education he was required to pay R10.00 merely as an entrance fee.

General Matters

The President directed attention to the following matters of general interest:

(i) The improved appearance of the Journal.

(ii) The inclination to form splinter Branches which could easily embarrass or even destroy established Branches.
Such a case was presently being experienced where a Highveld Branch was contemplated merely to coincide with a Departmental Veterinary Region. The S.A.V.M.A. developed its own Branches and these did not have to coincide with regions created by the State.
Members had a duty to the Association. They could decide to attend Branch Meetings at their own expense if the State arrangement prevented their attendance at certain Branch Meetings of the Association.

Continuation as President

The President referred to the fact that he was now entering his seventh year of office and felt that he should now give way to the next candidate. He would only be prepared to continue if there was a very specific request for him to do so. He had enjoyed the wholehearted and loyal support of members during his period of office and he would not remain until he became part of the furniture. He considered that he had been able to exercise some influence in a number of quarters.

He left these thoughts with members and would like to know how they felt well in advance. He had sacrificed a great deal during this time but would be prepared to continue even though the demands on his time and finances were considerable.

4. Membership

Deaths

The President referred to the deaths of Major D. D. Morton, Dr. R. J. Ortlepp, Dr. P. R. Viljoen, Dr. L. R. Morford and also of Mr. J. J. Adams who had been a very staunch friend of the profession when he was under Secretary for Agriculture.

Resignations

Dr. F. W. Langbridge.

New Members

The following thirty-six members had applied for membership and were recommended by Council.

Their applications were acceptable to Council and were endorsed by the Meeting:

Anema, S. G.	Nilsen, C. T.
Austin, J. C.	Pullinger, P. J.
Beverley, G. H.	Redelinghuys, E. L.
Bilbrough, R. B.	Screiber, U.
Brightman, M. P.	Sykes, R. D.
Crewe, Georgina.	Sutherland, R. J.
Deacon, C.	Thomson, M.
Du Preez, J. H.	Veary, C. M.
Downes, S. J. T.	Von Ludwiger, F. W. J.
Fleming, J. M. P.	Van der Merwe, C. P.

Freeman, F. M.
Gerdes, G. H.
Gillespie, A. E.
Harte, C. P.
Immelman, A.
Jordaan, A. J.
Lombard, S. S.
McLauchlan, I.
Van der Vyver, J. W.
Vogelsang, G. H.
Visser, C. J.
Weaver, D. B.
Wellington, A. C.
Wilson, R. A.
Young, E.
Zumpt, I. F.

Cadet membership was approved in respect of the following students:

H. R. Cable.
I. Firth.
T. O. Gurnell.
D. T. Longland.
A. M. Simon.
O. T. van Niekerk.
P. Wisselwenste.

Associate Members

Associate membership was unanimously conferred by the meeting on the following two persons, on the recommendation of Council.

(i) Mr. William Henry Gerneke, M.Sc.
(ii) Dr. Francis Mary Combes Gilchrist, B.Sc., Dip. Bact., Ph.D.

Details of the proposals appear elsewhere in this issue.

It was agreed that Scrolls would be prepared for presentation to these two Associate members at a forthcoming meeting of Council.

5. Consideration of Reports

(i) *Financial Statement and Auditor's Report*

The Secretary explained certain items in the Balance Sheet and Income and Expenditure account for the year ending 31st March, 1964, and in particular a suggestion by the Auditors that the sources of income of the Benevolent Fund be improved.

The adoption of the Balance Sheet and Financial Statement was unanimously approved by the meeting.

Dr. Sutton thought that the allocation to the Benevolent Fund of R1.00 per subscription should be increased to R2.00 per member subscription.

Prof. S. van Heerden spoke at length on the need to improve finances of the Benevolent Fund and suggested the formation of a Congress Fund.

He proposed the following *two resolutions*. They were seconded by Dr. L. W. van den Heever and agreed to unanimously.

Resolution No. 1

This Fifty-ninth Annual General meeting of the South African Veterinary Medical Association requests Council to consider ways and means of swelling the resources of the Benevolent Fund such as further annual contributions from members, special Branch functions etc.

Resolution No. 2

This Fifty-ninth Annual General Meeting of the South African Veterinary Medical Association requests Council to consider the creation of a Congress Fund from surplus monies derived from Congress income; such a Fund to be utilized for financing further Congresses in general and for covering the costs of visiting specialists and guests lectures in particular.

(ii) *Reports of Standing Committees*

The Secretary reported briefly on the activities of the Finance Committee, the Editorial Committee, the General Purposes Committee, and the Disciplinary Committee, all of which reflected the usual matters attended to during the year. The Secretary reminded the meeting that the activities of the Sub-committees of Council were reported on by the President in his Annual Report on the work of the Association and its Executive Council.

6. World Veterinary Association

The President advised Council that the Membership fee to the World Veterinary Association was presently 1/– (one shilling) per member but that it would be increased to 1½ shillings per member from 1965. The Association had just paid its subscription of R53.10 for 1963/64 in respect of 528 members.

The next International Veterinary Congress was to be held in Paris from 17–22 July, 1967. A bicentenary celebration would be held in respect of the Alfort Veterinary Institute, near Paris.

7. Congress Matters

(i) *Revision of Congress Fees*

After a brief discussion, *Prof. R. Clark* proposed that the matter be left to the discretion of Council in the light of the general financial position, and in particular the donation of any surplusses to the Benevolent Fund.

The President felt that the disposal of surplusses should be left to the discretion of Council.

Dr. van den Heever considered that in principle there should be no budgeting for surplusses but should there be a surplus this would be quite incidental.

(ii) *Publication of Congress Papers*

Congress agreed that the method adopted for the present Congress appeared to be the most suitable, the papers being published in the post-Congress issue of the Journal and summaries of papers circulated in advance of Congress.

The President felt that more time should be allocated to the presentation of papers and to the discussion.

Dr. Muller suggested the utilization of galley proofs of articles as a means of notifying members of the contents of papers.

Prof. Clark pointed out that this system had been tried previously and found to have no merit.

Congress agreed that it was inadvisable to publish papers in one issue and the discussions in subsequent issues.

The President felt that contributors should be advised in advance how much time each was allowed for the delivery of his paper.

(iii) *Proposed Biennial Scientific Congress for the S.A.V.M.A.*

Professor B. C. Jansen spoke on the subject and analysed the pros and cons. After a brief discussion the meeting decided to continue with the Annual Congresses as at present.

(iv) *Medical Exhibitors Association*

The Secretary intimated that the Medical Exhibitors Association had been disbanded, but that the firms concerned would continue to support the Congress Exhibitions.

The meeting expressed its appreciation to the Exhibitors and in particular to Mr. Veitch and Mr. Stabler of Chas. F. Thackray.

(v) *Venue of future Congresses*

Prof. Clark considered that there should be one or two Congresses at Onderstepoort and then one away. This was agreed to.

Prof. Jansen invited the Association to hold its next Congress at Onderstepoort.

8. Administration of the Association

(i) *Private Practitioners to take a greater interest in the affairs of the Association*

This was agreed to.

(ii) *Disbursements from the Benevolent Fund*

There were no further comments.

(iii) *Proposed Veterinary Foundation*

Prof. K. van der Walt verwys na die beleid in belang van die Fakulteits Fonds en stel die moontlikheid dat die twee fondse mekaar kan help.

Die President onderskei die twee fondse en meen die Fakulteits Fonds is huishoudelik, terwyl die ander as 'n Nasionale Fonds beoog word.

(iv) *Committees of Enquiry*

This matter was referred to by the President in his report to the Annual General Meeting.

Dr. Thorold thought that veterinary employment could be arranged by the Military, the Police and the Railways.

The President informed the meeting about the endeavours made by Council to revive the Veterinary Corps and that a meeting was soon to take place with the Commandant General.

He agreed that avenues of employment should be explored in the Police and Railways.

(v) *Record of a list of Veterinarians*

Prof. Jansen referred to this matter when speaking on item 2 (*c*) — registration of Veterinarians under the Veterinary Act and the titles which were to be included in such registration.

The Association would undertake the keeping of records of achievement of its members and a curriculum vita for each member.

(vi) *Increase in membership fees for those residing overseas*

The President explained that Council considered that members residing outside the Republic would have to pay a membership fee of R8.00 instead of R6.30 as from next year. — Agreed.

(vii) *Veterinary Hospitals in Built-up areas*

This matter had already been dealt with by the President when referring to the case of the Pretoria Municipality vs Dr. Craig.

(viii) *Work of the Associations Secretarial Staff*

The President explained that the typiste to the Secretary had now been permitted to work 5 days a week and was working additional hours to make up the necessary time.

(ix) *The Publication of the Journal*

No matters were raised in this regard.

9. Veterinary Board Matters

(*a*) *Preparation of a list of Veterinarians*

Prof. Jansen indicated that the publication of the list of registered veterinarians would take place annually. He dealt with other items concerned with the Veterinary Board under Item 2 (*c*). Authorization under Section 89 of the Medical Dental and Pharmacy Act would be negotiated by the Registrar of Veterinarians with the Personnel Clerk in the Department of Health.

(*b*) *Items of interest to the Association*

Prof. Jansen intimated that usual matters dealt with by the Board would be passed on to the Association for its information and if need by publication.

Details of complaints brought before the Board would be briefly summarised in such communications. Unusual cases, cases held in camera and confidential matters would remain the exclusive property of the Board.

10. Matters Concerning the Council of the Association

(*a*) *Mannekrag Navorsing*

The President asked Prof. Jansen to explain the request by the Department of Education, Arts and Science that investigation into manpower be conducted.

Prof. Jansen briefly referred to the matter and indicated that Council was dealing with the matter.

11. Notification of Election of Office Bearers 1964

The Secretary at the request of the President informed the meeting that the following members had been elected as office bearers for the ensuing year.

President: Dr. H. P. Steyn (unopposed).

Vice-President: Prof. R. Clark (three candidates).

Members of Council:
Dr. J. D. Coles
Dr. J. L. Doré.
Dr. M. C. Lambrechts.
Dr. A. F. Tarr.

There were seven candidates for the four vacancies on Council. Dr. J. L. Doré replaced Dr. M. de Lange.

11. Other Matters of Importance

None were raised.

13. General

Prof. O. T. de Villiers complimented the President on the work he had done for the Association and considered he should not think of resigning.

He had accomplished a monumental task and should follow it up. He would like to see him as President for a few years longer. He thanked President for the adroit manner in which he had conducted the present meeting.

He complimented Mr. de Villiers Loubser on his excellent opening address.

Dr. J. F. Brownlie raised the matter of interference by animal welfare societies with private practitioners in their advertisements to conduct spaying operations and X-ray examinations.

He would supply cuttings of the advertisements to the Secretary.

Prof. Jansen referred to the provisions of the Veterinary Amendment Act which allow prosecution in the cases mentioned by Dr. Brownlie.

Prof. B. C. Jansen meld dat as Dekaan hy die Vergadering graag in kennis wil stel insake sekere beurse wat nou beskikbaar is deur die Universiteit van Pretoria vir spesiale studie op die gebied van slagvee produksie. Hierdie beurse is deur die Lewendehawe Afslaers aangebied en die Universiteit is angstig om applikante so spoedig moontlik te kry. Verdere besonderhede is van Prof. Jansen of van die Universiteit van Pretoria of van die Sekretaris van die S.A.V.M.V. verkrygbaar.

Dr. K. Shone advised that a group was being formed to incorporate the interests of Veterinarians in Commerce. Anyone interested should contact either Dr. Purchase of Cooper & Nephews or himself at A. S. Ruffel, Johannesburg.

Dr. R. A. Solomon intimated that he had been approached to ascertain what the position was regarding:

(*a*) The payment of a bonus on milk from T.B. free cows — in other words an increase in price for milk from T.B. free herds.

(*b*) The role of the private practitioner in the T.B. eradication scheme.

He had been informed that a Committee on Bovine Tuberculosis had sat and produced recommendations some ten years ago.

Dr. G. F. van der Merwe advised the meeting that Dr. Mansvelt was busy arranging details of the forthcoming T.B. control scheme and that the profession could expect these details quite soon. Matters in this connection were now moving rather fast.

The President in reply to a question informed the meeting that the Secretary was busy preparing a short article on the keeping of records under the laws relating to the custody of Poisons, Habit Forming Drugs and Potentially Harmful Drugs. This would be published in the Journal.

Prof. S. van Heerden drew the attention of the availability of a Minikopter and suggested that it should be demonstrated at the Congress next year.

Dr. J. F. Brownlie advised that the formation of a Practitioners Group was now being considered and requested all interested to get in touch with him.

A Special Function

The President informed the meeting that he was now about to perform a very special function. The members present had been informed that the Secretary had become an "*Oupa*". He heartily congratulated the Secretary on his new and elevated status and asked him to accept a special gift from the members of the 59th A.G.M. (applause).

At the request of the President, the Secretary undid the wrapping and revealed a very attractive miniature "*Pottie*" and "*Dummie*", smartly decorated with pink ribbon — (further applause).

In thanking the members (through the President) Dr. Diesel expressed his sincere appreciated for the very thoughtful gesture and for the great honour paid to him by the members in attendance at the meeting — (still further applause).

14. Preparation and Arrangements for the 1965 Congress

The meeting adopted a vote of appreciation and thanks to the Congress Committee and requested that the Committee undertake the planning of the 1965 Congress and continue the good work.

15. Resolutions

The following two resolutions were proposed by Professor S. van Heerden, seconded by Dr. L. W. van den Heever and adopted unanimously.

Resolution 1

This Fifty-ninth Annual General Meeting of the South African Veterinary Medical Association requests Council to consider ways and means of swelling the resources of the Benevolent Fund such as further annual contributions from members, special Branch functions etc.

Resolution No. 2

The Fifty-ninth Annual General Meeting of the South African Veterinary Medical Association requests Council to consider the creation of a Congress Fund from surplus monies derived from Congress income; such a Fund to be utilized for financing further Congresses in general and for covering the costs of visiting specialists and guests lectures in particular.

15. Adjournment

The President received a hearty vote of thanks and the meeting was adjourned until next year.

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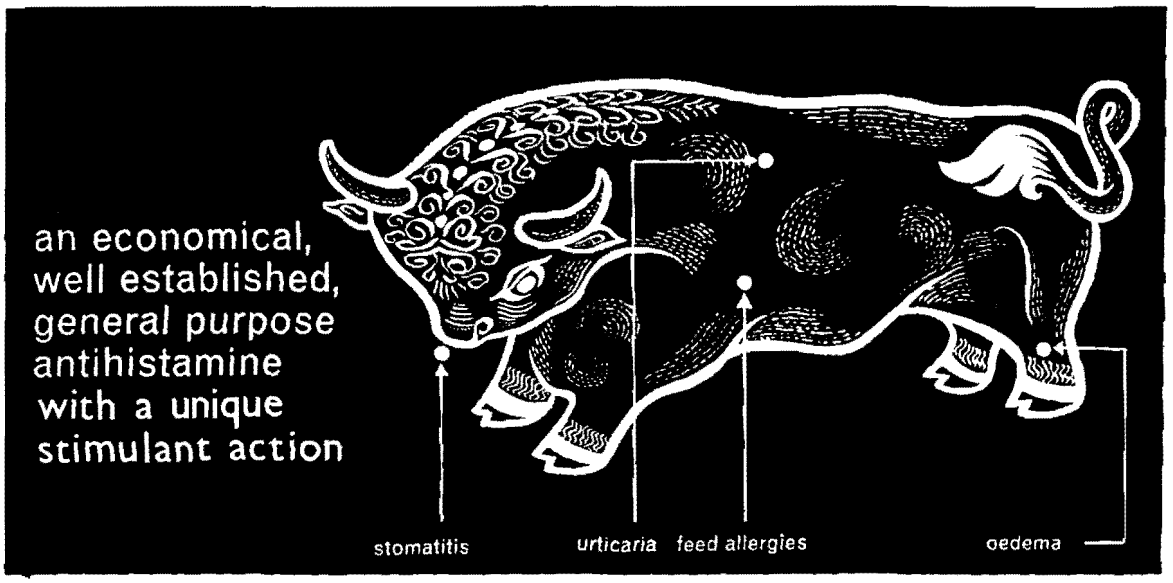

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ASSOCIATE MEMBERSHIP IS CONFERRED ON MR. WILLIAM HENRY GERNEKE, M.SC. AND ON DR. FRANCES MARY COMBES GILCHRIST, B.SC. DIP BACT. PH.D.

The bestowal of Associate Membership on Mr. W. H. Gerneke and Dr. F. M. C. Gilchrist was unanimously approved at the 59th Annual General Meeting of the Association.

The testimonies which were presented in support of the proposals received the recommendation of Council and are reproduced below.

WILLIAM HENRY GERNEKE, M.SC.

Professor Clark proposed and Professor Jansen seconded a motion to confer an Honorary Associate membership on Mr. William Henry Gerneke M.Sc.

W. H. GERNEKE

Professor Clark presented the following testimony for the information of Council.

Mr. Gerneke was born in Johannesburg in 1924. After studying at the University College of the Orange Free State (Grey's), he obtained the degree of B.Sc., with distinction in chemistry and zoology, in 1944. He was appointed to the section of pathology at Onderstepoort in 1946 and promoted to Senior Professional Officer in 1959. In 1957 he was appointed as Part-time Lecturer in histology in the Faculty of Veterinary Science and was promoted to Senior Lecturer (full-time) in 1963.

In September 1963, Mr. Gerneke was awarded the degree of M.Sc. (cum laude) by the University of Pretoria, his thesis being on the embryological development of the pharangeal region of sheep.

The proposed award of an Honorary Associate Membership to Mr. Gerneke is in recognition of his outstanding contributions to veterinary science over the past eighteen years as a teacher, research worker and specialist consultant. He has always shown the greatest collaboration with his veterinary colleagues and his election into the Association will only formally and fittingly establish the fact that he has long been one of us.

FRANCES MARY COMBES GILCHRIST, B.SC., DIP. BACT., PH.D.

Professor B. C. Jansen proposed and Prof. R. Clark and Dr. M. de Lange seconded a motion to confer an Honorary Associate membership on Dr. Frances Mary Combes Gilchrist, B.Sc., Dip. Bact., Ph.D., and provided the following testimony:

Dr. Gilchrist was born in 1915 at Worcester, Cape Province. She was educated at Saint Cyprian's Dioscesan School for Girls in Cape Town. From there she proceeded on a matriculation scholarship to the University of Cape Town where she obtained the degree of Bachelor

of Science in zoology, chemistry, and general bacteriology. She also obtained a Diploma in medical bacteriology. In 1938 she joined the staff of the University and held the posts of junior lecturer and research assistant to Professor W. C. Campbell in the Department of Bacteriology at the Medical School, and of tutor to the women's residence, Fuller Hall. In 1948 she worked for the Agricultural Research Council in Britain as research assistant to Professor S. R. Elsden, head of the Council's Unit for Microbiology at the University of Sheffield, where she obtained the degree of Doctor of Philosophy. The title of her thesis was "Studies in Hydrogen Production by Bacteria". She returned to South Africa in 1951 to take up a Sewer Corrosion Fellowship with the Council of Scientific and Industrial Research at Pretoria, and worked in the Biochemistry Section of the National Chemical Research Laboratory under Dr. H. M. Schwartz. In 1953 she became a Senior Research Fellow of the Stock Diseases Research Fund of the Wool Board, the Dairy Board and the Meat Board, and started the Unit for Research on Digestion and Metabolism of Ruminants at Onderstepoort.

Dr. F. M. C. GILCHRIST

The object of the work of the Unit is to increase the fundamental knowledge of ruminant digestion and metabolism to provide a basis for planning and interpreting practical experiments.

The research work conducted by Dr. Gilchrist from the time of joining Onderstepoort is of vital importance to veterinary science in this country. Her association with veterinarians has always been most cordial and many positive ideas were derived from discussions with her.

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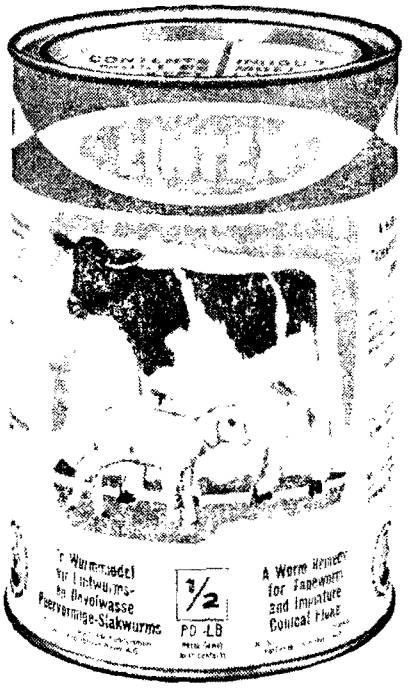

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THE SOCIAL FUNCTIONS AND ENTERTAINMENTS

The social functions arranged for the ladies were most successful and very much appreciated. The ladies were delighted with the visit to the Administrator's home and the cordial welcome they received from Mrs. Malan.

They were most grateful to Mrs. Faull for her part in the excursion.

The visit to Kirstenbosch was a special treat.

The Mayoral Cocktail was indeed a most pleasant function, where everyone was sorry when the time came to go home. The President thanked the Mayor and Mrs. Peters very warmly for their exceptional attention to the members and their wives who attended Congress.

The days touring of the Western Province by Railway bus was thoroughly enjoyable. It was most instructive to see the Western Province farmer at home and to see his methods of applied agriculture.

The carpets of wild Cape flora and their manner of protection by farmers was something everyone will remember for a long time.

Mr. Smit, M.P. and Dr. Laurie Muller are heatily congratulated on arranging such a treat for us. The braaivleis-lunch at Mrs. Basson's farm had to be experienced to be believed. We sincerely thank the Malmesbury Farmers' Association for this special treat.

Members are most grateful to the Cape Western Branch for the thought they put into this tour.

The "get-together" on 27th September was a very pleasant function and Dr. Thomson is particularly thanked for arranging the projection of the two films "To catch a Rhino" and "Lake Wilderness".

The Conference lecture by Prof. Kipps was something that had to be listened to to be appreciated. The written version appears in this issue; the tape recording is preserved at the secretary's office.

The Buffet-Supper was well attended; very well served by the Management of the Arthur's Seat Hotel. Everyone thoroughly enjoyed this very successful function.

The sport functions were not well patronised, but the entertainment by Dr. and Mrs. Faull at their home as an occasion for the presentation of the trophies was most enjoyable and one that will long be remembered by all.

THOSE WHO ATTENDED CONGRESS

A List of members and visitors who registered and attended the Opening Ceremony is given below:

MEMBERS

Abrams, L., Adelaar, T. F., Albertyn, A., Alderman, W. S., Anema, S. G., Azzie, M. A. J., Bacher, I., Baker, B., Basson, C. H., Belonje, P. C., Bisschop, J. H. R., Bothma, R., Brown, J. M. M., Brownlie, J. F., Buchalter, R., Cavanagh, F. E., Coetzee, C. J., Coetzee, J. D., Coetzee, H. G. J., Colly, P. L., Clark, R., De la Rey, R., De Kock, G., De Villiers, O. T., De Wet, G. J., Dickson, J. L., Diesel, A. M., Doré, J. L., Du Plessis, W. A. J., Ebedes, H., Erasmus, C. J., Faull, G. L., Fourie, J. M., Freeman, F. M., Gilchrist, F. M. C., Goosen, P. J., Greathead, M. M., Groenewald, J. M., Hellig, H., Hempstead, F. D. J., Hofmeyr, C. F. B., Horwitz, B. M., Howell, C. J., Jansen, B. C., Jensen, C. A. S., Krog, F., Kronsbein, G., Kriel, J. P., Le Riche, E. O., Le Roux, P. H., Louw, D. J., Louw, P. L., Loveday, R. K., Malan, W. du Toit, Malherbe, W. D., Maree, J., Marlow, C. H. B., Marnewick, J. J., Masters, P. M. S., Meara, P. J., Meredith, C. D., Morkel, D., McHardy, W. M., Muller, G. L., Naude, L. F., Nelson, E. C., O'Brien, S. V., Philip, J. R., Pols, J. W., Pretorius, J. L., Reinecke, R. K., Rous, R. C., Ryksen, W. J., Schneider, P. J., Scholtz, H. E., Schulz, K. C. A., Shone, D. K., Smit, J. D., Smit, J. P., Snijders, A. J., Smuts, T., Snyman, P. S., Solomon, R. A., Solomon, S., Starke, N. C., Stephan, S. A. R., Steyn, H. P., Sutton, G. D., Tabic, D., Terblanche, H. J. J., Theron, H. C., Thomson, J. K., Thornton, D. J., Thorold, P., Trichard, C. J. V., Turner, S. G., Van Blerk, J. A., Van Heerden, K. M., Van Heerden, S., Van Heerden, W. W., Von Ludwiger, F. W. J., Van Maltitz, L., Van Wyk, J. A., Van Rensburg, G. F., Van den Heever, L. W., Van der Merwe, G. F., Van der Walt, K., Vincent, Z. D., Walters, D. M., Wege, D., Wege, R., Wessels, C. C., Williamson, J. D., Wheeler, W. J., Winterbach, P. B.

VISITORS

Bride, H. D., Burger, P. J., Frey, M., Dr. and Mrs., Lochner, J. L., Marais, V., McCully, Maj. R. M., Ritchie, J., Thornton, H., Scott, D. B., Slabber, M. J., van der Walt, J. P.

THE FIRMS WHICH EXHIBITED AT THE CONGRESS

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The President Thanks Everyone

The President thanked everyone and used the following words:

Well Gentlemen we have come to the end of another very memorable meeting and we have certain duties to perform by way of closing the meeting.

There is a long list of people who must be thanked for contributing to the success of this meeting.

First of all I think I should thank all *contributor's and delegates*, people who have delivered papers and delegates who came from afar to swell the numbers and make it very successful.

We have representatives here from Bechuanaland, South West Africa, Southern Rhodesia and as you know even from Ethiopia and the United States.

It is quite an effort very often to come and attend, being such a long way from your home, but I think the surroundings and the general atmosphere of this meeting have been adequate compensation for those people who undertook to come and attend. Then the contributors particularly must be thanked *for the very high standard they maintained in the papers they presented.*

Now we have a number of people who have performed very onerous duties. Firstly I think I must thanked the *Cape Western Branch of the S.A.V.M.A.* for undertaking the organization and administration of the meeting. The people particularly involved are the Chairman, Dr. Albertyn, the Secretary, Dr. Masters, and the Vice Chairman, Dr. Muller, and the Committee.

Now we want to thank the *Publicity Bureau* for their assistance, *Sanlam*, the *Shell Company*, very particularly Mr. Matthews, and Mrs. Joyce Moll, sitting in the back of the hall. *Then for the films which were presented, Dr. Brownlie and Dr. Singleton*, and the *Department of Nature Conservation through our colleague Pat Thomson.*

The Mayor, of course we have thanked and the *Administrator's wife, Mrs. Malan*, for I believe the wonderful party the ladies had at the Administrator's residence.

Then of course I have already thanked the people whom we visited on our tour and I repeat that we are particularly indebted to *Mr. Hennie Smit and Dr. Muller* for organizing the wonderful tour and to the various people who contributed to make the success of it en route.

You know who they all were, the *Malmesbury Farmer's Association, Mr. de Villiers Loubser, the Duckett Bros* and *Fanie Basson. Rothman's* for keeping us supplied in this poisonous drug which I use myself, the cigarette, and then of course we want to thank the *Press* and the *S.A.B.C.* for the publicity which they have given us.

I wish to thank *Dr. Horwitz and the various sporting clubs* for the facilities that they are going to give us this afternoon in anticipation for enjoying some sport.

There are the *Exhibitors*, we thank them for their assistance and aid. I haven't yet seen the usual cheque otherwise my thanks would have been a lot warmer.

Then there are of course *the people who have been on duty at the entrance hall all the time. This man Sutton*, is an amazing fellow, he came down here originally on his own steam, and we said this is not quite good enough we will at least pay your fare, and he sits in that cubicle there and he sees that all you fellows do your duty by the treasury and he gets a tremendous kick out of it — that's what's so funny to me.

Now to come to the end of this pleasant task, *Miss Theron* and *Miss Melville*, who are two lovely girls who have been aiding us and assisting our friend and Mr. Richard Horwitz. Then there is of course *Prof. Kipps* to whom you expressed your thanks in no uncertain manner by the ovation you gave him after the Conference Lecture.

Now there is the *projectionist* and the *sub-projectionist*, there is one there now sitting in the back of the hall, and we must not forget our dear friend *Oupa Diesel* — ons moet hom insluit.

Then in conclusion I want to thank the whole of the *Council of S.A.V.M.A.* and *all my colleaugues* for the wonderful support, the staunch and unstinting support which they give me in this rather onerous task.

Now as a final step I have great pleasure in asking *Mrs. Joyce Moll* to come up and receive something from me and I want something from her in exchange!! I don't know how to handle this but this comes from all the boys you see here.

Then finally we must thank again the management of this hotel for the wonderful assistance they have given us and the wonderful table they have given us and the wonderful party they gave us last night, and that Gentlemen concludes our *verrigtinge* — Dankie.

THE CONTRIBUTIONS ON SHEEP AND SHEEP DISEASES

INTRODUCTION BY DR. THOROLD

My purpose in suggesting a symposium on sheep diseases was to focus attention on an industry which accounts for a big slice of our national income, second only in importance to gold; an industry which to me and I think to all of us, must be maintained and improved if it is to compete with the ever increasing variety and versatility of artificial fibres. Most of you have seen this morning's Times in which mention is made of a nylon spinning plant now producing clothing materials — this constitutes a danger to the wool industry.

During 1958–1962 I spent a lot of time on Blowfly control and other sheep ailments and visited practically all the sheep growing areas in South Africa. It was most enlightening, particularly the management side. The merino in Natal and E. Griqualand is small; large and well grown out in the Cape; in the Transkei they are really small and stunted due to poverty and parasites. It may interest you to know that the average income per year per sheep for wool in the Transkei is 25c. From what I saw in the Western Cape wheat and fruit growing areas, sheep are a side line and as such get second grade treatment, whereas proper attention would increase production and profit. By arranging a routine dosing programme on a particular farm in the Eastern Cape, wool production was increased by a half pound per sheep.

I would reiterate what our President has said— we need more Veterinarians, but particularly in the sheep growing areas. We need men with a sound knowledge of husbandry and management who will bring the farmer up to date and keep him there.

This most important branch of our livestock industry is highly specialized today and in my opinion the faculty at present is simply unable to cater for this. On the research side we have made little or no headway in the last 20 years against the blowfly. If it were not for the dip and insecticide people keeping one jump ahead of the resistant blowfly, we would be in a very sad state indeed. We have heard mention of erosion diseases — how many of us as professional men have given thought to Jackals and their control, and yet they are responsible annually for heavy losses directly, and indirectly when sheep have to be shedded or kraaled at night resulting in the spread of jaagsiekte.

Finally I consider the methods of handling sheep in this country to be archaic, particularly the continual man-handling and turning up of sheep for inoculations, jetting etc. This is where we want men with a sound knowledge of sheep husbandry at the Agricultural Colleges and Schools.

I hope the papers will be of great interest to you and will stimulate a lot of thought about this particular industry.

Thank you Mr. President.

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LUNGWORM IN DOGS IN THE WESTERN CAPE PROVINCE

J. E. DORRINGTON

Private Practitioner, 4 Kort Street, Bellville, C.P.

SUMMARY

The incidence, breeds affected, symptoms, diagnosis and treatment of Lungworm due to *Filaroides osleri* in dogs in the Western Province is described.

HISTORY

Verminous bronchitis as it was originally called, was first described by Osler[1] in 1877 in Montreal, Canada. He gave a very complete and interesting account of this condition but it was Cobbold in 1879 who identified the nematode worm as belonging to the genus *Filaroides*. Since this original discovery amongst foxhounds, lungworm has been found in many other breeds of dogs in all parts of the world namely poodles, greyhounds, dachshunds, cairn terriers, spaniels, beagles, Irish setters, bull mastiffs, boxers, in countries such as Canada, U.S.A., Europe, United Kingdom, India, New Zealand and South Africa.

Prior to 1958 *F. osleri* had for all practical purposes been associated only with the bull mastiff breed in South Africa[2]. In 1958 however, I performed a post mortem on an adult boxer dog that had suddenly died of asphyxia and discovered extensive infestation. The dam of this pedigreed boxer had been seen by me many times prior to 1958 for an unexplained bronchotracheitis. She in turn originated from another breeder in the Western Province in whose kennel I subsequently established infection, although this breeder refused to acknowledge having any infected animals. I do know, however, that she had purchased dogs from the Transvaal and can only assume that some time earlier a boxer and bull mastiff had been reared in close association by a breeder in the Transvaal.

Since 1958 I have positively diagnosed lungworm infection in well over 50 boxer dogs (pedigreed and/or crossbreeds); a few pugs and bull mastiffs, while my colleagues here in Cape Town have discovered it in a French poodle and possibly in alsations as well. It is reasonable therefore to assume, judging by local as well as overseas reports, that *F. osleri* is not confined to the brachycephalic breeds of dogs and that it occurs sporadically throughout the Republic of South Africa.

In other countries it has been seen in almost all breeds and I would like here to sound a timely warning to would-be importers of breeding stock to make certain they eminate from studs free of lungworm. Once established in a breeding stud, it can easily become a ruinous problem since no normal breeding can take place until the infestation has been cleared up. In all cases of infected boxers I was easily able to trace the source of infection back to known infected stud breeders either here in the Western Province or elsewhere in the Republic.

SYMPTOMS AND PATHOGENESIS

A sporadic yet persistent tracheo-bronchitic cough mainly characterised by forced expiration, remains the most obvious symptom seen. The cough remains dry and must not be confused with a bronchitic one. Attempts at vomition are sometimes seen after a bad spell of coughing. While resting the average case will show normal respiration; this only becomes abnormal in advanced cases where forced expiration with a ruptured mediastinum is seen. Emaciation and death occurs in many young animals.

The age at which clinical symptoms are first seen may vary from as early as 2½ months to dogs well over 2 years.

In Table 1 the different classes of animals that have been encountered in a known infected breeding stud, are given.

(under 6 months) I would advise microscopic examination of some tracheal mucous simultaneously since in the very early stages, the not yet fully developed papillomata may not easily be visible to the inexperienced eye. In such early cases the eggs will be found long before gross lesions are visible.

TABLE 1.—THE CLASSES OF DOGS TO BE FOUND IN STUDS INFECTED WITH *F. osleri*.

Type of Dog	*Condition*	*Cough*	*Breathing*	*Prognosis*
non-infected	good	absent	normal	excellent
infected	good	absent	normal	good
infected	good	sporadic	normal	good
infected	emaciated	persistant	normal	adult — fair puppies — hopeless
infected	emaciated	absent	laboured, forced expiration	hopeless

From this table one can clearly see that one cannot necessarily clinically diagnose a positive dog. The futility of attempts at eradication on clinical grounds alone, is therefore quite obvious.

DIAGNOSIS

A tentative diagnosis of lungworm can be made on clinical grounds only in cases where the origin of the dog is well known to the veterinarian. I have on several occasions had socalled positive cases referred to me by my colleagues who have arrived at their diagnosis on the clinical cough alone and have had the embarrassing situation to prove my colleagues in error after bronchoscopy. Remember not every coughing boxer, bull mastiff or some other breed must necessarily have lungworm.

Other rapid but unreliable methods of diagnosis are microscopic examination of laryngeal swabs and faeces for the typical embryoanted eggs, or lateral X-ray of the chest where the nodules are visible in well developed cases at the bifurcation of the trachea.

However, a definite diagnosis is only possible when using an illuminated bronchoscope intratracheally — the reddish nodules are clearly visible in the region of the bifurcation. Where examinations are made on very young dogs

DIFFERENTIAL DIAGNOSIS

Confusing symptoms may arise in cases of tonsillitis, bronchitis, pneumonia and cardiac failure in aged dogs.

LIFE CYCLE

While the life cycle of *F. osleri* remains as yet unsolved, those of closely related species occurring in badgers, polecats and mink are indeed known and may serve as an indication of what that of *F. osleri* may be like. In these animals the intermediate hosts appear to be slugs (*Deoceras falciformes*) and land snails (*Fricticola hispida*)[3].

However from my observations and investigations to date, it would appear to me that this mode of transmission in *F. osleri* is most unlikely. Numerous arthropods found in and around infected studs have been examined without any success.

I have found that where known infected bitches are discharging eggs at the time of nursing their litter, it is highly likely that the whole litter will become infected even though some may later never develop clinical symptoms. On the other hand where known infected bitches happen not to discharge eggs at this particular

time, I have found that such a bitch can rear a litter free from lungworm. I have also found that by the time 6 week old pups are weaned from their infected mothers they have already picked up the infection. A reassuring fact, however for breeders who are unfortunately afflicted with this disease in their stud is the fact that if they remove the puppies at the time of birth and have foster mothers rear the puppies, these pups will then be free of this infection — i.e. there is no intra-uterine transmission. However, I have experienced practical difficulties here in attempting to have two bitches whelping on or about the same day.

TREATMENT

Footnotes:

Dictycide (I.C.I.) (Cyanacethydrazide).

Helmox (I.C.I.) (Cyanacethydrazide and phenothiazine).

Franocide (Burroughs Wellcome) (diethylcarbamazine).

Nankor (Nicholas) (Ronnel — organic Phosphate).

Triostam (Burroughs Wellcome) (Sodium Antimonygluconate).

Caparsolate (Abbotts) (Thiacetarsamide).

TABLE 2.—DRUGS USED AND RESULTS OBTAINED AS A THERAPY FOR *F. osleri* IN DOGS.

Drug	*Dose*	*Duration*	*Clinical effect*	*Curative effect*
Dictycide	1 gr/5 kg i.m.	3 consecutive days	Reduces cough	None
Helmox	5 gr/5 kg per os	3 consecutive days	Reduces cough	None
Franocide	1 ml/5 kg i.m.	3 or more days	Variable	None
Nankor	2 gm/5 kg per os	6 times on alternate days	None	None
Triostam	1 ml/20 kg i.v.	6-12 days	Variable	Unreliable
Caparsolate	1 ml/5 kg i.v.	3-21 days	Good	Unreliable

Conversely I have found adult fully grown dogs most resistant to a natural infection; there is therefore very little danger of adult dogs picking up the infection at show gatherings or boarding kennels even if kenneled with known positive dogs for a considerable time. This would then also be another important fact for breeders to bear in mind when showing their dogs.

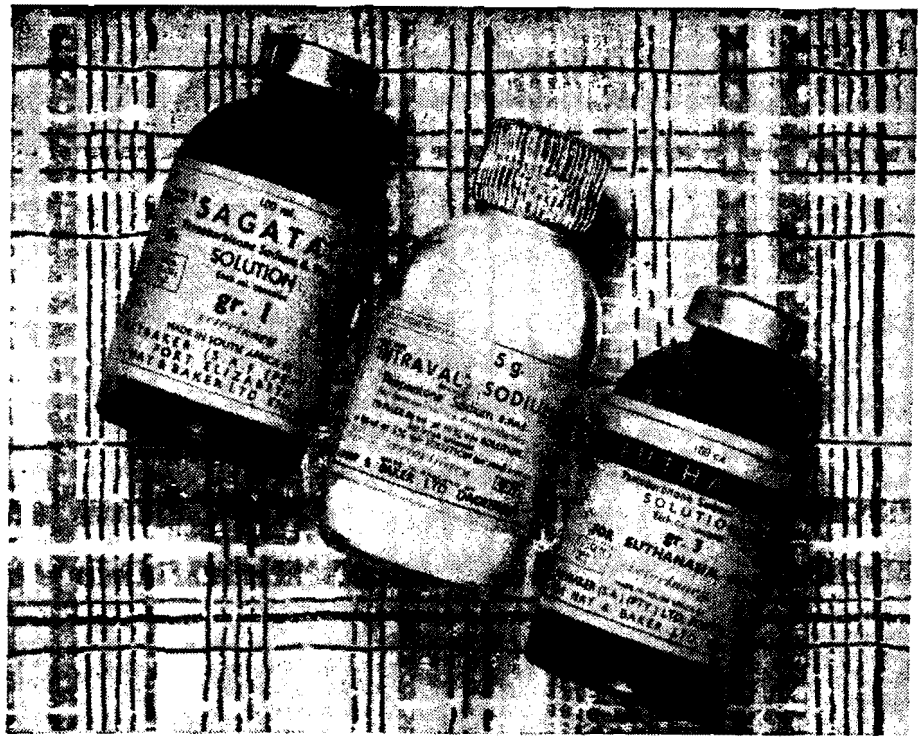
From the above table it is clear that no single drug has given consistently good results. Although subsequent research has yielded more encouraging results, I would advise the routine use of Caparsolate as indicated above, for the present[4].

ACKNOWLEDGEMENTS

I wish to thank the various firms listed above for making trial samples of their drugs available; also Prof. H. D. Brede of the Stellenbosch University Medical Faculty and his microbiological staff of the Karl Bremer Hospital for their valuable assistance.

REFERENCES

1. OSLER, W. (1877). Verminous bronchitis in Dogs. *The Veterinarian*, **12**, 387–397.
2. MALHERBE, W. D. (1954). The Chemotherapy of *Filaroides osleri* infestation in dogs. *J.S. Afr. Vet. med. Ass.*, **25**, 9–12.
3. OLSEN, O. W. (1959). Lungworm, *Filaroides osleri*, in a dog in Colorado. J.A.V.M.A., **134**, 330–334.
4. DORRINGTON, J. E. (1963). *Filaroides osleri*. The success of Thiacetarsamide Sodium Therapy. J.S. Afr. vet. med. Ass., **34**, 435–438.



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HYDROCYANIC ACID POISONING

A Note on the HCN Content of Animal Tissue at Various Stages of Decomposition

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SUMMARY

Five sheep were killed by dosing them each with 5.5 mgm KCN per Kg. body weight. Specimens of muscle, liver and rumen contents were taken every 4 hours and the HCN content thereof determined.

It was found that immediately after death the HCN content was the highest in the stomach contents intermediate in the liver and lowest in the muscle. However, the HCN disappeared from the liver twice as fast as from the muscular tissue, the periods in which total disappearance occurred being 12 and 28 hours respectively.

It is suggested that 0.63 ug/g HCN in muscular tissue be taken as the lowest amount on which a positive diagnosis should be made. Confirmation of a diagnosis of HCN poisoning may be possible by analyses of liver and muscular tissue taken up to 20 hours after death, whereas liver specimens alone must be taken within 4 hours after death, especially during the summer.

INTRODUCTION

To confirm a diagnosis of hydrocyanic acid poisoning it is necessary to demonstrate HCN in the tissues. According to van der Walt[1] the liver is the most suitable organ for this purpose as it contains the highest concentration of HCN. Unfortunately this organ decomposes rapidly and specimens must be taken soon after death. He found that in samples taken immediately after death and kept in closed flasks in the laboratory at room temperature more than half of the original amount of HCN present in the liver disappeared between the 5th and 11th hour, and that practically all had disappeared after 24 hours. Coop[2] found that muscle and blood retained HCN much better than liver and kidney after decomposition at room temperature in the laboratory, over a 24 hour period. For this reason he was of the opinion that muscle specimens were preferable to those of liver.

In this laboratory stomach contents and liver, or liver only, are received for confirmation of diagnosis.

Many cases have been encountered where the stomach contents contained a significant amount of HCN but none was found in the liver. Or, when liver alone was received it gave a negative test despite a history typical of HCN poisoning. The liver was decomposed in all such cases. In view of the fact that a diagnosis was recently made on muscular tissue only where no liver was available[3], it was decided to compare the rate of disappearance of HCN from liver, muscle and stomach contents under more natural conditions and in the intact sheep.

METHODS

The test animals were poisoned with KCN of 97 per cent purity (BDH Analar). Immediately after death an incision was made in each flank and specimens of liver and ruminal contents removed. The ruminal wall and skin wounds were sutured with string in such a way as to allow of easy subsequent opening and closing. At the same time samples of muscle tissue from the leg were collected. The carcasses were

stored in a gauze cage to allow of decomposition and specimens collected as above periodically. Each individual specimen consisted of approximately 75g of tissue. These were preserved in a similar weight of 1 per cent mercuric chloride solution and stored in the refrigerator until analysed. The determination of HCN was done by the method of Gettler and Goldbaum[4].

Results

Experiment 1.

A six tooth Merino ewe was dosed KCN by stomach tube at a rate of 2.2 mgm HCN/kg. The animal showed marked symptoms but recovered and was given a further 4.4 mgm HCN/kg to which it succumbed. Specimens of liver, muscle and rumen contents were taken immediately, 48 and 72 hours after death.

Result:

Table 1

Days	Liver	Muscle	Stomach-contents	
0	1.5	Negative	5.0	μg/g
2	Negative	0.3	0.5	μg/g
3	Negative	Negative	0.2	μg/g

Experiment 2.

An adult Merino ewe was dosed with KCN by stomach tube at a rate of 4.4 mgm HCN/kg. Marked symptoms were seen within 10 minutes but the animal recovered. The next day it was dosed 5 mgm HCN/kg. Marked symptoms occurred up to a half an hour after dosing again followed by recovery. It was then given 12.5 mgm HCN/kg an hour after the first dose and died.

Table 2

Hours after death	Liver	Muscle	Stomach-contents	
0	6.0	2.0	13.0	μg/g
6	Negative	5.0	12.0	μg/g
21	Negative	1.0	2.0	μg/g
27	Negative	Negative	1.6	μg/g
45	Negative	Negative	1.0	μg/g
51	Negative	Negative	0.7	μg/g
99	Negative	Negative	0.2	μg/g

Experiment 3.

Five adult sheep, 3 ewes and 2 wethers, weighing from 40 to 69 kg were dosed KCN by stomach tube at a rate of 5.5 mgm HCN/kg i.e. at a rate of $2\frac{1}{2}$ x M.L.D. (1 M.L.D. = 2.2 mgm/kg[1, 2]). The animals all died 33, 39, 53, 62 and 94 minutes respectively after dosing.

Results:

See tables 1 and 2 and graph 1 drawn from the mean figures.

Discussion

If specimens can be taken very soon after death, the liver remains the best organ from which to take specimens because of its high HCN content. However, although it might still be possible to make a diagnosis on this organ 8 hours after death in some animals (see table 1 sheep No. 1, 3 and 5). In other cases (see table 1 sheep No. 2 and 4 and experiment 2) this would not be possible in as short a period as four hours.

In six sheep dosed with minimum lethal dosages Coop[2] found 0.7; 0.7; 0.5; 0.7; 0.6 and 0.6 μg HCN/g in the muscular tissue. If the mean value 0.63 μg HCN/g is taken as the minimum amount of HCN present in muscular tissue to render a positive justifiable diagnosis, then such a diagnosis could have been clearly made on all live sheep mentioned in tables 1 and 2 after 8 hours, and actually even up to 20 hours after death.

Although it is now clear that HCN remains at least twice as long in muscle as in liver tissue, it is recommended that both be submitted for analysis as suggested by Blood[5]. The reasons being that too few figures are available on levels of HCN in muscular tissue which would justify a positive diagnosis, and the unexplained findings on muscle tissue shown in tables 1 and 2. (Negative at 0 hours and 0.3 μg/g at 2 hours and 2.0 μg/g at 0 hours and 5.0 μg/g at six hours respectively). These findings can probably be explained as due to uneven distribution, as is well established in ruminal contents and liver.

There was still an appreciable amount of HCN in the rumen contents after 56 hours, although it was below the 10 μg/g[1] diagnostic level after 16 hours.

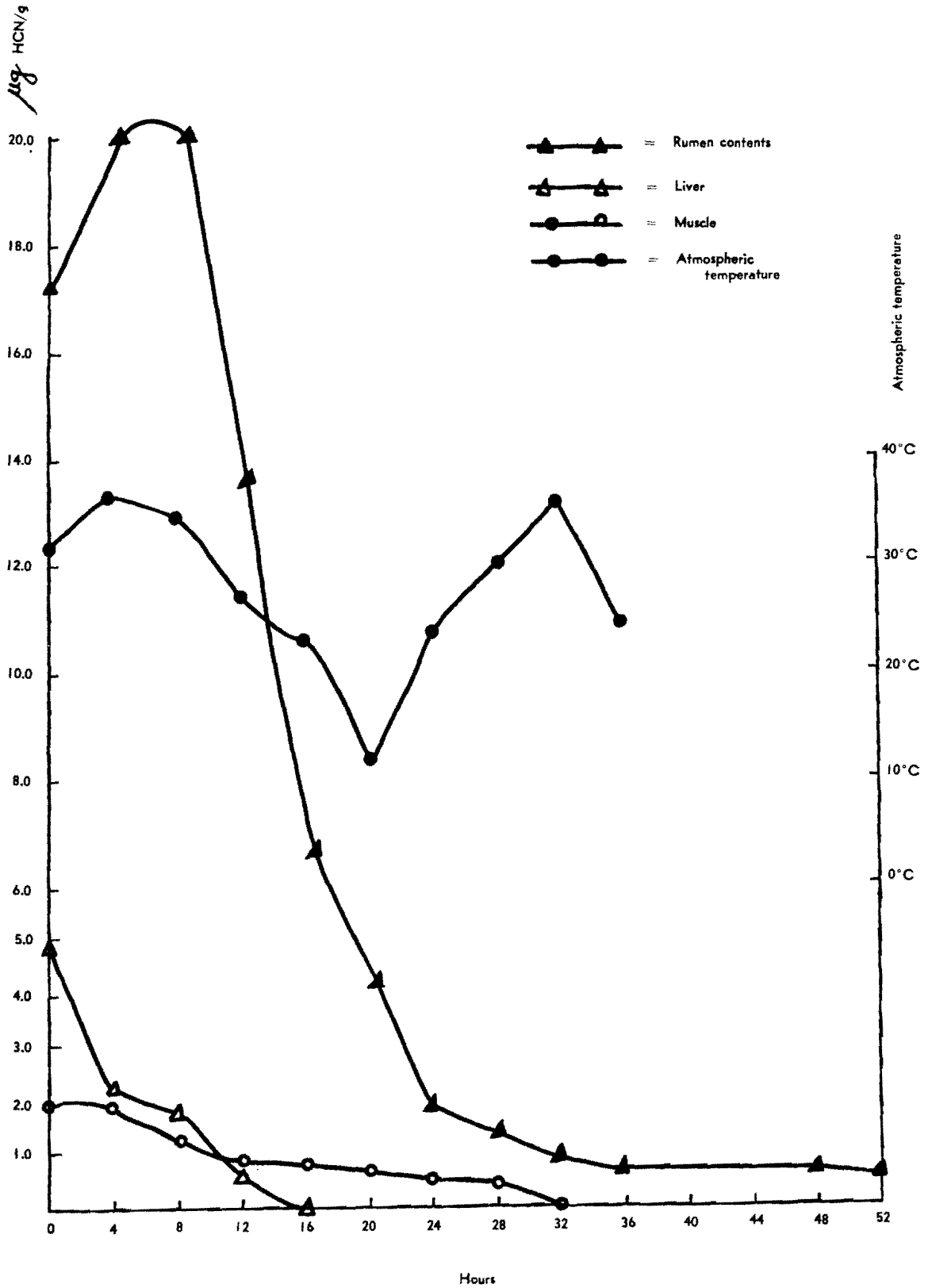

TABLE 3.—HCN CONTENT OF THE LIVER

Hours after death	Sheep number					Mean	
	1	2	3	4	5		
0 hours	5.0	3.0	6.00	3.00	7.00	4.80	µg/g
4 hours	4.0	1.0	2.25	0.50	3.00	2.15	µg/g
8 hours	2.0	0.58	3.00	0.25	2.00	1.57	µg/g
12 hours	1.0	Neg.	0.67	Neg.	Neg.	0.33	µg/g
16 hours	Neg.	Neg.	Neg.	Neg.	Neg.	Neg.	µg/g
20 hours	Neg.	Neg.	Neg.	Neg.	Neg.	Neg.	µg/g

TABLE 4.—HCN CONTENT OF THE MUSCLE

Hours after death	Sheep number					Mean	
	1	2	3	4	5		
0	1.50	2.00	2.50	2.00	1.50	1.90	µg/g
4	3.00	1.00	2.50	1.50	1.50	1.90	µg/g
8	2.00	1.00	1.00	1.00	1.00	1.20	µg/g
12	1.00	0.67	0.83	0.83	0.83	0.83	µg/g
16	1.00	0.67	1.00	0.83	0.50	0.80	µg/g
20	0.67	0.67	0.67	0.83	0.67	0.70	µg/g
24	0.50	0.50	0.33	0.83	0.50	0.53	µg/g
28	0.67	0.38	0.83	0.25	0.25	0.48	µg/g
32	Neg.	Neg.	Neg.	Neg.	Neg.	Neg.	µg/g
36	Neg.	Neg.	Neg.	Neg.	Neg.	Neg.	µg/g

TABLE 5.—HCN CONTENT OF THE STOMACH-CONTENTS

Hours after death	Sheep number					Mean	
	1	2	3	4	5		
0 hours	12.00	12.00	20.00	20.00	22.00	17.20	µg/g
4 hours	14.00	12.00	24.00	24.00	26.00	20.00	µg/g
8 hours	16.00	8.00	28.00	20.00	28.00	20.00	µg/g
12 hours	5.00	4.00	16.00	20.00	24.00	13.80	µg/g
16 hours	1.67	2.00	6.00	14.00	10.00	6.73	µg/g
20 hours	0.83	1.50	3.00	8.00	8.00	4.26	µg/g
24 hours	0.67	1.00	2.00	3.33	3.33	2.07	µg/g
28 hours	0.67	0.50	1.00	3.00	2.00	1.43	µg/g
32 hours	0.67	0.67	1.00	1.00	1.67	1.00	µg/g
36 hours	0.50	0.67	1.00	1.00	1.00	0.83	µg/g
48 hours	0.33	0.67	1.00	1.00	0.83	0.77	µg/g
52 hours	0.67	0.67	0.83	0.67	0.67	0.70	µg/g
56 hours	0.50	0.33	0.83	0.83	0.67	0.63	µg/g

ACKNOWLEDGEMENTS

The Chief of the Veterinary Research Institute, Onderstepoort is thanked for permission to publish this paper. Mr. B. P. Maartens is thanked for his technical assistance. Prof. R. Clark is thanked for assisting in preparing the text.

REFERENCES

1. VAN DER WALT, S. J. (1944). Some aspects of the toxicology of hydrocyanic acid in Ruminants. *O.P. J. Vet. Sci. An. Ind.*, **19** (1 & 2), 79.
2. COOP, I. E. and BLAKELEY, R. L. (1950). The metabolism and toxicity of cyanides and cyanogenetic glucosides in sheep. III. The toxicity of cyanides and cyanogenetic glucosides. *N.Z. J. Sci. & Technol.*, **31A** (5), 44.
3. TERBLANCHE, M., MINNE, J. A. and ADELAAR, T. F. (1964). A diagnosis of Prussic-acid poisoning in cattle made from a sample of muscular tissue. *J.S. Afr. Vet. Med. Ass.*, **35** (2), 99.
4. GRADWOHL, R. B. H. (1948). Clinical Laboratory Methods and Diagnosis (The Gettler & Goldbaum method), 4th Ed., 2: 2103. C. C. Mosby Co., St. Louis, U.S.A.
5. BLOOD, D. C. and HENDERSON, J. A. (1963). Veterinary Medicine p. 1052, 2nd Edition, Bailliere, Tindall and Cox, London.

ADVANCES IN GEELDIKKOP (TRIBULOSIS OVIS) RESEARCH

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Section of Physiology, Veterinary Research Institute

6: STUDIES ON SELECTED ASPECTS OF THE BIOCHEMISTRY OF GEELDIKKOP AND ENZOOTIC ICTERUS

Received for Publication August, 1964

SUMMARY

Certain aspects of the biochemistry of Geeldikkop and enzootic icterus are discussed. Previous observations with regard to increased levels of selenium in the livers of affected animals have been confirmed. The lesions in biliary excretion described earlier have been found to extend to bile acids, other glucuronides and alkaline phosphatase. Maximum retention of biliary components occurs during the second and third days of illness. Profound disturbances of carbohydrate metabolism exist in geeldikkop as evidenced by elevated plasma lactate and liver glucose-6-phosphatase values, markedly lowered values of activity for glyceraldehyde phosphate dehydrogenase and a considerably retarded rate of clearance of intravenously administered glucose from the circulation. Marked elevations of the activity of certain enzymes in plasma, indicative of liver injury have been observed. Increased levels of ascorbic acid in affected animals together with increased glutathione reductase activity in liver have been noted. The nature of the erythrocyte lesions in geeldikkop and enzootic icterus have been defined as increased fragility consequent to decreased glyceralde-phosphate dehydrogenase activity. Geeldikkop has been defined as a special type of cutaneous hepatic porphyria and is associated with marked coproprophyrinuria in its early stages.

Marked inhibition of liver succinic dehydrogenase activity has been observed, unassociated with damage to terminal electron transfer chains. The essential disturbance in the disease is believed to be a failure in the energy supply to systems responsible for transport of bilirubin glucuronides and other compounds across the hepatic cell membranes associated with a decrease in the selective permeability of these structures. Some thoughts are advanced regarding the aetiology of geeldikkop.

INTRODUCTION

The essential primary disturbance thought to occur in Geeldikkop is an interference in the passage of bilirubin glucuronides, porphyrins and sulphonphthalein dyes across the hepatic cell membranes. Retention of these compounds in the body is aggravated by renal involvement. The lesions in enzootic icterus are basically similar but much more severe[1, 2, 3, 4, 5]. On the basis of these studies it has been concluded that the two conditions represent extreme syndromes of a single basic disease entity, the nature of which is not yet clear although a low grade subsclinical selenium intoxication has been incriminated in the aetiology of these conditions[2, 6].

During the course of outbreaks of geeldikkop during the latter half of the summer of this year investigations were performed on many aspects of the biochemistry of this condition. These studies have been extended and largely confirmed by more recent work. Certain of the most interesting aspects of this work have been selected for discussion in this paper. Detailed reports of all the investigations made to date will appear in print elsewhere.

Materials and Methods

The sheep used in these studies were typical cases of geeldikkop collected from farms where the disease was prevalent within hours of the appearance of the first symptoms. They were in general young merinos of both sexes and were housed in sheltered sheep pens situated on the showgrounds at Victoria West. The facilities offered by these showgrounds were converted for use as a laboratory in which the biochemical work reported could be done. The experimental animals were given dry lucerne hay and water *ad lib.*

Twenty-seven cases of the disease were collected for study together with six clinically healthy sheep for purposes of comparison. The latter were obtained from two farms where the disease was active.

The investigation was planned to cover all stages of the disease from the appearance of the first symptoms until the subsidence of these at about eight days after onset. Some of the sheep were sacrificed for biochemical work immediately on arrival at the laboratory the remainder being slaughtered at regular intervals thereafter during the test period. Sheep classed as "recovered" cases were those which appeared clinically normal once more apart from actively healing lesions on their faces and ears. They were slaughtered three weeks after the appearance of the first symptoms. Control animals were sacrificed at intervals throughout this work.

The animals used have been grouped for the purpose of this discussion according to the severity and duration of illness as follows:—

Group 1.—Clinically normal, no visible symptoms of either syndrome.

Group 2.—Severely photosensitive cases of one day's standing.

Group 3.—Photosensitive cases of 1–2 day's standing with severe oedema of the head.

Group 4.—Mildly photosensitive cases of 2–3 day's standing with oedema of the head.

Group 5.—Non-photosensitive cases of 4–5 day's standing. Some residual oedema of the ears.

Group 6.—Non-photosensitive cases of 5–8 day's standing. Sloughing of affected skin on face and ears.

Group 7.—Recovered cases, actively healing lesions apparently normal once more.

Blood specimens were collected from the various animals immediately prior to slaughter or at intervals throughout the period of study as demanded by the work. Anticoagulants used were as prescribed in the methods used for the determinations concerned.

Studies on urine and faecal porphyrin excretion patterns were performed on urine collected from wethers using the collecting bottle described earlier,[7] and the standard faeces bags designed for metabolic studies in sheep. Studies on bile pigment and bromsulphalein metabolism were conducted on animals in which the common bile duct was cannulated as described earlier.[7]

Experiments on the biochemical action of icterogenin were performed on rabbits with biliary cannulae as reported elsewhere.[8] At the time of introduction of the cannula into the common bile duct about four grams of liver tissue were removed for estimation of enzyme activity levels before intoxication. The results obtained were compared with those obtained from liver removed from the animal at the end of the test period, usually 4–5 hours after intra-abdominal administration of the triterpene at the time of operation. Each animal therefore served as it's own control with respect to the activity levels of liver enzymes.

Chromatographically pure icterogenin (22 β-angeloyloxy-24-hydroxy-3-oxo-olean-12-en-28-oic acid) was kindly provided by my co-workers. Dr. W. T. de Kock and Mr. L. A. P. Anderson of the National Chemical Research Laboratories, Pretoria. The compound was finely powdered and suspended for administration in 2 per cent (W/V) aqueous Cellophas B (medium viscosity, Imperial Chemical Industries) solution.

The methods used for assay of the various liver and plasma enzymes studied together with those used for other estimations on blood or tissues are given in Table 1 below:

TABLE 1.—METHODS USED IN THIS INVESTIGATION

Coproporphyrin (44).	Glucose — 6 — phosphate dehydrogenase (58).
Uroporhyrin (44).	Uridine diphospho-glucose (59).
Faecal protoporphyrin (44).	Uridine diphospho-glucose dehydrogenase (26).
RBC protoporphyrin (45).	Bilirubin conjugation (8).
Phylloerythrin (46).	Adenosine triphosphatase (60).
Alkaline phosphatase (42).	Diphospho-pyridine nucleotidase (61).
Plasma Isocitric dehydrogenase (47).	Cytochrome — c — reductase (62).
Liver Isocitric dehydrogenase (48).	Diaphorase (62).
Lactic dehydrogenase (49).	Lactate (63).
Glutamic oxalacetic transaminase (50).	Sugar (64).
Glutamic pyruvic transminase (50).	Glucuronides (65).
Phospho hexose isomerase (51).	Total plasma proteins (66).
Amylase (42).	Electrophoresis (42).
Cholinesterase (52).	Ascorbate (42).
Aldolase (53).	Bile salts (67).
Succinic dehydrogenase (54).	Bilirubin (68).
Glucose — 6 — phosphatase (55).	Red cell fragility (2).
Glutathione reductase (56).	Selenium (69).
Glyceraldehyde — 3 — phosphate dehydrogenase (57).	

NOTE:

(*a*) In the text Cytochrome — c — reductase and diphospho — pyridine nucleotidase have been abbreviated to Cyto — c — red. and DPN — ase respectively. All other abbreviations used are indicated in the text where they occur.

(*b*) The method used for alkaline phosphatase was the AAP one in ref. 42; that for plasma ascorbate used indophenol (ref. 42). while that used for cholinesterase (ref. 52) depended upon the hydroxamate — ferric-chloride reaction.

All enzyme assays done on liver tissue, except where noted otherwise, were performed using 10 per cent homogenates in ice cold 0.25 M sucrose solution made in a manual all glass Potter type homogenizer. All homogenates were kept in ice baths at 0°C during use. Liver specimens for enzyme assays were collected immediately after slaughter of the sheep concerned and homogenized at once, the assays being completed as soon as possible thereafter. At the same time specimens of various organs were removed for histopathological studies and a full autopsy examination was performed on each case.

A Unicam S.P. 500 spectrophotometer was used for all the enzyme studies; other photometric procedures were carried out on an Evans Electroselenium (E.E.L.) portable Model A photoelectric colorimeter. Paper electrophoresis of plasma proteins was performed using the E.E.L. paper electrophoresis apparatus and 34 x 5 cm Whatman No. 1 filter paper strips. Proteins were separated in a veronal:acetate buffer pH 8.6, over 16 hours with a current setting of 2 milliamps per strip.

Chemicals used throughout were of "analytical reagent" grade. Compounds used for the preparation of substrates for enzyme assays or pure enzymes used for the standardization of these were obtained from the Sigma Chem Co. (St. Louis Mo.) or from C. F. Boehringer (Mannheim, West Germany) and were of the highest purity offered by these manufacturers.

THE MAJOR METABOLIC PATHWAYS

Before passing on to a consideration of the biochemical studies presented in this paper it is not out of place to review very briefly the major metabolic pathways in mammalian cells. This will assist in following the discussion and help the reader to visualize the location of the various enzymes studied in the general scheme of metabolism.

The enzymes of particular interest in the problem of the aetiology of geeldikkop and enzootic icterus are the dehydrogenases and particularly those containing sulphydryl (SH) groups which are known to be sensitive to inactivators like selenium. Dehydrogenases catalyze the removal of two atoms of hydrogen from a substrate (or it's addition to the substrate in reversible reactions) and their transferance to a co-enzyme hydrogen acceptor, e.g. Lactic dehydrogenase (LDH.) brings about the transfer of 2H from lactic acid to diphosphopyridine nucleotide (Coenzyme 1, DPN) thus:—

$$CH_3-CH(OH)-COOH+DPN \xrightarrow{LDH} CH_3-CO-COOH+DPNH_2$$

By definition dehydrogenases cannot transfer hydrogen atoms directly to molecular oxygen. They are therefore linked through a coenzyme, which may be DPN as above or triphosphopyridine nucleotide (TPN), to a terminal respiratory or electron transfer chain consisting of a series of acceptors at the end of which water is produced by combination of hydrogen with oxygen. The dehydrogenases and their associated respiratory chains are thus intimately concerned in the oxidation of nutrient substances, the generation of energy and the trapping of this for physiological purposes.

The major metabolic pathways are presented for consideration in highly schematic form in Figure 1:

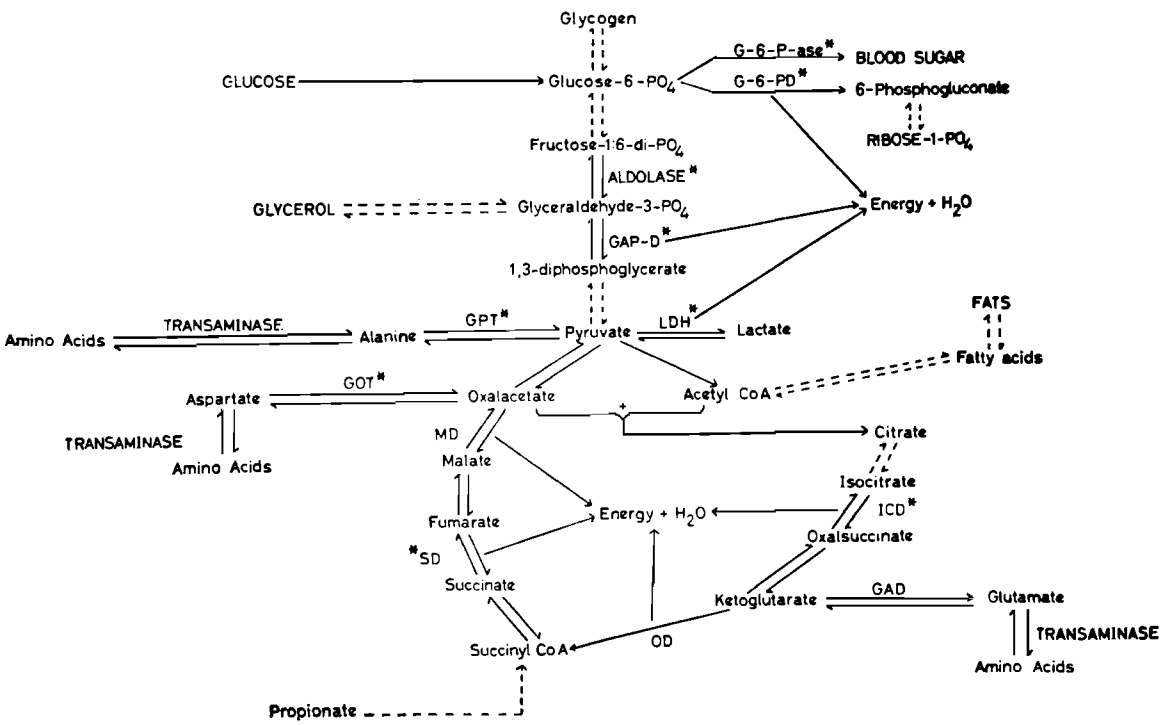


Fig. 1: The major metabolic pathways. (Enzymes marked with an asterisk were studied in this investigation.)

Carbohydrates are degraded to pyruvic acid by a sequence of reactions known as glycolysis, during the course of which the enzyme aldolase splits fructose — 1,6-diphosphate into two molecules of glyceraldehyde — 3-phosphate. This substrate is oxidized in a reaction catalysed by glyceraldehyde-phosphate dehydrogenase (GAP-D) to phosphoglyceric acid, the reaction representing the only oxidative step in the direct pathway for metabolism of glucose to pyruvate. In the ovine erythrocyte the hydrogen which is removed is linked i.a. to the reduction of methaemoglobin *in vivo* (see later). The enzyme glucose-6-phosphatase (G-6-P-ase) present in liver is responsible for the conversion of glucose-6-phosphate (G-6-P) to glucose (blood sugar), while glucose-6-phosphate dehydrogenase (G-6-P-D) provides the link between the glycolytic cycle and the monophosphate shunt — a pathway for the synthesis of many useful chemicals, e.g. ribose-phosphate. In certain species mentioned later in the text glucose is utilized by the erythrocytes via this monophosphatepathway, the hydrogen removed by G-6-P-D being used in the reduction of methaemoglobin *in vivo*.

Fatty acids are degraded to acetyl-Coenzyme A (acetyl-CoA) by repeated oxidative cleavage at the β-Carbon atom. Each β-oxidation step involves two dehydrogenations coupled with very active respiratory chains. The formed acetyl-CoA is disposed of as shown in Figure 1.

Most amino acids are handled by oxidative deamination reactions, which involve transaminations to produce one of three animo acids, viz. aspartic acid, glutamic acid or alanine. Aspartate and alanine enter into two highly specific transaminations catalyzed by glutamic-oxalacetic transaminase (GOT) and glutamic-pyruvic transaminase (G-PT.) yielding the corresponding keto-acids and glutamic acid. The latter is oxidatively deaminated in a highly specific reaction catalyzed by glutamic acid dehydrogenase (G-AD.) yielding α-ketoglutarate. The points of entry of the keto-acids formed in these reactions into the final common pathway of metabolism are indicated in Figure 1.

The tricarboxylic acid cycle (TCA. cycle, Krebs cycle) is the final common oxidative pathway for the complete degradation of carbohydrates, fats and protein to CO_2 and water with the release of energy inherent in their intramolecular bonds as indicated in Figure 1. The cycle involves i.a. four separate oxidations in which 8 atoms of hydrogen are taken up by respiratory chains for oxidation to water and energy.

The most important sequence of biochemical events in the mammalian cell is that of oxidative phosphorylation. Most of the cell's adenosine triphosphate (ATP) is synthesized in the process. A tremendous amount of energy is liberated in energy yielding reactions in the cell as noted above. In the absence of some mechanism which conserves or traps some of this released energy for use in driving energy requiring reactions, the chemical constituents of the cell would rapidly be broken down to simple waste products accompanied by the evolution of energy as heat. A certain amount of this heat energy can be used for the maintenance of body temperature but the rest would be dissipated uselessly. An imperative function of mammalian cells is therefore the trapping and securing of some of this energy, which would otherwise be lost, in a form in which it can be used for work, e.g. chemical synthesis, nerve impulse transmission, secretion, absorbtion, muscle contraction, mitosis etc. This is achieved by trapping this energy in the energy-rich pyrophosphate bonds of ATP formed during the process of oxidative phosphorylation. Unless ATP is synthesised at a rate which is rapid enough to replace the large and constant drain on it cellular metabolism begins to fail, the cell dying quickly once it's store of ATP is depleted.

Oxidative phosphorylation can be defined as the formation of the high energy pyrophosphate bonds of ATP during the passage of electrons along the terminal respiratory chains associated with the oxidation of substrates by enzymes e.g. the various dehydrogenases mentioned, particularly those of, or associated with, the Krebs cycle. A typical terminal respiratory chain is shown in schematic form in Figure 2.

Hydrogen is removed from the substrate by the dehydrogenase and transferred to the first coenzyme of the chain, which is thus reduced. The hydrogen (or electrons) is then passed down a series of acceptors or hydrogen carriers which are alternately reduced and re-oxidized by the

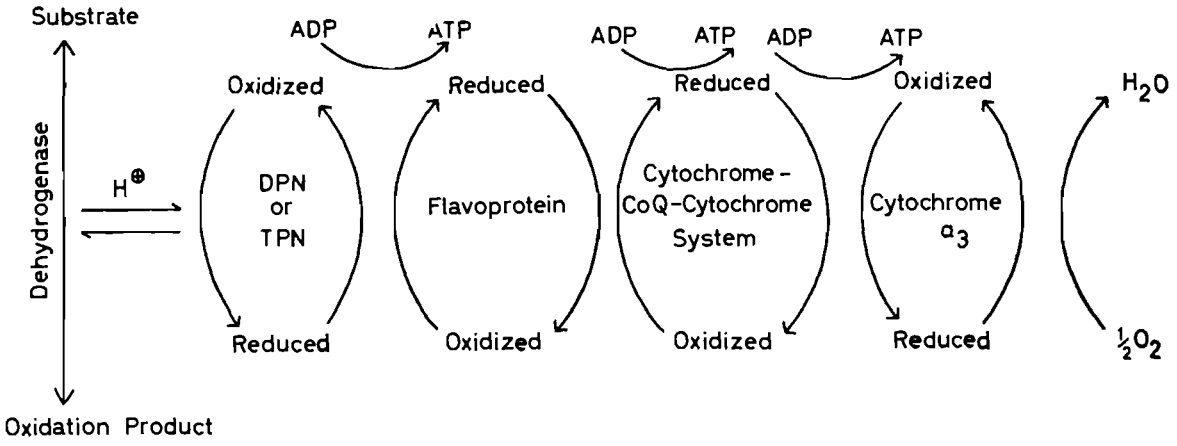


Fig. 2: A typical terminal electron transfer chain associated with the function of oxidative phosphorylation.

passage of electrons, the process culminating in the reduction of molecular oxygen to water. Energy is trapped at three points in the repiratory chain to form ATP as shown in Figure 2.

Most but not all of the cells glycolytic enzymes are present in the cytoplasmic sap, except in the cells of the brain, the mitochondria of which contain a full complement of glycolytic enzymes and cofactors[9]. The mitochondria of mammalian cells contain the greatest concentration and variety of intracullular enzymes. Although some of the enzymes of the TCA cycle occur in other parts of the cell it is only in the mitochondria that all of the enzymes, co-enzymes, co-factors and carrier systems of the cycle are found together and hence only the mitochondria can perform the vital function of oxidative phosphorylation. They can be regarded as the dynamos responsible for the generation of by far the largest part of the cells ATP[9]. It follows then that any damage to these structures, whether physical or chemical will have a profound influence on cell metabolism as a whole, affecting in particular those reactions which are ATP dependant. For the sake of the discussion which follows it should be remembered that one such ATP-energy dependant function is the maintenance of the selective permeability of cell membranes.

The Role of Selenium in the Aetiology of Geeldikkop and Enzootic Icterus

A low grade subclinical and chronic selenium intoxication has been incriminated as the possible disturbance underlying these conditions either syndrome being precipitated by a variety of severe and non-specific stress conditions[1, 2, 6]. Tissues from cases of geeldikkop and enzootic icterus were found to contain dangerous amounts of this element, the range of values found being in excellent accord with the severity of either syndrome[1, 2, 6]. These findings have to a large extent been confirmed by recent work. Further analytical data regarding the amounts of selenium present in the livers of cases of either condition are presented in Table 2 below. The animals studied in this respect include not only those used in the present investigation but also numerous others which have been examined since the publication of the initial report[6]. A survey of the occurrence of selenium in Karoo vegetation, confirming the findings in this regard has been completed and submitted for publication elsewhere.

The role played by this element in the genesis of these two conditions is still not clear. It is known to be a powerful inhibitor of numerous dehydrogenases in the body notably succinic dehydrogenase (SD.) GAP-D, pyruvic oxidase

TABLE 2.—GEELDIKKOP AND ENZOOTIC ICTERUS. VALUES FOR SELENIUM FOUND IN THE LIVERS OF TYPICAL CASES. (Values are expressed as Micrograms Se per gm. of liver tissue on wet weight basis).

GEELDIKKOP			ENZOOTIC ICTERUS		
Number of cases	Mean value	Range	Number of cases	Mean value	Range
41	8.89	1.0–26.8	26	16.2	3.0–29.6

and many others[10]. It has been suggested that herein may be at least part of the answer to this problem[1,2]. SD and GAP-D activities have been studied in the livers of typical cases of Geeldikkop and the results obtained (and presented later in this text) are most enlightening.

Brown and Abrams[11] have recently made the interesting observation that in ducklings and chickens exposed to various forms of mycotoxicosis, e.g. by *Alternaria, Fusarium* and *Aspergillus* species, there appear to occur a marked accumulation of selenium in the livers of affected birds, which is apparently unrelated to the dietary intake. Livers from affected birds were found to contain 10–25 ppm of the element in fresh liver as compared with the normal range of 0.5–3 ppm for chickens and ducklings kept under natural conditions. The significance of these findings is not at all clear. Since the apparent levels of selenium are unrelated to the dietary intake, it is thought that the fungal intoxication may lead to either increased absorbtion or decreased utilization or excretion of this element with consequent storage of higher amounts than usual.

The possible role of fungal toxins in the aetiology of geeldikkop, similar to that played by sporidesmin, the toxin elaborated by *Pithomyces chartarum* and believed to be the causal agent of Facial Eczema in New Zealand and Australia has been considered[3,6]. As yet we have been unable to obtain any evidence that a mycotoxicosis in concerned in the appearance of this disease. The epizootiology of both geeldikkop and enzootic icterus is in most ways inconsistent with a mycotoxicosis.

During the course of previous investigations twenty-three different fungi have been isolated from *Tribulus terrestris* growing on farms in the presence of severe outbreaks of geeldikkop. These fungi, which included *Culvularia, Penicillium, Fusarium, Helminthosporium, Rhizopus, Alternaria, Myrothecium* and *Hendersonia* species were grown in pure culture on a wide variety of media, including minced *Tribulus* and tribulus saponins, under an equally wide variety of conditions. The various cultures were dosed to at least twelve sheep at different times. The amount of culture given which included mycelial mat, spores, and fluid medium varied from 3–5 litres given either in single or split doses over one week. At no time was any evidence produced of either the symptoms or biochemical lesions so characteristic of geeldikkop and enzootic icterus.

More detailed studies of this nature are at present being conducted by officers of the National Chemical Research Laboratories (Pretoria) using cultures of fungi isolated from various plants in the affected areas during the recent investigations. We must await the results of this work before making a definite decision about the possible role of mycotoxicosis in the aetiology of geeldikkop, either as a primary factor or as a potent secondary stressor.

BLOOD CHEMISTRY AND ENSYMOLOGY

Previous studies of this nature have brought to light many interesting facts indicating the complexity of the metabolic disturbances in geeldikkop. The recent investigations have added much to this knowledge but it is beyond the scope of this paper to do more than touch briefly on some of the more interesting findings.

In the first instance it is clear from the data which will be presented that the crisis, if it is permissable to use the word in this context, in the metabolic disturbances which are seen in uncomplicated geeldikkop is reached during the second or third day of illness. This period

coincides with maximum photosensitivity and intensity of the icterus and is generally followed by a slow return to normal of the metabolic systems most affected. Some of the biochemical lesions may persist however right through into the period of apparent recovery.

The disturbances in biliary excretion seen in geeldikkop have been reported elsewhere[1, 2, 5]. In severe cases of this disease a small amount of almost colourless bile is frequently found in the gallbladder[3, 5]. Besides the previously noted failures in the biliary excretion of bilirubin glucuronides, phylloerythrin and sulphonephthalein dyes, there is also a marked inability to secrete certain substances like the bile salts and alkaline phosphase.

Plasma bile salt concentrations were studied throughout the course of the disease and the relevant data is reproduced in histogram form in figure 3 below. Huge increments in the total bile acid concentration in the plasma are evident in the early stages of the disease particularly during the second day of illness. A rapid decrease in the plasma concentrations of these compounds is seen as the animal recovers.

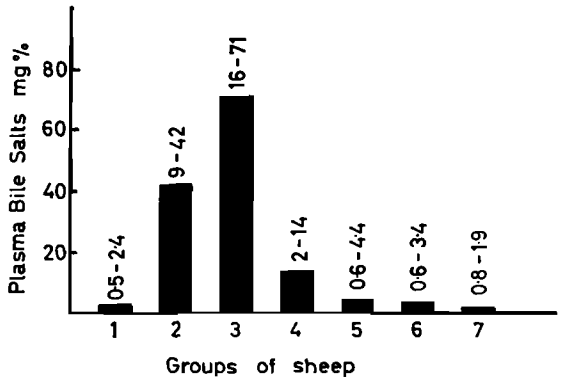


Fig. 3: Plasma Bile Salt concentrations in Geeldikkop.

Figure 4 is a schematic representation of the bile salt, bile pigment and phylloerythrin (phyllo.) content of the plasma of affected animals at various times throughout the course of the disease. Maximum values for the plasma concentration of all three types of compounds are found during the second and third days of illness at which time transfer of these compounds across the hepatic cell membrance must be virtually non existent. So far these disturbances have not been correlated with a hypercholesterolaemia[2] and it appears therefore that not all the constituents normally present in bile are equally affected. Electrophoretograms made from the bile of early cases of the disease according to the method of Verschure and Hoefsmit[12] indicate the presence of four protein bands of varying polarity and mobility as well as two bands of lipid material. These bands were also observed in electrophoretograms of bile from control animals. It is possible that these proteinaceous and lipoidal components are added to the bile by the gallbladder mucosa and not secreted by the bile duct epithelium.

The total glucuronide concentration in plasma follows the concentration of bile pigments bile salts and phyllo almost exactly, maximum levels being encountered in animals of group 3.

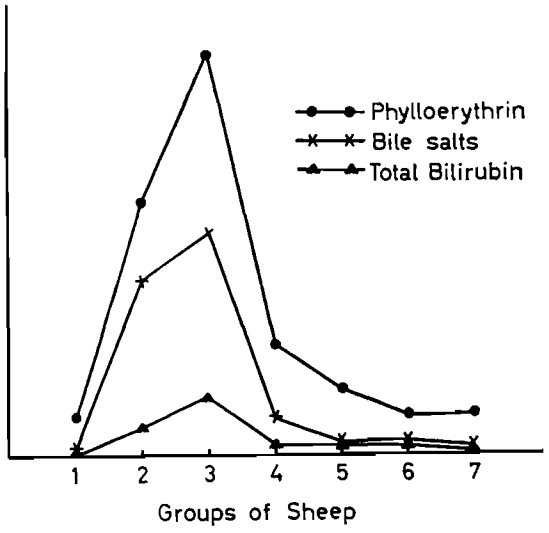


Fig. 4: Schematic representation of phylloerythrin, bile salts and total bilirubin levels in the plasma of geeldikkop cases.

The severity of the bile acid retention is noteworthy. Plasma concentrations in clinically normal sheep have been found to range from 0.5–2.4 mgm% whereas values of up to 71 mgm% were encountered in the animals of group 3. This is similar to the severity of bilirubin retention where values for total bilirubin of up to 35–40 mgm% are not uncommon in early cases of the disease under field conditions.[2]

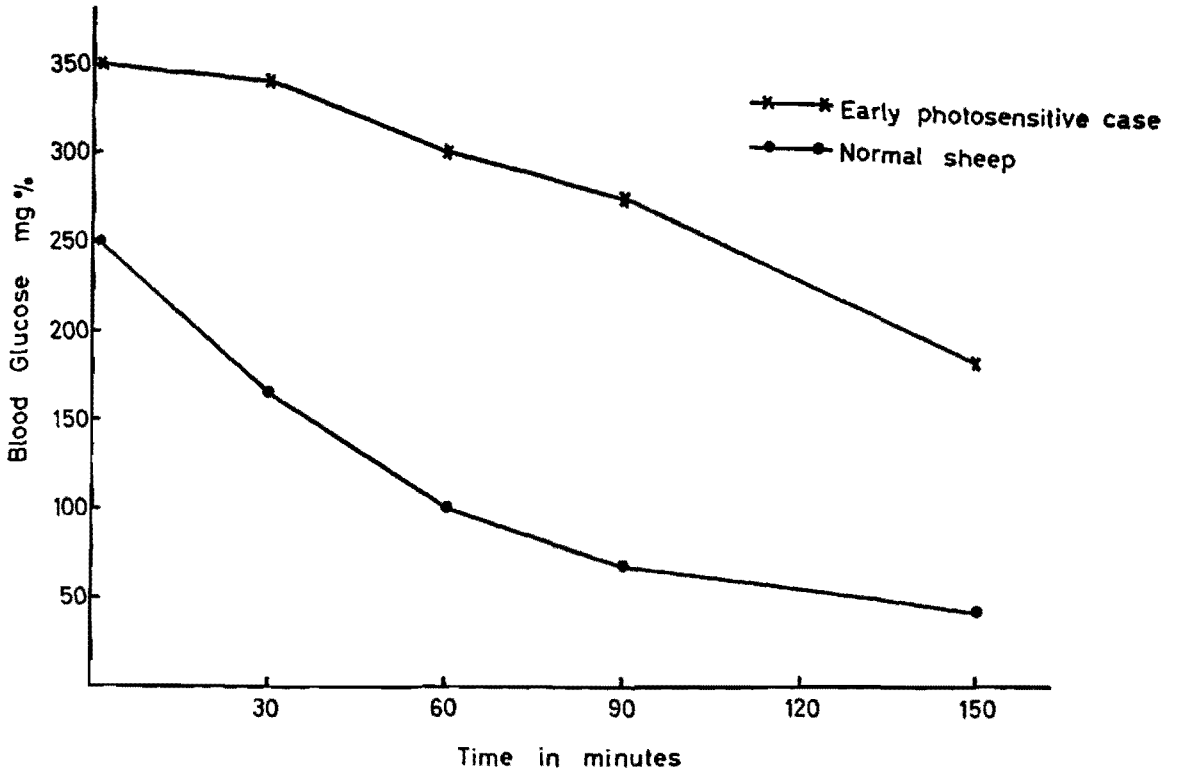


Fig. 5: Rate of clearance of intravenously injected glucose from the blod of a typical early case of geeldikkop compared with that shown by a clinically normal animal.

A most profound disturbance in carbohydrate metabolism exists in the early cases of geeldikkop. Severe hypoglycaemia is a frequent finding particularly in these early cases and in fat animals is frequently complicated by ketosis[2].

The rate of clearance from the blood of intravenously administered glucose is abnormally and markedly retarded in early cases of the disease presenting a picture reminiscent of diabetic states. Typical results are presented in figure 5 in which the glucose clearance curve from an animal of group 3 is compared with that shown by a clinically normal control sheep taken from the same farm.

The values found for lactic acid in the plasma of affected animals are equally interesting. Negligable amounts of this acid are normally present in the blood of healthy sheep, e.g. in the control animals maintained on lucerne hay the range was found to be from traces to 0.6 mg.% with the method used. Extremely high values are encountered from the second to the fifth day of illness in affected animals and

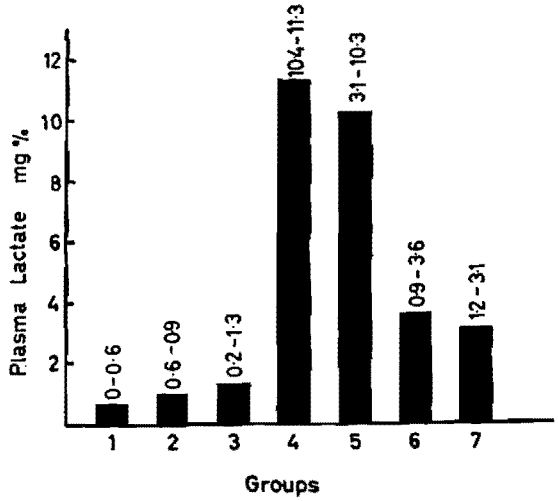


Fig. 6: Plasma lactic acid levels in the different groups of geeldikkop cases.

although these are followed by a decline towards the normal range, recovered animals still present abnormally high plasma levels for this compound. These results are presented in histogram form in figure 6.

A number of enzymes normally present in plasma and which are conventionally assayed for the diagnosis of hepatic pathology were studied in this and previous investigations, viz. alkaline phosphatase, GOT., GPT., isocitric dehydrogenase (ICD), lactic dehydrogenase (LDH) and amylase. Included in the present study as well were phosphohexose isomerase (PHI), aldolase and cholinesterase.

No significant variations were seen between control and affected animals as regards GPT., amylase and cholinesterase, but contrary to previous findings[2] it has been established that moderate elevations in plasma alkaline phosphatase activity do in fact occur in the early stages of the disease. The relevant figures are presented in Table 3 below.

The activity levels of GOT., ICD., LDH., PHI and aldolase in the plasma of affected animals are markedly raised in the earliest stages of the disease. Although after the second day of illness there is a decline in the activity of these enzymes in this medium, moderately elevated values with the exception of those for aldolase persist even in apparently recovered animals. This feature is particularly striking in

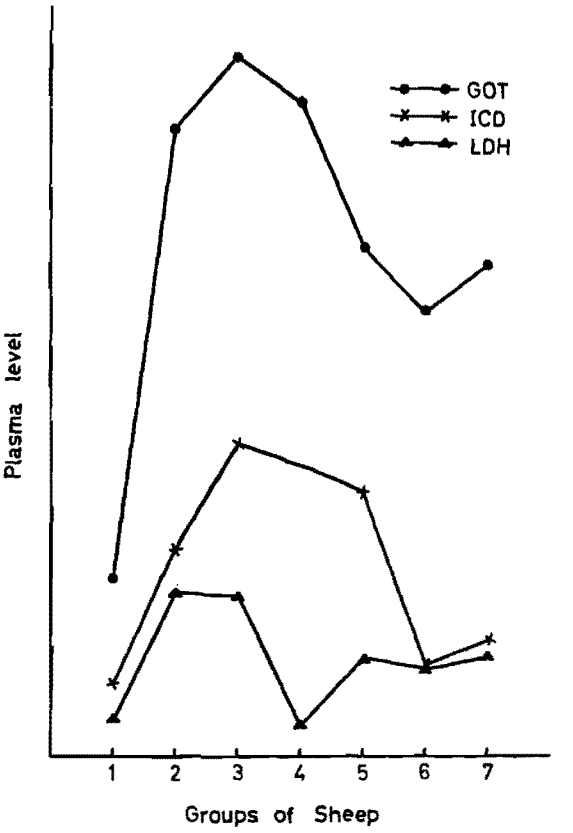


Fig. 7: Schematic representation of glutamic-oxalacetic transaminase (GOT), lactic dehydrogenase (LDH) and isocitric dehydrogenase (ICD) activity levels in the plasma of typical cases of geeldikkop.

TABLE 3.—ALKALINE PHOSPHATASE ACTIVITY IN PLASMA.
(Figures represent units of enzyme/100 ml of plasma).

Groups of sheep	1	2	3	4	5	6	7
Range of activity	5–10	17–61	13–48	40–45	15–81	13–38	10–36
Mean value	7	33	26	43	33	24	22

TABLE 4.—GOT., ICD., LDH., PHI., and ALDOLASE ACTIVITY IN PLASMA OF CONTROL SHEEP AND CASES OF GEELDIKKOP OF 1–2 DAYS STANDING.
(Figures represent units of enzyme/100 ml. of plasma.

Enzyme	GOT	ICD	LDH	PHI	Aldolase
Group 1 animals—					
Range	50–162	50–250	410–590	51–107	9–34
Mean value	114	130	461	76	22
Group 3 animals—					
Range	382–487	275–598	1020–2140	331–910	48–132
Mean value	448	514	1604	545	91

the case of GOT. The values found for the activity of these enzymes in the plasma of control animals and sheep of group 3 are presented in Table 4 while the trends mentioned are depicted schematically in figures 7 and 8.

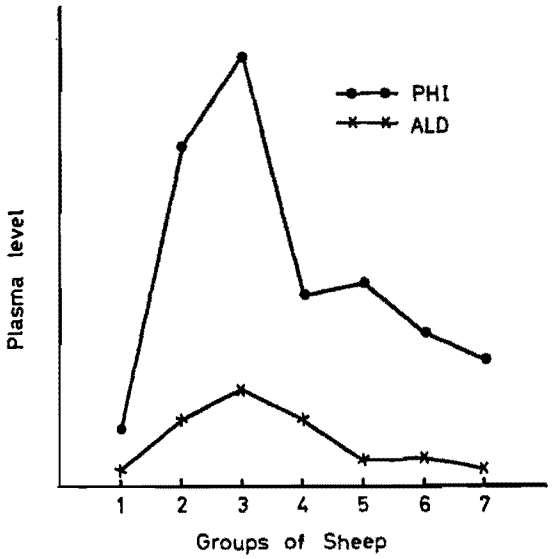


Fig. 8: Schematic representation of phosphohexose isomerase (PHI) and aldolase (ALD) activity levels in the plasma of typical cases of geeldikkop.

Plasma ascorbic acid levels have been found to be moderately increased during the early stages of geeldikkop. Advanced cases of this syndrome or enzootic icterus present either slightly elevated plasma figures, values in the same order as the control animals or lower values depending upon the severity of the condition.

Figures representative of these findings are presented in Table 5 below. The enzootic icterus cases were studied in a previous investigation.

The figures obtained for the total plasma proteins of affected animals show mild variations from normal in many instances. These can generally be related to water loss or retention and form part of the stress syndrome.[2] Mild elevations in the γ-globulin fraction and quantitative changes in other globulin fractions have been noted in most cases studied earlier[2]. During the present study the pattern of plasma proteins was followed throughout the course of the disease in typical geeldikkop cases. Results fully representative of those found in the various groups of sheep studied are presented for consideration in Table 6, while electrophoretograms representing the pattern in different stages of the disease are depicted in figures 9–15 below.

TABLE 5.—PLASMA ASCORBIC ACID LEVELS IN CONTROL ANIMALS AND CASES OF GEELDIKKOP OR ENZOOTIC ICTERUS. (Figures represent mgm. ascorbic acid/100 ml. plasma).

Control Sheep	Early Geeldikkop	Advanced Geeldikkop	Early Enzootic Icterus	Advanced Enzootic Icterus
Sheep 12221: 1.94	Sheep F1: 3.88	Sheep F4: 2.02	Sheep F7: 2.24	Sheep F10: 0.39
Sheep 12222: 1.51	Sheep F2: 3.11	Sheep F5: 2.03	Sheep F8: 1.44	Sheep F11: 0.63
Sheep K2: 0.98	Sheep F3: 3.07	Sheep F6: 1.97	Sheep F9: 1.57	Sheep F12: 0.56

TABLE 6.—PLASMA PROTEINS OF CASES REPRESENTING VARIOUS STAGES OF GEELDIKKOP. (Figures represent gm. protein/100 ml. plasma).

Protein Fraction	Group 1 Sheep 5	Group 2 Sheep 21	Group 3 Sheep 20	Group 4 Sheep 15	Group 5 Sheep 16	Group 6 Sheep 2	Group 7 Sheep 3
Total Plasma proteins.....	7.0	9.15	8.6	7.9	9.28	8.6	8.26
Albumins.....	2.94	2.39	2.36	1.83	1.54	1.93	2.89
Globulins.....	3.40	6.12	5.73	5.17	6.96	6.03	4.45
Undifferentiated fraction.....	0.64	0.66	0.49	0.87	0.74	0.62	0.91
α and β-Globulins.....	1.47	1.94	1.83	3.08	3.86	2.69	1.39
γ-Globulins.....	1.93	4.17	3.89	2.09	3.10	3.33	3.06
A:G ratio.....	0.86	0.23	0.61	0.35	0.22	0.32	0.65
Electrophoretogram.....	Fig. 8	Fig. 9	Fig. 10	Fig. 11	Fig. 12	Fig. 13	Fig. 14

NOTE: The electrophoretograms are depicted in the text below according to the figures noted in this table.

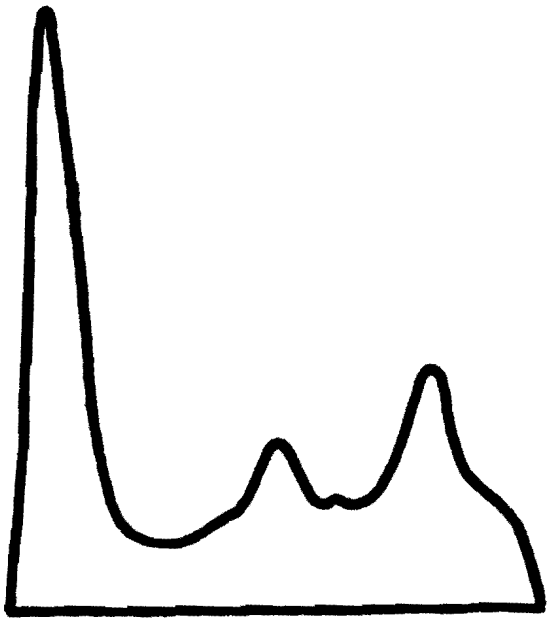


Fig. 9: Plasma protein pattern in healthy control sheep.

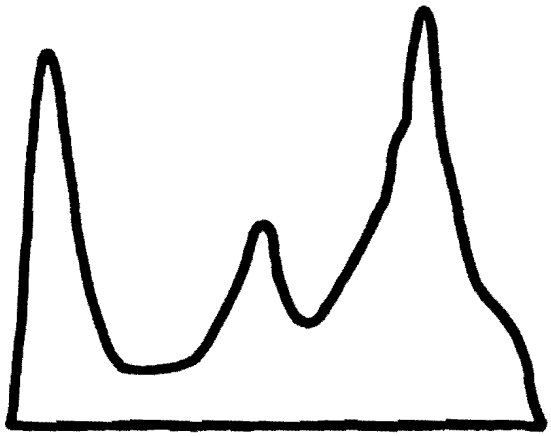


Fig. 11: Plasma protein pattern in a typical case from group 3.

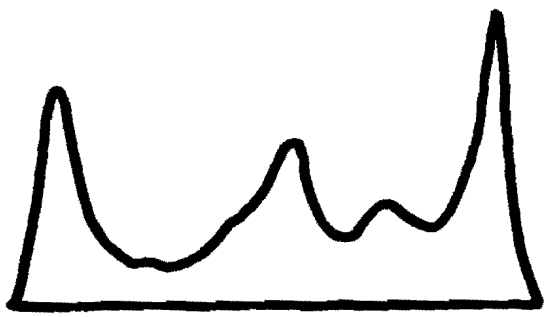


Fig. 12: Plasma protein pattern in a typical case from group 4.

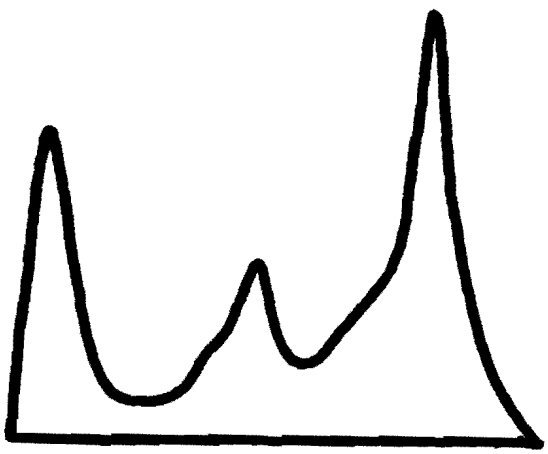


Fig. 10: Plasma protein pattern in a typical case from group 2.

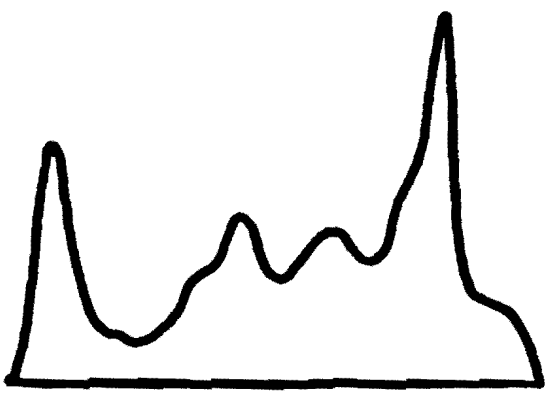


Fig. 13: Plasma protein pattern in a typical case from group 5.

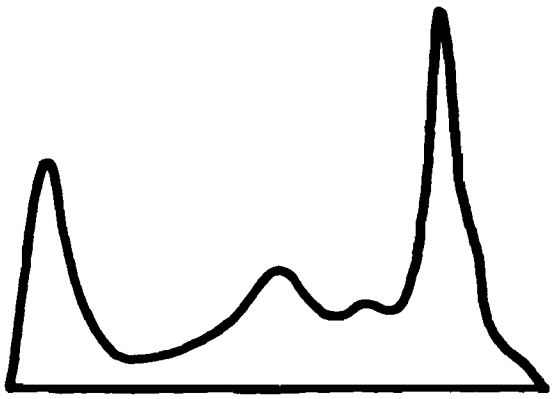


Fig. 14: Plasma protein pattern in a typical case from group 6.

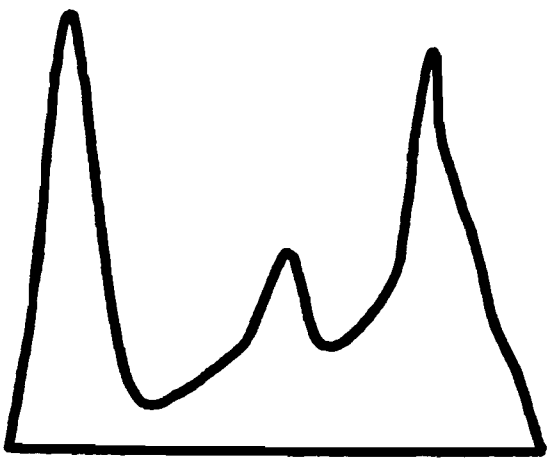


Fig. 15: Plasma protein pattern in a typical recovered animal from group 7.

The normal A/G ratio has been given for sheep raised outside the areas where geeldikkop is prevalent by Horak and Clark[13] as 0.76. The figures found for our control sheep taken from farms where the disease was active are in excellent accord with their findings. As the disease progresses there is a steady fall in the values found for plasma albumin accompanied by a marked and sustained rise in the globulin fractions. In recovered animals the A/G ration approaches the accepted normal figure once more. The marked increment in the globulin fractions is due in the first instance to a dramatic and sharp increase in the γ-globulins followed after the third day of illness by a similar but less marked increase in the α and β-globulins. These fractions are also far more clearly defined in the respective electrophoretograms.

Liver Enzymology

Since the greater part of this work will be published elsewhere only a few of the most noteworthy results have been selected for discussion in this paper.

(*a*) *Succinic dehydrogenase* (*SD.*)

The activity levels of this enzyme in the livers of clinically normal sheep born and raised outside the areas where geeldikkop is enzootic have been provisionally established as 161–350 units/100 mgm of fresh liver with a mean value of 230 units. In the livers of apparently normal sheep from farms where the disease was prevalent the range was found to be 149–265 units/100 mgm of fresh liver, with a mean value of 205 units.

The range and mean values of SD. activity encountered in the various groups of affected sheep are presented in Table 7.

During the first two days of illness there is a marked decrease in liver SD. activity. This is followed by a definite increase in the activity of this enzyme well above the levels seen in the control animals. From about 8 days after the appearance of the initial symptoms, the values

Table 7.—Succinic dehydrogenase activity in livers of natural cases of Geeldikkop.
(Activity is expressed in units/100 mgm. of fresh liver.)

Groups of sheep.....	1	2	3	4	5	6	7
Range of activity.....	149–265	162–178	129–226	226–291	129–291	204–307	194–226
Mean value.....	205	170	174	260	219	254	200

obtained for the activity of SD. approach those found in the control sheep livers once more.

(*b*) *Isocitric dehydrogenase* (ICD)

In the livers of sheep from areas other than the karoo the activity of this enzyme has been found to assay at 4.0–6.0 units/100 mgm fresh liver (mean value 4.3 units) whereas in sheep from the affected areas the range is 4.0–8.0 units with a slightly higher mean of 6.0 units/100 mgm of liver. The corresponding values found in livers of the different groups of affected animals studied are presented in Table 8.

TABLE 8.—ISOCITRIC DEHYDROGENASE ACTIVITY IN LIVERS OF NATURAL CASES OF GEELDIKKOP.
(Activity is expressed in units/100 mgm. of fresh liver).

Groups of sheep.....	1	2	3	4	5	6	7
Range of activity.....	4–8	6–10	5-18	12–26	6-12	4–14	8–9
Mean value.....	6.0	7.3	10.8	18.3	8.0	10.5	8.5

It is apparent from these results that a steady and marked increase of activity occurs in the early stages of the disease reaching its peak on the fourth day of illness and subsequently falling off slowly towards the normal levels as the sheep recover.

(*c*) *Glucose–6–phosphatase* (*G–6–P–ase*)

In Table 9 below results representative of the activity levels of this enzyme in the livers of the various groups of affected animals are presented.

TABLE 9.—GLUCOSE –6– PHOSPHATASE ACTIVITY IN LIVERS OF NATURAL CASES OF GEELDIKKOP.
(Activity is expressed in units/100 mgm. of fresh liver).

Groups of sheep.....	1	2	3	4	5	6	7
Range of activity.....	20–26	20–34	14–38	56–74	14–74	32–40	40–45
Mean value.....	23	28	22.3	64.3	31.8	35.6	42.5

As in the case of ICD there is a marked increase in G–6–P–ase activity in the livers of animals of groups 4. This activity declines somewhat thereafter to reach a fairly constant level in the remainder of the period of study, which is noticeably higher than that found in control animals.

(*d*) *Glutathione reductase* (G–R)

The tripeptide glutathione has long been known to occur in appreciable amounts in the cells of mammalian tissues e.g. erythrocytes, liver etc. In general it acts as a hydrogen carrier in cellular oxidation-reduction systems but many other important functions have recently been attached to it. Amongst the better known of these are the following: it acts as a specific coezyme activator of the enzyme glyoxalase which converts methylglyoxal to lactic acid; it is a specific coenzyme for formaldehyde dehydrogenase and maleylacetoacetate isomerase; it forms the active centre of GAP–D; it plays a special role in transpeptidation reactions where it serves as a donor of glutamic acid; it is a reducing agent in the reduction of nitrate *in vivo* and participates in the hepatic conjugation of substances like bromsulphalein.

Since SH group containing enzymes are only active in the thiol (SH) or reduced state it has been suggested that an important function of glutathione (GSH) is to keep these enzymes in the reduced form[14]. A further important function of GSH in animals is to protect ascorbic acid from oxidation. Recent work[15] seems to indicate that GSH-ascorbic acid systems may have considerable influence in controlling redox potentials in cells. Above pH 6.5 the reaction between GSH and the oxidized form of ascorbic acid viz. dehydroascorbic acid is instantaneous

and probably not reversible in tissues[16]

dehydroascorbate + 2GSH ——> Ascorbate + GSSG

This reaction occurs spontaneously, is non enzymatic and could under certain conditions act as an oxidizing system for reduced TPN (TPNH), this latter reaction being linked to glutathione reductase (GR).

GR. is an oxidized glutathione (GSSG) specific TPNH linked dehydrogenase first found in animal tissues by Meldrum and Tarr[17] and catalyses the following reaction:

TPNH + H + GSSG ——> TPN + 2 GSH

It reacts with reduced DPN (DPNH) in the same way but at a slower rate[18] GR is present mainly in the cytoplasmic sap.

zyme in the livers of these sheep. In few instances GR activity was so increased that it could not be measured accurately by the method used. The results from these animals were therefore omitted when computing the figures given for this group.

The results of the assays of these four liver enzymes which have been discussed are taken together for consideration in figure 16. The sharp decrease in SD activity in the early and latter stage of the disease is clearly discernable while in all four cases the marked increase in activity during the second and third days of illness is most prominent. This coincides with the period of maximum metabolic derangement as revealed by the blood chemistry discussed earlier.

TABLE 10.—GLUTHATHIONE REDUCTASE ACTIVITY IN LIVERS OF NATURAL CASES OF GEELDIKKOP.
(Activity is expressed in units/100 mgm. of fresh liver).

Groups of sheep.....	1	2	3	4	5	6	7
Range of activity.....	66–93	186–233	159–306	159–506	150–386	61–300	133–213
Mean value.....	80.5	210	213	382	246	284	173

In clinically normal sheep from the pool of available animals at this Institute GR activity in liver is in the order of 106–213 units/100 mgm of fresh liver (mean value 164 units). The values found in the different groups of affected animals and the control sheep used in this investigation are presented in Table 10.

Some interesting points are immediately apparent from this table. The liver activity levels of this enzyme are considerably lower in the control sheep from farms where geeldikkop is prevalent than in those clinically normal sheep studied at this Institute. With the appearance of the first symptoms of geeldikkop there is a dramatic rise in GR. activity which is sustained throughout the first week of the illness declining only slowly as the animals recover. The peak of activity is reached during the second or third day of illness. There is good correlation between the increments of GR activity and the elevations of plasma ascorbic acid. The figures given for the animals of group 4 are not a true reflection of the actual range and mean value of this en-

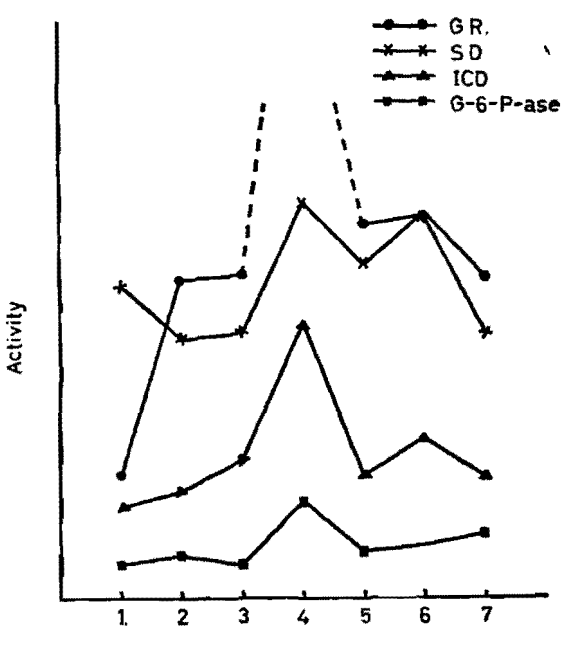


Fig. 16: Schematic representation of the levels of activity of glutathione reductase (GRD), succinic dehydrogenase (SD), isocitric dehydrogenase (ICD) and glucose-6-phosphatase (G-6-P-ase) in the livers of typical cases of geeldikkop.

The Nature of the Erythrocyte Lesions

A marked increase in erythrocyte fragility is a constant feature of both geeldikkop and enzootic icterus. It is related to the methaemoglobin-cythaemia which is sometimes seen in these syndromes and was thought to be due to inactivation of G–6–P–D[1, 2].

It has now been demonstrated that G–6–P–D activity is normally negligable in ovine erythrocytes and that in these cells glycolysis proceeds actively through the direct pathway. Methaemoglobin reductase in sheep red bloodcells has been shown to be DPN linked and dependant on GAP-D for hydrogen supply. The essential erythrocyte lesion in either syndrome appears to be marked suppression of GAP-D activity.

Since this subject will be treated in greater detail in a separate paper only the findings relative to the general discussion in this paper will be present at this stage. Figure 17 below represents results indicating the levels of G–6–P–D activity found in the erythrocytes of different species. The assay method depends on the rate of reduction of TPN by the enzyme in the presence of G–6–P, and the figure depicts decrease in TPN concentration plotted against time in seconds.

The negligable activity of G–6–P–D in the erythrocytes of sheep and goats is obvious compared with that in the red cells of other species.

The erythrocytes of clinically normal sheep raised outside of the areas in which geeldikkop is enzootic are very resistant to the hypotonic effects of 0.7 per cent saline. Significant differences have been detected in this respect between these animals and those from the areas in which the disease occurs[19]. The same differences have been found to pertain in the case of GAP-D activity in erythrocytes[20].

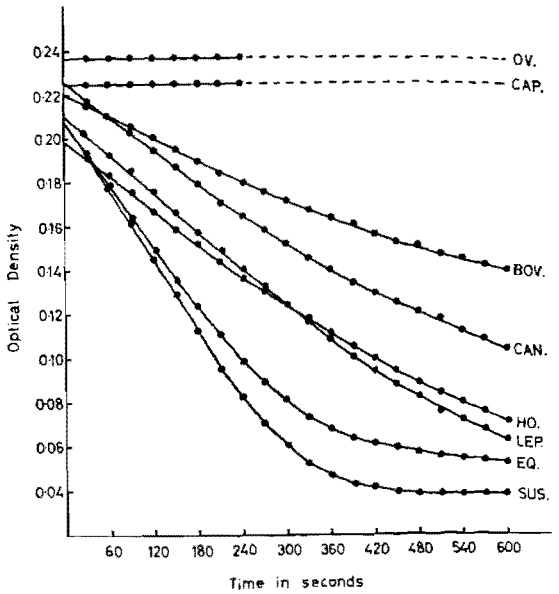


Fig. 17: Glucose-6-phosphate dehydrogenase activity in the erythrocytes of different species — typical analytical data.
OV = Sheep, CAP = goat, BOV = bovine, CAN = dog, HO = Human, EQ = horse, SUS = pig.

A marked and progressive decrease in GAP-D activity is obvious in early cases of geeldikkop and this persists even in recovered animals. Excellent correlation of this with increased red cell fragility has been found, both disturbances being maximal during the second and third days of illness. In Table 11 a comparison is drawn between the data obtained from the control animals of group 1 and those of group 4 in which the lesions are most prominent. The trends seen in red cell fragility and GAP-D activity throughout the course of the disease are portrayed schematically in Figure 18 which follows:

TABLE 11.—ERYTHROCYTE FRAGILITY AND GLYCERALDEHYDE-PHOSPHATE DEHYDROGENASE ACTIVITY IN CLINICALLY NORMAL ANIMALS AND CASES OF GEELDIKKOP.

Groups of Animals	rbc. fragility	GAP-D activity
Group 1	Range 0.2–14 mean 4.1	Range 225–530 mean 440
Group 4	Range 55–80 mean 73	Range 250–375 mean 317

NOTE: Fragility is expressed as % of cells haemolysed in 0.7% saline and GAP-D activity as units/100 ml. of washed erythrocytes.

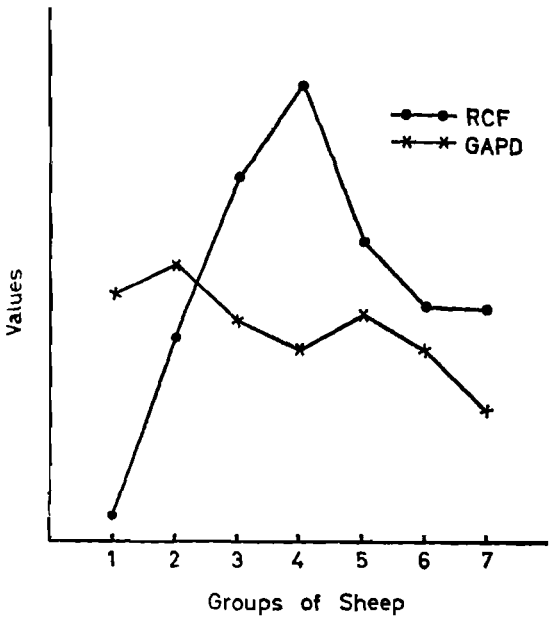


Fig. 18: Schematic representation of glyceraldehyde fragility (RCF) in the erythrocytes of typical cases of geeldikkop.

Porphyrin Metabolism in Geeldikkop

Wethers representing various stages of the disease were chosen for urinary and faecal porphyrin excretion studies, phylloerythrin (phyllo) and erythrocyte protoporphyrin (proto) being determined in all the experimental animals.

In general no significant variations in red cell proto could be detected between the various groups of experimental animals. Values found in the control sheep ranged from 0.05–0.12 mcg./100 ml washed erythrocytes (mean 0.08 mcg%) as compared with a range of 0.02–0.22 mcg% (mean 0.07 mcg%) in all the affected animals.

The values found for plasma phyllo are most interesting in view of the postulated role of this porphyrin as the photodynamic agent in geeldikkop. The relevant data for each group are presented in Table 12.

An extremely sharp rise in plasma phyllo values is seen during the first day of illness, when the animals are most severely photosensitive. Maximum levels are attained two days after the appearance of the first symptoms. Thereafter there is a decline towards normal levels. Values considerably higher than in the control animals are found in individuals which show no signs of photosensitivity and even in some apparently recovered animals. There is an excellent correlation between the values for plasma phyllo, bile salts and bilirubin throughout the course of the disease but this does not extend to attempts to relate clinical photosentivity to plasma phyllo levels.

The pattern of urinary excretion of coproporphyrin (copro) can be perfectly related to photosensitivity and its disappearance during recovery. No attempt was made during this investigation to separate the copro. isomers I and III. The figures presented here represent total urinary coproporphyrins.

The daily urinary excretion of copro. in young wethers was found to be in the order of 10–32 mcg. A marked rise in the amount excreted occurs on the first day of illness, maximum levels of excretion being observed during the second day. The range of copro excretion at this time was found to be 450–4089 mcg *per diem* in severe photosensitive wethers. Disappearance of photosensitivity was accompanied by a rapid fall in the amount of copro being excreted, the normal excretory pattern being attained on the fourth day of illness. The pattern of urinary copro excretion is shown in histogram form in figure 19.

The marked increase in urinary copro excretion is accompanied by an equally rapid decline

Table 12.—Plasma phylloerythrin levels in clinically normal animals and cases of geeldikkop. (Values are expressed as mcg./100 ml. plasma).

Groups of sheep	1	2	3	4	5	6	7
Range	0.9–5.8	22.0–47.0	10.0–119.0	3.6–28.0	2.9–23.0	2.2–9.6	0.98–13.0
Mean	4.4	38.0	60.0	16.7	9.9	5.8	6.16

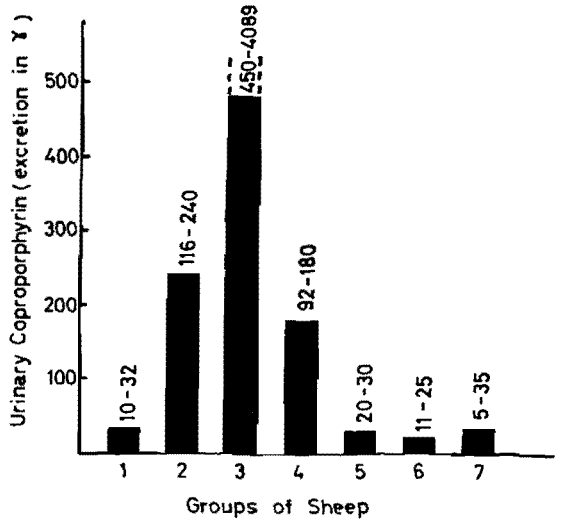


Fig. 19: Urinary coproporhyrin excretion patterns in geeldikkop.

in faecal copro excretion. Faecal proto. values were variable throughout the course of the disease and at no time did any of the sheep excrete more than traces of uroprophyrin in the urine.

Bilirubin Conjugation and the Hepatic Transfer of Bilirubin

The conjugation of bilirubin, various phenols and similar aglycones with glucuronic acid occurs in the microsomes of the liver cell[21, 22, 23] and in the case of bilirubin is believed to take place according to the sequence of events shown in figure 20 below[21, 24, 25, 26].

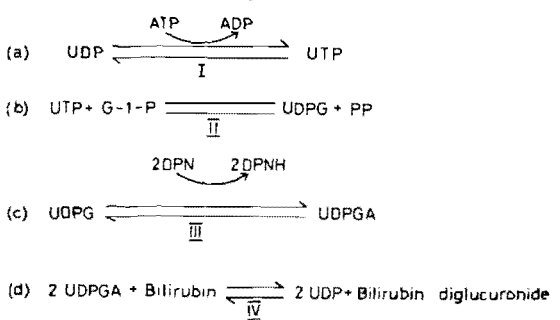
The overall conjugation of bilirubin was studied in control and affected animals *in vitro* using liver homogenates, the appropriate co-factors and pure crystalline bilirubin bound to albumin as performed by Grodsky and Carbone[27]. Using the same homogenate, assays were made of UDPG-D activity and UDPG content. The results are presented in Table 13 below.

There appears to be some decrease in the conjugating ability of the liver during the initial three days of illness. Thereafter there is a marked increase in the efficiency of the conjugation systems in the affected animals, particularly noticeable in the recovered animals. UDPG formation appears to be unimpaired and maintained at a steady rate, whereas UDPG-D activity appears to increase markedly in the early stages of the disease returning to control levels of activity as the clinical symptoms and biochemical disturbances subside.

Bromsulphalein (BSP) Metabolism

Earlier findings with respect to BSP metabolism[2] have been confirmed. Affected sheep of Group 3 with common bile duct cannulae fail to excrete any BSP conjugates in the bile up to 24 hours after intravenous administration of loading doses of the dye. BSP is rapidly cleared from the systemic circulation in these cases, negligable amounts being detected in the plasma at 30 minutes after injection. In affected animals where renal function is unimpaired fair amounts of BSP conjugates previously designated BSP. A and BSP. B[2] appear in the urine within four hours of administration of the dye.

Since these conjugates appear to be the major metabolites of this dye in sheep, conjugation of BSP appears to be unimpaired. Its transfer across the hepatic cell membranes appears however to be effectively blocked.



[abbreviations used :- UDP = uridine diphosphate

UTP = uridine triphosphate ; G-1-P = glucose-1-PO_4

UDPG = uridine diphosphoglucose

UDPGA = uridine diphosphoglucuronic acid

I = nucleoside diphosphate kinase

II = UDPG-pyrophosphorylase ; III = UDPG-dehydrogenase

IV = UDP-transglucuronylase or glucuronyl transferase]

Fig. 20: The mechanism of bilirubin conjugation in the liver.

TABLE 13.—BILIRUBIN CONJUGATING ACTIVITY UDPG CONTENT AND UDPG-D ACTIVITY IN THE LIVERS OF CONTROL ANIMALS AND CASES OF GEELDIKKOP

Group of Sheep	Percentage of bilirubin conjugated (range)	UDPG (mean)	UDPG-D (mean)
1	7–10	0·004	66
2	3–7	0·004	182
3	2–9	0·001	98
4	1–15	0.005	83
5	10–20	0.005	130
6	6–16	0.007	69
7	20–21	0.003	70

NOTE: UDPG values are mcg/100 mgm. liver; UDPG-D values are units/100 mgm. fresh liver. Percentage of bilirubin conjugated is the percentage of added bilirubin recovered as glucuronide at the end of incubation.

LIVER ENZYMOLOGY IN ICTEROGENIN INTOXICATION

The icterogenic triterpenes induce a syndrome in sheep clinically similar to natural geeldikkop yet differing from it in many important respects[2, 3]. Since the biochemical nature of the icterus and photosensitivity and the hepatic histopathology are essentially similar in both instances[2, 5] a considerable amount of attention has been paid to the icterogenic effects of these compounds[8, 28, 29, 30] in the hope of elucidating the disturbances of biliary excretion in diseases of this nature.

SD., GAP-D, bilirubin conjugation and the levels of activity of various enzymes associated with terminal electron transfer chains and oxidative phosphorylation were studied in the livers of rabbits before and after intoxication as explained earlier. Icterogenin was administered intra-abdominally at a dosage level of 100 mgm/Kg. body weight. In all cases the typical icterogenic effects were observed[8, 29, 30].

The results of the liver enzyme assays in four typical experiments are presented in Table 14.

TABLE 14.—LIVER ENZYMOLOGY IN RABBITS POISONED WITH ICTEROGENIN.
(Enzyme activities are expressed as units/100 mgm. of fresh liver tissue. Bilirubin conjugating ability is expressed as the percentage of bilirubin added to the reaction medium recovered as glucuronide)

Rabbit No.		SD.	GAP-D	DPN-ase	Cyto-c-red	Diaphorase	ATP-ase	UDPG-D	Per cent Bilirubin conjugation
1	Control values	356	76	2.70	1.20	4.4	279	100	4.86
	After intoxication	259	69	2.30	0.72	5.0	321	100	3.57
2	Control values	259	118	2.90	1.96	3.9	276	100	7.84
	After intoxication	226	72	2.90	1.68	3.3	207	95	5.45
3	Control values	450	102	2.47	2.22	4.8	339	135	3.75
	After intoxication	291	88	2.37	2.48	4.4	237	145	1.81
4	Control values	424	92	3.07	1.64	1.0	264	125	5.88
	After intoxication	259	76	2.90	1.28	1.3	210	175	3.33

Some reduction in the ability of the liver to conjugate bilirubin following intoxication by icterogenin is evident in all the experimental animals. UDPG-D activity is in general either unaffected or increased at the end of the test period. The most noteworthy effects of the intoxication are a marked decrease in the activity of SD and GAP-D, the two dehydrogenases most severely affected in natural geeldikkop. The various enzymes associated with the respiratory chain and oxidative phosphorylation do not appear to be significantly affected, although some reduction in adenosine triphosphatase (ATP-ase) activity is evident in most cases. It would appear at this stage of our work that the icterogenic triterpenes do not exert any significant effect on mitochondrial respiratory chains but have rather a selective depressant effect on the activity of certain vital dehydrogenases e.g. SD and GAP-D. The nett effect of this action would be to deprive the cell of a large amount of energy which could be derived from direct glycolysis or TCA cycle oxidations. The decrease in conjugating ability with respect to bilirubin is probably associated with this failure in energy production.

Discussion

Since the sheep used in the present investigation were maintained under closely controlled conditions the greatest care was taken to prevent the appearance of secondary complications such as extensive skin necrosis in affected animals, infection of the lesions of photosensitization, and severe gastro-intestinal atony. The careful handling of these animals served to arrest the progress of the kidney lesions generally seen[1,2,5] and in most cases prevented the catastrophic adrenal collapse seen in so many cases in the field[1,2].

The biochemical changes which have been observed throughout the course of the disease are thus probably a true reflection of the metabolic disorder in uncomplicated geeldikkop.

The primary biochemical lesions in geeldikkop are of a highly selective nature. Only those dehydrogenases known to contain SH groups appear to be affected, while the activity of SH-group free enzymes, e.g. ICD and LDH is markedly increased. Whether the high levels of selenium present in the livers of affected animals are involved in the supression of activity of enzymes like SD and GAP-D must still be proved.

The biochemical nature of the icterus and disturbances in porphyrin metabolism are essentially similar in geeldikkop and icterogenin intoxication and in both instances the same dehydrogenases are affected. One could by analogy infer that as in the latter case the metabolic disturbances seen in geeldikkop are not due to mitochondrial disruption or damage to the electron transfer chain but are of a much more subtle nature. That the selective permeability of the hepatic cell membrane is undoubtedly altered is illustrated by the marked leakage of certain enzymes out into the plasma e.g. ICD, LDH, GOT, PHI and aldolase during the highly photosensitive stages of the disease. I do not propose to deal in detail with the significance of the elevations in plasma activity of these enzymes. Such findings are frequent in acute hepatic disease and in many conditions involving tisse necrosis[31–34]. A quotation from a recent paper by Sibley[32] is most relevant to this discussion particularly in view of the hepatic histopathology of geeldikkop[5]: ". . . abnormally high serum aldolase content results from the rather sudden injury of many cells. It is probably more exact to speak of tissue injury which would include relatively mild and sometimes reversible changes in the cell membrane, rather than to use the term necrosis, which implies a destruction of cells demonstrable by histological examinations. This concept would explain those instances of elevation of serum enzyme values without finding gross or microscopic evidence of necrosis of some tissue."

The study of intrahepatic cholestatic states has evoked considerable universal interest within recent years particularly with regard to those seen in the new-born. These conditions involve in general failure of bilirubin conjugation or interference with its passage through the liver cell wall. Some of these states have a heriditary or teratological background, others are essentially of toxic origin. The mass of relevant literature is formidable. Suffice it to

say that the list of substances now known to induce icteric states highly reminiscent of geeldikkop is a long one and includes substances of widely differing natures and structures, e.g. novobiocin, numerous steroids, pentacyclic triterpene acids, vitamin K analogues and certain iodinated cholecystographic contrast media. In general the mode of action of these compounds in producing hyperbilirubinaemic states appears to be (*a*) competition with bilirubin for gluronide conjugation, (*b*) inhibition of UDPG-D, (*c*) inhibition of glucuronyl transferase, (*d*) competition for excretion of glucuronides in the liver cell, (*e*) alterations in the permeability of the cell wall to these conjugates[1, 2, 8, 22, 23, 28, 35–38].

Although some evidence of decreased hepatic conjugating ability is seen in the case of bilirubin in both geeldikkop and icterogenin poisoning, there is no evidence of failure of UDPG supply to the conjugation systems or of direct inhibition of UDPG-D or glucuronyl transferase. The present work has largely confirmed that the essential lesion must be one in the transport of conjugates through the liver cell wall. The interference in excretion extends to quite a few compounds of different natures, e.g. bilirubin glucuronides, BSP conjugates, porphyrins, bile acids, alkaline phosphatase and copper[2]. It is not known whether the mechanism of excretion is the same in all instances.

Since the selective permeability of cell membranes is known to be ATP dependent in many cases, it is tempting to suggest that in the cases of the compounds known to be retained in geeldikkop and icterogenin intoxication the energy required for excretion is somehow directly linked with the dehydrogenases which are affected viz. SD and GAP-D and possibly the latter in particular since the greatest metabolic disturbances appear to lie in carbohydrate metabolism. Most of the glycolytic processes are known to proceed outside of the mitochondria[9].

The markedly retarded removal from the circulation of parenterally administered glucose and the gross elevations of plasma lactate seen during the early stage of the disease are of great interest. These findings are difficult to explain at the moment. Since the activity of both SD and GAP-D is severely depressed the tissue cells will be deprived of a large amount of energy normally derived from the action of these enzymes and their associated electron transfer chains. It is therefore not surprising that there is a corresponding increase in the activity of dehydrogenases like ICD which are unaffected. The apparent increase in G–6–P–ase activity in liver is probably likewise a compensation on the part of this organ to provide blood-sugar at a faster rate than usual from any G–6–P which is being formed.

The significance of the huge increase in GR. activity in the livers of affected animals is most difficult to interpret without indulging in speculation at this stage. If this is permissable then it is tempting to couple this fact with the lactic acidaemia and the elevated plasma ascorbate values seen in early cases in the manner indicated in Figure 21.

The scheme presented here is in part an established metabolic pathway,[15, 25] the significance of which is not fully understood at the moment. If the rate of glucose oxidation or of glycogen formation from pyruvate is effectively reduced by lowered GAP-D activity then this pathway could provide a convenient bypass of the metabolic block assuming that the unknown mechanism of the first is reversible. Should this be the case one could expect a compensatory increase in the activity of the catalysts concerned in the various steps shown in figure 21.

Since GSH is dependant on either ascorbate or TPNH linked GR to keep it in its reduced form a compensatory increase in the effective levels of ascorbate and GR is logical. If, as is likely, dehydrogenases like the "malic enzyme", ICD (as demonstrated) or GAD, which are either wholly TPN dependant or TPN or DPN linked, show a marked increase in activity then the reaction;

$$\text{TPNH} + \text{H} + \text{GSSG} \longrightarrow 2\text{TPN} + 2\text{ GSH}$$

catalysed by GR may assume far greater importance than is usually the case.

It has for long been known that increased urinary porphyrin levels are associated with hepatic disease. Coproporphyrinuria is a feature of hepatic cirrhosis of alcoholic or non-alcoholic origin, infectious hepatitis, extra-

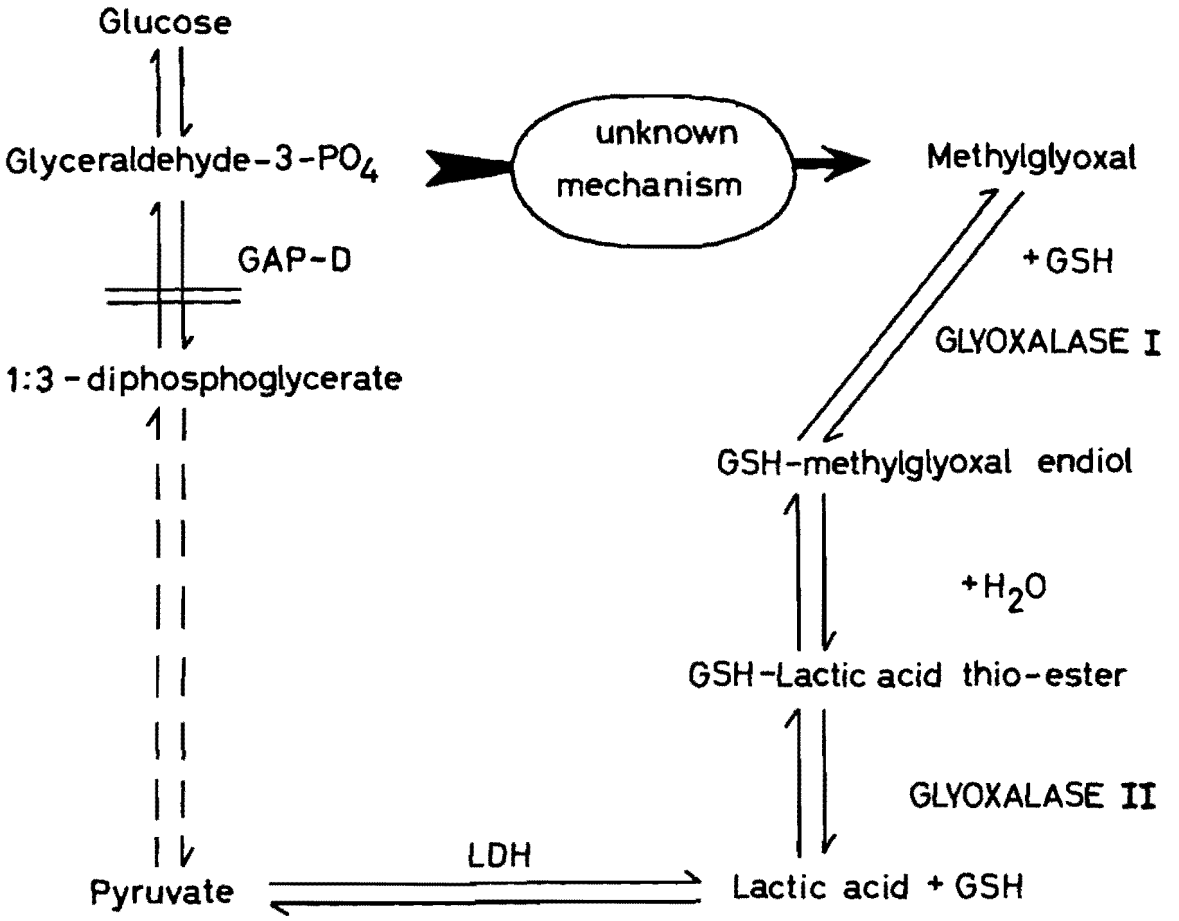


Fig. 21: The glyoxalase pathway from glyceraldehyde phosphate to lactic acid or pyruvate.

hepatic biliary obstruction and various intoxications, e.g. with lead, arsenic, beryllium, aniline, nitrobenzene, sulphonamides etc[39]. Roets[40] has demonstrated increased urinary coproporphyrin excretion in East Coast Fever.

On the face of it the coproporphyrinuria seen in geeldikkop suggests that this syndrome is a further example of a symptomatic hepatic porphyria. When the known epizootology, the liver pathology and peculiar nature of the biochemical lesions are carefully considered and taking the associations between this syndrome and enzootic icterus into account the entire disease complex must be classified as a special type of cutaneous hepatic porphyria (CHP). Porphyrias of this group may be the result of many different aetiological factors — a hereditary defect of porphyrin metabolism in the liver, a variety of hepatotoxic processes, a simple liver neoplasm and many more. All have however a number of important features in common, viz. (i) in all cases the liver is assumed to *form* excessive amounts of porphyrins which are normally excreted via the bile. As long as this route of excretion is efficient, symptoms are unlikely. In the event of hepatic damage porphyrin excretion is deviated through the kidneys and while large amounts of these porphyrins are in the blood skin manifestations are evident; (ii) in all cases acute attacks are precipitated in asymptomatic individuals by a variety of conditions which can be regarded as stress factors, e.g. excessive alcohol consumption, barbiturate administration etc. Severe icterus is often but not always associated with the acute episodes;

(iii) the liver pathology encountered in CHP has been reviewed by Rimington[39] and varies from mild fatty infiltration or minor reticulin changes (cf. geeldikkop) to frank cirrhosis in advanced cases (cf. enzootic icterus).

The essential biochemical abnormality in CHP is excessive hepatic porphyrin production induced by the aetiological factors mentioned and coupled with a temporary and unexplained inability of the liver to excrete these porphyrins during acute episodes. In the case of geeldikkop the liver does not suddenly elaborate more porphyrins than usual. The analogy lies in the extra porphyrin load in the form of phyllo. which is being continually formed in large amounts in the digestive tract of the ruminant. Once this is appreciated the similarity between geeldikkop and CHP is obvious.

From the data presented earlier there seems little doubt that when phyllo is present in very large amounts in the plasma it acts as a potent photodynamic agent. The better correlation between urinary coproporphyrin and clinical photosensitivity indicates that this porphyrin may be quantitatively more important in this respect.

One of the most interesting aspects of the present study is the plasma protein pattern throughout the course of the disease. Margen and Tarver[41] have calculated, by isotopic work on humans, the half life of albumin in the body to be 26 days. Assuming that the rate of protein catabolism does not change, it will take 16 days before a 50 per cent reduction in the rate of albumin synthesis leads to a decrease of 20 per cent in the plasma albumin concentration. Even a complete cessation of albumin production will result only in a 20 per cent decrease in plasma albumin after about 8 days (their figures). There is no reason to assume that this situation is peculiar to man only. Horak and Clark[13] have deduced a similar turnover rate in sheep. In the light of these findings it is inconceivable that the marked alterations in the A/G ratio can be related to liver cell injury. It must be borne in mind that the changes indicative of hepatic disturbances are observed virtually simultaneously with the rises in plasma globulins.

While interpreting the results presented in table 6 the following points must be borne in mind. Horak and Clark[13] have commented on the poor resolution of the globulin fractions in the method of paper electrophoresis which has been used. Earlier in the text it has been noted that sharper definition of these fractions is obtained as the disease progresses. This is readily seen by inspection of the electrophoretograms shown earlier. In groups 4 and 5 there appears to be some reduction in the albumin content of plasma. This is accompanied by an increase in the undifferentiated protein fraction. Since the amounts of each protein fraction are calculated planimetrically from the electrophoretogram and the total protein figure[42] any increase in the undifferentiated fraction occasioned by a greater spread of the protein bands on the electrophocetogram will lead to lower figures being obtained for the albumins. When the globulin peaks are clearly defined much of the undifferentiated fraction will undoubtedly be unresolved albumins since these are not separated at all by this method. The noted alterations in albumin levels are therefore more apparent than real. Since albuminuria is negligable in geeldikkop in spite of the nephrosis generally observed[2] loss of albumin from the circulation must be negligible. One must conclude therefore that there is an absolute increase in globulins, this being due largely to γ-globulins and therefore indicative in this instance of an immune body reaction.

Theiler in his initial studies[43] drew attention to the high temperatures observed in many of his experimental animals and the similarity of the temperature charts of these animals to those seen in cases of ovine bluetongue. The possible role of virus disease participating as stressors in the aetiology of geeldikkop has been noted previously[1, 2, 5]. During the present investigation temperatures of 103–105° were noted in all the experimental animals during the first three days of illness. Temperatures were usually taken at 8 a.m. and 10 p.m. The presence of coronitis in early cases of geeldikkop has been noted earlier[5]. Close study of all the affected animals used in this investigation showed that this feature was in actual fact not a pododermatitis but diffuse coronary band haemorrhages highly reminiscent of bluetongue. Although this

lesion was a constant feature of the cases studied no mouth lesions typical of the latter disease were found. In some animals a fair to marked pulmonary oedema was evident. Most of the cases studied had not been innoculated against bluetongue, the remainder had been within a week before developing geeldikkop.

As yet we have been unsuccessful in isolating any infectious agent from the blood and tissues of all the cases studied and this has been the experience of workers in the past[3].

Notwithstanding these failures the evidence in favour of an infectious agent acting as a potent stressor is now considerable. The epizootology is largely in favour of this, the plasma protein patterns support it, the leukopaenia observed in early cases is another consideration,[5] and Pienaar of this Institute has found inclusion bodies in the nuclei of liver cells in typical cases of enzootic icterus. (Personal communication). Previous work has shown that profound biochemical and haematological disturbances are evident in affected animals even before the appearance of the typical symptoms of geeldikkop[2, 5].

In conclusion some remarks regarding the known effects of some fungal toxins on hepatic cells are not out of place in view of the possible role of these in the aetiology of geeldikkop. Brown and Abrams[11] working with the potent hepatotoxins of *Aspergillus flavus* and *A. ampstelodami* have demonstrated severe inhibition of SD. and certain enzymes of the respiratory chain, e.g. Cyto-c-red. diaphorase and DPN-ase in the livers of Pekin ducklings and New Hampshire chickens. There is severe mitochondrial damage, failure of ATP synthesis and marked reduction in protein synthesis which is ATP dependant. Gallagher and others[9] working with Sporidesmin, the toxin elaborated by *Pithomyces chartarum* have demonstrated severe mitochondrial damage in the livers of animals affected by this toxin, with consequent failure of succinoxidase activity and cellular respiration. The disturbances noted by Brown and Abrams[11] can be detected long before visible structural damage to the liver is apparent. The nature of fungal toxins varies tremendously and their action on mammalian cells can be expected to vary just as much. Such compounds which are able to reproduce the disturbed biochemistry of geeldikkop may yet be found.

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REFERENCES

1. BROWN, J. M. M. (1962). *J.S. Afr. vet. med. Ass.*, **33,** 493.
2. BROWN, J. M. M. (1963). *Ann. N.Y. Acad. Sci.*, **104,** 2, 504.
3. BROWN, J. M. M. (1959). *J.S. Afr. vet. Med. Ass.*, **30,** 97.
4. BROWN J. M. M. (1959). *J.S. Afr. vet. Med. Ass.*, **30,** 403.
5. BROWN, J. M. M., LE ROUX, J. M. W. and TUSTIN, R. C. (1960). *J.S. Afr. Vet. med. Ass.*, **31,** 179.
6. BROWN, J. M. M. and DE WET, P. J. (1962). *Ond. J. Vet. Res.*, **29,** 111.
7. BROWN, J. M. M. (1959). *J.S. Afr. vet. med. Ass.*, **30,** 4.
8. BROWN J. M. M., RIMINGTON, C. and SAWYER, B. C. (1963). *Proc. Roy. Soc. B.*, **157,** 473.
9. GALLAGHER, C. H. (1964). Nutritional factors and enzymological disturbances in animals. Crosby Lockwood and Son, London.
10. KLUG, H. L., MOXON, A. L., PETERSEN, D. F. and POTTER, van R. (1950). *Arch. Biochem.*, **28,** 253.
11. BROWN, J. M. M. and ABRAMS, L. (1964). *Ond. J. Vet. Res.* — in Press.
12. VERSCHURE, H. and HOEFSMIT, C. (1956). *Clin. Chim. Acta.*, **1,** 38.
13. HORAK, I. G. and CLARK, R. (1963). *Ond. J. Vet. Res.*, **30,** 145.
14. DIXON, M. and WEBB, E. C. (1958). Enzymes. Longmans, Green and Co., London, N.Y., Toronto.
15. WEST, E. S. and TODD, W. R. (1952). Textbook of biochemistry, MacMillan Co., N.Y.
16. BURNS, J. J. (1960). In Metabolic Pathways, Vol. I ed. D. M. Greenberg. Academic Press, N.Y. and London.
17. MELDRUM, N. V. and TARR, H. L. A. (1935). *Biochem. J.*, **29,** 108.
18. KUN, E. (1961). In Metabolic Pathways, Vol. 2, ed. D. M. Greenberg. Academic Press, N.Y. and London.
19. WAGNER, A. M. (1964). *Ond. J. Vet. Res.*, **31,** 77.
20. WAGNER, A. M. — unpublished observations.
21. DUTTON, G. J. (1963). *Ann N.Y. Acad. Sci.*, **111,** 1, 259.
22. BILLING, B. H., MAGGIORE, Q. and CARTTER, M. (1963). *Ann. N.Y. Acad. Sci.*, **111,** 1, 319.
23. ARIAS, I. M., GARTNER, L., FURMAN, M. and WOLFSON, S. (1963). *Ann. N.Y. Acad. Sci.*, **111,** 1, 274.
24. GRANICK, S. and MAUZERALL, D. (1961). In Metabolic Pathways, Vol. 2 ed. D. M. Greenberg. Academic Press, N.Y., London.
25. UMBREIT, W. W. (1960). Metabolic Maps. Vol. 2. Burgess Publishing Co., Minneapolis.
26. PONTIS, H. G., and LELOIR, L. F. (1962). In Methods of Biochemical Analysis, Vol. 10, ed. D. Glick. Interscience Publishers, N.Y. and London.
27. GRODSKY, G. M. and CARBONE, J. V. (1957). *J. Biol. Chem.*, **226,** 449.
28. HEIKEL, T., KNIGHT, B. C., RIMINGTON, C., RITCHIE, H. D. and WILLIAMS, E. J. (1960). *Proc. Roy. Soc. B.*, **153,** 47.
29. BROWN, J. M. M. and RIMINGTON, C. (1964). *Proc. Roy. Soc. B.*, **160,** 246.
30. BROWN, J. M. M., ANDERSON, L. A. P. and DE KOCK, W. T. (1963). *J. S .Afr. Lab. Clin. Med.*, **9,** 263.
31. WROBLEWSKI, F. (1958). *Ann. N.Y. Acad. Sci.*, **75,** 1, 322.
32. SIBLEY, J. A. (1958). *Ann. N.Y. Acad. Sci.*, **75,** 1, 399.
33. WHITE, L. P. (1958). *Ann. N.Y. Acad. Sci.*, **75,** 1, 349.
34. WOLFSON, S. K., SPENCER, J. A., STERKEL, R. L. and WILLIAMS-ASHMAN, H. G. (1958). *Ann. N.Y. Acad. Sci.*, **75,** 1, 260.
35. FLINT, M., LATHE, G. H. and RICKETTS, T. R. (1963). *Ann. N.Y. Acad. Sci.*, **111,** 1, 295.
36. YI-YUNG HSIA, D., DOWBEN, R. M., and RIABOV, S. (1963). *Ann. N.Y. Acad. Sci.*, **111,** 1, 326.
37. SUTHERLAND, J. M. (1963). *Ann. N.Y. Acad. Sci.*, **111,** 1, 461.
38. SILVERBERG, M., DESFORGES, J. and GELLIS, S. (1963). *Ann. N.Y. Acad. Sci.*, **111,** 1, 472.
39. GOLDBERG, A. and RIMINGTON, C. (1962). Diseases of Porphyrin Metabolism. Charles C. Thomas. Springfield III.
40. ROETS, G. C. S. (1938). *Ond. J. Vet. Sci.* and *An. Ind.*, **11,** 2, 385.
41. MORGEN, S. and TARVER, H. (1956). *J. Clin. Invest.*, **35,** 1161.
42. KING, E. and WOOTTON, I. D. P. (1958). Microanalysis in Medical Biochemistry. 3rd Ed. J.A. Churchill, London.
43. THEILER, A. (1918). 7th and 8th Repts., *Dir. Vet. Res.*, **1,** 55.
44. RIMINGTON, C. (1958). Broadsheet No. 21 (New Series), Assoc. of Clinical Pathologists London.
45. GRINSTEIN, M. and WATSON, C. J. (1943). *J. Biol. Chem.*, **147,** 675.
46. PERRIN, D. D. (1958). *Biochem. J.*, **68,** 314.
47. TAYLOR, T. H. and FRIEDMAN, M. E. (1960). *Clin. Chem.*, **6,** 208.
48. OCHOA, S. (1948). *J. Biol. Chem.*, **174,** 133.
49. WROBLEWSKI, F. and LA DUE, S. (1955). *Proc. Soc. Exptl. Biol. Med.*, **90,** 210.
50. KING, J. (1958). *J. Med. Lab. Technol.*, **15,** 17.
51. BODANSKY, O. (1954). *Cancer*, 7, 1191.
52. AUGUSTINSON, K. B. (1957). In Methods of Biochemical Analysis, Vol. 5, ed. D. Glick. Interscience, N.Y. and London.
53. SIBLEY, J. A. and LEHNINGER, A. L. (1949). *J. Biol. Chem.*, **177,** 859.
54. SCLATER, E. C. and BONNER, W. D. (1952). *Biochem. J.*, **52,** 185.
55. SWANSON, M. A. (1950). *J. Biol. Chem.*, **184,** 647.
56. RACKER, E. (1957). In Methods in Enzymology, Vol. 2 ed. Colowick, S. P. and Kaplan, N. O. Academic Press, N.Y.
57. Sigma Tech. Bull. No. 10 (1961); Sigma Chemical Co., St. Louis, Mo.
58. KORNBERG, A. and HORECKER, B. L. (1957). In Methods in Enzymology, Vol. 1, ed. Colowick, S. P. and Kaplan, N. O., Academic Press, N.Y.

59. STROMINGER, J. L., MAXWELL, E. S. and KALCKAR, H. M. (1957). In Methods in Enzymology, Vol. 3, ed. Colowick, S. P. and Kaplan, N. O., Academic Press, N.Y.
60. UMBREIT, W. W., BURRIS, R. H., and STAUFFER, J. F. (1964). Manometric Techniques, Burgess Publishing Co. Minneapolis.
61. ZATMAN, L. J., KAPLAN, N. O., and COLOWICK, S. P. (1953). *J. Biol. Chem.*, **200**, 197.
62. MAHLER, H. R., SARKAR, N. K., VERNON, L. P. and ALBERTY, R. A. (1952). *J. Biol. Chem.*, **199**, 585.
63. BARKER, S. B. and SUMMERSON, W. H. (1941). *J. Biol. Chem.*, **138**, 535.
64. LEHMAN, H. and SILK, E. (1952). *Biochem. J.*, **50**, 31.
65. FISHMAN, W. H. and GREEN, D. E. (1955). *J. Biol. Chem.*, **215**, 527.
66. WEICHSELBAUM, T. E. (1946). *Am. J. Clin. Path.*, **16**, 40.
67. IRVIN, J. L., JOHNSTON, C. G. and KOPALA, J. (1944). *J. Biol. Chem.*, **153**, 439.
68. MALLOY, H. I. and EVELYN, K. A. (1937). *J. Biol. Chem.*, **119**, 481.
69. BROWN, J. M. M. — Unpublished method.

THE RELATIONSHIPS BETWEEN HELMINTH INFESTATION, PRODUCTION AND NUTRITION

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INTRODUCTION

The sheep and cattle populations in South Africa are of enormous value; the former are primarily responsible for a conserable percentage of the valuta earnings while the latter make a considerable contribution to the production of essential foods for the inhabitants of this country.

Pasture growth in South Africa is characterized by seasonal non-uniformity both in quantity and quality mainly as the result of climatic vagaries. The efficient conversion of pasture herbage by cattle and sheep into products of agricultural value is a matter of prime importance. One of the most important factors militating against efficient feed conversion is helminth infestation.

Nematodes parasitic in the alimentary tract have been shown adversely to affect the productivity of the host (Gibson 1951,[11] Spedding 1954,[25] Andrews *et al* 1944[2]). There is a significant depression of growth rate associated with subclinical worm burdens (Spedding and Brown 1957[27]). The effects of a worm infestation can persist for up to a year and infected sheep do not catch up even if the infestation is eliminated. As stated by Hammond (1932)[17] "A check in growth received early in life affects the ultimate size of the animal".

THE EFFECT OF HELMINTH INFESTATION ON THE ANIMAL

The deleterious effects of a helminth infestation on the host is envinced by:

(*a*) Reduction of appetite.
(*b*) Impaired digestive efficiency.
(*c*) Pathogenesis.

REDUCTION OF APPETITE

Experiments by Gibson (1951),[11] Spedding (1954)[25] have shown that the depression of the appetite is a major factor of damage as the result of infection with *Trichostrongylus axei* in sheep, confirming the findings of Bennets (1933),[3] Andrews (1939)[1] and Gordon (1948, 1950).[14, 15] Spedding found that the appetite depression was about 8 per cent compared to the feed intake of the controls, while Gordon (1964)[16] found that the daily food intake in sheep infected with *T. colubriformis* dropped from 2 lbs. to less than 0.5 lb.

IMPAIRED DIGESTIVE EFFICIENCY

Impaired digestive efficiency can be defined as impaired digestive economy and not necessarily interference with the physiological digestive processes. The apparent digestibility coefficient is a measure of the amount of feed absorbed (Spedding 1954)[25]. Franklin *et al* (1946)[9] found that in moderate infestations with helminths there was a depression of protein digestibility and poorer utilisation of calcium and phosphorus. Gordon (1950)[15] states: "The appetite continues to decline, and when diarrhoea supervenes, the animal is virtually starved to death, because the small amount of food ingested passes through the alimentary canal too fast for digestive processes to be completed."

PATHOGENESIS

Each parasite has its predilection site in the host, and this strict location of the parasite is important in the relationship to the pathogenic

effects. The habits of the various parasites vary considerably. *Haemonchus contortus* is a blood sucker and causes a haemorrhagic anaemia. Fourie (1931)[8] found that out of 38 animals infected with *H. contortus* 9 developed a fatal anaemia, 6 recovered from the subsequent anaemia, while the rest showed no symptoms. *T. axei*, another inhabitant of the abomasum produces an oligocythaemia with the absence of regenerative forms. This anaemia is purely the result of a failure in the production of erythrocytes by the erythroblastic tissues, the normal destruction of the red cells resulting in a gradual decrease in the total number of erythrocytes in the blood (Gibson, 1954).[13]

Because of its high biotic potential, *H. contortus* is able to take the fullest advantage of short term favourable conditions. Heavy infestations develop rapidly and sheep may succumb while in fat condition. A high plane of nutrition, usually the basis of control measures for parasitic diseases of the gastro-intestinal tract, is not of great significance in controlling haemonchosis, and outbreaks often occur when pastures are very good.

Trichostrongylosis: The disease has an insidious onset and its earliest manifestations are often confused with unthriftiness and malnutrition. It is not unusual for trichostrongylosis and malnutrition to be concurrent. The effects of trichostrongylosis are long-lasting; symptoms are not striking and unless carefully conducted, the *post mortem* examination may fail to show even a fatal infestation. In acute infections, although there may be a watery diarrhoea, there is little loss in condition, which is not surprising in view of the early mortality.

Oesophagostomum columbianum produces pathogenic effects in two distinct ways. The young parasitic stages in the wall of both the small and large intestines produce nodular lesions, which undoubtedly interfere with bowel movements and with digestion and absorption of food materials, thus essentially an anorexia. Nodules may rupture resulting in peritonitis or ulcerous lesions in the lumen of the bowel. The presence of adult worms in the colon is associated with a thickening of the bowel and a marked secretion of mucus.

Ostertagia spp. causes progressive loss of condition with intermittent scouring while a severe gastritis is common in severe infestations. Anaemia with dehydration occurs in heavy infestations while there is severe loss of condition even in moderate infestations.

Chabertia ovina destroys the glandular epithelium of the colon while infestations with immature worms result in haemorrhagic mucoid diarrhoea.

Bunostomum spp. and *Gaigeria pachyscelis* are blood suckers of the small intestine and primarily cause haemorrhagic anaemia.

Loss of Production

There can be no doubt that helminth infestation can, and in fact does result in considerable loss of production of wool, meat and milk.

Wool Production

Clunies Ross and Graham (1933)[6] found in a field trial in Tasmania that the percentage of tender fleeces in treated sheep ranged from 6.0–17.9, while in untreated sheep it was 58.3. These authors make the points that "improving wool quality by decreasing the number of tender fleeces indicates that quality of wool may be a more sensitive indicator of adverse conditions than the weight of fleece produced" and "that under improved pasture conditions fleece weight was a more sensitive indicator of the effects of internal parasitism than body weight".

Various authors have published results of controlled experiments which proved that worm infested sheep produce from 15 to 40 per cent less wool than worm-free controls (Kauzal 1936,[18] Sarles 1944,[23] Carter, Franklin and Gordon 1946,[4] Spedding and Brown 1957[27]). There is naturally a greater depression of wool production if there is a high level of helminth infection and a lower plane of nutrition. Under these conditions the effect is very marked, whether measured by sampling (over 40 per cent) or by total fleece weight (38.8 per cent) (Spedding and Brown 1957).[27] The precise effect of worm

infestation on wool growth will depend on the age of the animal at the time when the infestation is at its maximum, since wool growth does not proceed uniformly during the year.

Live bodyweight is synonymous with meat production. Numerous experiments have proved that there is a considerable loss of live bodyweight when animals have been infested with helminths. Spedding (1954)[25] has shown that over a period of 315 days there was a carcase weight difference of 17 lbs. between infected and worm-free lambs; carcase quality measurements indicating a reduction in the amount of bone, fat and chiefly muscle. In this experiment the effects of the worm infestation, which had occurred shortly after weaning, persisted in sheep for up to a year.

At a low level of nutrition infected sheep suffer more at all levels of worm infestation (Clunies Ross and Gordon 1934,[5] Culbertson 1938,[7] Wetzel 1952[31]).

Vegors et al (1955)[30] found that heavily infected calves gained only 1.27 lb. daily compared to 1.45 lb. of the controls; Gordon (1964)[16] found that sheep, experimentally infected with *T. colubriformis*, had declined in live bodyweight from 64.3 lb. to 50.9 lb.

Snijders and his co-workers (1964)[24] using an efficient anthelmintic in sheep, found that there was an increase in live weight of from 17 to 24 per cent over the controls.

Milk

Muller (unpublished results) found that the drenching of cows in a commercial dairy herd resulted in an increase of 12 per cent in the milk production.

Gordon (1950)[15] showed that in ewes infected with *H. contortus* there was a marked depression in milk yield, with a resultant loss in weight by the lambs. The milk yield declined from a daily maximum of 53 fluid ounces to 11.5 fl. oz. within 50 days after infection.

This decline in the milk yield will have a disastrous effect on the lambs. The total quantity of nutrients supplied by the milk exercises a dominant influence on the growth of the lamb expecially so in the first 6 to 8 weeks. It therefore, very probable that a lamb receivi less milk will consume more grass or pastur and, where this is the case, the intake of larv on a pasture, with any given number of larv per pound of herbage, will be negatively relat to the quantity of milk consumed, and mo worms were found *post mortem* in lambs rear as twins than in lambs reared as singles (Spe ding, Brown and Large 1962).[28] It is axiomat that twin lambs receive less milk individual than do single lambs. The changes in nutritic will thus not only influence the number parasites ingested by the lamb, but influen the growth rate and the period over which t lamb is exposed to infection (Spedding 1962).

The Effect of Nutrition on Helminth Infestation

Many workers have studied the effects nutrition on gastro-intestinal worm infestatio in both sheep and cattle, and the results of the experiments show that animals on a high pla of nutrition suffer less ill-effects than tho animals on a low plane of nutrition. Fraser an Robertson (1933)[10] reported a heavier wor burden in poorly fed lambs than in well fed on grazing on the same pasture. Taylor (1943) found larger numbers of parasites in anima kept on a diet of straw and hay, than in tho receiving both hay and concentrates. Simil results were obtained by Gibson (1954),[12] wh found profound changes in the blood picture i animals on a low level diet; similar chang were absent in the high level diet animals.

Roberts *et al* (1952)[22] stated that good nutr tion is mainly concerned with the developmer and maintainance of a resistance to the effec of the infection rather than to the infection itsel Lawrence and his co-workers (1951)[19] clearl demonstrated the beneficial effects of a high maize intake on the response of sheep to he minthosis, and Gordon (1948)[14] showed th sheep grazing on oats threw off their nodul worm infestations quicker than comparabl sheep grazing on natural pastures. Lucker an Neumeyer (1947)[20] reported that lambs on diet deficient in protein, iron, calcium and ce tain vitamins were most susceptible to infectio

with hookworm, and less able to compensate for blood loss than comparable animals on a good ration of lucerne and grain.

Whitlock (1949)[32] indicated that the overcrowding of lambs on a pasture was of less significance than the caloric intake in the prevention and control of trichostrongylosis. Vegors *et al* (1955)[30] found that the addition of maize to the rations of beef calves resulted in a statistically significant reduction in the worm burden (5 per cent level of probability). On some pastures the animals receiving supplements had only one third as many worms as their control companions and made much larger daily gains.

It is, therefore, quite evident that the large number of anthelmintics, some very good, some not so good, which are to-day available to the stock owner, are not the complete answer to the problem of helminth infestation. The veterinarian must of necessity take cognisance of the beneficial effects of good nutrition and proper husbandry methods, which, when combined with strategic drenching with a highly efficient anthelmintic, will certainly go a long way to the goal of increased production per animal unit, which in effect will lower the cost of production.

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REFERENCES

1. ANDREWS, J. C. (1939). *J. Agric. Res.*, **58**, 761.
2. ANDREW, J. S., KAUFMAN, A. B. and DAVIS, R. E. (1944). *Amer. J. Vet. Res.*, **5**, 22.
3. BENNETS, H. W. (1933). *J. Agric. Res.*, **58**, 761.
4. CARTER, H. B., FRANKLIN, M. C., GORDON, H. Mc L. (1946). *J. Counc. sci. indust. Res. Aust.*, **19**, 61.
5. CLUNIES ROSS, I. and GORDON, H. Mc L. (1934). *Aust. vet. J.*, **11**, 106.
6. CLUNIES ROSS, I. and GRAHAM, N. P. H. (1933). *J. Counc. sci. indust. Res. Aust.*, 6, 191.
7. CULBERTSON, J. T. (1938). *Arch. Path. Lab. Med.*, **25**, 85.
8. FOURIE, P. J. J. (1931). *Rep. Dir. Vet. Serv. O.P.*. **17**, 495.
9. FRANKLIN, M. C., GORDON, H. Mc L., and MACGREGOR, C. H. (1946). *J. Counc. sci. indust. Res. Aust.*, **19**, 46.
10. FRASER, A. M. M. and ROBERTSON, D. (1933). *Emp. J. exp. Agric.*, I, 17.
11. GIBSON, T. E. (1951). *State Vet. J.*, **6**, 22.
12. GIBSON, T. E. (1954). Studies on Trichostrongylosis. I. The pathogenesis of *T. axei* in sheep maintained on a low plane of nutrition. *J. Comp. Path.*, **64**, 127–140.
13. GIBSON, T. E. (1954). Studies on Trichostrongylosis. II. The pathogenesis of *T. axei* in sheep maintained on a high plane of nutrition. *J. Comp. Path.*, **64**, 360–370.
14. GORDON, H. Mc L. (1948). The epidemiology of parasitic diseases, with special reference to Studies with nematode parasites of sheep. *Aust. Vet. J.*, **24**, 17–45.
15. GORDON, H. Mc L. (1950). Some aspects of parasitic Gastroenteritis of sheep. *Aust. Vet. J.*, **26**, 14–98.
16. GORDON, H. Mc. L. (1964). Studies on resistance to *Trichostrongylus colubriformis* in sheep. *Aust. Vet. J.* **40**, 55–61.
17. HAMMOND, J. (1932). Growth and development of mutton qualities in the sheep. *Biol. Monographs and Manuals*. Edinburgh & London. Oliver & Boyd.
18. KAUZAL, G. (1936). *Aust. Vet. J.*, **12**, 107–110.
19. LAURENCE, G. B., GROENEWALD, J. W., QUIN, J. J., CLARKE, R., ORTLEPP, R. J. and BOSMAN, S. W. (1951). The influence of nutritional level on Verminosis in Merino lambs. *Onderstepoort J. Vet. Res.*, **25**, 121–132.
20. LUCKER, J. T. and NEUMAYER, E. M. (1947). An experiment on the relationship of diet to hookworm disease in lambs. *Amer. J. Vet. Res.*, **8**, 400.

21. MULLER, G. L. Unpublished.
22. ROBERTS, F. H. S., O'SULLIVAN, P. J., and RIEK, R. F. (1952). The epidemiology of parasitic gastro-enteritis of cattle. *Aust. J. Agric. Res.*, **3**, 187.
23. SARLES, M. P. (1944). *Tech. Bull. U.S. Dept. Agric.* No. 875.
24. SNIJDERS, A. J., STAPELBERG, J. H., and MULLER, G. L. (1964). *J.S. Afr. Vet. Med. Ass.*, **35**, 83–88.
25. SPEDDING, C. R. W. (1954). The persistence of the effects of worm infestation in sheep. *Empire J. exp. Agric.*, **22**, 55–58.
26. SPEDDING, C. R. W. (1962). Modern trends in animal health and husbandry. The agricultural ecology of sheep grazing. *Brit. Vet. J.*, **118**, 461–481.
27. SPEDDING, C. R. W. and BROWN, T. H. (1957). A Study of Sub-clinical worm infestation in Sheep. I. The effect of level of infestation on the growth of the lamb. *J. Agric. Sci.*, **48**, 286–293.
28. SPEDDING, C. R. W., BROWN, T. H. and LARGE, R. V. (1962). (Proceedings of Nutrition Society.)
29. TAYLOR, E. L. (1943). *Vet. Rec.*, **55**, 117.
30. VEGORS, H. H., SELL, O. E., BAIRD, D. M. and STEWART, T. B. (1955). Internal parasitism of beef yearlings as affected by type of pasture, supplemental corn feeding and age of calf. *J. Anim. Sci.*, **14**, 256–267.
31. WETZEL, R. (1952). *Zbl. Bakt. Abt. I.*, **158**, 199.
32. WHITLOCK, J. H. (1949). The relationship of nutrition to the development of the trichostrongylidosis. *Cornell Vet.*, **39**, 146.

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THE REPRODUCTIVE ASPECT OF THE SHEEP ENTERPRISE

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INTRODUCTION

The emphasis in livestock production has shifted to the fields of nutrition, genetics and scientific animal production. Economically speaking maximum efficiency from each animal unit is needed and therefore from the sum total of animal units which constitute any given livestock operation or enterprise.

A flock therefore shows impaired efficiency when its production falls under the accepted optimal standards. This may be the result of lowgrade, chronic or illdefined disease syndromes the aetiological components of which are a mixture of infective, genetic, nutritional and managerial factors. These call for study, firstly to define their importance in terms of economics, secondly to segregate and evaluate the components to establish diagnosis and thirdly to institute treatment. Treatment may involve changes of management, feed or breeding practice rather than, or ancillary to medicinal therapy.

A good example which may be mentioned in this respect is that of a large flock running under range conditions where some ewes do not come in contact with a ram during the mating period. These ewes may be perfectly healthy but the flock as a whole is not, because its lambing percentage has been decreased in that some of the ewes failed to lamb.

The small stock enterprise is based on an adequate lamb production and it is estimated that an increase of only 5 per cent in the lambing percentage will increase the lamb crop of this country by more than half a million lambs per annum.

In this paper it will be endeavoured to analise the breeding aspect of the sheep enterprise and indicate at what stages, from mating of ewes to weaning of lambs, losses may occur.

MATING MANAGEMENT

PERIOD BEFORE MATING

The necessary preparation of ewes and rams before joining them in the breeding season is of paramount importance and negligence during this early stage is one of the most important causes of poor lamb crops. It is essential to allow enough time for some of the undermentioned pre-breeding preparatory measures to be applied, and these should be attended to 6–8 weeks before mating.

RAMS

1. *Examination for fertility and freedom from infection*

Over 50 per cent infertile and clinically affected rams have been found on many properties and it is obvious that these should be eliminated as early as possible to avoid the expense of extra feeding and later, the danger of joining infertile rams to the ewes.

By means of a clinical examination for fertility rams may be sub-divided into three groups namely sound, temporarily unsound (e.g. skin conditions like mange and dermatitis, fly strike, trauma etc.) and permanently unsound (e.g. penile adhesions or distortions, varicocoele,

hydrocoele, Tunica vaginalitis, cryptorchidism, orchitis, epididymitis, hypoplasia etc.). Before the clinically sound group of rams are given preferential treatment and extra attention, they should be subjected to a semen test for fertility and freedom from infection.

The main object of the fertility test is to detect those rams which may be suffering from temporary infertility due to a variety of factors such as high environmental temperatures, fever conditions, fly strike, bluetongue inoculations etc. It must be remembered that a ram giving a poor semen sample is not necessarily infertile and may only be considered infertile after several unsatisfactory collections have been made over a prolonged period.

When making a semen collection to examine for infection the technique must be such that the operator can certify, that if infection is found, it originated from some part of the genital tract of the animal in question and not from the prepuce or other outside sources.

The following organisms have recently been isolated from the semen of rams immunised against *Brucella ovis* at our diagnostic laboratory: *Corynebacterium pyogenes*; *Pseudotuberculosis-ovis*, *ulcerans* and *haemolyticum*; as well as a number of non-pathogenic organisms. The significance of these infections is being investigated.

By means of these examinations and tests we may therefore ensure that only fertile rams and those rams that are free from infection will qualify for preferential treatment. Only one or two infertile rams amongst the group may have a pronounced effect on the reproductive performance of the flock, as such a ram might pair off with a group of ewes or hinder other rams to serve ewes in oestrum. The fertility of rams becomes progressively more important where owners use a lower proportion of rams.

Although regular semen testing of rams is indicated, it is not an economical venture for most flock farmers and in these instances, provided the rams have been immunised with Rev. I vaccine, regular palpation of the testicles with semen testing of a percentage of rams as well as those feeling suspicious on palpation should suffice.

Amongst sale rams it is essential that those intended for export and stud purposes are subjected to a thorough semen test, while careful palpation and testing of only a percentage might suffice with flock rams. It is important to note that genital palpation has a limited value as a test for fertility as not all rams with lesions are infertile or infected and not all normal rams are fertile or free of infection.

2. *Inoculations*

The necessary inoculations, depending upon the time of year when mating is carried out, should be done approximately 2 months before joining. In the case of bluetongue inoculations for example, we have established that a percentage of rams might become temporarily infertile for a period of several months following inoculation.

Rev. I immunization against ovine brucellosis should be done on properties where rams have not been inoculated previously. By the use of this vaccine alone it has been possible to decrease the occurence of epididymitis from 70 per cent to 1 and 2 per cent on some properties. The percentage lambs born on infected farms have increased from under 30 to well over 100 per cent. For the effective control of this disease breeders should be encouraged to immunise their ram lambs at or before weaning as one inoculation will suffice for life.

3. *Dosing*

Rams should be dosed against internal parasites before supplementary feeding starts to ensure maximum utilisation of the additional food. If necessary rams can be dosed again just before joining. We have not been able to incriminate infertility in rams due to various internal parasite remedies up to the present. Full scale tests are, however, envisaged.

4. *The wool must be short* as the efficiency of mating a ram with long wool is very much reduced.

5. *Hooves must be trimmed and be sound*

Enough time should be given for any lameness that might occur to disappear.

6. *Nutrition*

With the increased emphasis on wool production, the unfortunate habit of some farmers to run their rams in a veld camp without any supplementary feed and join them directly to the ewes, should be discouraged.

Rams should receive supplementary feed from 6–8 weeks before joining to activate the genital organs and get the rams ready for mating as soon as they are put to the ewes. Half to 1 lb of a ration consiting of 5 parts yellow mealies, 3 parts oats and 1 part wheaten bran will give good results.

Rams that have been to shows or bought on a sale are usually too fat and should be given time to lose condition before joining.

Before joining the testicles of these rams should be firm but resilient on palpation and a purplish colouration of the thighs is as excellent indication of health and such a ram should work well.

7. *Genetics*

A large group of farmers breed their own flock rams. For this purpose they run a small stud of 50 to 60 ewes. The few stud rams used are usually only replaced when they are old. The coefficient of inbreeding therefore increases at an alarming rate and this may result in poor conception figures on a number of properties.

Testicular development of rams of various age groups are of the utmost importance and should receive special attention as the incidence of hypoplasia is increasing and rams with underdeveloped or slow developing testicles may possibly be grouped under the term "sensitive rams". These rams are the first to become temporarily infertile under adverse conditions. This may be a manifestation of a possible hypopheseal inadequacy similar to that found in Angora goats by van Heerden (1963) or by Doney (1959) where he found a highly significan tincrease in growth rate by injecting one group of inbred Merino lambs with crude pituitary extract. This aspect needs special investigation as it might be of paramount importance to the sheep industry.

8. If vasectomised rams are to be used they must receive the same treatment as the fertile rams.

Ewes

Special attention should be paid to the ewes before mating and the following are the most important points to consider.

1. *Time of year*

Most farmers prefer their ewes to lamb in April or May, but during spring and the early summer months there is a period of reduced sexual activity and most ewes might even go into anoestrus, whereas in autumn sexual activity of ewes reaches its peak with the result that fewer matings are required for conception and a larger percentage twin births occur.

This depression in sexual activity in spring and early summer is limited to some extent by various factors like breed, nutrition, climate, presence of rams and can to some extent be overcome by removal of the casual factors.

In temperate areas late autumn and winter rains ensure good conception rates with spring mating. By putting ewes on green food shortly before joining a flushing effect is achieved, but if the surrounding country is dry at that time of the year, this effect is minimal.

The introduction of vasectomised rams approximately 17 days before joining has a stimulating effect and is recommended when mating in spring.

2. *Nutrition*

Ewes should not be too fat during mating but, if possible, in a gaining condition. The onset of sexual activity is greatly favoured when ewes are putting on weight and condition, after being in poor condition during and after winter.

An endeavour should be made to determine whether an im-balance of trace elements occurs in a particular area as improved lambing rates have been obtained by supplementing the deficient minerals and trace elements.

3. *Inoculations*

Taking into consideration that spring mating usually takes place from the middle of September to the end of October and autumn mating, from he middle of February to the end of March,

the following inoculation schedule is recommended.

Bluetongue: Ewes three to six weeks before spring mating. It is best not to immunise pregnant ewes, but if the need arises one may inoculate during late pregnancy. Rams are to be inoculated after spring mating.

Enterotoxaemia: Best results in the Karoo areas are obtained if the annual inoculation is done during May. In certain areas twice a year immunisation may be necessary. No harmful effects have yet been noted by immunising pregnant ewes.

Rift Valley Fever and Wesselbron: In areas where these diseases are prevalent vaccinate ewes before mating in spring or before autumn mating, but not later than January. This vaccine should not be given in conjunction with bluetongue inoculations.

Blackquarter: Immunise 2–3 weeks prior to shearing.

Rev. I vaccine: Young ewe lambs at weaning may be inoculated. In a recent survey, however, we could find no difference in lambing percentage on Brucella ovis infected properties where ewes and rams were inoculated as against rams only.

Lamb Dysentery: The first inoculation should be given approximately 4 weeks prior to lambing and the second 14 days later. During subsequent years only one inoculation is necessary namely approximately 2 months prior to lambing.

4. *Dosing*

Ewes must be healthy to exhibit regular oestrous periods and care must be taken that the flocks does not suffer from verminosis. In a recent experiment three internal parasite remedies were administered a week prior to hand serving, but no significant difference in the oestrus cycles of mated ewes compared to the control group could be noted.

In the more arid regions of the Cape Province some farmers tend to dose too much, at wrong intervals and with less effective remedies. The effect of this on fertility needs investigation and concerted attempts should be made to eliminate these faulty management practices.

5. *Wool*

Ewes with long wool should be crutched before the mating period.

6. *Feet*

The flock should be sound on its feet and any indication of lameness treated.

7. *Udders*

Examine the udders of the ewes carefully before mating and cull all ewes with faulty udders or teats.

8. *Mothering ability*

Ewes that show poor mothering ability should be identified and care taken not to include them in the breeding flock. Similarly ewes which fail to lamb should also be identified in order to eliminate those that have not produced during two consecutive seasons.

During investigations on this aspect on a number of properties we found an average of 70 per cent of those ewes which had been marked for not having produced a lamb during a previous season once again failed to produce a lamb.

9. *Conformation and wool quality*

Young ewes introduced into the breeding flock should be classed carefully on wool and conformation and mature ewes examined for other faults such as defective teeth, diseased udders etc.

Mating Period

During this period every endeavour should be made to ensure that a ewe exhibiting oestrous will be mated by a fertile ram.

Handserving and artificial insemination is used on a relatively small scale in our sheep enterprise at present and we will therefore confine ourselves to extensive mating which will continue to be the basis of reproduction in South Africa for a long time.

A number of points of importance are the following:—

1. Rams should be used in rotation as it is unlikely that the semen quality will be maintained at a satisfactory level for a long period. In spring mating a smaller percentage rams might be introduced initially and vice versa with autumn mating.
2. Inspect rams regularly as lame or sick rams will not mate ewes. Lazy rams that have no or little inclination to breed should be eliminated and replaced by vigorous rams. Such inactive rams might cause over work of the fertile rams.
3. When mating during the warm time of the year adequate provision of shade is essential. Infertility in rams due to high environmental temperatures have not yet been demonstrated in South Africa although it is a common complaint in the more tropical zones.
4. Best results are obtained when mating is conducted in relatively small camps with only one watering point. If this is impossible the watering points in a bigger camp should be closed leaving only one open for a period, or preferably the flock should be bunched a few times a week.
5. With extensive mating the percentage rams to ewes should not be too small.
6. If possible young ewes should be mated separately to obtain a better conception rate amongst this group as they cannot compete with the more experienced older ewes.
7. By providing favourable conditions for ewes during this period losses at lambing will be reduced.

One Month before Lambing

1. *Nutrition*

It must be remembered that 2/3 of the lamb's prenatal growth takes place during the last six weeks of pregnancy. This is a most critical period for trouble free lambing to follow.

If the ewe receives insufficient feed, milk production will be affected and lamb losses might occur. On the other hand uterine inertia is a common complaint with ewes in too good a condition and receiving little exercise, with the result that a large number of ewes must be helped to lamb.

2. *Dosing*

Ewes in advanced pregnancy should be dosed and moved to the lambing paddock which has been rested for some time to limit infestation of the new born lamb. An effective remedy that may be dosed to pregnant ewes should be used.

3. *Inoculations*

At present only lamb dysentary vaccine is used as has been mentioned earlier.

4. *Abortions*

Ewes should be inspected for possible abortions or the birth of premature lambs. Such cases should be reported to the nearest diagnostic laboratory and every effort should be made to determine the cause.

5. *Shearing*

The udders of ewes with long wool should be inspected and cleaned. Ewes should be crutched.

6. *Culling*

As mentioned previously ewes which fail to lamb during two breeding seasons should be discriminated against.

According to the replies from a questionnaire the percentage difference in lambing between farmers that culled on wool and conformation only as against those that took lambing performance into consideration was 20 per cent. In the first group only 30 per cent of the farmers had a lambing percentage of 80 per cent or more as compared to 78 per cent of the farmers in the second group. In an analysis done on a number of farms an average of 70 per cent of the ewes that failed to produce a lamb during one season again failed to do so the next season.

Lambing

1. *Inability to lamb*

An experienced operator should always be available to help ewes with dystokia or uterine inertia. It is estimated that if a farmer uses 4 per cent rams for an average of 4 years and pays R100.00 each for them with 80 per cent lambing, each lamb born will cost him R1.50. By saving a few lambs and ewes the wages of the operator can therefore easily be covered.

2. From birth most lambs die between the first and third day of life mostly due to—

(*a*) no or too little feed;

(*b*) environmental conditions such as cold or heat;

(*c*) infections; and

(*d*) predatory animals.

In some countries a loss of 20 per cent at this stage is not exceptional.

The body temperature of the new born lamb drops initially and recovers again after a couple of hours. It continues dropping, causing death of the lamb, in exceptionally cold weather or where the lamb does not suckle during the first few hours of life.

It is of special importance to notice the mothering ability of young ewes as those that fail to raise their first-born are poor risks for the future. Care should be taken, however, not to become prejudiced too soon as ewes tend to hide their lambs carefully and in the case of twins, even at seperate places.

3. In the case of infected paddocks preventive inoculations against some diseases may be done, the naval of new born lambs treated with iodine, or the lambing paddock should be changed.

Disease of Young Lambs

Special attention should be paid to prevent or treat the following conditions: Lamb dysentery, naval ill, tetanus, arthritis due to various organisms, enterotoxaemia, Vit. E deficiency, coccidiosis and verminosis.

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REFERENCES.

DONEY, J. M. (1959). The effects of inbreeding on four families of Peppin Merinos. 3. The influence of crude pituitary extract on inbred lambs. *Austr. J. Agric. Res.*, **10**, 97–107.

VAN HEERDEN, K. M. (1963). In vestigations into the cause of abortions in Angora Goats in South Africa. *Onderstepoort J. Vet. Res.*, **30** (1), 23–84.

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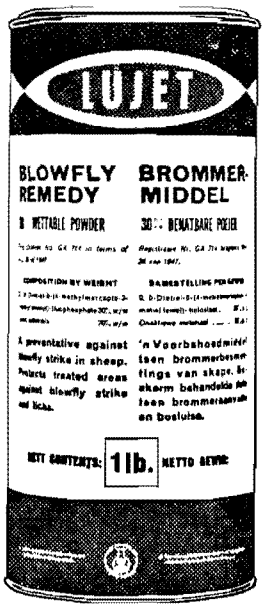

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SOME COMMON BACTERIAL INFECTIONS OF SHEEP IN SOUTH AFRICA

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SUMMARY

This paper is concerned with reviewing some of the more important lethal and economic diseases of sheep and particularly those caused by the sporing anaerobes:

ENTEROTOXAEMIAS DUE TO *Clostridium welchii*, DISEASES CAUSED BY THE GAS-GANGRENE GROUP OF MICROORGANISMS, ALSO TETANUS, BOTULISM, BRUCELLOSIS, CASEOUS LYMPHADENITIS, INFECTIOUS OPTHALMIA, LUMPY WOOL, FLEECE ROT, AND FOOT-ROT.

If it were not for highly efficient vaccines, the stock population in South Africa would be decimated annually. By far the greatest bulk of vaccine issued from Onderstepoort is that for the anaerobic group *Cl. welchii* B and D, *Cl. botulinum* C and D., Cl. *chauvoei*, *Cl. oedematiens*, *Cl. septicum* and *Cl. tetanus*.

A group of potent killing diseases and, coupled with diseases such as heartwater, blue tongue, Rift Valley fever and brucellosis, the sheep farmer would likely have very little left over to profit the purveyors of dips and dosing remedies, if they were not controlled.

To describe these conditions and causative organisms in their entirety would be so much duplication of information already available (Henning[37], Gaiger & Davis[30], Belschner[6], Marsh[45], Topley & Wilson[82], MacKie & McCartney's[26], Hagan & Bruner[34]). Instead, for the purposes of this congress, emphasis has been laid on those aspects which would be of particular value and interest to the field veterinarian and husbandryman, namely, infection, symptoms, diagnosis and control.

1. ENTEROTOXAEMIAS DUE TO *Cl. welchii* (PERFRINGENS)

There are six recognised strains or types of *Cl. welchii* all potent toxin producers. According to Oakley & Warrack, 1953[52] and Brooks et al, 1957[14], 12 different toxic components can be identified and the strains specified by antibody neutralisation tests for the lethal or necrotic factors of these toxins.

Types A—Mainly human infections (gas-gangrene following war wounds, puerperal infections, etc.) but also enterotoxaemia in cattle, horses and goats.

B—Lamb Dysentery or "Bloedpens".

C—Struck of sheep, enterotoxaemia of calves, lambs and piglets. (Not been diagnosed in South Africa).

D—Enterotoxaemia, Pulpy Kidney or "Bloednier" of sheep.

E—Enterotoxaemia of lambs and calves.

F—Necrotic enteritis of man.

Three of these strains are responsible for probably the heaviest losses sustained by sheep farmers annually throughout the world. In South Africa Types B and D are of importance and were it not for the control given by vaccines it is doubtful whether the annual wool cheque would be one half of the ± R100 million it is at present. Enterotoxaemias caused by other *welchii* types have not as yet appeared or been diagnosed in South Africa, their incidence etc. have recently been reviewed by the author, 1963[73].

Lamb Dysentery or Bloedpens (*Cl. welchii B or Cl. agni*).

An acute infectious and highly fatal disease of lambs during the first 2–3 weeks of life.

Infection

The infective organism gains entrance per os with the lamb sucking and nuzzling the ewes teats which have been contaminated by faecal material, soil etc. The disease appears to be favoured by cold weather and rages with extra virulence when animals are herded together in sheds, kraals or cultivated paddocks especially reserved for lambing. Once it has made its appearance the disease may persist indefinitely. The disease affects lambs mainly during the first week of life; deaths in animals over 2 weeks are rare except in protracted outbreaks or where the condition appears year after year when the organism seems to gain in virulence and older lambs are affected and may show a chronic form of the disease.

Symptoms

The incubation period varies from about 10 hours to 2 days, the more virulent the strain the quicker the onset of symptoms. In acute cases the course is about 12 hours, the lamb will appear listless, lie down a lot and not make any effort to move or feed. Faeces become pasty and grey eventually liquid and blood-stained, may be almost pure blood, there may also be straining with a tense abdomen, the eyes are sunken, the lamb becomes comatose and dies.

The less acute cases may have a course of 2–3 days with severe diarrhoea, the evacuations eventually being dark, blood-stained, foetid and passed with severe straining. The lamb is dull, lags and refuses to drink, tail and crutch usually packed with yellowish-brown blood-stained faecal material and the abdomen distended with gas. The lamb will eventually lie down and refuse to move, eyes are sunken and the animal is very weak with a subnormal temperature, becomes comatose and dies.

A chronic form has been described, Henning 1949[37], affecting older lambs of 2–3 weeks age. These lambs are unthrifty, inactive, lie down a great deal and show a persistent diarrhoea with straining and take 3–4 days to die, Belschner, 1959[6]. Some cases seen recently showed a posterior paralysis and death after 2–3 days.

The mortality during an outbreak of lamb dysentery is usually in the region of 90–95 per cent, very few infected animals ever recovering.

Morbid Anatomy

Tail and crutch are stained and/or packed with pasty, faecal material, carcase usually in good state of nourishment but dehydrated, chronic cases will be emaciated. There may be serous or blood-stained fluid in the peritoneal cavity.

The typical lesion encountered is an acute ulcerative enteritis which may extend into the large intestine. The ulcers or necrotic spots are discrete round or irregular and surrounded by a zone of haermorrhage, they may coallesce and form large necrotic patches with adhesions between loops of the bowel. The mucous membrane of the intestine is markedly congested and swollen and a scraping from the necrotic patches will reveal almost a pure culture of Welch organisms.

The mesenteric lymph nodes are usually hyperaemic and oedematous, the liver pale and friable showing fatty degeneration, the heart shows endo- and epi-cardial haemorrhages and pericard may contain blood-stained serous fluid or a gelatinous exudate, the kidneys may be enlarged and hyperaemic.

The evacuations of an infected lamb are a rich source of the organism so that pastures, paddocks, sheds etc. become heavily contaminated in a short period thus causing a fulminating infection.

Diagnosis

This is not usually difficult when large numbers of young lambs succumb to an acute ulcerative enteritis. Confirmation can be obtained by the following laboratory tests:—

(i) Toxin neutralisation by specific antiserum.

(ii) Cultural examination.

(iii) Specimens—The contents of 1–2 feet of the affected portion of the small intestine are milked into a bottle and preserved with 2–3 drops chloroform; unstained smears of the necrotic or ulcerated intestinal lesions should also be submitted to the laboratory.

TREATMENT

No satisfactory treatment is known at present.

IMMUNITY

It was shown by Dalling, Mason, Gordon & Paul, 1926–28[27, 29] that lamb dysentery could be prevented either, by active immunisation of pregnant ewes with anaculture and transmission of antibody to the lamb through the colostrum or, by passive immunisation of newborn lambs with hyper-immune serum. Mason, 1935[46], showed that the greater the anti-toxin combining power of a toxoid the better an antigen it proved and Jansen, 1961[41], that alum precipitated toxoids are better antigens than toxoid alone. Various vaccines are available either anacultures or toxoids, alum precipitated or adsorbed with aluminium phosphate or hydroxide which should all conform to the standard laid down by the British Veterinary Codex, 1953[13].

For field control ewes are inoculated initially 6 weeks and again 2 weeks before lambing, thereafter receiving an annual booster dose 2 weeks before lambing.

ENTEROTOXAEMIA (Pulpy Kidney, Bloednier, Geilsiekte) caused by *Cl. welchii* D, Wilsdon, or *Cl. ovitoxicum*, Bennetts)

An acute infectious enterotoxaemia of sheep of all ages. The condition had been known under a variety of names in different countries before the aetiology was elucidated by Bennetts, 1932[7]. The disease is world-wide in distribution, affecting both sheep and goats, and was first identified in South Africa by Schultz & McIntyre, 1948[58].

Reports from various countries agree that there must be some form of digestive disturbance as a predisposing factor. Bullen, 1952[16], showed, that many normal sheep carry the organism in their intestinal tracts, and Bullen & Batty, 1957[18], that enterotoxaemia may occur spontaneously in sheep if the amount of starch in their diets is suddenly increased by allowing them to overeat on wheat and then introducing Welch D culture into the duodenum. Jansen, 1960[40], showed that dextrin stimulates toxin production and developed a technique for the reproduction of enterotoxaemia. Experimental reproduction of enterotoxaemia is not easily achieved as unlike Welchii B and C, Type D does not depend on any invasive capacity but upon a favourable medium in the gut contents for growth and toxin production.

Any drastic change in the normal functioning of the bowel such as a change from poor to lush grazing, overeating in pastures, oats, barley, etc. as well as cereals put out for winter feed, too much concentrated food and insufficient roughage, treatment for internal parasites, can precipitate enterotoxaemia. Cases have also been observed in sheep in very poor condition probably as a result of bowel stasis which, according to most workers, is a constant finding. Once the prototoxin has been formed its toxicity is activated and enhanced by trypsin, Bosworth & Glover, 1934[12]. The toxin acts on the intestinal mucosa increasing its permeability, Bullen & Batty, 1956[17], so allowing greater absorption of toxin with fatal results caused by direct action on the central nervous system. The toxin has been purified, Verwoerd, 1960[81], and its amino acid composition determined, Thomson, 1963[70].

SYMPTOMS

Sudden deaths are most characteristic of the disease, if clinical cases are observed they may follow either of 2 syndromes, one showing depression and coma, the other, nervous symptoms and convulsions.

In the depressive form the affected animal lags and tires easily, the gait becomes staggery and it may knuckle over. Progressively, the sheep will lie down, be disinclined to move, become dull and listless, the head will swing over onto the flank or the animal becomes recumbent on its flank, sinks into a coma and dies. The nervous form on the other hand is characterised by restlessness, staggering gait, wandering into objects, champing of jaws and salivation, depraved appetite, increased and irregular respirations. An animal so affected may suddenly leap into the air and fall down in a fit of convulsions lying on its side, paddling strenuously, rolling its eyes, grinding its teeth and frothing at the mouth with muscles twitching, finally the head may be pushed back and the legs stretched out in opisthotonus resembling Tetanus.

Some animals may show a subacute or chronic form characterised by a severe foetid diarrhoea, dullness and disinclination to move and graze — a number of these cases will recover particularly if the grazing is changed.

Morbid Anatomy

Carcases of the per-acute and acute cases are usually in good condition and if examination occurs 2–3 hours after death then the cadaver will be greatly distended with gas, the mucous membranes will be congested and bluish, wool will pull easily and the skin at wool free parts of the axilla and underside of thighs will probably be a blue or purplish colour. The blood vessels of the skin and sub-cutis are congested and the inner surface of the skin may show patchy haemorrhages and the sub-cutis, oedematous and gaseous infiltration. Putrefaction sets in rapidly after death, intestines are distended with gas and the kidneys are dark red or greyish and completely soft or pulpy. This putrefaction and pulpiness of the kidneys is characteristic but is not evident immediately after death, an hour or two must elapse before the changes occur.

The liver is usually enlarged and congested and may show degenerative changes, the lungs are congested and show a variable amount of oedema with blood-stained froth in the trachea and oozing out of the nostrils. The pleural cavity contains a variable amount of fluid and the pericard is usually distended with blood-stained fluid which may have jelled. There are usually haemorrhages on the epi- and endo-card and may be on the diaphragm. The small intestines may be distended with gas and the mesenteric vessels congested and petechiae or echymoses on the intestine under the serous coat, there may be a variable degree of enteritis. The kidneys immediately after death are usually markedly congested and may show petechiae, the putrefactive changes developing later as mentioned above.

Diagnosis

In the field the acute disease must be differentiated from anthrax, heartwater (*Rickettsia ruminantium*), malignant oedema (*Cl. septicum*), black quarter (*Cl. chauvoei*), tetanus, haemorrhagic septicaemia (*Pasteurella septicum*), plant, fungus and chorinated-hydrocarbon insecticide poisoning, and the chronic form from verminosis. On more than one occasion the author has investigated deaths in sheep suspected of being due to insecticide poisoning, particularly when animals have shown aimless wandering, champing of the jaws and convulsive siezures typical of gamma BHC and Dieldrin poisoning, only to find, following examinations outlined below, that Enterotoxaemia was the cause of death.

Ths history of an outbreak of enterotoxaemia will usually give an indication of some environmental change in, or reflected in, the alimentary tract. The diagnosis and differential diagnosis have been well described by Stevens, 1959[64].

Aids

(*a*) Demonstration of the toxin in the contents of the small intestine. The contents of 1–2 feet of the small intestine are milked into a suitable bottle with a screwcap and preserved if necessary with 2–3 drops of chloroform. Such material maintains its toxicity for a considerable time and can be tested for epsilon toxin by serum neutralisation tests, Bennetts, 1932[7], Jansen, 1960[39].

(*b*) Smears from the selected portion of the small intestine usually show almost a pure culture of Welchii type organisms. Bennetts, 1932[7].

(*c*) Welchii Type D can usually be isolated from the intestine, liver, kidneys and mesenteric lymph nodes, Jansen, 1960[39].

(*d*) Hyperglycaemia and glycosuria. It has been shown that the sugar content of both the blood and the urine is high in cases of enterotoxaemia, Gordon et al, 1940[31], Bullen & Batty, 1957[18]. The glycosuria can very easily be determined in the field by Benedicts qualitative test otherwise specimens can be preserved for despatch to a laboratory by adding a crystal of phenol, Belschner[6]. A positive sugar test, is not pathognomonic as sheep dying from alkalosis after overeating protein rich feeds, enzootic icterus and pregnancy toxaemia, also show a positive test, a negative test does however exclude enterotoxaemia.

Treatment

No satisfactory drug treatment is known. The disease however, being associated with lush grazing or concentrated feeding, an immediate change to grazing with a higher roughage content to stimulate bowel action, is usually found to be beneficial. In South Africa where the condition was confused with hydrocyanic acid poisoning dosing with sulphur was resorted to with apparent success — according to Hagan & Bruner, 1957[34], sulphur tends to reduce feed intake. Jordan et al, 1956[42] found that Aureomycin at 10 mg. per lb. total feed reduced losses in feed-lot lambs. The most important control measure other than immunisation is dietary management of rich pastures and concentrated feeds.

Immunity.

Sheep can be protected against enterotoxaemia either by vaccination with a trypsinized, alum precipitated, formoltoxoid or by inoculation with anti-toxin. The latter method is uneconomical where the disease occurs all the year round and not really applicable under management conditions where sheep are run in camps of a few hundred to a few thousand acres and only seen once in 2–3 weeks.

Bennetts in Australia 1932–63[7-9] and Bythel and Parker, 1946[18], in Britain, found that a formalised anaculture gave good protection, which could be transmitted to lambs, Oxer, 1936[53]. Smith & Marsh, 1953[62], found that the minimal age at which all lambs responded to active immunisation was 60 days.

It is evident from the use of specific antiserum and toxoid that the most important factor in controlling enterotoxaemia is an adequate level of epsilon anti-toxin and this can be ensured by giving 2 inoculations of alum precipitated trypsinised-toxoid at an interval of not less than 4 weeks and a maintenance dose every 6 months. The minimal level of antibody to give field protection has been estimated at 0.15–0.3 units/ml.

2. Gas Gangrene Group

(*a*) *Cl. chauvoei* (black quarter, black leg or "sponssiekte")

(*b*) *Cl. septicum* (malignant oedema or "sponssiekte").

(*c*) *Cl. oedematiens* (swelled head or "dikkopsiekte").

Infection

In sheep infection by one or other of this group is almost always associated with wound contamination, i.e. shearing cuts, inoculations, rams fighting, gashes and penetrating wounds from wire, dosing guns, etc., which become sullied.

The gas gangrene group and tetanus are dependent on tissue damage for the production of favourable conditions for the establishment of a nidus of infection. Conditions which are met as described above when there is a lowered oxidation-reduction potential and the introduction of infected earth and faecal material, etc.

Symptoms

Infections by this group cause high mortalities, usually following shearing, and are characterised by sudden onset, short course, high temperature 106°–108°, crepitating and/or oedematous swellings.

Chauvoei attacks muscle, penetrating deep into the tissue producing the characteristic dark, almost black, spongy appearance of the musculature. There is usually fairly severe dependant oedema. *Septicum* and *Oedematiens* are not so fastidious; infections running in the subcutis and intermuscular tissue and not penetrating deep into the muscle. A severe semi-fluid oedema develops and spreads from the site of infection in the sub-cutis and intermuscular tissue. Both *Oedematiens* and *Septicum* are highly toxigenic, the affected animal succumbing to a toxaemia comparatively early in the infection whereas a *chauvoei* infection appears to have to develop considerably more before sufficient toxin is generated. *Cl. chauvoei* is the most frequently diagnosed in South Africa, *Septicum* and *Oedematiens* being encountered only occasionally.

If a sick animal is observed it will usually stand around, be disinclined to move or graze and show a high temperature 106°–108°C. There is a progressive swelling and oedema

around and extending from the site of infection, which may reach gigantic proportions and if a limb is affected, a progressive lameness, eventually the animal goes down, mucous membranes become infected, animal becomes comatose with subnormal temperatures and expires.

Diagnosis

Sudden deaths, occurring within 3–4 days of shearing, dosing, inoculating or lambing, with animals showing oedematous swellings and rapid putrefaction of the carcase is the usual history of infection by the gas gangrene group. The oedema fluid in a *Chauvoei* infection is dark red with numerous organisms, that of *Oedematiens* clear and contains few, if any, organisms away from the site of infection, that of *Septicum* may be clear or reddish coloured and contains a variable number of organisms, the oedema is however much more extensive than in either of the other 2 infections, underrunning the subcutis, filling the inter-muscular tissues with a slightly gelatinous fluid. Liver impression smears are diagnostic showing *Septicum* in chains, *Chauvoei* in pairs or singles and *Oedematiens* (if present, usually not) as large paired or single organisms. Pairs of organisms showing a spectacle or dumbbell formation are pathognomonic for *Chauvoei*. In the majority of field cases unless examined within a short while after death the picture will be clouded by numerous contaminating putrefactive bacteria so that a specific diagnosis on clinical and microscopical grounds alone is liable to be misleading.

A definite diagnosis can be made by cultural examination and sub-inoculation of material from:

(*a*) *Oedematiens*—the site of infection, as oedema fluid may be sterile.

(*b*) *Chauvoei* and *Septicum*—oedema fluid, affected muscle or a bone. Material should either be preserved in salt or glycerine, a bone, with tissue removed, can be packed without preservative. Unstained liver impression smears and from the local lesion should be sent with the above material.

A definite diagnosis ultimately rests on isolation of the organism in pure culture, specific toxin—anti-toxin neutralisation tests, staining of smears with fluorescent antibody, Batty & Walker, 1963[2].

Treatment and Control

It is very seldom that an infected animal in the field recovers from any of these infections without treatment. Prior to the advent of sulpha drugs and antibiotics the only treatment was amputation and/or curettage and debridement with local application of carbolic or iodine disinfectants—heroic treatment for both man and animal alike. Once the organism had been identified specific anti-serum could be used—rapidity of diagnosis was therefore essential. The sulpha drugs, particularly Sulphamezathine and antibiotics, such as penicillin are most effective for the treatment of the gas gangrene group infections. Many cases can be saved in spite of extensive involvement of musculature, in fact, unless the animal is in a coma it is worth treating. An outbreak in a flock can now be controlled immediately by drug treatment until a definite diagnosis has been made when the flock can be immunised.

Immunity

All three of the above infections can be controlled by vaccines, either ana-cultures (*Chauvoei*) or toxoids (*Oedematiens* and *Septicum*) alum precipitated or adsorbed with aluminium phosphate or hydroxide as adjuvants.

Immunity to *Oedematiens* and *Septicum* infections is dependent on toxoid alone whereas for *Chauvoei* the important antigen is the bacterial cell itself, Henderson, 1932[36], Mason, 1936[47], Thorold 1953[72], and it has recently been shown by the author (1963 unpublished) that sheep can be protected against at least 50 LD of culture by as little as 1 mg of dried cellular material.

Post Mortem Examination

It is seldom that a full post mortem examination is conducted due to the obvious clinical symptoms and the danger of spreading around the area highly resistant spores. To obtain material for diagnosis the smallest possible incisions are made to obtain liver impression smears and infective tissue. The carcase is then, ideally, burnt *in situ* or buried in lime at least 6 feet below ground level.

Tetanus and Botulism

Cl. tetanus and *Cl. botulinum* are without doubt the two most potent exo-toxin producers known, doses of the order .000001 ml. and .0000000001 ml respectively being the MLD for mice of crude toxins and considerably less for purified toxins. Of particular interest in these two conditions is the method of infection and mode of action of the toxins. Tetanus being a true infection and requiring exactly the same conditions as the gas gangrene group to establish itself, Botulism, however, not being an infection at all but a food poisoning.

3. Tetanus.

Tetanus or lockjaw has been one of the most dreaded diseases of man since early times; it was known and described by the early Greeks but it was not until about 1889 that it was finally isolated in pure culture. The disease has not been a serious threat to livestock in this country but of recent years has been occurring with increasing frequency in flocks following navel infections, docking and castrating of lambs and in some cases following inoculations for other conditions and dosing, when sheep have been roughly handled and the nozzle of the dosing gun penetrated the retropharyngeal tissues.

As in gas gangrene the condition is always associated with an infected wound and tissue damage, spores being found in the soil and faecal material. It has been noted that the organism may multiply in the alimentary tract of man and animals, Noble 1915[51], Ten Broek & Bauer, 1924[65]. Dangerous sites thus being kraals and sheds where there is an accumulation of dung.

Favourable conditions obtaining the spore germinates and the bacilli multiply and produce toxin. The bacilli are not invasive and remain confined to the local lesion.

There are a number of theories explaining how the toxin is absorbed either by the nerve tissue itself, blood or lymphatic systems. The most likely seems to be that advanced by Bayliss, et al, 1952[3] that the toxin is absorbed and carried by the neural fluid in the space between the nerve sheath and axone. Support is given to this theory by the use of sclerosing agents to produce an anulus round the axone which prevents the development of tetanus following inoculation of toxin distal to the anulus. But if the animal received an intra-neural injection above the site of sclerosis the lower limb develops spasticity at the expected time following action of the toxin on the cells in the cerebro-spinal axis.

Symptoms

In sheep the first noticeable symptom is stiffness of the limbs and difficulty in walking resembling a beast with "3-day" or ephemeral fever and there may be severe diarrhoea. Lambs particularly may not show the tetanic spasms, paralysis of the 3rd eye-lid and trismus but, following the stiffness of the limbs, the head and neck become twisted to one side, the animal falls over and becomes quite incapable of movement — death follows due to paralysis of the respiratory muscles.

When adult animals are affected some are bound to show the typical tetanic spasms, however, lambs may not and it is quite possible that the diagnosis may be missed.

Diagnosis

As the organism is not invasive, it is to be found only at the infection site. For a diagnosis when typical symptoms are not in evidence, scrapings and material from a local lesion are:

(*a*) Examined microscopically for the drumstick shaped organism.

(*b*) Subinoculated in mice or guinea-pigs.

(*c*) Cultured following heating for isolation in pure culture.

(*d*) Blood or serum can be sent to the laboratory for toxicity test.

Treatment

The prognosis in cases of tetanus is always guarded due to the affinity of the toxin for the cells of the cerebrospinal column between which and the vascular system there is an effective barrier thus hindering the action of antibody.

The use of an anaesthetic and/or tranquiliser to control the muscular contractions, opening up, cleansing and disinfection of local lesion plus dressing with antibiotic or sulpha drug

should control further toxin production. An established intoxication cannot be controlled by antiserum indicating that either anti-toxin cannot reach the site of action or that the toxic action may be an enzymic process which is not inhibited by the specific antibody, Marack, 1950[44], Cinader, 1953[23].

Experience has shown however that flock infections, in lambs and weaners following marking and inoculating, can be controlled with anti-serum and antibiotics — untreated controls continuing to succumb whilst deaths in the treated group stop after 5–6 days.

Immunity

Immunisation can be carried out with alum precipitated or a. hydroxide adsorbed toxoid, Chodnik et al, 1959[21]. Lambs are the most constantly exposed group which need protection and they can be given a colostral immunity lasting for 10–12 weeks, the best results being obtained by inoculating the ewes when non-pregnant then giving a second dose about 2 weeks before parturition, subsequently an annual booster dose during late pregnancy will suffice, Chodnik et al, 1960[22].

4. Botulism or Lamsiekte, *Cl. botulinus*

There are five spp. of *Cl. botulinus*, A, B, C, D and E which all produce specific toxins not neutralisable by anti-toxins of the other spp. Types A, B and E produce food poisoning in man and C and D are responsible for poisoning in animals. *Botulinus* is unique in the anaerobic group in not being invasive but producing its toxin in decaying organic matter which is then ingested and the toxin absorbed through the intact mucus membrane of the alimentary tract. After absorbtion the toxin acts very widely on all parts of the peripheral nervous system that are cholinergic in character both of the autonomic system and motor nerves of the skeletal musculature. There is no indication that the toxin has any direct injurious effect on the brain or spinal cord.

Infection

In South Africa Botulism is of comparatively rare occurrence in sheep and goats — the disease however has been reported from South West Africa and the Bredasdorp sandveld and two outbreaks have been seen by the author in the Border and East Cape following feeding of bonemeal. Sheep and goats are highly susceptible to the toxin but do not appear to suffer so severely from pica and osteophagia as do cattle. Unless an outbreak occurs, as in the case of contaminated feeds, the diagnosis may be missed in the odd few cases occurring during the dry season. Sheep farmers accept as a normal risk a certain percentage of deaths and in many instances carcases are only found 2–3 days after death when putrid and unsatisfactory for examination.

Symptoms

One of the first symptoms seen in sheep is a wriggling of the tail and constant urination typical of crutch strike by blowfly. General weakness and paresis may then ensue with the animal lying on its sternum with its head over a flank, the neck muscles being too weak to hold the head up, masseter and tongue paresis with salivation. Sigwart, 1929[60], describes a peculiar arching of the neck with the head being held higher than normal in the preliminary stages of neck weakness. Sheep and goats do not seem to show the subacute and chronic forms seen in cattle, mainly the acute form—once the animal shows general weakness and paresis it is unlikely to recover. A post mortem examination reveals no pathognomonic changes and as the toxic material may have been ingested 3–4 days previously examination of stomach contents may draw a blank. If lamsiekte is suspected however, it is always advisable to look for foreign and unnatural materials in the rumen. Also of course any suspected feedstuff, carcase material etc. should be examined for the toxin.

Diagnosis

A diagnosis of lamsiekte depends almost entirely on the history and clinical symptoms exhibited as there are no specific lesions developed. There may be a history of aphosphorosis, osteophagia and pica. The organism is apparently widely spread with cases reported in East Province, West Province, West Transvaal and South West Africa. An outbreak investigated by the author in the East Cape occurred when

Angora goats were given a bonemeal supplement which had been put out for 2–3 days in troughs and then been thoroughly wetted by rain.

Treatment and Control

There is no suitable treatment known at present. Control is effected by phosphate supplementation and the use of vaccines. Onderstepoort puts out a mixed C and D vaccine, developed by Sterne & Wentzel, 1950[63], which gives satisfactory field results.

5. Ovine Brucellosis

This condition comparatively recently discovered is of considerable economic importance. As in cattle abortion storms may follow initial infection with *Brucella melitensis* or *Br. abortus* but in long standing infections the main loss is through infertility. To help combat the spread of this insidious infection a veterinary certificate should accompany each animal sold for breeding and particularly stud rams.

The prevalence of epididymitis in infertile rams and its association with abortions and infertility in ewes was noted in various countries from about 1942 onwards (Gunn, Saunders and Granger, 1942[33], Moule, 1950[50], McFarlane, 1952,[48], McGowan and Schultz, 1956[49], Belonje, 1951[5]). A brucella-like organism, eventually named *Br. ovis*, was subsequently isolated (Simmons & Hall, 1953[61], Buddle & Boyes, 1953[15], Hoptman, 1959[38], Van Drimmelen, 1959[74], Van Rensburg, 1958[79]) from cases of this infectious infertility.

Infection

Infection may take place:

(*a*) From an infected male or female during copulation.

(*b*) Directly from ram to ram by contact, particularly during the off season when rams are run together and mount one another, infective ejaculate may soil the breech and crutch regions. Van Rensburg, 1958[79], has shown that a ram may become infected by placing a drop of infective semen on the glans penis.

(*c*) By the ingestion of infective material such as foetal membranes, fluids and foetuses.

(*d*) Transmission by infected shears, Clapp *et al*, 1962[24].

Most breeds of sheep appear to be equally susceptible — the apparent higher incidence in some breeds probably being due to methods of breeding and management.

Symptoms

The epizootiology and symptoms of infectious infertility of sheep (*Brucella ovis*) follow very closely the pattern of infectious epididymitis and vaginitis of cattle (epivag) of which the author had considerable field experience in Kenya. The clinical manifestations of an anterior cervicovaginitis which subsides and recurs at oestrus and after parturition. The lesions in the female genitalia found by Van Rensburg, 1958[79], of salpingitis, oophoritis and resorption of the foetus, the development in the ram of an epididymitis with subsequent degeneration of the testes are typical of Epivag. A brucella infection was often associated with epivag but the disease was mainly one of infertility and not abortions. *Br. melitensis* and *abortus* infections in sheep show the same symptoms as caprine and bovine brucellosis.

Diagnosis

To make a diagnosis in the field direct microscopical examination of foetus, foetal membranes, colostrum and semen suitably stained by one of the 4 methods described by Van Drimmelen, 1959[74], is resorted to. It may not always be possible to demonstrate Brucella from a case of epididymitis as secondary infection by *Corynebact. ovis* may have caused them to disappear.

A laboratory diagnosis is made by examining the same materials culturally and microscopically but with the very much improved technique of fluorescent antibody staining, Van Drimmelen et al, 1963[77], which attains a high degree of specificity.

Where no material for direct cultural or microscopical examination is available then serolo-

gical tests such as complement fixation, Coombs antiglobulin, blocking antibody test and 5 per cent saline agglutination test have given good results when used on a flock basis to determine infection, where the routine agglutination test has been inadequate, Van Drimmelen, 1962[76].

Treatment

No suitable treatment known at present.

Control

Effective control can be obtained by the use of vaccines, Van Drimmelen, 1960[75], Van Heerden & Van Rensburg 1962[78]. It appears that a high degree of resistance to infection can be stimulated in sheep and goats by the use of live vaccines. In weaner rams Elberg Rev 1 confers a high degree of immunity against all brucella infections for life. Vaccines plus proper control of breeding stock, care when introducing new stock and rigid isolation of old infected rams from clean or vaccinated rams are the prerequisites for a clean flock today where a survey has shown that the condition is wide spread in South Africa. Vaccinated rams can if necessary be put to ewes that have been tupped by infected rams but have since lambed and been isolated, Van Drimmelen (personal comm.)

6. Caseous Lymphadenitis

This infection caused by *Corynebacterium ovis* (*Bac. pseudotuberculosis* or *bac. of Preisz Nocard*) is more a chronic disease characterised by abscess formation in the lymph nodes. The health of the animal is unaffected, unless the infection becomes general through metastasis, but there is economic loss through portions of the carcase being condemned for human consumption. The aesthetic aspect of cutting through an abscess on the Sunday dinner joint cannot be ignored.

C. ovis and the related *C. pyogenes* are world wide in distribution contaminating wounds and abrasions producing pus and abscesses. The organism is capable of surviving outside the animal body in contaminated material, where it probably leads a saprophylic existence, for periods of a year or more, it may also be present in faecal material.

Infection and Symptoms

The most common method of introduction is by wound infection, shearing cuts, docking and castrating, penetration of grass seeds, navel infection of new-borne. Glands commonly affected are the pre-scapular, precrural and popliteal, abscesses however, may develop in any of the internal organs, particularly in the lungs and liver, joints and tendon-sheaths. A generalised fatal toxaemia may develop following a massive infection at birth or following inoculations, Robinson, 1928[56], or metastatic abscesses in the lungs, liver and kidneys breaking down. The course is then acute with high temperature, jaundice and general toxaemia, mucous membranes injected, haemorrhagic enteritis, the animal seldom survives longer than 48 hours. Belonje, 1951[5] describes cases of epidemic pneumonia, bacterial icterus, epididymitis in rams, emaciation and cachexia.

Treatment

The acute condition does not respond to any form of drug treatment as far as the author is aware, however, antitoxin is specific if given in time. Superficial glandular abscesses can be treated and cauterised if discharging or if not can be extirpated.

Control

Shearing wounds are probably the commonest means of infection — there is a higher incidence of the disease in older sheep than in lambs — so that steps taken to reduce infection are:—

(*a*) Proper supervision in shearing sheds to reduce wounding.

(*b*) Disinfection of cutters and shears and shear cuts.

(*c*) Clean pens and dust control.

Belonje[5] and Cameron and Hugo, 1962[20] lay particular stress on hygiene in the control of this condition, the latter authors also consider the Cl. Hydrocarbon and Phosphatic dips to be a source of infection.

An experimental vaccine is being tested in the field at present.

Mastitis

Mastitis in sheep is not nearly as common as it is in cattle. Organisms that have been isolated from affected udders are *Staphylococcus*, *Streptococcus* and *Corynebacterium*. *Corynebacterium* is usually associated with an evil smelling, gangrenous mastitis frequently resulting in the death of the animal.

Infection is through some injury to the udder, tick bites (particularly the *Amblyomma* and *Hyalomma* spp), sore teats, and bruising by suckling lambs, etc.

Gangrenous Mastitis or "Blou Uier" (Blue Bag)

This condition is occurring with increasing frequency in the Karroo and East Cape and in the majority of cases seen by the author was as a result of infected tick bites. The organisms isolated are either *Corynebacterium* or *Staphylococci*. The staphylococcal infection is not usually as severe as the *Corynebacterium*. The animal recovering slowly with the gangrenous quarter/s sloughing off. The condition develops rapidly, the quarter becoming red, swollen, blue to almost black, with extension forward over the belly within 24 hours; death may occur within 2–3 days of symptoms being noted. Initially the ewe will stand around, not eat and have a high temperature.

Treatment

Cases of acute gangrenous mastitis are usually seem too late for treatment to be of any avail. If a *Corynebacterium* infection, anti-toxin can be life saving followed by energetic local and general treatment, heat, udder infusions, antibiotics and may be amputation.

Control

Where ticks are the exciting cause regular treatment of the udder with a suitable tick grease is the most effective method of control. Regular examination and treatment of udders for wounds, sore teats, etc.

7. Infectious Opthalmia

Opthalmia not being a killer nor particularly debilitating, other than in severe cases, has never received the attention it warrants. There are probably more suggested causes, and certainly more remedies recommended, for it than any other condition.

The specific *Rickettsiae conjunctivae* infection was first described by Coles, 1931[25] and since then has been described as occurring in all classes of stock throughout the world.

A large number of organisms have been isolated from infected eyes but are not consistently present and do not reproduce the condition as does *Rickettsiae conjunctivae*.

Exciting and contributory causes are what were formerly thought to be the cause, i.e. foreign bodies, pollen, dust, grass, seeds, sun glare and wind, flies, moths, etc.

Infection and Symptoms

The infective organism does not survive for more than a few days in the dry state so that the main source of infection in an outbreak is from the carrier animal, Beveridge, 1942[11], infection can then be spread by mechanical means, direct contact, flies and moths and by atomisation following sneezing.

An ourbreak may assume severe proportions affecting a large number of animals particularly when stock are brought together and concentrated, e.g. lambs for fattening or ewes for lambing.

Initially there is a watery discharge with the development of an acute conjunctivitis, the mucous membrane becoming bright red, swollen and painful with partial closing of the lids. An opaque film rapidly forms over the cornea which may develop into a suppurative keratitis or corneal ulcer. The discharge becomes thick and yellowish glueing the lids together and may become purulent particularly if there is secondary bacterial infection. It is at this stage with the eye swollen, painful and partially blind, if both eyes affected then animal will be blind, that the animal will be disinclined to graze and starts losing condition. In bad cases the corneal ulcer may extend into the anterior chamber with abscessation and loss of the eye.

Once the opaque film has developed over the cornea no amount of treatment will hasten resolution — treatment is then aimed at con-

trolling secondary infection. If treatment can be commenced in the early stages before the development of the acute conjunctivitis and keratitis then in many cases the disease can be controlled and the acute keratitis and organisation of the cornea with opaque film prevented. Chronic cases occur which do not respond well to treatment.

Treatment and Control

As previously mentioned there exists today a veritable pharmocopeia of remedies for opthalmia alone — most of them beneficial in some way or another, even the most drastic, such as the hot iron (used by some African tribes) or irrigation with power-paraffin producing results by shock treatment.

In controlling an outbreak, affected animals should be isolated and treated and the remainder of the flock, should they have been concentrated, dispersed if possible and the heads sprayed with insecticide to control flies which are the most important vectors. Aureomycin and Terramycin seem to be almost specific for *R. conjunctivae* and if treatment is commenced early there is every possibility that the condition will be aborted. The host of other remedies are of value in controlling bacterial infection, reducing inflammatory changes and resolving the corneal opacity. The following list gives some idea of the preparations in use:

> Silver nitrate 1%; corrosive sublimate .1%; copper sulphate 1.2%; sodium sulfacetamide 30%; protargol; powdered sulfonamide 10% in cod liver oil; tinct. iodine in milk; antibiotics (Penicillin, Aureomycin, Terramycin, etc.); paraffin; aerosols.

This is by no means all, one could quite easily list another ½ dozen remedies of equally unfailing virtue.

8. Lumpy Wool

This is essentially an exudative dermatitis followed by scab formation caused by *Dermatophilus dermatonomus*, Austwick, 1958[1], formerly known as *Actinomyces dermatonomus*. The condition of "lumpy wool" was first described by Bekker, 1928[4] in South Africa and in the same year a similar disease "mycotic dermatitis" was described in Australia by Seddon, 1928[59]. The disease was first described in cattle by Van Sacegham, 1915[80], in the Belgian Congo, and Lane 1915[43], mentioned Senkobo skin disease in Northern Rhodesia; this condition in cattle eventually became known as "streptothricosis" and the causal organism variously as *Dermatophilus-* or *Streptothrix-* or *Actinomyces-*, *congolensis.*

Another condition of sheep "strawberry foot rot" appeared in 1946 and was described by Harris, 1948[35] — its bacterial nature was first described by Thompson, 1954[68], who then placed it in a new sub-order *Polysepta*, Thompson & Bisset, 1957[69].

From the description of isolates from all three conditions it is evident, as outlined by Austwick, that these are different species of the same genus and that his proposed nomenclature appears both reasonable and valid.

Order: *Actinomycetales*
Family: *Dermatophilaceae*
Genus: *Dermatophilus*
Species: *D. congolensis*

Streptothricosis in cattle, horse, sheep, goat, game.

Species: *D. dermatonomus.*
Lumpy Wool of sheep or Mycotic Dermatitis.
Species: *D. pedis.*
Strawberry foot rot in sheep.

The life cycle has been well described by Roberts, 1961[55] and the micromorphology by Gordon & Edwards 1963[32].

Lumpy Wool or Mycotic Dermatitis of Sheep.

In South Africa this condition is caused by *D. dermatonomus* affecting mainly wooled sheep. At present strains are being collected for typing.

Infection

A stained preparation of a culture will show a mass of Gr. + branching filaments dividing longitudinally and horizontally into tetrads from which motile coccal forms are released.

The motile coccal forms, following desquamation of scabs, will be present in soil and dust etc. and so easily contaminate fleeces. Unless

the organism is introduced into the superficial layers of the epidermis by tick bites, Plowright 1956[54], or scarification, then to penetrate to its substrate moisture is required. Investigation of outbreaks in South Africa has shown that they occur in areas of higher rainfall or mist belts, or in dry areas (i.e. Kimberley) following a rainy period when fleeces were wet for a few days. Benzene hexachloride dips were at one time suspected of being a cause of spread but this was found to be unlikely, Thorold 1950[71].

The condition is always present and may flare up in areas such as the Eastern Transvaal, Natal highlands, East Griqualand and Border. In some parts of the Border and Transkei farmers have given up Spring lambing because of the high incidence of the disease during the rainy period. I have examined lamb flocks in the Cala-Engcobo districts in which 80–90 per cent of lambs have shown severe disseminated lesions on ears and over backline and flanks. In the higher rainfall areas of East Africa the condition is frequently seen in haired sheep, also outbreaks of a fulminating nature may occur in cattle with 70–80 per cent of a herd affected. The condition was diagnosed by the author in cattle, sheep, goats, horses and game, i.e. zebra, topi, Thomson's gazelle and oribi.

Symptoms

The organism penetrates the superficial layers of the dermis and proliferates causing a dermatitis with exudate. About 24–36 hours after artificial infection a yellowish pustule surrounded by an erythematous zone is evident. The pustule then bursts discharging its contents into the fleece where it dries and gums the fibres together. Typically discrete lesions are ½–1 in. in diameter and when the scab is removed it appears pyramidal with the base hollowed. The scabs are in the fleece and are only detected at shearing or when the fleece is handled. In severe cases lesions coalesce and the fleece over the flanks and back may become hardened like a suit of armour which breaks away easily, leaving a large raw granulating surface, such cases usually succumb to secondary bacterial invasion.

A few lesions do not upset any animal but when there is extensive involvement of the skin over the back and flanks the animal walks stiffly, has difficulty in grazing and falls off in condition. Affected lambs suffer a setback and do not grow out as well as they should. The lesions are attractive to the sheep blowfly and body strike, particularly in the higher rainfall areas, is initiated by Dermatophilus lesions.

Should affected animals be dipped in Arsenic preparations, there is a danger of poisoning by absorption through the lesions.

Diagnosis

If scab material is crushed on a slide with a few drops of water, fixed by heat and stained with methylene blue numerous coccal forms will be noted but diagnostically branching filaments and sections of filaments dividing into tetrads as mentioned above, should be demonstrated. Scab material without preservative can be sent to the laboratory for microscopical and cultural examination.

Treatment

This is of little avail as by the time the condition is noted the scabs have developed and the dermatitis per se subsided. Once the scabs have been removed and an open lesion results then it is treated like any surface wound. Scabs should be burnt to destroy the infective agent.

Immunity and Control

As with other skin infections, there appears to be some immunity developed following infection, or age resistance. In an outbreak the lambs and weaners are severely affected showing an incidence rate of over 60% whereas the adult sheep will only show about a 5 per cent infection rate.

In South Africa the incidence has been considerably reduced on farms where the stock are regularly dipped in Arsenical preparations or if .03 per cent copper sulphate is added to the dipping fluid. Some of the quaternary ammonium disinfectants have given promising results but have not as yet been sufficiently tested out, Rossiter 1956[57].

Fleece Rot

The fleece of a sheep contains numerous bacteria which do no harm as long as the fleece is dry. Following prolonged wetting of the fleece, as occurs in continued wet weather or in a mist belt, the fleece, skin detritus, moisture and body temperature provide the medium and temperature for bacterial growth. The products of bacterial growth and moisture soften the superficial layers of the skin and produce a degree of dermatitis with exudation, matting of the fibres and crust formation. When the fleece dries out bacterial activity ceases and subsequent wool growth is unaffected. With the growth of the wool the matted and crusted material is raised from the skin and appears as a band across the lower end of the staples in the affected region—there is usually no break in the staple.

A particular pigment producing organism may be present and the affected part of the fleece become coloured a bright blue, green or red. In the Border region of the East Cape following prolonged rain and mist I have seen on a number of occasions sheep with the fleece over the back and flanks a bright green or blue, appearing as though they had been dipped in dye.

The usual rot however, shows no marked coloration being grey-brown or a dirty yellow.

Predisposition and Susceptibility

An open fleece which allows of the penetration of moisture is a predisposing factor. Young sheep are more susceptible than adult because of the openness of the fleece. Conformation of the shoulder-blades, high shoulder-blades which open up the fleece during movement, broad withers with a hollow between the shoulder blades, any factor which allows water to penetrate and wet the fleece will predispose to fleece-rot. The type and quality of the fleece also has a bearing on this problem, poor quality with slackness of the wool being more susceptible than a good quality dense fleece.

Fleece rot is of considerable economic importance as bacterial action produces putrefactive odours attractive to blow-fly and a strike initiated at the withers very rapidly runs right round the girth and takes out the "heart" of the fleece. Body strike is not so easily detected as is the common crutch strike so that usually extensive damage is done before it is detected.

The value of the wool is also decreased if it shows banding of the staple and a definite colouration.

Control

The only method of control is prevention by rigorous culling of sheep showing faulty conformation and poor quality wool.

9. Foot Rot

(*a*) Contagious Foot Rot caused by *Fusiformis nodosus*.

(*b*) Interdigital and coronet abscesses caused by ticks.

Contagious Foot Rot

A highly contagious infection of the soft tissues of the foot caused by *F. nodosus*, Beveridge, 1941[10]. Other tissue such as bones, tendon, sheaths etc. may also be involved.

For many years this condition was thought to be caused by *F. necorphorus* and other organisms but the aetiological agent was finally demonstrated by Beveridge who showed that the disease was transmitted from diseased to healthy sheep when wet conditions favoured pasture contamination.

Infection

According to Beveridge, *F. nodosus* does not survive away from the host in mud, discharges, foot clippings etc. for longer than 3 weeks, however it may survive in lesions in carrier animals for 3–4 years. The disease is seasonal being associated with periods of high rainfall with conditions of mud and slush when the continual wetness, mud and grit packing between the claves, softens the skin and produces injuries through which infection gains entrance; the same conditions allow of the setting free and rapid dissemination of infective material from carriers.

Symptoms

The first sign of infection in a flock is the appearance of lame sheep, the examination of which will probably show nothing further than that the affected feet are hot and painful with swelling between the claves. This develops and there is a break in the skin-horn junction which will extend both forwards and backwards to the heel. Progressively the infection underruns the sole and wall of the foot so that the hoof gradually becomes separated from the underlying tissue. There is usually a small amount of grey-black necrotic muck between the layers of separating horn and tissue and eventually the whole hoof may be shed. If untreated the contion may exist for months and then heal spontaneously. Usually both claves of a foot are affected and frequently two feet may be affected. The condition is very painful, the animal often just standing or kneeling and falling off severely in condition. The hooves may become overgrown and misshapen and due to a general fever there will be a "break" in the wool.

Diagnosis

After paring away the superficial horn, scrapings, swabs and smears from the depth of the lesion should be taken (the swabs and scrapings should be kept moist) and sent for laboratory diagnosis. The differential diagnosis has recently been described by Thomas, 1962[66]. *F. nodosus* is a difficult organism to culture, initial isolation is done anaerobically and for further culture Thomas, 1963[67], recommends a liquid media containing trypsin, hoof-acid hydrolysate and thioglychollate.

Treatment

The only really efficient way of treating and controlling an outbreak of foot rot in a flock is to turn up every sheep in the flock and examine its feet, pare and treat the feet of those affected, isolate them and put the flock through a copper sulphate 10 per cent or formalin 6 per cent foot bath regularly. Good results have been obtained by painting the lesions after paring with 10 per cent tinct. Chloromycetin or preparations of Dichlorophen. Individual cases can be treated with Sulpha drugs and antibiotics.

Interdigital and Coronet abscesses

Severe and painful abscesses caused by ticks are of frequent, seasonal occurrence in the Karoo and East Cape areas. The two main species of ticks involved being the Bontpoot (*Hyalomma spp.*) and Brown (*Rhipicephalus glabroscutatum*). The Bontpoot particularly, with its large mouth parts causing extensive damage to the integument and opening the way for infection by Corynebacteria. Both these ticks will attach either between the claves, under the dew-claws or round the coronet.

Any tick attaching between the claves will almost immediately cause lameness and in a short while the interdigital space is inflamed, swollen and painful. Between the claves appears to be the predilection site of *R. glabro.*, the *Hyalomma spp.* more often attaching round the coronet causing the development of suppurating abscesses.

As previously mentioned Bontpoot ticks are one of the main causes of mastitis and loss of teats.

Control

Regular routine treatment of feet and crutch with insecticides by means of foot-baths, dips, power-spray units and tick grease. There are various methods of application and numerous insecticides at the farmers' disposal today so that with proper routine treatment tick infestations can be reduced to a minimum.

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REFERENCES

1. AUSTWICK, P. (1958). *Vet. Reviews and Annots*, **4**, 33.
2. BATTY, IRENE and WALKER, P. D. (1963). *J. Path. and Bact.*, **85**, 517.
3. BAYLISS, J. H. *et al* (1952). *J. Path. Bact.*, **64**, 33.
4. BEKKER, J. G. (1928). *J.S. Afr. Vet. Med. Ass.*, **1**, 51.
5. BELONJE, C. W. A. (1951). *J.S. Afr. Vet. Med. Ass.*, **22**, 165.
6. BELSCHNER, H. G. (1959). Sheep Management and Diseases, 6th Ed. Angus & Robertson.
7. BENNETTS, H. W. (1932). Bull. 57, C.S.I.R., Australia.
8. BENNETTS, H. W. (1933). *J.C.S.I.R., Australia*, **6**, 92.
9. BENNETTS, H. W. (1963). *Austr. Vet. J.*, **12**, 196.
10. BEVERIDGE, W. I. B. (1941). *C.S.I.R. (Austr.) Bull.* No. 140.
11. BEVERIDGE, W. I. B. (1942). *Aust. Vet. J.*, **18**, 155.
12. BOSWORTH, T. J. and GLOVER (1934). *Rep. Dir. Inst. An. Path. Cambridge*, **4**, 79.
13. Brit. Vet., Codex (1953).
14. BROOKS, M. E., STERNE, M., WARRACK, J. H. (1957). *J. Path. Bact.*, **74**, 185.
15. BUDDLE, M. B. and BOYES, B. V. (1953). *Aust. Vet. J.*, **29**, 33.
16. BULLEN, J. J. (1952). *J. Path. Bact.*, **64**, 20.
17. BULLEN, J. J. and BATTY, IRENE (1956). *J. Path. Bact.*, **71**, 311.
18. BULLEN, J. J. and BATTY, IRENE (1957). *Vet. Rec.*, **69**, 1268.
19. BYTHELL, D. and BARKER, W. (1946). *Vet. Rec.*, **58**, 367.
20. CAMERON, C. & HUGO, P. (1962). *Farming in S.A.*, **39**, 21.
21. CHODNIK, K. S. *et al.* (1959). *Vet. Rec.*, **71**, 904.
22. CHODNIK, K. S. *et al.* (1960). *Vet. Rec.*, **72**, 277.
23. CINADER, B. (1953). Biochem. Soc. Symp. No. 10. Immuno Chemistry 16.
24. CLAPP, K. H. *et al.* (1962). *Aust. Vet. J.*, **38**, 482.
25. COLES, J. D. W. A. (1931). *17th Rep. Vet. Ser. S.A.*, 175.
26. CRUIKSHANK, R. (1962). Mackie & McCartney's Handbook of Bact. 10th Ed.
27. DALLING, T., MASON, J. H. and PAUL, H. (1926). *J. Comp. Path. & Therap.*, **29**, 153.
28. DALLING, T., MASON, J. H. and GORDON, W. S. (1927). *Ibid.*, **4**, 217.
29. DALLING, T., MASON, J. H. and GORDON, W. S. (1928). *Vet. J.*, **84**, 640.
30. DAVIES, G. O. (1957). "Gaiger & Davies *Vet. Path. & Bact.*", 4th Ed. B.T. & Cox, London.
31. GORDON, W. S. *et al.* (1940). *J. Path. Bact.*, **50**, 251.
32. GORDON, M. A. and EDWARDS, M. R. (1963). *J. Bact.*, **86**, 1101.
33. GUNN, R. M. G., SAUNDERS, R. N. and GRANGER, W. (1942). Bull. C.S.I.R. Aust. No. 148.
34. HAGAN, W. A. and BRUNER, D. W. (1957). Infect. Dis. of Domestic Animals. 3rd Ed. B.T. & Cox. London.
35. HARRISS, S. T. (1948). *J. Comp. Path.*, **58**, 314.
36. HENDERSON, D. W. (1932). *B.J. Exp. Path.*, **13**, 421.
37. HENNING, M. W. (1949). "*Animal Diseases in South Africa.*" 2nd Ed.
38. HOPTMAN, J. (1959) — Quoted by v. Drimmelen (1960). *J.S. Afr. Vet. Med. Ass.*, **31**, 129.
39. JANSEN, B. C. (1960). *J.S. Afr. Vet. Med. Ass.*, **31**, 15.
40. ——— —— *Ibid.*, **31**, 205.
41. ——— (1961). *O.P. Jl. Vet. Res.*, **28**, 495.
42. JORDAN, R. M. *et al.* (1956). *J. Animal Sc.*, **11**, 566.
43. LANE (1915). Quoted by Hornby H. E. (1920). *Vet. J.* **76**, **210**.
44. MARACK, J. R. (1950). Summer & Myrback "The Enzymes" 1. 343. Academic Press. Inc. N. York.
45. MARSH, H. (1958). "Newsom's Sheep Diseases". 2nd Ed.
46. MASON, J. H. (1935). *O.P. Jnl.*, **5**, 391.
47. ——— (1936). *O.P. Jnl.*, **7**, 433.
48. McFARLANE *et al.* (1952). *Aust. Vet. J.*, **28**, 221.
49. McGOWAN, B. and SCHULZ, G. (1956). *Cornell Vet.*, **46**, 227.
50. MOULE, G. R. (1950). *Aust. Vet. J.*, **26**, 26.
51. NOBLE, W. (1915). *J. Infect. Dis.*, **16**, 132.
52. OAKLEY, C. L. and WARRACK, J. H. (1953). *J. Hyg.*, **51**, 102.
53. OXER, D. T. (1936). *Aust. Vet. J.*, **12**, 54.
54. PLOWRIGHT, W. (1956). *Vet. Rec.*, **68**, 350.
55. ROBERTS, D. S. (1961). *Aust. J. Exp. Biol. & Med. Sc.*, **39**, 463.
56. ROBINSON, E. M. (1928). 15th Rep., *Dir. Vet. Ed. & Res.*, V.I.O.P., S.A.
57. ROSSITER, L. W. (1956). *J.S. Afr. Vet. Med. Ass.*, **27**, 179.
58. SCHULTZ, K. & McINTYRE, C. (1948). *J.S. Afr. Vet. Med. Ass.*, **19**, 93.
59. SEDDON, H. R. (1928). Pastoral Review, p. 1067.
60. SIGWART, H. (1929). *J.S. Afr. Vet. Med. Ass.*, **1**, 25.
61. SIMMONS, G. C. & HALL, W. T. K. (1953). *Aust. Vet. J.*, **29**, 33.
62. SMITH, L. D. & MARSH, H. (1953). *Amer. J. Vet. Res.*, **14**, 408.
63. STERNE, M. and WENTZEL, L. M. (1950). *J. Immunol.*, **65**, 175.
64. STEVENS, A. J. (1959). *Vet. Rec.*, **71**, 692.
65. TEN BROEK, C. and BAUER, J. H. (1924). *Proc. Soc. Exp. Biol. (N.Y.)*, **21**, 267.
66. THOMAS, J. H. (1962). *Austr. Vet. J.*, **38**, 159.
67. ——— (1963). *Ibid.*, **39**, 434.
68. THOMPSON, R. E. M. (1954). *J. Path. Bact.*, **68**, 445.
69. ——— & BISSET, K. A. (1957). *Nature.*, Lond., **179**, 590.

70. THOMSON, R. O. (1963). *J. Gen. Microbiol.*, **31,** 79.
71. THOROLD, P. W. (1950). *J.S. Afr. Vet. Med. Ass.*, **21,** 30.
72. ——— (1953). *Brit. Vet. J.*, **109,** 302.
73. ——— (1963). F. A. O. Agric. Studies No. 61, 203.
74. VAN DRIMMELEN, G. C. (1959). *J.S. Afr. Vet. Med. Ass.*, **30,** 259.
75. ——— (1960). Ibid., **31,** 129.
76. ——— (1962). Ibd., **33,** 151.
77. ——— *et al.* (1963). Ibid., **34,** 265.
78. VAN HEERDEN, K. M. and VAN RENSBURG, S. W. J. (1962). *J.S. Afr. Vet. Med. Ass.*, **33,** 143.
79. VAN RENSBURG, S. W. J. *et al.* (1958). *Ibid.*, **29,** 223.
80. V. SACEGHEM, R. (1915). *Bull. Soc. Pat. Exot.*, **8,** 354.
81. VERWOERD, D. W. (1960). *J.S. Afr. Vet. Med. Ass.*, **31,** 195.
82. WILSON, G. S. and MILES, A. A. (1948). "Topley & Wilson's Princips. Bact. & Imm., 3rd Ed. Arnold & Co. London.

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AFRICAN HORSE-SICKNESS WITH EMPHASIS ON PATHOLOGY

MAJOR R. M. MCCULLY

U.S. Air Force

SUMMARY

Major McCully, a guest worker at Onderstepoort from the Department of Pathology of the U.S. Air Force, delivered a most interesting illustrated address on the pathology of African Horsesickness.

Major McCully illustrated his talk by presenting many 35 mm. colour slides depicting both the macroscopic and microscopic pathology of the disease.

The material obtained from horses in the 1961 outbreak of the disease in the Middle East (investigated by Dr. Peter Howell of Onderstepoort) formed the basis of this study.

The entire paper was published and may be seen, in the American Journal of Veterinary Research, Vol. 24 (99) — March, 1963 — 235–266. This study was made, and the collaborative paper written, by Dr. Fred D. Maurer D.V.M. Ph.D. and Dr. R. M. McCully D.V.M.

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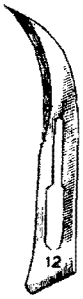

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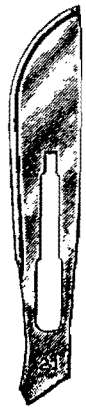

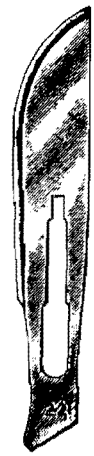

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C. F. B. HOFMEYR

Departement van Snykunde

Fakulteit van Veeartsenykunde, Onderstepoort

OPSOMMING

Hoewel Hippocrates (370 v.C.) heupdisplasie in die mens beskryf het, is die eerste verwysing betreffende die toestand in die hond eers in 1936 deur Schnelle gedoen. Die kondisie kan definieer word as 'n erflike displasie van die acetabulum verbonde met subluksasie of luksasie van die caput femoris.

Heupdisplasie is reeds in meer as 40 honderasse beskryf met 'n verspreiding byna uitsluitend beperk tot die swaarder rasse. Aangesien Duitse herderhonde dikwels gebruik word vir militêre doeleindes, is opnames van die voorkoms van heupdisplasie veral gedoen in hierdie ras en wel in die Skandinawiese lande en die V.S.A. Die syfers wissel van 20 tot oor die 50 persent.

Heupdisplasie word geneties oorgedra as 'n dominante eienskap met onvolledige deurdringing. 'n Groot persentasie gevalle wat radiologies heupdisplasties is, is klinies normaal. Primêr is heupmisvorming nie vasstelbaar nie, maar ontwikkel gedurende die eerste ses maande van leeftyd waarskynlik as gevolg van slapheid of swakheid van die gewrigsomgewende bande en spiere.

Klinies word die toestand beskou as swakheid of mankheid in die heup(e). Daar is spieratrofie en, veral in gevorderde gevalle, word die trochanter major te hoog gedra. Daar is 'n neiging om te galop met die agterlyf terwyl die voorlyf draf.

Finale diagnose kan alleen radiologies gemaak word met presiese posisionering van deurslaggewende belang — die hond presies dorsaal met die heupe in volle buiging en dan in volle strekking met die tone effens na binne gedraai. Die diepte van die acetabula word bepaal deur pelvimetrie; die eenvoudigste metode is volgens die metode van Norberg. Behalwe vervlakking van die heuppotjie word afplatting van die caput waargeneem sowel as peri-artikulêre eksostosisse. Die grade is soos volg:

1. Effens — caput en acetabulum pas swak.
2. Gemiddeld — duidelike vervlakking van die acetabulum.
3. Erg — die acetabulum en die caput is baie plat. Daar is subluksasie.
4. Baie erg — die caput is ontwrig.

Alle aangetaste gevalle (ook die klinies negatief en radiologies positief) moet van teling weerhou word. Geen heeltemaal effektiewe behandeling is bekend nie. In huishonde is amputasie van die kop van die femur aangedui om 'n pynlose en relatief goeie bestaan te verseker.

In die differensiaal diagnose kom die volgende in oorweging:

1. Perthe se siekte.
2. Epihisiolyse.
3. Traumatiese coxitis.

'n Teorie ten opsigte van moontlik twee toestande wat gesamentlik as heupdisplasie beken is, word gedoen.

SUMMARY

Prof. Hofmeyr in his talk presented the known features of the disease, its prevention and treatment.

He illustrated his talk by the projection of a film on the amputation of the neck of the femur.

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GEORGANISEERDE VARKGESONDHEIDSBEHEER

G. F. VAN DER MERWE

Veeartsenykundige Velddienste, Privaatsak 138, Pretoria

Vir publikasie in September 1964 ontvang

SUMMARY

A review is given of the functioning and application of organized health control schemes which have been instituted in certain overseas countries to combat swine diseases with the object of establishing healthy herds. These schemes have in all cases principally been inaugurated to ensure freedom from enzoötic pneumonia and atrophic rhinitis in participating herds but at the same time an opportunity is afforded to evaluate the incidence of, and to combat, other disease conditions.

The inauguration and functioning of a Pig Recording and Health Scheme to promote and ensure a healthy pig industry in South Africa but on which can be greatly improved, is briefly discussed.

OPSOMMING

'n Oorsig word gegee van die funksionering en toepassing van georganiseerde gesondheidsbeheerskemas wat in sekere oorsese lande ingestel is om varksiektes te bestry en daardeur gesonde kuddes op te bou. Die skemas is in alle gevalle hoofsaaklik in die lewe geroep om vryheid van ensoötiese pneumonie en atrofiese rinitis in deelnemende kuddes te verseker maar dit bied ook terselfdertyd 'n geleentheid om die voorkome van ander siektetoestande te bestry.

Die instelling en funksionering van 'n Varkaantekening en -gesondheidskema ter bevordering en versekering van die gesondheid van die varkbedryf in Suid-Afrika maar waarop nog heelwat verbeter kan word, word kortliks bespreek.

INLEIDING

Dit ly geen twyfel dat die benadering van bevordering van algemene dieregesondheid op 'n kudde grondslag jongsjare alhoemeer deur veeartsenykundiges dwarsdeur die wêreld gepropageer en uitgebou word in 'n poging om in besonder die produksie potensiaal van die verskillende soorte dierenywerhede tot hulle maksimum te ontwikkel en daardeur die aanbod van voedselbronne vir menslike verbruik dermate te probeer verhoog dat dit tred kan hou met die steeds toenemende benodigdhede van die immer aanwassende bevolkingsgetalle.

So vind mens tans in verskeie Europese lande, na die suksesvolle deurvoering van hulle nasionale uitroeiingsskemas ten opsigte van beestering en -brusellose wat as 'n bepaalde opdrag van die Marshall-plan na die Tweede Wêreldoorlog uitgevloei het, dat die funksies van die onderskeie veeartsenydienste geleidelik uitgebrei is om bykomstige algemene dieregesondheidsskemas in die lewe te roep en uit te bou.

In Wes-Duitsland is daar byvoorbeeld reeds 'n reeks sodanige dienste ingestel, t.w.:

(*a*) *'n beesgesondheidsdiens* wat daarop gemik is om dekinfeksies en alle vrugbaarheidsteurnisse in kuddes te bekamp en dusdanig die verlengde bruikbaarheid van diere vir teelt-, reproduksie- en produksie doeleindes te verseker;

(*b*) op die gebied van melk higiëne 'n *uiergesondheidsdiens* waarby aandag gegee word aan die bestryding van mastitis om sodoende die kwantiteit en kwaliteit van suiwelprodukte te verhoog;

(c) *'n skaapgesondheidsdiens* wat veral te make het met die bestryding van inwendige parasiete asook die vroeë diagnose en regstelling van ander kudde-siektetoestande;

(d) *'n pluimveegesondheidsdiens* vir die georganiseerde bestryding van Salmonella besmettings en ander aansteeklike siektes; en

(e) *'n varkgesondheidsdiens* wat aanvanklik ingestel is om oor die gesondheidstoestande van stoetkuddes te waak maar met die toenemende uitbreiding van die bedryf op landelike gebied is die diens dan ook geleidelik uitgebrei om op 'n steeds wyer front uitskakeling te probeer verseker van veral daardie siektetoestande wat die behoorlike opgroei van jong diere ondermyn soos bv. ensoötiese pneumonie (E.P.), atrofiese rinitis (A.R.), bloedarmoede, skurfte, wurmbesmetting, ens.

Varkgesondheidsskemas in enkele lande

Swede

Sweedse veeartsenykundiges het gedurende 1940/41, die voortou geneem met die uitbouing van 'n georganiseerde varkgesondheidskema wat daarop gemik was om siekte-vry kuddes daar te stel nadat 'n landswye opname getoon het dat nie minder nie dan 35 persent van alle varkies wat aankom vrek voordat hulle slagouderdom bereik en die oorgrote meerderheid daarvan voordat hulle gespeen word. Behalwe E.P. en A.R. was 'n hele reeks ander bakteriese-, parasitêre- en tekortsiektes vir hierdie vrektes verantwoordelik.

'n Oorsig oor die toepassing van die skema in Swede tot ongeveer 1955/6 is reeds deur Loveday[1] verstrek. Dit kan net bykomstig hier gemeld word dat daardie land sedertdien dusdanig geslaag het met die bevordering van varkgesondheid dat sy 214 teeltbedrywe tans byna 100 persent siekte-vry verklaar is en voortdurend sulks gehou kan word terwyl die sowat 50,000 slagvarkproduksieplase sedert 1962 almal by die skema ingeskakel is en die getal „premie" varke wat vanaf hierdie plase verkoop word reeds die 90 persent kerf oorskry.

Statistieke toon verder dat waar 3.5 persent van alle varkies afkomstig vanaf produksieplase voorheen binne die eerste paar weke gevrek het as gevolg van besmetlike- en tekortsiektes, die vrektesyfer tans omlaag gebring is na 0.9 persent wat van geweldige ekonomiese belangrikheid is in 'n produksie bedryf met 'n jaarlikse omset van oor 'n miljoen slagvarke. Volgens Swahn[2] beteken die suksesvolle deurvoering van die Sweedse skema in sy geheel 'n jaarlikse finansiële gewin van sowat R7 miljoen vir die varkbedryf in daardie land.

Nederland

In Nederland waar alle aspekte van dieresiektebestryding, -voorkoming en -diagnose op 'n gebiedsbasis onder elf Provinsiale Dieregesondheidsdienste (P.D.G.D.) ressorteer en deur die onderskeie boeregemeenskappe finansier word, is vanaf 1958 'n begin gemaak met georganiseerde bestryding van varksiektes, die opbouing van siekte-vry houdings en die instelling van die sg. „Geslote Kuddes."

Deelname aan die skema is op 'n vrywillige basis en beide stoettelers sowel as kommersiële varkbedrywe kan daaraan deelneem. Veeartsenykundige kontrole-inspeksies word viermaal jaarliks uitgevoer en so gereël dat dit saamval met dié tyd wanneer die grootste getalle jong varkies (4–7 weke oud) gesien kan word. 'n Bedryf word as gesond verklaar na minstens 3 agtereenvolgende „skoon" inspeksies waarvan 2 verkieslik in die winter uitgevoer moet wees. By sodanige kontrole-inspeksies moet daar geen aanduidings wees van E.P., A.R., varkpes en skurfte nie. Elke hoes word aangeteken en indien daar enige suspisie van 'n asemhalingsiekte is word een of meer diere geslag vir makro- en mikroskopiese nadoodse ondersoeke waarbenewens oordragingsproewe by die Rotterdamse Dieregeneeskundige Instituut uitgevoer word. Long-ondersoeke van slagvarke uit deelnemende kuddes word bykomstig by slagpale uitgevoer. Dit dien ook gemeld te word dat die verantwoordelike veeartse tydens inspeksies hulle nie alleen bepaal by die kliniese vasstelling van siektetoestande nie maar die eienaars ook met advies bedien oor die nuutste ontwikkelings en toepassings op die gebied van higiëne, huisvesting, voeding en algemene bestuurspraktyke.

By die sanering van kronies besmette bedrywe word die bewese Sweedse patroon grootliks nagevolg, d.w.s. totale ontvolking gepaard met ontsmetting en leeglê van bestaande hokke vir sowat 2 maande daarna maar met behoud van 'n paar ouer klinies-gesonde sôe wat reeds twee- of meermale gejong het vir verdere teling in algehele afsondering op aparte persele en kontrolering daarna van die nageslagte. Dit word as belangrik beskou dat teeltbere altyd in afsondering gehou moet word en waar K.I. nie toegepas word nie, moet sôe in die ope en nie in die hok van die beer nie, uitgevoer word.

Die P.D.G.D. in Nederland is besonder streng sover dit die behoud van die gesondheidsstatus van deelnemende kuddes betref en 'n kudde word onmiddellik van die sertifiseeringsskema onttrek indien 'n aansteeklike of besmetlike siekte daarin vasgestel sou word. Daar is tans sowat 6,000 deelnemende varkkuddes waarvan ruim 60 persent ten volle gesond verklaar is as Geslote Bedrywe.

Wes-Duitsland

Die uitvoering van georganiseerde skemas om varkgesondheid te bevorder word in die verskillende „Länder" van Wes-Duitsland deur bemiddeling van die organisasies van die onderskeie Landboukamers moontlik gemaak. Kuddes word op 'n vrywillige grondslag opgeneem indien hulle vry gevind word van E.P. en A.R. na twee kliniese ondersoeke gepaard met negatiewe patologies-anatomiese en histologiese laboratorium ondersoeke in verdagte gevalle en met 'n tussenpose van 3 maande tussen sulke ondersoeke. Indien E.P. of A.R. vasgestel word, word die kuddes gekwarantyn en mag geen varke daarvandaan tentoongestel word of aan slagprestasietoetse deelneem alvorens eers ten volle van die besmettings ontslae geraak is nie.

Om varkhoudings te bevry van E.P. en A.R. word die volgende maatreëls aanbeveel en/of voorgeskryf:—

(*a*) algehele uitslagting van die hele bedryf en heropbou met invoere vanaf skoon, gesonde kuddes; of

(*b*) geleidelike ontvolking van die besmette kudde terwyl 'n gesonde kudde tegelykertyd op nuwe persele opgebou word met, waar moontlik, aparte werkkragte, toerusting, ens.; of

(*c*) die opbou van 'n skoon kudde uit ou sôe op die houding self in aparte enkelhokke aangehou met daaropvolgende kontrolering van die werpsels vir vryheid van kliniese tekens en serologiese afwesigheid van virus.

Switserland

Aan die begin van 1962 is deur die Switserse Varktelersvereniging in samewerking met die Staatsveeartsenydiens, die ambulatoriese kliniek van die Universiteit Bern en die Bernse Kantonveeartse 'n georganiseerde varkgesondheidsdiens in Switserland in die lewe geroep. Dit het 'n noodsaaklike vereiste geword omdat opnames aan die lig gebring het dat tot 20 persent van alle varke wat aldaar in slagpale ondersoek is longveranderings aanduidend op E.P. getoon het en dit ook daar aanvaar word dat sodanig-aangetaste diere se daaglikse gewigstoename en voeromsetting met 15–20 persent verminder word wat 'n tydsverlenging van sowat een maand meebring om slagvarke markgereed te kry en assulks 'n beraamde jaarlikse ekonomiese verlies van minstens een miljoen rand vir daardie land meebring.

Die doelstellings hier is om aanvanklik 40–50 E.P.-vry teeltkuddes daar te stel en opvolgend mettertyd die orige bedrywe skoon te maak en met siekte-vry diere afkomstig van die gesertifiseerde houdings op te bou. In die beplanning van die skema is voorgelê dat vier veeartsenykundige inspeksies jaarliks uitgevoer moet word en 'n volwaardige siekte-vry sertifikaat word uitgereik indien 'n houding kwalifiseer uit oorde van negatiewe kliniese inspeksies, lykskouings op varke wat vrek of „opgeoffer" moet word vir laboratorium-ondersoeke en ook op diere wat by slagpale geslag word.

Die opbouing van skoon kuddes word op tweërlei wyse bereik, t.w.:

(*a*) weereens die toepassing van die Sweedse sisteem waarby die voorwaarde van „opoffering" as 'n bykomstige versekeringsmaatreël vir die daarstelling van 'n besmetting-vry nageslag verpligtend gemaak is. Dit bestaan daaruit dat voornemende deelnemers aan die skema onderneem om

gedurende die eerste twee inspeksiejare minstens dubbeld soveel jong varkies van 4 weke oud as moedersôe wat aangehou word, moet opoffer vir 'n volledige histopatologiese, bakteriologiese en virologiese laboratoriumondersoek en daarna een afstammeling van elke teelsog jaarliks; en

(*b*) die verkryging van patogeen-vrye nageslagte deurmiddel van histerektomie. 'n Sodanige onderneming is op 'n uitgebreide kommersiële skaal teen 'n aansienlike koste op 'n afgesonderde plaas by Hendschiken in Oos-Switserland deur 'n veevoedingsmaatskappy aangepak. Geen besoekers word daar toegelaat nie en die strengste higiëne word gehandhaaf om die inbring van enige vorm van besmetting te verhoed. Vir die eerste week word sodanige varkies, elkeen apart, in 'n broeikas gehou en met oporiseerde (steriele) melk gevoed. Daarna word hulle in groepies van 5–6 in steriele hokkies gehou totdat hulle 'n maand oud is en dan uitgeplaas na die groter permanente persele. Die eerste histerektomie is hier in Maart 1962 gedoen en presies 'n jaar later is die eerste nageslag uit hierdie kunsmatig-verkreë kern gebore. Huidig word die kudde op 'n natuurlike wyse verder opgebou en bestaan reeds uit sowat 600–700 diere wat geleidelik beskikbaar gestel sal word as teeltkerne aan belangstellende eienaars wat intussen hulle eie bedrywe ontvolk en die persele saneer het vir die aanhou voorts van patogeen-vrye houdings.

In die Verenigde Koninkryk bestaan 'n soortgelyke skema waarby histerektomie-verkreë varke op 'n kommersiële skaal deur twee maatskappye produseer en distribueer word. Hulle is onderworpe aan die strengste veeartsenykundige beheer waarby bepaalde standaarde van behuising, voeding, higiëne en bestuur opgelê en stiptelik gehandhaaf moet word. Die kuddes wat aldus ontstaan word erken as „Minimale Siekte Kuddes" en benewens die verspreiding daarvan onder telers, word van hierdie varke ook grootliks gebruik gemaak by Navorsings Institute in oordragingseksperimente by die studie van besmetlike of aansteeklike siektes.

Hoewel die gebruik van spesifieke patogeenvry (S.P.V.) varke vir hierdie doel seer sekerlik 'n goeie doel dien, bestaan daar nog bedenkinge oor die suksesvolle aanwending van hierdie tipe dier as vervangingsmateriaal in die algemene boerderypatroon. Afgesien van die hoë koste daaraan verbonde moet opvolgende aankope en introduksies uitsluitlik van kuddes met 'n soortgelyke status geskied want vermenging met ‚normale' varke mag onder geen omstandighede plaasvind nie. Daarbenewens moet bere en sôe apart gehou word, geen diere wat na markte gestuur word moet teruggebring word nie terwyl vir skou-doeleindes diere op die persele self beoordeel moet word. Die gevaar van besmetting inbring d.m.v. persone, toerusting, voedingstowwe, ens. skep natuurlik voortdurend 'n probleem.

Volgens Abelseth *et al*[3] was herbevolking van varkbedrywe in Kanada volgens die isolasie metode, veral sover dit die uitskakeling van A.R. betref, nie baie suksesvol nie en groter verwagtings word daar gekoester van vervangings met S.P.V. varke waarmee sowat twee jaar gelede begin is. Die outeurs bevestig nietemin dat sukses met die grootmaak van sulke diere die grootste oppassendheid, omsigtigheid, aseptiese en antiseptiese tegnieke vereis in aansig van die afwesigheid van kolostrale teenliggaampies. Besondere voorsorg is ook nodig om die behoud van S.P.V. varke te verseker na uitplasing vanaf die laboratorium bv. die voorsiening van 'n egalige warm temperatuur sonder enige trekke in die hokke asook goeie voedsame kos wat absoluut vry moet wees van enige kontaminante. Probleme wat by die eerste 1,100 sodanig uitgeplaasde varke ontstaan het was o.a. verlaagde vrugbaarheid by sommige en die voorkome van siektetoestande soos ingewandsedeem, disenterie (vibrio en E. coli), virus enkefalitis en erisipel by andere.

Suid-Afrika

Die konsep van algemene bevordering van dieregesondheid waarby die uitbouing van gesondheidsdienste en -skemas ten nouste ingeskakel is, geniet ook onlangse jare in die Republiek van Suid-Afrika progressief meer aandag. In belang van die varknywerheid in die algemeen en ter verbetering van produksie, genetiese waarde en gesondheidsbeheer by kuddes van

belangstellende telers in die besonder, het die Varktoetssentrumkomitee van Suidelike Afrika reeds 'n Varkaantekening en -gesondheidskema ontwerp en ingestel. Die handhawing van die gesondheidsaspekte berus by die Afdeling Veldveeartsenykundigedienste en bestaan daaruit dat voornemende deelnemers aan die skema voor aanvaarding aan sekere gestelde minimum standaarde van behuising en sanitasie moet voldoen waarna hulle persele minstens tweemaal jaarliks deur die plaaslike Staatsveearts besoek word om die gesondheidstatus van die kuddes te evalueer en die eienaars met toepaslike veterinêre advies te bedien. Waar moontlik word longondersoeke op slagvarke van deelnemers by slagpale uitgevoer.

Tans is daar 26 telers wat as deelnemers toegelaat is en hoewel vryheid van E.P., en A.R. nog nie soos in oorsese lande as 'n voorvereiste vir sodanige toelating neergelê is nie, word dit beskou dat veel goeds reeds bereik is met hierdie skema insoverre dat—

(*a*) 'n duideliker opname-prent van die mees algemene en belangrikste siektetoestande wat by varke op 'n gebiedsbasis verwag kan word reeds verkry is;

(*b*) siektebeheermaatreëls daarooreenkomstig ingestel en aangepas kan word;

(*c*) 'n bewustheid van algemene siektevoorkoming en -bestryding in verhouding tot belangrikheid van goeie behuising, higiëne, voeding en bestuur by die deelnemers opgewek is;

(*d*) relatief siekte-vry varke na die Varktoetssentrums gestuur en/of aan kopers verskaf word.

Toestande wat reeds tot 'n mindere of meerdere mate gedurende die kontrole inspeksies ontbloot is en progressief uitgeskakel probeer word is o.a. tuberkulose, wurmbesmetting, anemie by jong varkies, enteritis, omlope, melkloosheid, aflatoksikose asook E.P. en A.R.

Sover dit die laasgenoemde twee siektes betref is dit bekend dat—

1. E.P. wydverspreid voorkom by varkerye in sommige dele van die land maar blykbaar onder Suid-Afrikaanse toestande nie dieselfde nadelige ekonomiese omvang aanneem as in ander wêrelddele nie mits optimale bestuurs-standaarde gehandhaaf word. Daar is nietemin 'n verminderde gewigstoename by die aangetaste jong dier wat noodwendig die voedingstydperk vanaf speen- tot slagouderdom verleng en assulks 'n ekonomiese verlies daarstel. Hoewel die etiologie van die siekte nog nie bevredigend gedefineer is nie en daar ook geen spesifieke toetse is om die moeilike kwessie van diagnose enigsins te vergemaklik nie, is dit my beskouing dat in kuddes waar E.P. 'n probleem skep en die voorkome bevestig is op gronde van kliniese verskynsels, epidemiologie en herhaalde makro- sowel as mikroskopiese longondersoeke, daadwerklik pogings aangewend moet word om van die siekte ontslae te raak. Tot sodanige tyd dat meer doeltreffende uitroeiingsmetodes beskikbaar is kan ons die varknywerheid probeer help deur oorweging te skenk aan een van die volgende moontlikhede om skoon kuddes en dusdanig vervangingsmateriaal vir die herbevolking van besmette bedrywe daar te stel:—

(i) totale ontvolking met behoorlike ontsmetting van hokke en persele en leeglê daarvan vir minstens 4 weke waarna herbevolk kan word met ingevoerde varke vanaf 'n E.P. — vry bron;

(ii) deur die teel uit 'n paar uitgesoekte klinies-gesonde ou sôe in afgesonderde enkelhokke en kontrolering van die nageslag deur 'n volledige laboratorium ondersoek op 1–2 varkies van 4 weke ouderdom en herhaalde kliniese ondersoeke elke 4 weke daarna. Normaalweg kan die aanteel gebruik word vir die opbouing van skoon kuddes indien die laboratorium bevindings negatief is en geen hoes of ander kliniese tekens, veral na geforseerde oefening, op 8 weke ouderdom aanwesig is nie; of

(iii) beheerde geboorte in isolasie en die hansgrootmaak van varkies nadat hulle onder streng gekontroleerde toestande toegelaat is om 1–2 maal biesmelk te suip en veral voorsorg te tref dat hulle nie met die sog se kop in aanraking kom nie. Hierdie metode verg ook besondere hoë higiëniese standaarde, die gebruik van infra-rooi lampe, kunsmatige voeding elke 4 ure

met daaglikse kook van suiptrôe, ens. maar is reeds op Onderstepoort en deur boere suksesvol toegepas.

2. As gevolg van 'n geïsoleerde geval van asemhalingsmoeilikheid wat gepaard gegaan het met nies- en snuifsimptome by 3-week oud varkies op 'n plaas in Natal gedurende 1962 en inklusie-liggaam rinitis histologies in monsters op Onderstepoort vasgestel is, is gedurende 1963 besluit om 'n landswye slagpale-opname van varksnoetseksies te onderneem. By die sowat 9,000 seksies wat ondersoek is van varke afkomstig van 180 verskillende varkbedrywe is variërende grade van atrofie van die snoetkraakbene in 4.5 persent van die gevalle gevind. Uitwendige gesigsabnormaliteite soos verwronge snoete, ens. was egter nie bespeurbaar nie terwyl die karkasse van aangetaste diere van ewe goeie kwaliteit, gewig en gradering was en dié van dieselfde werpsels wat nie atrofiese veranderinge getoon het nie. Met latere opvolgingspeksies op 'n aantal plase van herkoms van die atrofie gevalle kon geen geskiedenis van asemhalingsmoeilikhede by jong of ouer varke verkry word nie. Die etiologie en belangrikheid van hierdie toestand in Suid-Afrika is dus nog ietwat onduidelik en hoewel dit voorasnog aanvaar word om die patogeen of van ekonomiese belangrikheid te wees nie, verg dit beslis verdere opklaring.

'n Redelik ontstellende faktor wat ook onder aandag gekom het by die kontrolering van deelnemende kuddes aan die Varkaantekening en -gesondheidskema en van geweldige ekonomiese belangrikheid is, is die ontsettend hoë mortaliteit wat byna sonder uitsondering voorkom by pasgebore en jong varkies voor speenouderdom. Hierdie verliese varieer van 20 tot 35 persent en dit moet aanvaar word dat dit moontlik nog hoër kan wees in bedrywe waar geen beredeneerde bestuursstandaarde neergelê is of nagevolg word nie. Dit word beskou dat alle moontlike pogings aangewend moet word om die oorsake van hierdie verliese by varkboerderye te bepaal en reg te stel.

Alles in aanmerking genome en in weerwil van die feit dat na die beskouing van baie persone varkboerdery 'n minder belangrike rol speel in die veenywerheid van die Republiek van Suid-Afrika, word dit voorgelê dat dit ons professie as die bewaarders oor algemene dieregesondheid in die land, se dure plig is om meer aandag te begin gee aan die ontwikkeling van beheerprogramme wat die gesondheidspeil van ons sowat 600,000 varke sal verhoog en ook voorkombare verliese sal uitskakel.

ERKENNING

Graag betuig ek my dank aan die Hoof van die Afdeling Veeartsenykundige Velddienste vir sy toestemming om hierdie referaat te publiseer.

VERWYSINGS

1. LOVEDAY, R. K. (1958). "Swine Health Control", *J.S. Afr. vet. med. Ass.*, **29**, 201–207.
2. SWAHN, O. (1963). Persoonlike mededeling.
3. ABELSETH, M. K. *et al.* (1963). "Controlled Studies on Swine Repopulation in Canada", XVIIde Veeartsenykundige Wêreldkongresverslag, **1**, 251–255.

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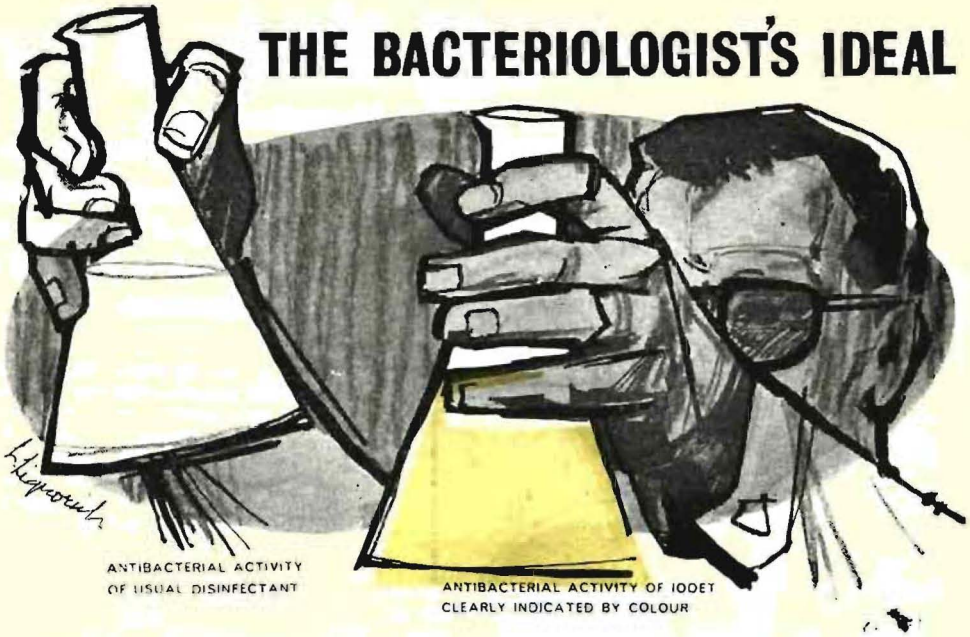

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OESOPHAGEAL FISTULATION OF SHEEP

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SUMMARY

A two stage operation for fistulation of the oesophagus and insertion of a special designed split-plug is described in order to prepare sheep for experimental sampling of grazing. Of 43 sheep operated upon by this method 25 survived for at least three months after discharge from hospital.

INTRODUCTION

Oesophageal fistulation is used for the collection of samples representative of material eaten by grazing animals. The method of Torell[1] as used by Lombard and van Schalkwyk[2] was first tried but proved unsatisfactory, due to continual wound infection and migration of the pins in the plastic tubes closing the fistula. The methods described by Cook *et al*[3] and Lesperance *et al*[4] were abandoned because of repeated blocking of the cannulae. Although Hamilton *et al*[5] found their technique effective in eight out of nine sheep, the oesophageal mucosa sloughed and produced fistuale. McManus used split plugs[6] and after inserting them into the oesophagi of thirty-seven sheep, he and his coworkers were left with fifteen which could be used for grazing experiments six months after the operation[7].

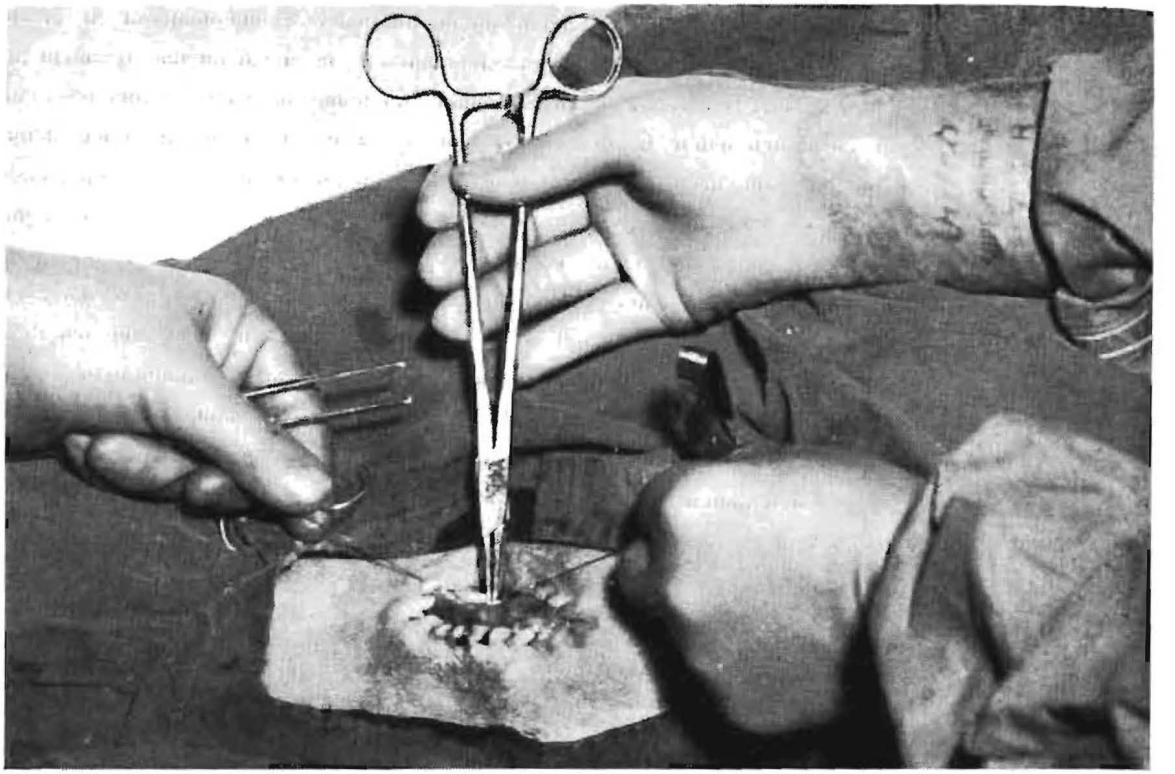
OPERATIVE TECHNIQUE

The operative technique eventually evolved was done in two stages: First fixation of the oesophagus to the skin and then insertion of the plug by means of which the fistula is closed and contraction prevented between collection periods.

First stage

Slow intravenous injection of chlorpromazine at the rate of four to five mg/kg body weight as well as local infiltration analgesia (subcutaneous and deep) are employed and the operative field is prepared (clipping being preferable to shaving).

With the sheep on its right side the mouth is gagged and a stomach tube is passed. An incision exceeding the size of the plug by two cm is made through the skin and subcutis in the middle third of the neck between the trachea and sternomastoid muscle. (Incisions dorsal to the sternomastoid muscle are more prone to leak. Muscular movements are also held responsible for injury and stenosis observed in all 13 cases so operated). By blunt dissection and haemostatic procedures, the oesophagus is exposed and delivered by means of gallbladder forceps introduced behind the oesophagus. At either end of the commissure, the fascia, usually

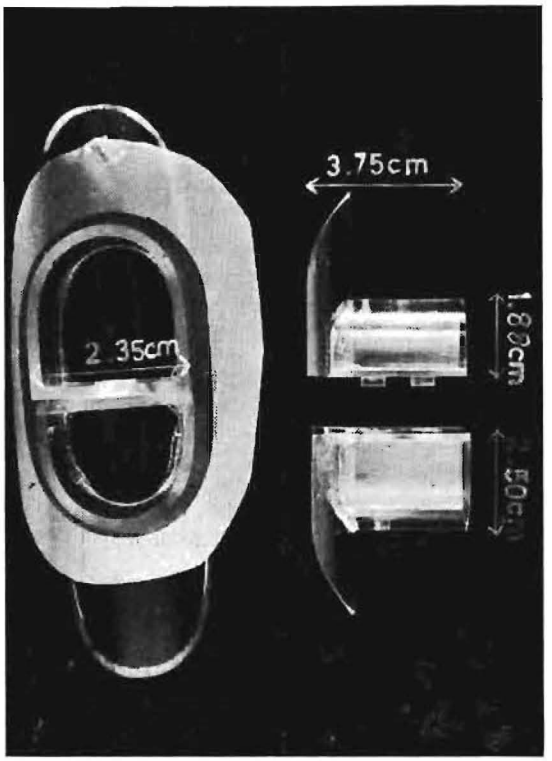


containing fairly large bloodvessels, must be divided to effect full delivery. The wall of the oesophagus is secured to the skin over an elliptical area by continuous chronic catgut suture, beginning and ending at the holding forceps and passing only as deep as the submucosa. The suture must be left long at the knot for final tying. Daily sulphanilamide dusting constitutes the after-treatment.

Second stage

After an optimum period of 12–15 days the oesophageal wall is incised under local anaesthetic — a stomach tube being passed as before — and the plug inserted. Any division of the newly formed granulation tissue to fit the stopper predisposes to infection, which may cause death. Before 12 days have elapsed the union between skin and oesophagus is not firm enough and after 20 days contraction of the wound prevents insertion of the plug.

The plugs are made from clear polymethyl methacrylate ("Perspex") each in two unequal sections as shown in the figure, so that three different sizes may be obtained by fitting the different halves together, thus allowing for contraction or dilatation of the fistula. The two sections are held together by a rubber band. At the first insertion one wide and one narrow section is used to form a medium-sized plug.



Experimental Procedure

This may be summarised as follows:

1. Number of sheep operated upon according to techniques outlined by other workers	11
2. Number of sheep with incisions dorsal to the M. sternomastoideus (Seven of this group received chlorpromazine and atropine i/v followed by nitrous oxide and either inhalation.)	13
3. Number of sheep operated according to present technique	30
Total	54

After fistulation the sheep were sent to various experimental stations, involving a train journey often over hundreds of miles. The conditions under which the sheep were kept at the various stations varied greatly.

Results

As the conditions under which this work was carried out were those attending routine surgery, neither experimental planning nor standardisation was possible, consequently results cannot be assessed critically. Taking the least favourable view, all the sheep operated upon according to the various modifications introduced by us, that is the total of 43 sheep of groups 2 and 3, may be classed together. Of these, six died from causes unrelated to the operation, seven from direct sequelae (purulent or gangrenous infection, dissolution of union between oesophagus, impaction of the oesophagus and consequent foreign body pneumonia, "loss of condition") and five from unfavourable post-operative conditions (exposure and privation during transit, loss of plug).

Twenty-five sheep could be used for sample collections for at least three months after discharge from hospital, i.e. 62.5 per cent. In contrast to two to three months' period elapsing before sheep were suitable for sampling of grazing after the older methods of operation, many of these sheep could be used for smapling three weeks after the first stage of the operation.

Loss of plugs, with rapid contraction of the fistulae so that tardy replacement became impossible, or loss with damage to the oesophagus leading to progressive stenosis, were late sequelae. The animals consequently became either useless for further sampling work, or they died.

Conclusions

1. The method outlined has given promising results which compares favourably with those obtained by other methods.
2. Use of the unequally split plug makes allowances for dilatation or contraction of the fistula.
3. The convalescent period is shorter than heretofore obtained.
4. Only sheep in good condition, free from debilitating disease, should be operated upon.
5. During the post-operative period sheep should be housed singly in loose boxes to avoid accidental tearing out of the plugs.
6. Sheep put out to grazing early, always do much better. Close confinement in wired pens is to be avoided, in order to obviate injury by the plug to the mucosa of the oesophagus.
7. Good supervision at all times is a requisite.
8. Debilitating conditions such as prolonged transport must be avoided as far as possible.

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REFERENCES

1. TORELL, D. T. (1954). An oesophageal fistula for animal nutrition studies. *J. Anim. Sci.*, **13**, 878.
2. LOMBARD, P. E. and VAN SCHALKWYK, A. S. (1963). Changes in the composition of Feedstuffs during sampling by means of an oesophageal fistula. *So. Afr. J. Agric. Sci.*, **6**, 205.
3. COOK, K. C. W., THORNE, J. L., BLAKE, J. T. and EDLEFSEN, J. (1958). Use of an oesophageal fistula cannula for collecting forage samples by grazing sheep. *J. Anim. Sci.*, **17**, 189.
4. LESPERANCE, A. L., BOHMAN, V. R. and MARBLE, D. W. (1960). Development of techniques for evaluating grazed forage. *J. Diary Sci.*, **43**, 682.
5. HAMILTON, F. J., McMANUS, W. R. and LARSEN, L. H. (1960). An improved method of oesophageal fistulation for food intake studies in the sheep. *Austral. Vet. J.*, **36**, 111.
6. McMANUS, W. R. (1962). Oesophageal fistulation studies in the sheep. *Austral. Vet. J.*, **38**, 85.
7. McMANUS, W. R., ARNOLD, G. W. and HAMILTON, F. J. (1962). Improved techniques in oesophageal fistulation of sheep. *Austral. Vet. J.*, **38**, 275.

NEMATODE PARASITISM OF SHEEP IN THE SOUTH WESTERN DISTRICTS OF THE CAPE PROVINCE

PART II—A SURVEY OF WORM EGG COUNTS IN EWES, YEARLINGS AND LAMBS

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SUMMARY

1. A seasonal incidence survey, based on differential worm egg counts, was carried out over a period of two years. Flocks of ewes, yearlings and lambs on three farms, each in a different ecological area of the South Western Districts of the Cape Province, were used.
2. *Trichostrongylus* was dominant and lambs showed a spring rise and a secondary peak in autumn.
3. *Ostertagia* showed no definite seasonal incidence patterns.
4. *Oesophagostomum*: Peak egg counts were recorded from April to July.
5. *Chabertia* reached a peak from November to March.
6. *Haemonchus* was only present at Outeniqua, primarily during summer. Lambs were heavily infested.
7. Severe clinical parasitism in the lambs at Outeniqua necessitated treatment in March.
8. "Strategic" drenching in March, August and December is recommended.

The South Western Districts comprise the coastal area of the Cape Province bordered by the Humansdorp district in the East and the Hottentots-Holland mountain range in the West (Fig. 1).

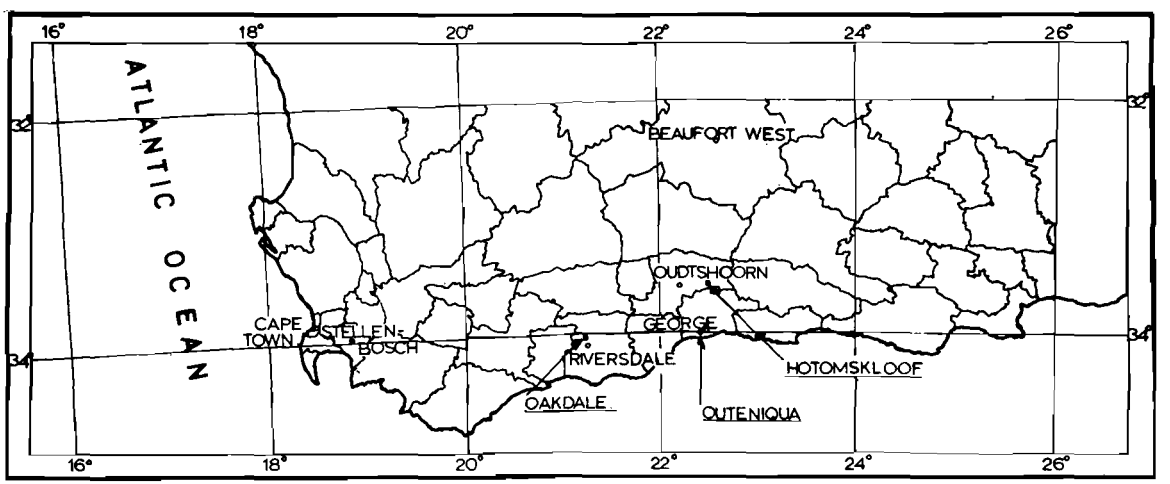
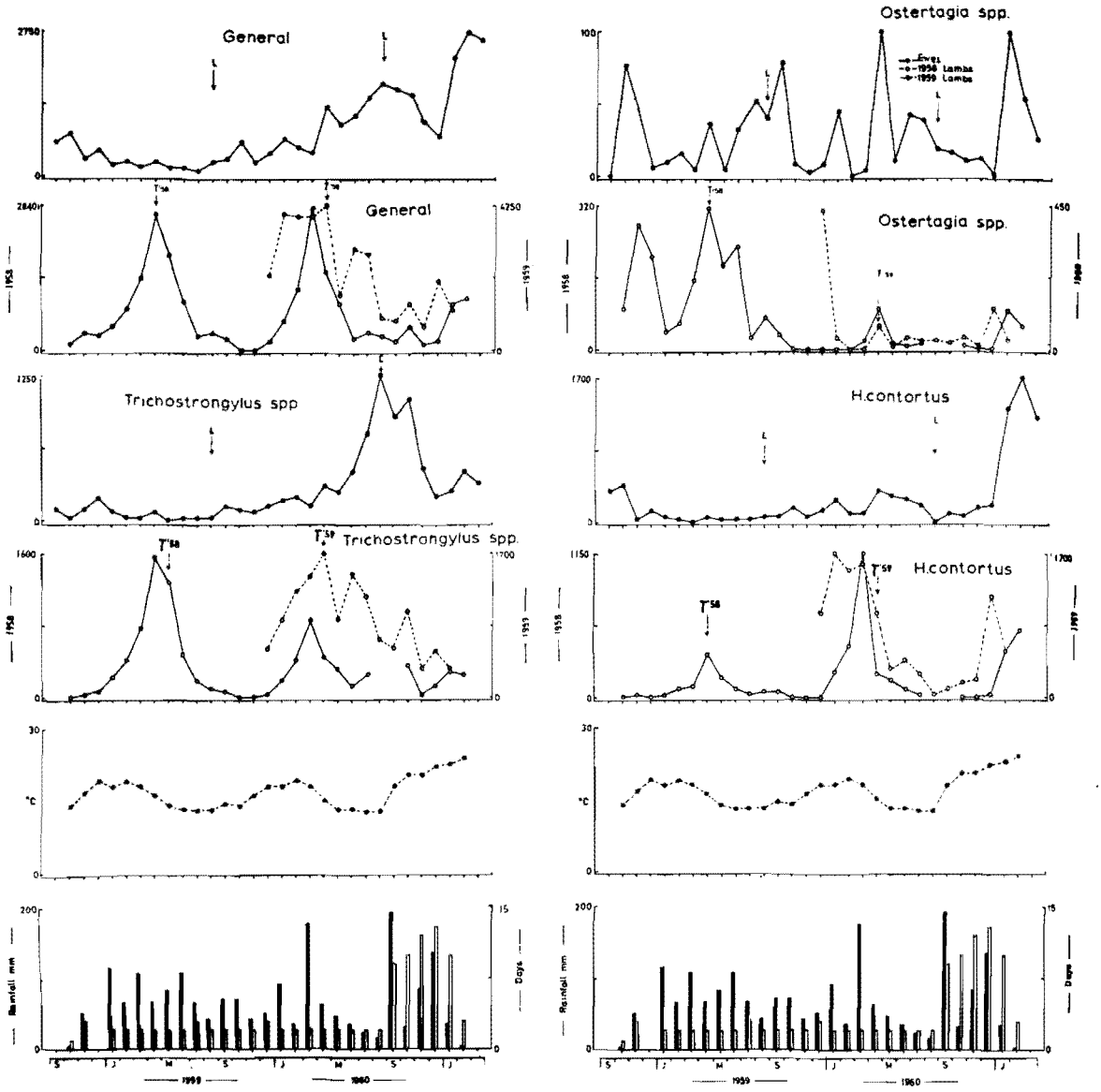


Fig. 1



NOTE — Average eggs per gram counts at monthly intervals. Left ordinants, ewes and 1958 lambs; right ordinants 1959 lambs. —●— = mean monthly temperatures; ▮ =monthly rainfall; ▯ =number of days on which rain fell.

Figure 2

GROUP A — OUTENIQUA

This survey was carried out in three distinct ecological areas viz. George, Riversdale and Oudtshoorn. George and Riversdale are situated between the Outeniqua mountain range and the sea; the average altitude of this coastal plain is 300 feet. The rolling dune-like country is broken by numerous rivers, streams and ravines. Oudtshoorn lies in the valley of the Olifants River, between the Swartberg mountains in the north and the Outeniqua mountains in the south. The average altitude of this valley is 1,300 to 1,500 feet.

George

The rainfall is non-seasonal, averaging 820 mm per annum. The driest months are January and July. The climate is temperate with little variation in the daily temperatures; frost is rare. The vegetation is mostly scrub, dominated by *Elytropappus rhinocerotis* (renosterbos) and poor quality grass. The rainfall and the climate favour the establishment of permanent pastures of grass-clover leys, rotated with annual cereals. Dairying and vegetable growing are carried out on a large scale.

Riversdale

Average annual rainfall is 500 mm, most of it falling during autumn and early spring. The winters are relatively mild, but moderate frosts occur; the summers are usually hot and dry. The main farming activities are wheat growing and wool production. Natural grazing, being sparse and inferior, is supplemented with annual cereals and lupins (*Lupinus* spp.) during winter and spring, and with stubble grazing on the harvested fields in summer and autumn.

Oudtshoorn

This is an arid area because the Outiniqua Range acts as a barrier to the moisture-laden South East tradewinds. The vegetation which is typically karoid and relatively sparse, consists mainly of succulents (*Mesembryanthemum* spp.)

The annual rainfall, averaging 125 mm, falls mainly in winter, with an occasional thunderstorm in summer. The climate is severe, with very short, mild winters and long summers, which are characterised by intense sunlight and extremely high maximum temperatures (35°C or higher). Livestock farming and the crops produced (lucerne, tobacco and fruit) are dependent on irrigation. This is the home of the ostrich industry; and the communal grazing of these birds with sheep, dairy cattle and horses, is normal practice.

Lucerne is either cut at the 10 per cent flowering stage for hay and the stubble grazed, or it is grown to the full flowering stage and then grazed simultaneously by all the animals mentioned. Pastures are always grazed down to the ground, exposing the soil to the hot sun. This results in a considerable degree of dessication.

Muller (1962)[10] recorded the following important parasites in these areas:—

Trichostrongylus spp.
Ostertagia spp.
Haemonchus contortus
Bunostomum trigonocephalum
Chabertia ovina
Oesophagostomum spp.
Cooperia spp.

Muller also recorded the following lesser parasites:—

Strongyloides papillosus
Nematodirus spp.
Marshallagia marshalli.

In this paper observations on the epizootiology of the nematode parasites of sheep, based on differential worm egg counts, will be presented.

Materials and Methods

Farms

Three flocks of 25 ewes each were used on the following farms (Fig. 1):

Group A: Outeniqua Experimental Farm, George.
Group B: Oakdale Agricultural School, Riversdale.
Group C: Hotomskloof Farm, Oudtshoorn.

Livestock

Group A: German Merinos and Dormers (Dorset Horn x German Merinos) were stocked at the rate of 3 ewes per acre. Both are dual-purpose breeds. They were kept mainly for fat

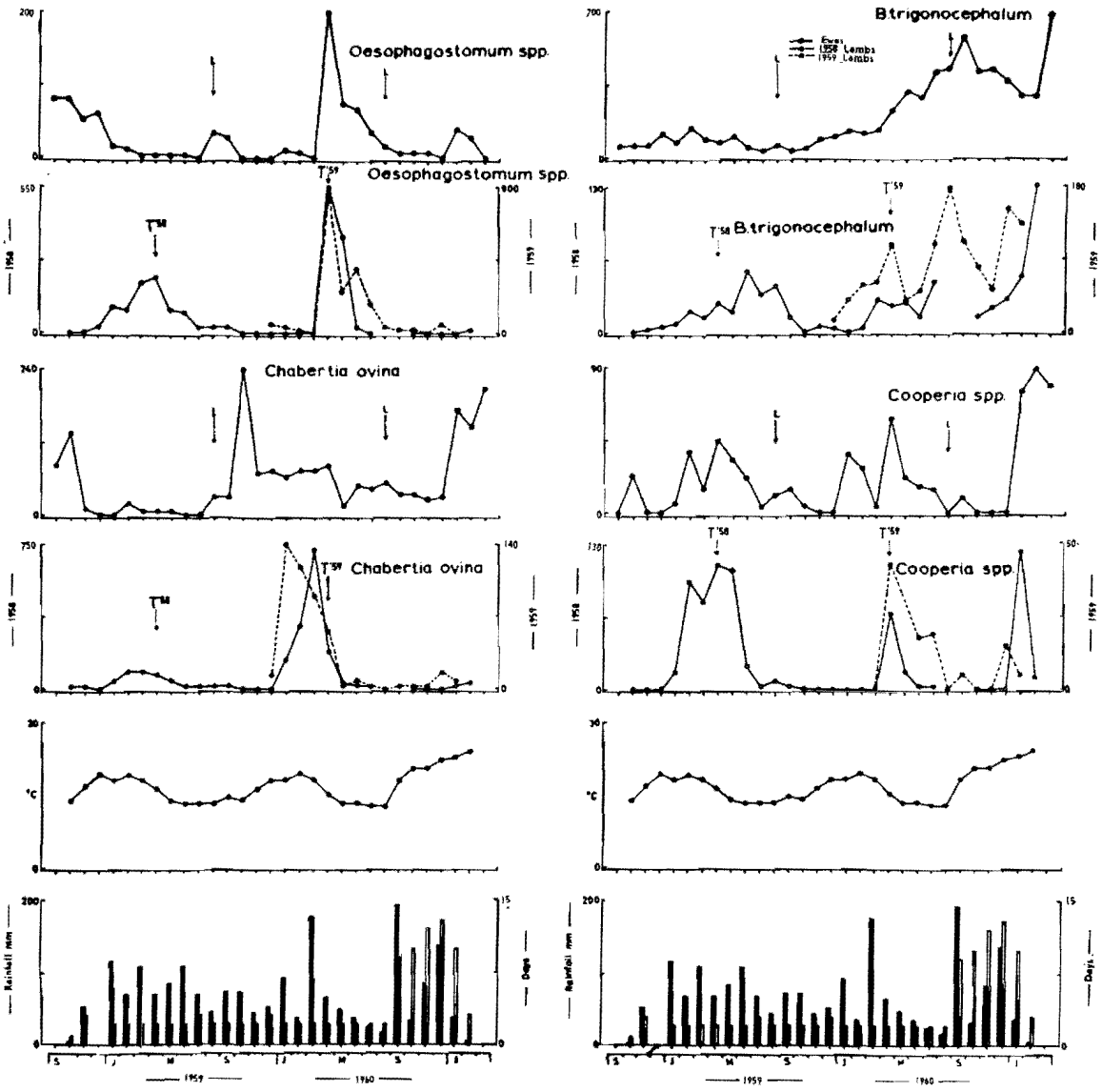


Figure 3

GROUP A — OUTENIQUA

lamb production. The ewes lambed in spring and the lambs were identified by the year of their birth. The survey was conducted from 26 September, 1958, to 9 March, 1961.

Group B: Pure-bred Merinos lambing in autumn, were kept at the rate of 1 ewe per acre. The survey period commenced on 26 January, 1959, and concluded on 23 March, 1961. Lambing occurred twice and the lambs were referred to as 1959 and 1960 lambs.

Group C: This group consisted of Dorpers (Dorset Horn x Blackhead Persian) which are extremely hardy, excellent foragers, particularly suitable for arid areas. They produced excellent fat lambs. Autumn lambing occurred twice during the survey period, running from 9 March 1959, to 20 February, 1961. The lambs were identified as 1959 and 1960 lambs.

Differential Egg Counts

Faeces samples were collected from the ewes throughout the experimental period; while the collection of samples from the lambs commenced at the age of 2 months. The methods of Reinecke (1961)[13] were used.

Husbandry Methods

The experimental groups ran with the flocks belonging to the respective farms. As an exception to normal husbandry practices, the experimental ewes were never treated with anthelmintics. At Outeniqua and Hotomskloof the sheep were confined to pens at night as a protection against jackals, whereas the flocks at Oakdale were on free range in jackal-proofed paddocks.

Results

Results, grouped at monthly intervals, are shown graphically in figures 2 to 8. These figures include details of rainfall and average monthly temperatures.

GROUP A: OUTENIQUA

General trends in egg counts

Ewes: From October, 1959, until January, 1960, egg counts were generally low, i.e. less than 1,000 e.p.g. (eggs per gram of faeces); thereafter the counts rose steadily till August, 1960 (2,730 e.p.g.), to fall again during the remainder of the year. During the first two months of 1961 the counts again rose sharply to reach 3,730 ep.g. at the end of the survey.

1958 Lambs: Egg counts rose steadily until the autumn of 1959, falling throughout the rest of the year and rising again to a similar peak (3,924 e.p.g.) in autumn (March), 1960. A similar rise and fall in the egg counts of these lambs was recorded in the following year. At the peak counts (3,305 e.p.g.) in April, 1959, these lambs showed clinical symptoms of parasitism, which necessitated maximum doses of liquid micro-fine phenothiazine. These lambs were erroneously given a second maximum dose of phenothiazine on 5 October, 1959. At the second peak in the autumn of 1960, severe clinical parasitism was again noted. No anthelmintics were given, and within 4 weeks the egg count dropped to 1,410 e.p.g. This fall continued until a count of 140 e.p.g. was recorded.

1959 Lambs: These lambs showed a steady rise in egg counts to reach a peak of 5,204 e.p.g. in April, 1960, when 13 out of 32 lambs died as a result of parasitism. Treatment with phenothiazine was essential to prevent further mortality. Although egg counts fell temporarily after treatment, there was a sharp resurgence to an average of 3,480 e.p.g. six weeks later, subsequently falling to a low level with the exception of a minor peak in October ,1960. During the hot months of January and February, 1961, egg counts started to rise again.

The order of prevalence of the genera were: *Trichostrongylus* spp.

Ewes: Egg counts remained low with a minor peak after lambing in the spring of 1959. There was a marked rise in 1960, starting in the autumn to reach a maximum peak of approximately 1,600 e.p.g. at the height of the lambing season (August). Subsequent counts fell to a low level in January, 1961.

1958 Lambs: Egg counts rose steadily until April, 1959, falling after treatment to a low level, with a minor peak at approximately the same period in 1960.

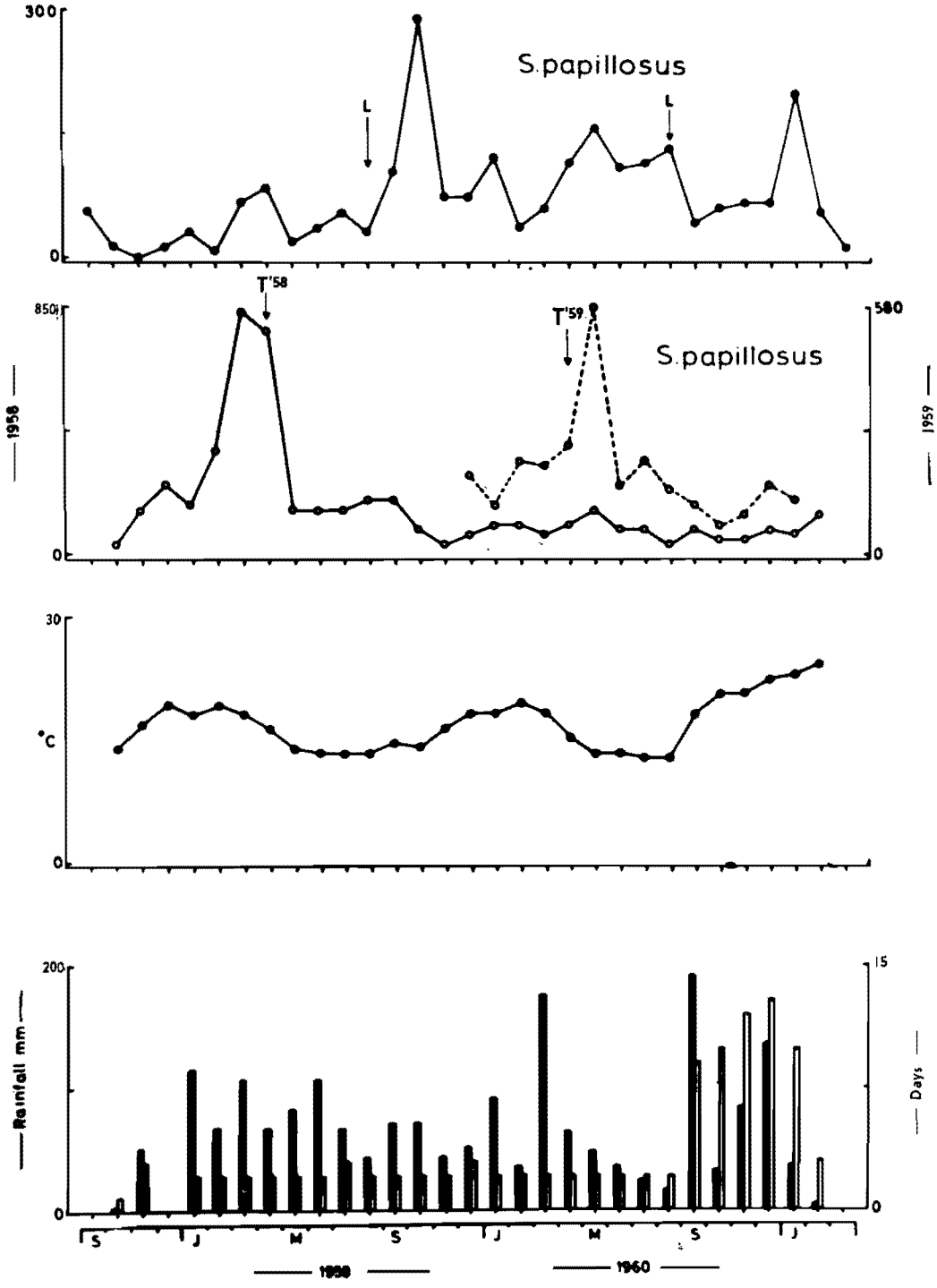
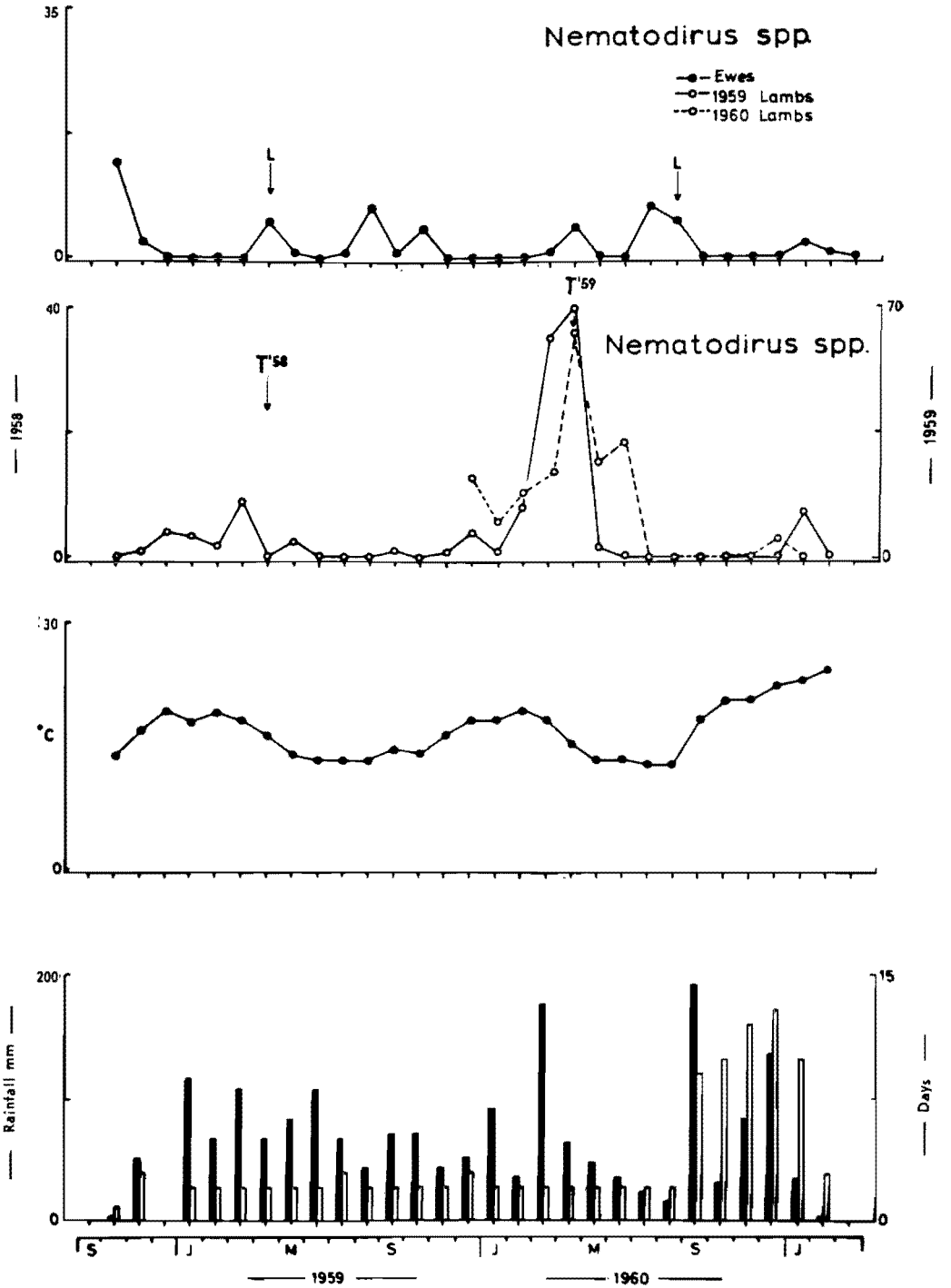


Figure 4



GROUP A — OUTENIQUA

1959 Lambs: These egg counts followed the same trends as shown by the 1958 lambs. The rapid drop after treatment was followed by a peak in June, 1960 (1,700 e.p.g.), falling steadily thereafter.

Haemonchus contortus

Ewes: Egg counts rose during 1960, to reach a maximum peak of 1,600 e.p.g. in January, 1961.

1958 Lambs: Egg counts were insignificant, the highest 1,600 e.p.g., being recorded in March, 1960.

1959 Lambs: Egg counts were high during the summer and the autumn of 1960, with a repetition of this pattern in the following summer of 1961.

Ostertagia spp.

Ewes: Although egg counts were relatively low, the general pattern closely followed that of *H. contortus.*

1958 Lambs: Egg counts rose to a peak (330 e.p.g.) in April, 1959, falling after treatment, to rise again in June of that year. Thereafter egg counts were low except for a minor peak in the following April, and a rising trend in January, 1961.

1959 Lambs: Apart from a minor peak in December, 1959, egg counts were insignificant.

Bunostomum trigonocephalum

Ewes: Egg counts showed a steady rise throughout the autumn and the winter of 1960 and reached a peak of 750 e.p.g. or higher in the spring (September); thereafter remaining at a fairly high level.

1958 Lambs: Egg counts were very low, reaching significant levels when the lambs were 12 months old, only to fall again. Counts started rising at the end of the survey period, when this group was 2½ years of age.

1959 Lambs: When this group reached the age of approximately 10–12 months, counts of 150 to 200 e.p.g. were recorded; falling during the late spring and the early summer, to rise again in January, 1961.

Chabertia ovina

Ewes: Egg counts rose every year in late spring and early summer, falling to low levels in the ensuing autumn and winter. The highest egg count was 220 e.p.g. in October, 1959.

1958 Lambs: Seasonal peaks were reached somewhat later, viz: in February ,1959, and in March, 1960, with a maximum count of 700 e.p.g.

1959 Lambs: Counts rose to a peak of 1,520 e.p.g. in January, 1960; thereafter falling to very low levels, with only a slight rise in the following summer.

Oesophagostomum spp.

Ewes and Lambs: Although egg counts were generally low, all groups reached maximum counts in the autumn of 1960. The highest was recorded in the 1959 lambs (1,300 e.p.g.)

Cooperia spp.

Ewes and Lambs: This genus was recovered only in small numbers from larval cultures every autumn.

Strongyloides papillosus

Ewes and Lambs: This genus was recorded in all groups, and counts followed a pattern similar to that of the overall counts.

Nematodirus spp.

Ewes and Lambs: Egg counts were insignificant in all three age groups.

Comment

The non-seasonal rainfall in this area accounts for the unusual seasonal incidence of the various genera.

Although *Trichostrongylus* spp. showed an autumn rise in the lambs, this pattern was not reflected in the egg counts of the ewes. There appeared to be a "*post partum*" rise in ewes in spring (Crofton, 1955).[2] The summer rainfall parasite, *H. contortus*, was prevalent from January to April, as was recorded by Gordon (1948)[6] in Ausutralia, and by Thomas (1959)[17] in a summer rainfall region (Transvaal) of South Africa.

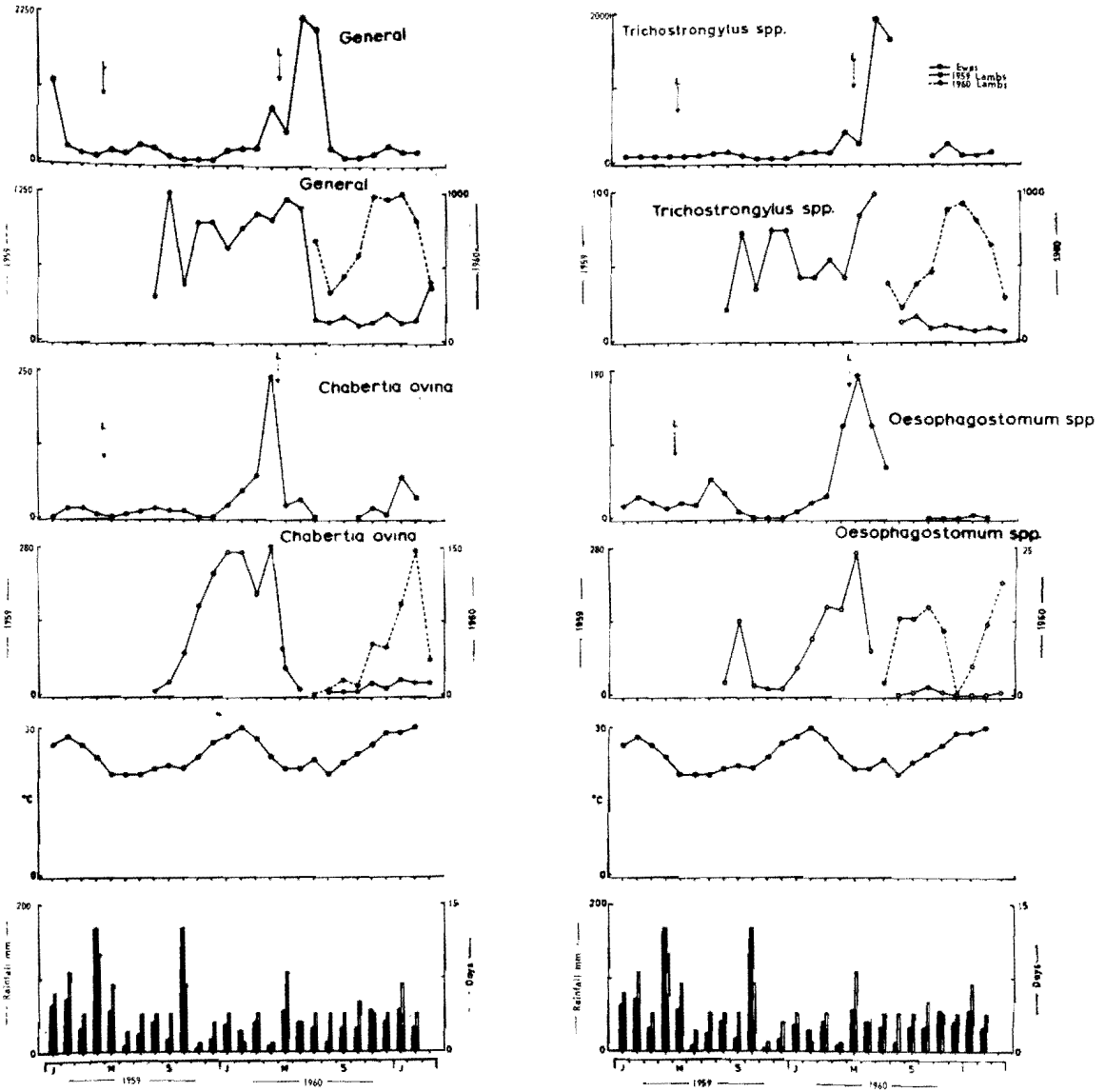


Figure 5

GROUP B — OAKDALE

Ostertagia spp. were largely responsible for the autumn rise in the egg counts of the lambs.

Apparently the importance of *B. trigonocephalum* increases with increasing age, as higher egg counts were recorded in the ewes towards the end of the survey, while the counts in the lambs reached significant levels only after the age of 18 months.

It is interesting to note than *Chabertia ovina* appeared only during the summer, while *Oesophagostomum* spp. showed a significant rise in egg counts in all animals in the autumn of 1960.

GROUP B: OAKDALE

General Trends

Throughout 1959 the egg counts at Oakdale were low, while appreciably higher counts were recorded in 1960.

Ewes: Egg counts remained at a very low level for the first year. The only appreciable rise occurred in the autumn and the winter of 1960, reaching a maximum of 2,480 e.p.g. in June, 1960.

1959 Lambs: Egg counts fluctuated noticeably in the first year with a series of peaks in the autumn. The highest count was 2,023 e.p.g. in May, 1960.

1960 Lambs: Egg counts were noticeably high at first, but fell in the early spring period, subsequently rising to a peak of 1,799 e.p.g. in November, 1960.

Trichostrongylus spp. and *Ostertagia* spp. were the genera of importance. *Ch. ovina*, *Oesophagostomum* spp. and *Nematodirus* spp. were present in moderate numbers only. *Cooperia* spp. were recovered in very small numbers in the summer of 1960 form the 1959 lambs, but were absent in the other animals of this group.

Neither *H. contortus* nor *B. trigonocephalum* were recovered from larval cultures.

Individual Genera

Trichostrongylus spp.

Ewes: Egg counts were very low with minor fluctuations during the first year, starting to rise when the ewes had lambed in April, 1960, reaching a maximum count of 2,030 e.p.g. in July. Egg counts decreased sharply in the following spring and summer.

1959 Lambs: Egg counts fluctuated noticeably during the first year, reaching peaks in March and May, 1960, of 847 and 1,400 e.p.g. respectively.

1960 Lambs: In this group the pattern was different from that found in the previous two groups. Egg counts rose in September, fell in October and early November, to rise again to a peak of 1,637 e.p.g. at the end of November.

Ostertagia spp.

Ewes: The trends were similar to those of *Trichostrongylus* spp. although peak counts were considerably lower (390 e.p.g.)

1959 Lambs: Noticeable fluctuations occurred with an initial peak of 600 e.p.g. in September, 1959. Minor peaks in November, 1959, February and March, 1960, were followed by low egg counts until March, 1961.

1960 Lambs: The highest egg count was recorded in July, 1960 (275 e.p.g.); thereafter counts decreased steadily except for occasional minor peaks.

Chabertia ovina.

In both ewes and lambs counts rose in November, to reach a peak in the lambs in January and February, while in the ewes, this was delayed until April.

Oesophagostomum spp.

The ewes and the 1959 lambs showed peak egg counts in April and May. Counts were insignificant in the 1960 lambs.

Nematodirus spp.

Ewes: Egg counts rose in April, 1960, to fall to insignificant levels by August.

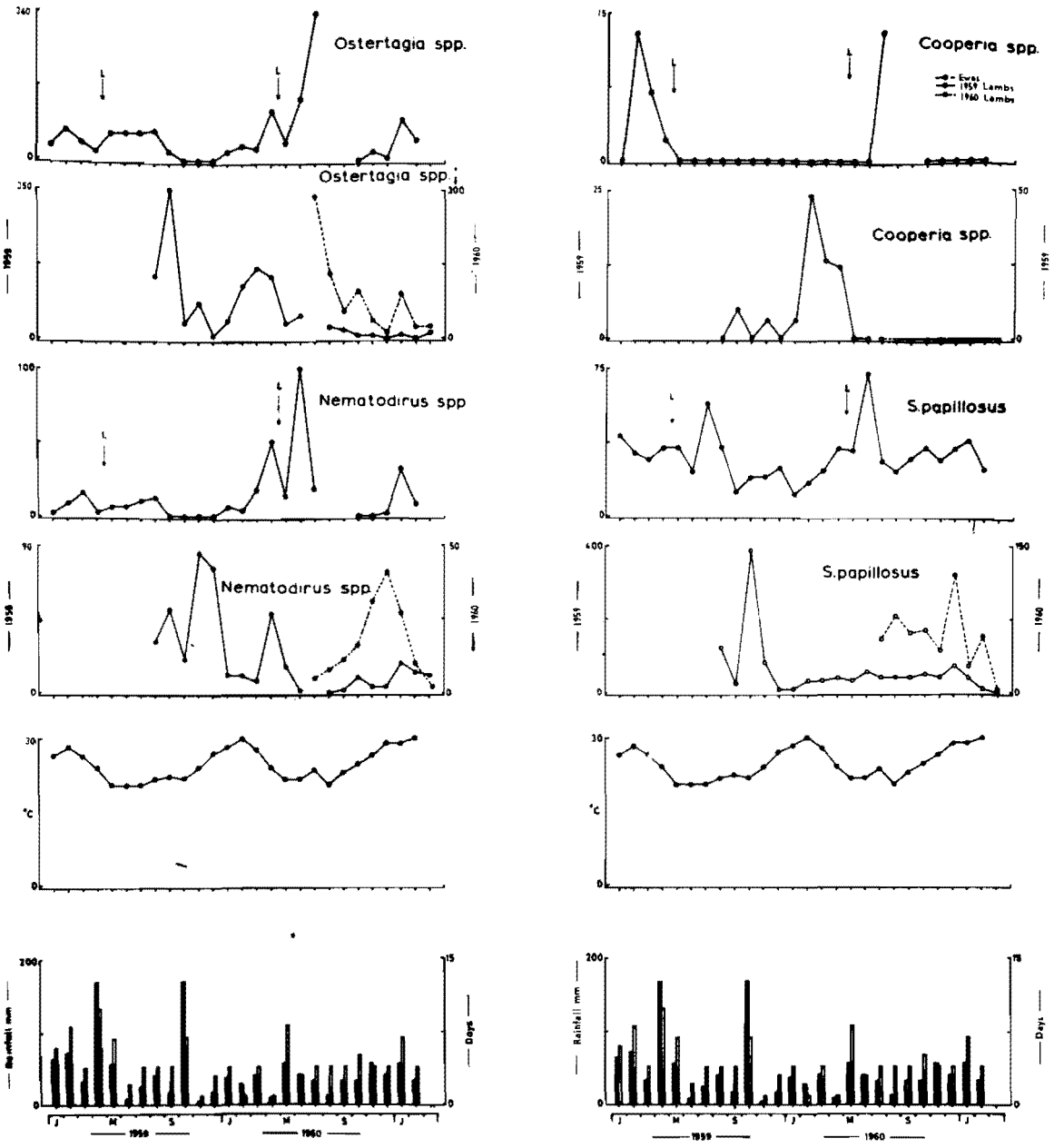


Figure 6

GROUP B — OAKDALE

1959 Lambs: Counts were characterised by a series of peaks and troughs, the highest egg counts being recorded from October to December.

1960 Lambs: The counts followed trends similar to those of the 1959 lambs.

Strongyloides papillosus

Ewes and Lambs: Counts in all three groups were insignificant.

Comment

In *Trichostrongylus* spp. no correlation of the egg counts in the three age groups was noted. The stress of lambing may explain the autumn rise in the egg counts of the ewes.

While the ewes showed a "*post partum*" rise of *Ostertagia*, the lambs started off with high egg counts in September, probably due to the high susceptibility of the new-born animal. There were, however, additional rises from March to May, which were similar to those encountered in the spring lambs of Group A. Both *Chabertia* and *Oesophagostomum* followed patterns similar to those of the flock at Outeniqua.

GROUP C: HOTOMSKLOOF

General Trends

In the ewes the general trends were similar to those of Group B, peak egg counts being noted from July to September, 1960.

During the first year of the survey, the egg counts in the 1959 lambs were very low, only rising to a series of peaks from June to September, 1960. The 1960 lambs initially showed higher counts in July; thereafter falling gradually with minor peaks.

The most important parasites were *Trichostrongylus* spp., *Oesophagostomum* spp. and *Ch. ovina*. *Ostertagia* spp. rarely exceeded 100 e.p.g., while *Cooperia* was recorded in the 1960 lambs only. Although few in number, eggs of *Marshallagia marshalli* were consistently recovered. This was the only group in which this genus was identified.

Individual Genera

Trichostrongylus spp.

Ewes: As at Oakdale, there was a rise during the period May to August, 1960, whereas the counts in the previous year were very low.

1959 Lambs: Until May, 1960, egg counts were low, seldom exceeding 100 e.p.g. but they rose from June to November, 1960, reaching 853 e.p.g.

1960 Lambs: High counts were recorded in July and August, 1960, but counts fell steadily thereafter, with a minor peak in November.

Ostertagia spp.

In all age groups the presence of this genus was insignificant.

Ch. ovina

Peak egg counts were noted in ewes and lambs in the summer.

Oesophagostomum spp.

Ewes and Lambs: Peak egg counts were recorded from April to October. These counts decreased gradually from November to February.

M. marshalli

This genus was recovered in small numbers throughout the period, particularly from the ewes. No seasonal trends were noted.

Comment

Chabertia ovina and *Oesophagostomum* spp. followed similar patterns to those recorded in the other groups, and were more prevalent than *Ostertagia* spp. This latter genus was not an important parasite at Hotomskloof, although adequate moisture, which is essential, was provided by flood irrigation of the pastures. This was not the case elsewhere. The mean temperatures at Hotomskloof were, however, extremely high with a very short "cool" period during June and July. The relative scarcity of this genus could, therefore, be accounted for, as the free-living stages of *Ostertagia* spp. and, to a

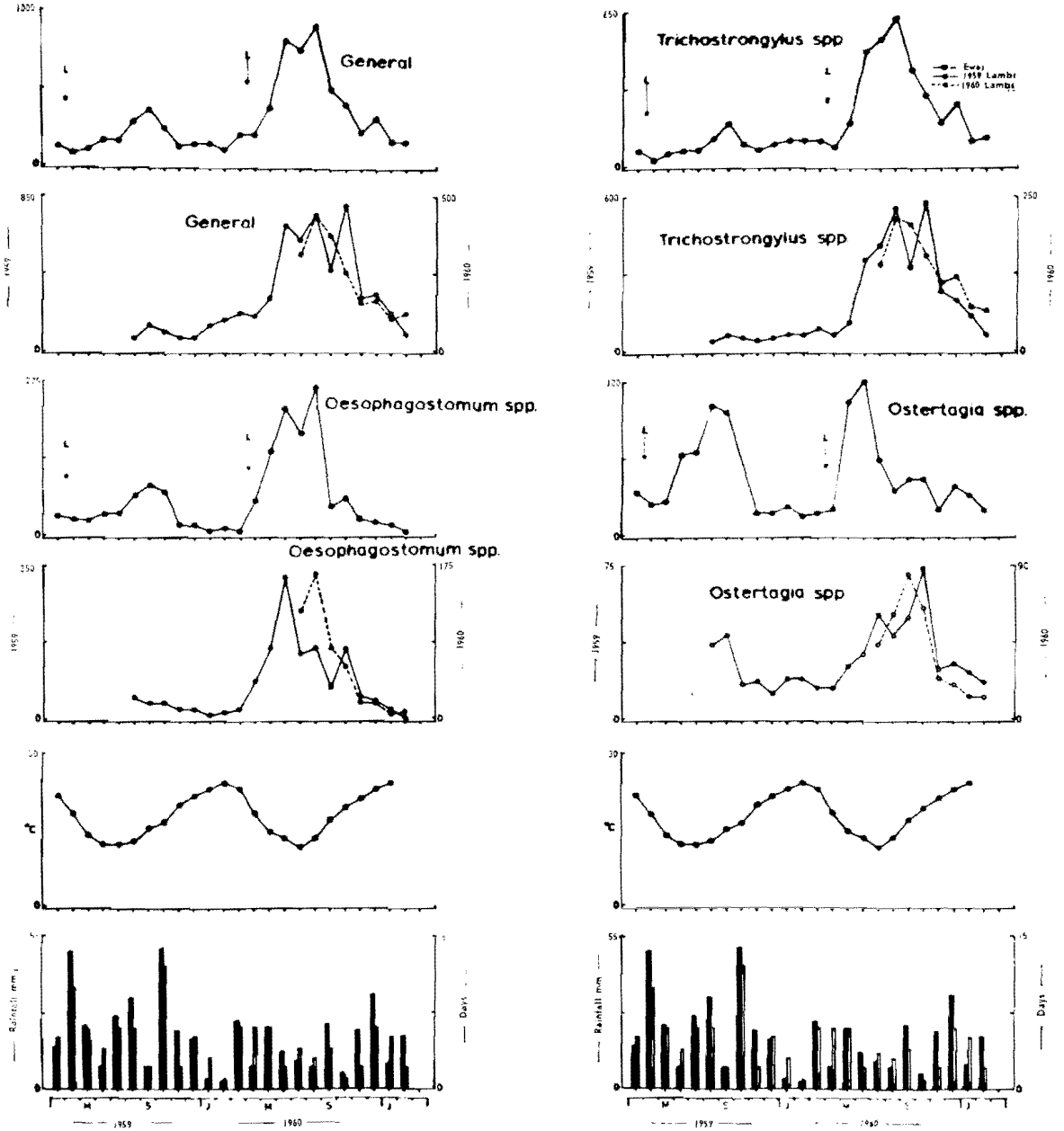


Figure 7

GROUP C — HOTOMSKLOOF

lesser extent those of *Trichostrongylus* spp., are sensitive to extremes of heat and dessication (Rogers 1940,[15] Furman 1944).[4] The cool and humid conditions at Outeniqua, and to a lesser extent at Oakdale, are more suitable for this genus.

Discussion

Because the climate and the husbandry methods on the three farms differ, the following observations are stressed:

Free-living stages: The survival rate of the free-living stages is determined by micro-climate, which is influenced by the state and condition of the pastures.

Outeniqua: Here the pastures are composed of dense-matting grass-clovers rotated with lush-growing cereals and lupins which, together with the higher rainfall and the equitable climate throughout the year, create optimum conditions for survival.

Oakdale: Although winter and early spring conditions are relatively favourable, the survival rate of the free-living stages in summer is low because of high temperatures and lack of moisture.

Hotomskloof: Despite periodic flood irrigation of the lucerne pastures, the arid, semi-desertlike conditions, coupled with very hot summers and sparse winter rainfall, militate against survival of the free-living stages.

Rainfall: A rainfall of 15 mm or more is usually followed by a significant rise in the egg counts of *Ostertagia* spp. and *Cooperia* spp. The graphs of the latter genus in the Group A lambs show high counts, which can be correlated with periods of heavy rain, e.g. 80 mm. in January, 1959, and 68 mm in March, 1960. Similar peaks in the autumn of 1960 and the summer of 1961 were preceded by heavy precipitations.

Age Susceptibility

Ewes: In all three groups low counts were recorded which increased only temporarily following the stress of parturition and lactation; nevertheless, the ewes remained excellent "carriers" of parasites, seeding the pastures and facilitating the infestation of succeeding generations of lambs.

Lambs: The high susceptibility of lambs to parasitic infestation is demonstrated by the high egg counts, which in all three groups exceeded those of the ewes, and also by the fact that the lambs in Group A were effected by clinical parasitism.

Prevalence of Different Genera

Trichostrongylus spp. and *Ostertagia* spp. are dominant at Outeniqua and Oakdale, while at Hotomskloof *Trichostrongylus* spp., *Ch. ovina* and *Oesophagostomum* spp. are the most important parasites.

The summer rise in egg counts in Group A is largely due to *H. contortus*. As both the rainfall and the temperatures during summer are favourable for the free-living stages of this parasite, its increase is understandable. The absence of *H. contortus* at Oakdale can be explained by the lack of moisture in summer, even though temperatures are high enough (Dinaberg, 1944).[3] At Hotomskloof where both temperature and moisture requirements of this parasite are met, the absence of *H. contortus* is puzzling and cannot be satisfactorily explained.

M. marshalli occurred only at Hotomskloof where the incidence of *Ostertagia* spp. was of a low order and *H. contortus* was absent. At Oakdale neither *H. contortus* nor *M. marshalli* was recorded, but *Ostertagia* spp. were relatively abundant; whereas at Outeniqua *H. contortus* and *Ostertagia* spp. were plentiful but *M. marshalli* was absent. The question can be posed to what extent *Ostertagia* spp. plays a role in provoking a cross-immunity to *Marshallagia marshalli*. This genus was previously classified as *Ostertagia marshalli* (Ransom, 1911).[11]

Seasonal Incidence

In Group A high counts were recorded in the ewes during summer months, and during autumn in the lambs. In both Group B and C the ewes had high counts throughout winter and spring, while the Group B lambs showed a spring and summer infestation (August to March); whereas the infestation in the Group

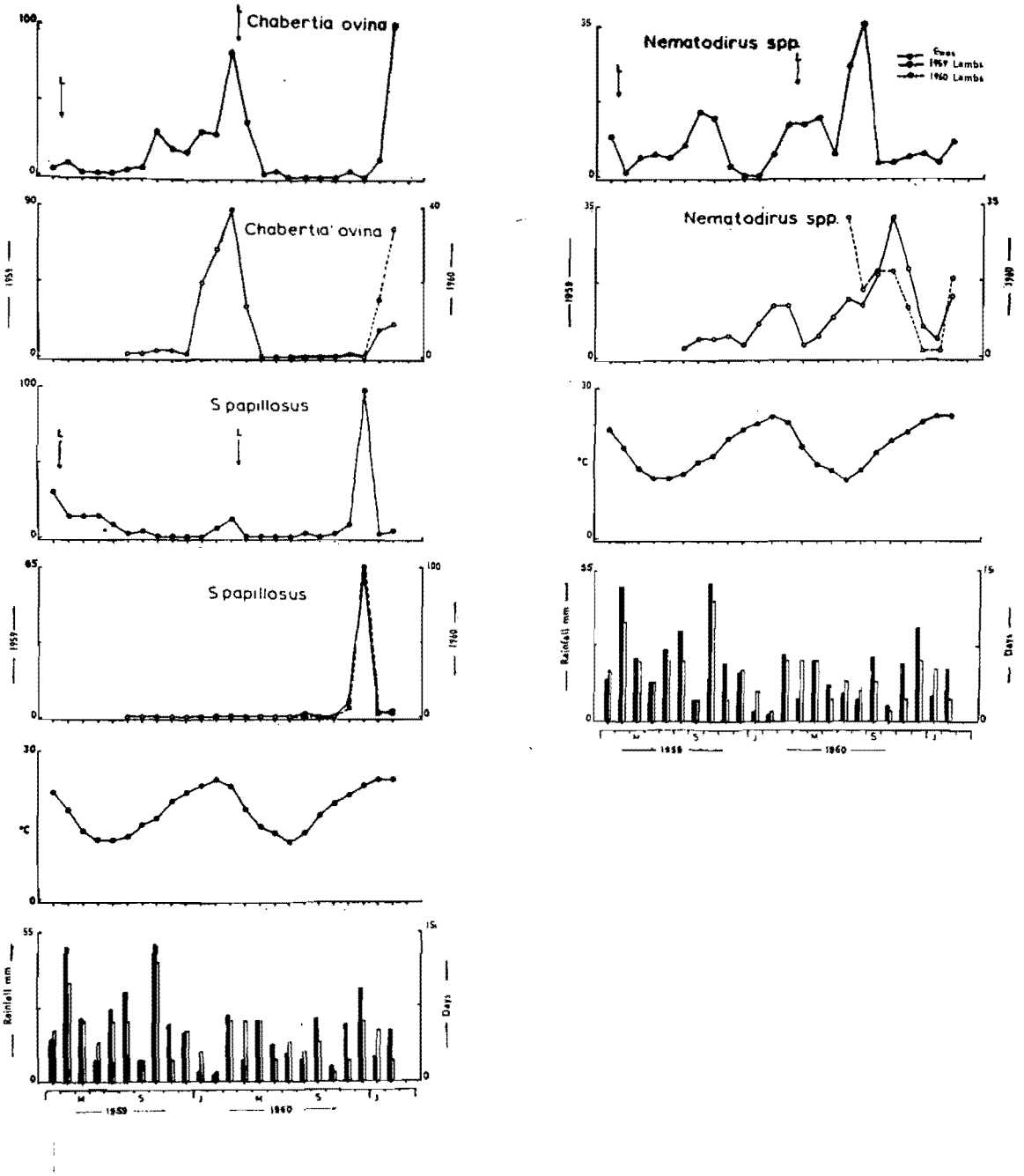


Figure 8

GROUP C — HOTOMSKLOOF

C lambs was recorded from midwinter to late spring (June to November).

Trichostrongylus spp. and *Ostertagia* spp. show very little correlation. The minor genera *Ch. ovina*, *Oesophagostomum* spp. and *Cooperia* spp., which are summer parasites common to all three groups, conform to a set pattern.

The autumn rise in the egg counts of the lambs in all groups can be ascribed to all parasites, while the spring rise, albeit of varying degree in the different groups, is in all probability due to *Trichostrongylus* spp. Similar tendencies in the epidemiology of *Trichostrongylus* have been described in New Zealand (Tetley, 1949)[16] and in Western Australia (Gordon, 1958).[8]

The graphs show the egg counts of various genera rising to a peak at the same time, despite the fact that the different genera all have varying prepatent periods e.g. *Strongyloides* 14 days and *Oesophagostomum* 35 days. This may be due to an oestrogenic substance in the pasture which plays a role in this synchornised rise in egg counts.

Reinfestations

In all three groups the lambs of the second season showed egg counts significantly higher than those of either their dams or the lambs of the first season. This could be the result of greater contamination of pastures on which heavily infected, untreated adult and yearling sheep had been concentrated. These higher counts were recorded at a very much younger age in the second crop of lambs in all three groups.

Animal Husbandry

At Outeniqua and at Oakdale the stocking rates were three and one ewe per acre respectively. This played a major role in the greater contamination of the pastures on the former farm.

At Hotomskloof the egg counts were significantly lower despite the concentration of the carriers on irrigated pastures. This is probably due to the fact that the sheep and cattle, horses and ostriches grazed the pastures simultaneously. The interaction of different animal species (cattle and sheep) on the epidemiology of various nematodes has been recordedby Tetley (1949)[16] in New Zealand.

In addition to the variety of animals, the grazing habits of the ostrich should be emphasised. This bird grasps the lucerne stalk very close to the ground, strips off the leaves, thus leaving the stem bare. This results in the breakdown of any favourable micro-climate which may have existed.

On a neighbouring property where only merino sheep were grazed continuously on lucerne lands, and the micro-climate was relatively favourable, the animals initially did extremely well. Within 12 months, however, heavy mortality was experienced as the result of severe parasitism. *Post mortem* examination showed very heavy infestation with *Trichostrongylus* spp., *Ostertagia* spp., and *Oesophagostomum* spp.

Treatment

Lambs of Group A showed clinical parasitism and resultant mortality to such an extent that dosing in autumn was necessary. Neither lambs nor ewes elsewhere had to be dosed.

The clinical parasitism at Outeniqua must be ascribed mainly to the presence of *H. contortus*, although there was a considerable rise in egg counts of both *Ostertagia* spp. and *Trichostrongylus* spp. At the same time a similar rise in egg counts was recorded in the lambs at Oakdale, but treatment of these lambs was not considered necessary.

'Strategic" Drenching

In the light of existing knowledge, the following "strategic" drenches are recommended to prevent the build-up of heavy worm burdens:

March
August
December

It would be wise to treat with either Methyridine or Thiabendazole because of their high efficacy against both adult and immature worms.

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REFERENCES

1. CROFTON, H. D. (1954). Nematode Populations in Sheep on Lowland Farms. I. Worm Egg counts in ewes. *Parasitology*, **44,** 465–477.
2. CROFTON, H. D. (1955). Nematode Populations in Sheep on Lowland Farms. II. Worm Egg Counts in Lambs. *Parasitology*, **45,** 99–115.
3. DINABERG, A. G. (1944). Development and survival under Outdoor conditions of eggs and larvae of the common ruminant stomach worm, *H. contortus*. *J. Agric. Res.*, **69,** 421–433.
4. FURMAN, D. P. (1944). Effect of environment upon the free-living stages of *Ostertagia circumcincta* (Stadelmann), *Trichostrongylidae* II Field Experiments. *Amer. J. Vet. Res.*, **5,** 147–153.
5. GORDON, H. McL. (1933). Differential diagnosis of the larvae of *Ostertagia* spp. and *Trichostrongylus* spp. of sheep. *Aust. Vet. J.*, **9,** 223–227.
6. GORDON, H. McL. (1948). The epidemiology of parasitic diseases, with special reference to studies with nematode parasites of sheep. *Aust. Vet. J.*, **24,** 17–45.
7. GORDON, H. McL. (1950). Some aspects of parasitic gastroenteritis of sheep. *Aust. Vet. J.*, **26,** 14–28.
8. GORDON, H. McL. (1958). The epidemiology of Helminthosis in sheep in winter rainfall regions of Australia. 2. Western Australia. *Aust. Vet. J.*, **34,** 5–19.
9. MULLER, G. L. (1961). Helminth Research in South Africa IV. Field trials on Thiabendazole as an anthelmintic for sheep; with a note on the assessment of diagnostic methods. *J.S. Afr. Vet. Med. Ass.*, **32,** 175–180.
10. MULLER, G. L. (1962). The seasonal incidence survey of internal parasites of sheep. *J.S. Afr. Vet. Med. Ass.*, **33,** 45–47.
11. RANSON, B. H. (1911). The nematodes parasitic in the alimentary tract of cattle, sheep, and other ruminants. *U.S.D.A. Bur. animal indust. Bull.*, **127,** pp. 132.
12. REINECKE, R. K. (1960). A field study of some nematode parasites of bovines in a semi-arid area, with special reference to their biology and possible methods of prophylaxis. *Onderstepoort J. Vet. Res.*, **28,** 365–464.
13. REINECKE, R. K. (1961). Helminth Research in South Africa III. The diagnosis of nematode parasites for worm survey purposes. *J. S.Afr. Vet. Med. Ass.*, **32,** 167–173.
14. ROBERTS, F. H. S. (1957). Reactions of calves to the stomach worm *Haemonchus placei* (Place 1893) Ransom, 1911. *Aust. J. Agric. Res.*, **8,** 740–767.
15. ROGERS, W. P. (1940). The effects of environmental conditions on the accessibility of third stage Trichostrongyle larvae to grazing animals. *Parasitology*, **32,** 208–225.
16. TETLEY, J. H. (1949). Rythms in nematode parasitism of sheep. *Dept. Sci. Ind. Res. New Zealand Bull. No.* 92, pp. 214.
17. THOMAS, R. J. (1959). Personal communication.

EPIZOOTIOLOGY AND CONTROL OF NEMATODE PARASITES OF SHEEP

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SUMMARY

(1) Critical slaughter experiments carried out by Rossiter (1964),[19] Barrow (1964)[1] and Viljoen (1964)[29] are reviewed and correlated.

(2) The seasonal incidence of the various genera is discussed.

(3) "Offensive" drenching, i.e. destruction of worms with anthelmintics when worm burdens are low, is recommended against *Haemoncus* in September; *Trichostrongylus* in December; *Oesophagostomum* in January and *Ostertagia* in May.

INTRODUCTION

Recently the seasonal incidence of parasitic nematodes of sheep has been investigated in the following summer rainfall areas of the Cape Province: the Karroo (Viljoen, 1964);[29] the Border Area (Barrow, 1964)[1] and the Eastern Coastal Area (Rossiter, 1964).[19] These surveys were based on differential worm egg counts and critical slaughter trials.

A correlation of the data collected by these workers shows general trends in the seasonal incidence of the various species. More effective control measures, based on the seasonal incidence, are possible with the use of the newly developed anthelmintics effective against all stages of development.

SEASONAL INCIDENCE

The seasonal incidence of the various species in the different areas has been summarized in Table I and represented schematically in Fig. I.

TABLE I.—MONTHS IN WHICH PEAK WORM BURDENS WERE RECOVERED IN SUMMER RAINFALL AREAS OF THE CAPE PROVINCE

Genus	Eastern Coastal Area		Border Area		Karroo	
	Fourth stage	Adults	Fourth stage	Adults	Fourth stage	Adults
Haemoncus	December August	December October	Negligible	May October	July	January October
Ostertagia	December November	October December	March	April	July	February
Trichostrongylus	November	*October June	March November	May	June	July
Oesophagostomum	April	December August	Negligible	May September	July	October July
Nematodirus	December January	January	November	April	January June	July
Trichuris	Absent	January	Absent	May	Absent	Absent

*Probably false peak due to 4,855 worms recovered in sheep 169. In the other three sheep the number of worms varied from 665 to 1,662.

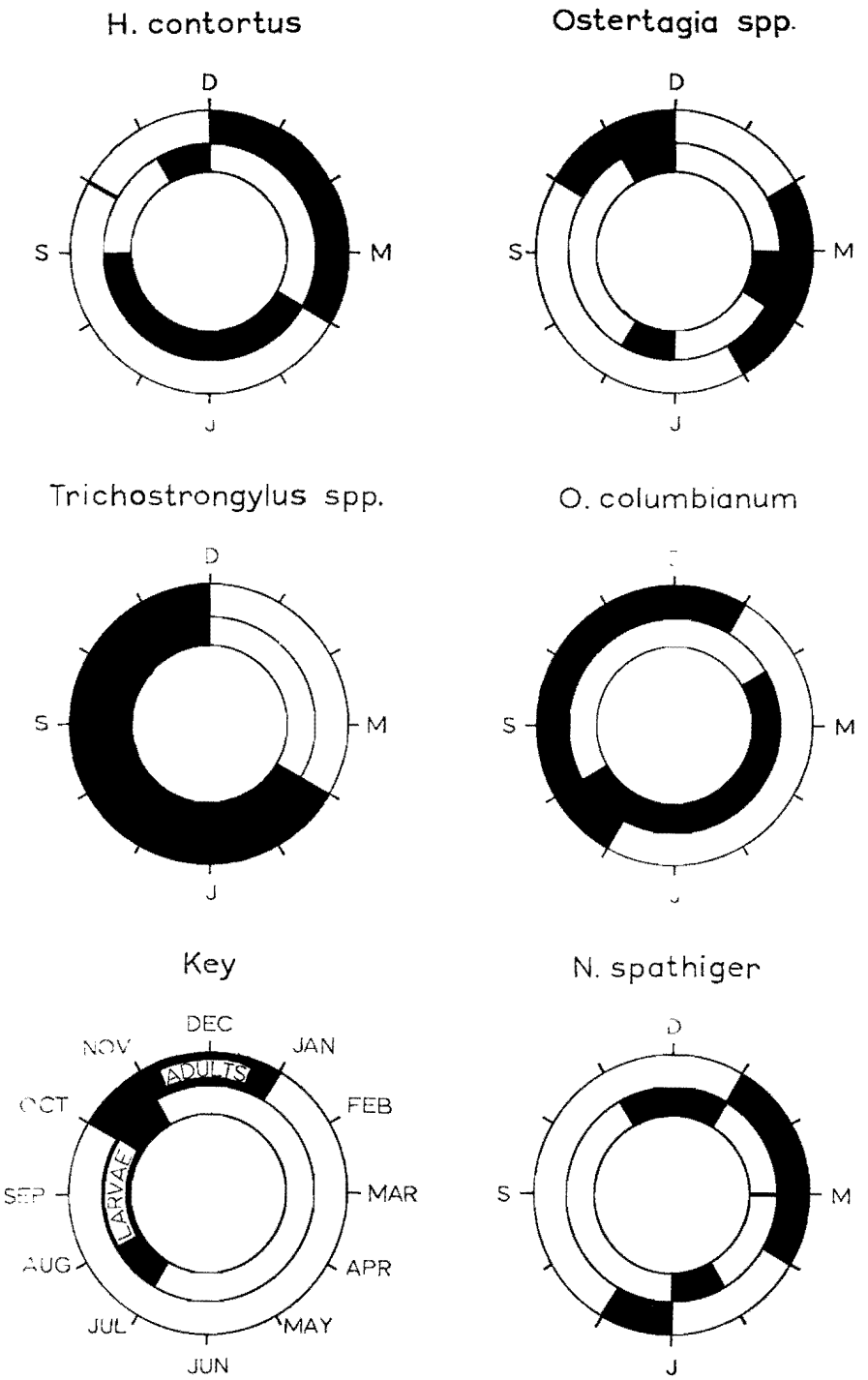

Fig. 1.—Correlation of autopsy results of Rossiter (1964)[18], Barrow (1964)[1] and Viljoen (1964)[29] showing the months when adults and larvae were most numerous. All species were present throughout the year and blank spaces do not indicate the complete absence of any species or genus.

In early summer (November and December) fourth stage *Haemoncus contortus* are plentiful and followed by increased adult worm burdens. This suggests that these larvae develop through to the adult stage. In April, however, adults are of minor significance and remain low until the following October, but fourth stage larvae are recovered in large numbers from April to September (Viljoen, 1964[29]; Rossiter, 1964).[19]

Ostertagia spp. larvae rarely precede (Barrow, 1964),[1] may fall between (Rossiter, 1964),[19] or be correlated with (Viljoen, 1964)[29] peak adult worm burdens.

It would seem that *Trichostrongylus* spp. larvae are acquired throughout winter, and develop rapidly to adults. Worm burdens increase rapidly from April onwards to peaks either in May, June or July; individual sheep may have heavy worm burdens as late as December.

Oesophagostomum columbianum has a prolonged histiotrophic phase lasting from February to August; adults only increase markedly from July onwards.

Nematodirus spathiger larval stages precede adult worm burdens by only one or two months.

Discussion

Tetley (1949),[25] Gordon (1948;[4] 1953;[5] 1958)[6] and Crofton (1955;[2] 1957)[3] found that the incidence of parasites differed with the seasons. This was confirmed by Rossiter (1964),[19] Barrow (1964)[1] and Viljoen (1964).[29]

Haemoncus contortus

Gordon (1948)[4] showed that optimal conditions for the free living stages were monthly rainfall in excess of 50 mm and mean temperatures above 17.7°C. In the areas covered by these surveys such conditions only prevail from November to April.

The spring rise of adults in October builds up pasture contamination; the free-living stages cannot develop to infective larvae before the rains in November or December. This is proved by the increased recovery of fourth stage larvae in November; these larvae develop rapidly to adults, peak burdens occurring as early as December and persisting until April.

From April to September fourth stage larvae predominate. Third stage larvae may be recovered as late as June (Viljoen, 1964)[29] indicating that there are still infective larvae on the pastures. These infective larvae probably develop before May when climatic conditions become unsuitable.

The retardation of development in the fourth larval stage is probably due to the host's resistance as is the case with *Haemoncus placei* in calves (Roberts, 1957).[16]

According to Stoll (1929)[24] immunity is dependant on a continual intake of infective larvae; these are at a low level in September and October and therefore the basic immunity of the sheep is lower. This allows some fourth stage larvae to develop to adults, thus accounting for the spring rise in October.

Ostertagia spp.

According to Rogers (1940)[18] and Gordon (1953[5]; 1958[6]) these are cool weather parasites and this accounts for the rise in worm burdens in early summer and autumn.

The retardation in the fourth stage coinciding with, and extending beyond, peak adult burdens is to be expected as these parasites have a prolonged histiotrophic phase (Sommerville, 1954).[21]

Trichostrongylus spp.

Roberts, O'Sullivan and Riek (1952)[17] stated that the optimal requirements for the free-living stages were mean maximum temperatures from 12.8 to 18.3°C and monthly rainfall in excess of 76.2 mm. Gordon (1953)[5] agreed with the temperature requirements but stated that monthly rainfall of 50.8 mm was adequate.

Rossiter (1964),[19] Barrow (1964)[1] and Viljoen (1964)[29] found that the temperature requirements were satisfied from May to October; the monthly rainfall, however, rarely exceeded 50 mm during this period.

One of the effects of immunity is retarded development in the larval stages (Urquhart, Jarrett and Mulligan, 1962).[28] At autopsy adults

invariably exceeded larvae, thus indicating susceptible sheep (Rossiter, 1964;[19] Barrow, 1964;[1] Viljoen, 1964).[29] This also confirms Gordon's (1953)[5] observation that sheep are highly susceptible to *Trichostrongylus* spp. in the autumn.

Oesophagostomum columbianum

Fourth stage larvae increase markedly from February onwards. Some spend long periods in the wall of the intestine, and are released into the lumen intermittently; this process increases rapidly from July onwards (Viljoen, 1964;[29] Rossiter, 1964).[19] Larvae either disappear or only a few are recovered from August to February. This does not entirely confirm Veglia's (1928)[30] observations that these larvae remain in the intestinal wall for as long as two years.

Nematodirus spathiger

This is known to be a parasite of young lambs which rapidly acquire resistance (Tetley, 1949;[25] Kates and Turner, 1953).[8] In sheep between the age of six and nine months egg counts decreased from December to March and the number positive from 52 to 5; thereafter only three were positive on isolated occasions (Viljoen, 1964).[29] Rossiter (1964)[19] and Barrow (1964)[1] made similar observations on older flocks. According to Viljoen (1964),[29] Rossiter (1964)[19] and Barrow (1964)[1] there were 11 per cent, 34 per cent and 55 per cent negative necropsies respectively, confirming that resistance increases with age.

Cross Immunity

On correlating the data there appeared to be a cross immunity between *H. contortus* and *Trichostrongylus* spp. and to a lesser extent between *H. contortus* and *Ostertagia* spp. This is clearly illustrated in Figure 1.

These results confirm Gordon's (1953)[5] field observations and the experimental results of Stewart (1953)[23] for *H. contortus* and *Trichostrongylus* spp. The cross immunity between *H. contortus* and *Ostertagia* spp. demonstrated by Kates, Wilson and Turner (1957)[9] and Turner, Kates and Wilson (1962)[27] was not as marked.

Species Common to Game and Sheep

Rossiter (1964)[19] diagnosed *T. pietersei*, which has also been recovered from *Lepus saxatilis*, the Southern bush or scrub hare (Ortlepp, 1964).[13] Viljoen (1964)[29] found *T. falculatus* the dominant *Trichostrongylus* spp. in the Karroo. This species has been recovered from *Antidorcas marsupialis*, the springbuck, *Damaliscus albifrons*, the blesbuck, and *Raphicer's campestris*, the steenbuck (Ortlepp, 1961).[12] *Trichostrongylus rugatus*, common to all three centres, has been recovered from *Connochaetes gnou*, the black wildebeest or gnu, and *Taurotragus oryx*, the eland (Ortlepp, 1961).[12] The ubiquitous *Trichostrongylus colubriformis* occurs in the springbuck, blesbuck as well as *Hippotragus niger*, the sable antelope, and *Kobus ellipsiprymnus* the waterbuck (Ortlepp, 1961).[12] *Trichostrongylus axei* has only been recovered from the blesbuck (Ortlepp, 1961).[12]

In the past these animals were common in the areas covered by the survey. At present only the scrub hare and steenbuck are widespread; while springbuck, blesbuck and other antelopes are confined to farms where they are protected. Game undoubtedly act as reservoir hosts of these species.

Geographical Distribution

Haemoncus contortus

This is rife in the Highveld (Thomas, 1959),[26] common in other summer rainfall areas (Barrow, 1964;[1] Rossiter, 1964;[19] Viljoen, 1964)[29] and rare in winter rainfall areas (Muller, 1962).[11]

Ostertagia spp.

These parasites are of major importance in the winter rainfall areas (Muller, 1962),[11] parts of the grassveld areas of the Border (Barrow, 1964),[1] and to a lesser extent, the coastal areas (Rossiter, 1964)[19] and the Karroo (Viljoen, 1964).[29] They are rarely encountered in the Highveld (Reinecke, 1964).[15]

Trichostrongylus spp.

This genus is widespread throughout South Africa (Rossiter, 1964;[19] Thomas, 1959;[26] Viljoen

1964),[29] being the major parasite in the winter rainfall areas (Muller, 1962),[11] as well as the grassveld areas (Barrow, 1964).[1]

Oesophagostomum columbianum

This parasite has a wide distribution (Barrow, 1964;[1] Rossiter, 1964;[19] Thomas, 1959;[26] Viljoen, 1964).[29] In the winter rainfall area, it is usually accompanied by *Oesophagostomum venulosum* (Muller, 1962).[11]

Nematodirus spp. Although *N. spathiger* occurs throughout the Cape Province, *Nematodirus filicollis* has only been found in winter rainfall areas (Muller, 1962)[11] and to a lesser extent in the Border area (Meldal-Johnson, 1961).[10] *Nematodirus* spp. are rarely encountered elsewhere (Reinecke, 1964).[15]

Prophylaxis

The general principles of epizootiology were set down by Gordon (1958),[6] which are summarized: "The basic ecological concept is that almost every animal in the flock is infested and that the environment is contaminated continuously".

(1) *Anthelmintics.*

The recently developed broad spectrum anthelmintics, which are also effective against immature stages, have made it possible to rid sheep of almost all their worms. Examples are the drugs thiabendazole and methyridine. Thiabendazole is ovicidal within 8 hours of drenching (Southcott, 1963).[22] Methyridine is erratic against abomasal parasites (Walley, 1961 a and b).[31, 32].

(2) "*Offensive*" *drenches*

There are certain periods of the year when worm burdens of the different genera are at a low level (Figure 1). It is logical to support the natural elimination of worms, with highly effective anthelmintics as follows:

(1) September — *H. contortus*
(2) December — *Trichostrongylus* spp.
(3) January — *O. columbianum*
(4) May — *Ostertagia* spp.

These treatments are an all-out "offensive" against worm burdens already at their ebb in the host. This "offensive" approach is in contrast to "defensive" measures such as the strategic and tactical drenches advocated by Gordon (1948).

(3) *Pasture control*

For maximum effect it is recommended that after treatment, sheep be kept overnight in a holding pen or paddock before being placed on a pasture that has been free of sheep for three months or longer. In this way worms are expelled, eggs sterilized and contaminated faeces left in the holding paddock. The "clean" paddock, which has been stock-free, has few available infective larvae and reinfestation is delayed.

The contamination of the environment may be controlled by low-level dosing with either thiabendazole (Reinecke, 1962;[14] Snijders, Stapelberg and Muller, 1964)[20] or phenothiazine (Thomas, 1959;[26] Snijders *et al*, 1964).[20] Low-level dosing has its real place in fat lamb production; on irrigated pastures or on natural pastures during exceptionally heavy rains.

(4) *Lambs and Yearlings*

As sheep under 18 months of age are more susceptible to infestation, lambs must be protected by treating ewes two to four weeks prior to lambing; lambs treated at weaning and from time to time when they show symptoms of parasitism.

It has been shown that treatment with phenothiazine predisposes to enterotoxaemia (Jansen, 1960). Other anthelmintics may have a similar effect and therefore all sheep should be vaccinated.

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REFERENCES

1. BARROW, D. B. (1964). The Epizootiology of nematode parasites of sheep in the Border area. *Onderstepoort J. Vet. Res.* (In press).
2. CROFTON, H. D. (1955). Nematode parasite populations in sheep on Lowland farms. III. Worm egg counts in lambs. *Parasitology*, **45**, 99–115.
3. CROFTON, H. D. (1957). Nematode parasite populations in sheep on Lowland farms. II The seasonal incidence of species. *Parasitology*, **47**, 304–318.
4. GORDON, H. McL. (1948). The epidemiology of parasitic diseases, with special reference to studies with nematode parasites of sheep. *Aust. Vet. J.*, **24**, 17–44.
5. GORDON, H. McL. (1953). The epidemiology of helminthosis in sheep in winter-rainfall regions of Australia 1. Preliminary observations. *Aust. Vet. J.*, **29**, 337–348.
6. GORDON, H. McL. (1958). The epidemiology of helminthosis in sheep in winter-rainfall regions of Australia 2. Western Australia. *Aust. Vet. J.*, **34**, 5–19.
7. JANSEN, B. C. (1960). The occurence of pulpy kidney in sheep dosed with phenothiazine. *J.S. Afr. Vet. Med. Ass.*, **31**, 209–210.
8. KATES, K. C. and TURNER, J. H. (1953). Experimental studies on the pathogenicity of *Nematodirus spathiger*, a Trichostrongylid parasite of sheep. *Am. J. Vet. Res.*, **14**, 72–81.
9. KATES, K. C., WILSON, G. I. and TURNER, J. H. (1957). Pathogenic effect of experimentally induced infections of three species of stomach worms which parasitize sheep. *Proc. Ass. South. Agric. Workers*, 54*th Ann. Conven.*, pp. 90–91.
10. MELDAL-JOHNSEN, C. M. T. (1961). Seasonal incidence of nematode parasites of sheep in the King William's Town area. *J.S. Afr. vet. med. Ass.*, **32**, 65–70.
11. MULLER, G. L. (1962). The seasonal incidence survey of internal parasites of sheep — part I. *J.S. Afr. Vet. Med. Ass.*, **33**, 45–47.
12. ORTLEPP, R. J. (1961). 'n Oorsig van Suid-Afrikaanse helminte veral met verwysing na die wat in ons wildherkouers voorkom. *Tydsk. Natuurwetenskappe*, **1**, 203–212.
13. ORTLEPP, R. J. (1964). Personal communication.
14. REINECKE, R. K. (1962). Three new anthelmintics. *J.S. Afr. Vet. Med. Ass.*, **33**, 245–247.
15. REINECKE, R. K. (1964). Unpublished data.
16. ROBERTS, F. H. S. (1957). Reactions of calves to infestation with the stomach worm, *Haemonchus placei* (Place, 1893) Ransom, 1911. *Aust. J. Agr. Res.*, **8**, 740–767.
17. ROBERTS, F. H. S., O'SULLIVAN, P. J. and RIEK, R. F. (1952). The epidemiology of parasitic gastro-enteritis of cattle. *Aust. J. Agr. Res.*, **3**, 187–226.
18. ROGERS, W. P. (1940). The effects of environmental conditions on the accessibility of third stage trichostrongyle larvae to grazing animals. *Parasitology*, **32**, 208–225.
19. ROSSITER, L. W. (1964). The Epizootiology of nematode parasites of sheep in the Coastal area of the Eastern Province. *Onderstepoort J. vet. Res.* (In press).
20. SNIJDERS, A. J., STAPELBERG, J. H. and MULLER, G. L. (1964). Comparative trials of phenothiazine and thiabendazole in worm control of sheep. *J.S. Afr. Vet. Ass.*, **35**, 83–88.
21. SOMMERVILLE, R. I. (1954). The histiotrophic phase of the nematode parasite *Ostertagia circumcincta*. *Aust. J. Agr. Res.*, **5**, 130–140.
22. SOUTHCOTT, W. H. (1963). Ovicidal effect of thiabendazole and its activity against immature helminths of sheep. *Aust. Vet. J.*, **39**, 452–458.
23. STEWART, D. F. (1953). Studies on resistance of sheep to infestations with *Haemonchus contortus* and *Trichostrongylus* spp. and on the immunological reactions of sheep exposed to infestation. V The nature of the "self-cure" phenomen. *Aust. J. Agr. Res.*, **4**, 100–117.
24. STOLL, N. R. (1929). Studies with the strongylid nematode, *Haemonchus contortus*. I. Acquired resistance of hosts under natural reinfection conditions out-of-doors. *Amer. J. Hyg.*, **10**, 384–418.
25. TETLEY, J. H. (1949). Rhythms in nematode parasitism of sheep. *New Zeal. Dept. Sci. Ind. Res. Bull. No.* **96**, pp. 214.
26. THOMAS, R. J. (1959). The use of low level phenothazine in a salt-bone meal lick for the control of internal parasites in sheep. *J.S. Afr. Vet. Med. Ass.*, **30**, 319–333.
27. TURNER, J. H., KATES, K. C. & WILSON, G. I. (1962). The interaction of concurrent infections of the abomasal nematodes, *Haemonchus contortus*, *Ostertagia circumcinta* and *Trichostrongylus axei*. *Proc. Helm. Soc. Wash.*, **29**, 210–216.
28. URQUHART, G. M., JARRETT, W. F. H. and MULLIGAN, W. (1962). Helminth immunity. *Advances Vet. Sci.*, **7**, 87–129.
29. VILJOEN J. H. (1964). The epizootiology of nematode parasites of sheep in the Karroo. *Onderstepoort J. vet. Res.* (In press).
30. VEGLIA, F. (1928). Oesophagostomiasis in sheep (Preliminary note). 13*th and* 14*th Rep. Dir. Vet. Ed. and Res. Un. S. Afr.*, 755–797.
31. WALLEY, J. K. (1961a). Promintic — a new anthelmintic for sheep and cattle. *Vet. Record*, **73**, 20.
32. WALLEY, J. K. (1961b). Methyridine — a new anthelmintic for sheep and cattle. *Vet. Record*, **73**, 159–168.

SPIROCERCA LUPI FROM THE AORTA OF A BULL

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Canines are the normal definitive hosts of *Spirocerca* spp., but this genus has also been recovered from man[1], goats and donkeys[3].

An 18 month old Jersey bull from Rosslyn, was submitted for autopsy on 24 May, 1963, and lumpy skin disease with fatal complications was diagnosed. In addition there was a nodule, 22 mm long, 16 mm wide and 5.4 mm deep in the wall of the first portion of the abdominal aorta. The middle portion of a worm was enclosed in the nodule while the anterior and posterior portions were free in the lumen.

The total length of the worm, a non-gravid female Spirurid, is 38.4 mm; the maximum width 0.65 mm. The buccal cavity is 0.74 mm in depth and width (Fig. 1). The oesophagus, 4.6 mm long, consists of the usual two parts. The vulva is situated just anterior to the end of the oesophagus, i.e. 4.17 mm from the anterior extremity (Fig. 2). It belongs to the genus *Spirocerca*, Railliet (Fig. 2) and Henry, 1911, and resembles *Spirocerca lupi* (Rud., 1819) of canines[4]. As this specimen was not gravid it is to be expected that the total length is less and the buccal cavity smaller than the measurements given by Neveu-Lemaire[2].

Histopathological examination of various sections of the aortic lesion, revealed several haemorrhagic tracts, involving the entire wall of the vessel (Fig. 3). These tracts were filled with blood, necrotic infiltrative cells and debris, and were surrounded by a zone of round cell leucocytes on the adventitial side. A significant feature of the host response was the extensive proliferation of the intima, which bulged into the lumen of the vessel (Fig. 4). A large tract was present in this part of the intima. Foci of early calcification were evident in the media. No parasitic tissue was found in the sections,

Fig. 1.—Buccal cavity of *Spirocerca lupi* from the aorta of a bull.

as the single parasite had been removed for identification.

This lesion differs from those found in dogs[4] and in goats and donkeys[3] in the marked proliferation of the intimal coat.

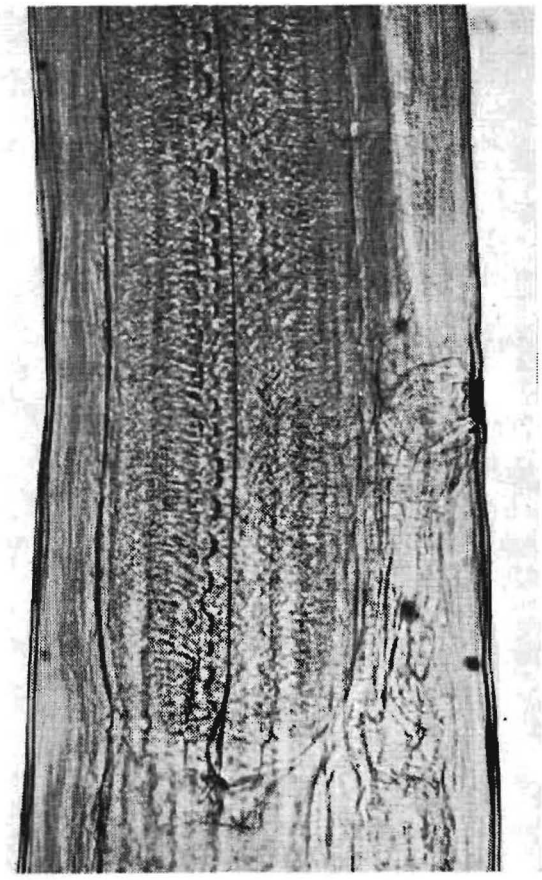

Fig. 2.—Position of the vulva in *Spirocerca lupi* from the aorta of a bull.

Fig. 3.—Photomicrograph of the aortic lesion.

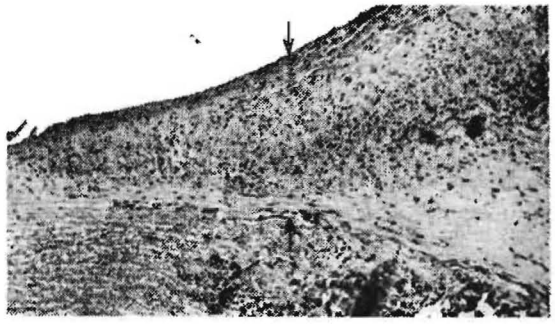

Fig. 4.—Photomicrograph showing proliferation of intima (arrows); calcification lower right. (X 75).

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REFERENCES

1. BIOCCA, E. (1959). Infestazione umana prenatale da *Spirocerca lupi*, (Rud., 1809). *Parassitologia. Rome*, **1** (2). 137–142. In *Helminth. Abstr.* (1960), **29**. 386.
2. NEVEU-LEMAIRE, M. (1936). *Traité d'Helminthologie Médicale et Vétérinaire*. Paris: Vigot Frères., pp. 1217–1224.
3. PANDA, B. P., RIA, PAND BHATIA, B. B. (1961). Nematode affecting the aorta in Indian caprine and equine hosts. *J. Parasit.*, **47**, 951–952.
4. SMITH, H. A. and JONES, T. C. (1961). *Veterinary Pathology*. London: Henry Kimpton, p. 522–525.
5. YAMAGUTI, S. (1961). Systema Helminthum, Vol. III. *The Nematodes of Vertebrates*. New York: Interscience Publishers, pp. 600–601.

REFERENCES

1. BIOCCA, E. (1959). Infestazione umana prenatale da *Spirocerca lupi*, (Rud., 1809). *Parassitologia. Rome*, **1** (2). 137–142. In *Helminth. Abstr.* (1960), **29**. 386.
2. NEVEU-LEMAIRE, M. (1936). *Traité d'Helminthologie Médicale et Vétérinaire*. Paris: Vigot Frères., pp. 1217–1224.
3. PANDA, B. P., RIA, PAND BHATIA, B. B. (1961). Nematode affecting the aorta in Indian caprine and equine hosts. *J. Parasit.*, **47**, 951–952.
4. SMITH, H. A. and JONES, T. C. (1961). *Veterinary Pathology*. London: Henry Kimpton, p. 522–525.
5. YAMAGUTI, S. (1961). Systema Helminthum, Vol. III. *The Nematodes of Vertebrates*. New York: Interscience Publishers, pp. 600–601.

TRANSVAAL ARTIFICIAL INSEMINATION COOPERATIVE LTD., IRENE, TRANSVAAL

GENERAL VETERINARY REPORT, 1963

We have been privileged to see a copy of the General Veterinary Report of the Transvaal Artificial Insemination Coop Ltd., for 1963.

This report makes very interesting reading and Dr. la Grange and his workers are to be congratulated on the work they are doing at Irene and the services they are giving to South Africa.

In parts the Report of the Transvaal A.I. Coop Ltd. shows a pessimistic trend, but the general impression is that progress is being satisfactorily maintained. An increase of 11,716 doses of semen is shown over the previous year — i.e. 10.8 per cent.

Substantial quantities of deep frozen semen were exported to Rhodesia with excellent results.

A total quantity of 142,756 doses of semen were disposed of during the year.

The A.I. Coop has at present 20 Sub-centres and 17 clubs and a gradual increase of private buyers of semen is taking place.

EDITOR.

FOOT AND MOUTH DISEASE OUTBREAK: KALKFELD AREA, SOUTH WEST AFRICA, IN MAY, 1964

SUPPLEMENTARY REPORT

By kind permission of the Director of Agriculture ,Windhoek

1. This report is intended to supplement the preliminary report on this outbreak issued on 3rd June, 1964.

2. There was no extension of infection from the four infected farms, to wit Wilhelm-Albrechtstal 84, Ondombo West 34, Okosongoro 46 and Elshorst 90.

3. All cloven-hoofed domestic stock on all farms within a radius of fifty miles of the infection were inoculated. Mostly modified strain R.V. 11 live vaccine prepared by Messrs Burroughs Wellcome was used in bovines and caprines and an inactivated vaccine prepared from Virus S.W.A. 40/1961 was used in ovines.

4. The inoculation programme with the live vaccine was completed on 29th June, 1964, and with the inactivated vaccine on 9th July, 1964. This time difference was due to the late receip of stocks of inactivated vaccine.

5. The campaign involved the inoculation/reinoculation of 222,511 cattle, 177,122 goats and 110,795 sheep on 539 farms.

6. Symptoms of Foot and Mouth Disease were last observed on Ondombo W and Wilhelm-Albrechtstal on 26th June, 1964. Repeated weekly interval mouthing of stock on infected farms, farms within a radius of twenty-five miles and seven-day interval inspection to a radius of 100 miles has failed to reveal any further sign of infection. The clinical endpoint of the outbreak is therefore regarded as 30th June, 1964.

7. It has not been possible to establish the source of infection. According to the Director

of the Virus Research Institute, Pirbright, the virus responsible closely resembles specimen S.W.A. 40/1961 collected on 7th October, 1961, on the farm 938, Gobabis District and which differs from the vaccine strain R.V. 11 used in combating that outbreak.

8. The lesson of this outbreak would appear to be that F.M.D. can be stopped in heavy game infested country by the early use of vaccines.

9. The object of this note is to report that South West Africa has been free of Foot and Mouth Disease since 30th June, 1964.

J. H. B. VILJOEN
for Director of Agriculture
Windhoek, S.W.A., 19th September, 1964

DIE HOOF NAVORSINGSINSTITUUT VIR VEEARTSENYKUNDE ONDERSTEPOORT KONDIG AAN

BRUCELLOSE IN SEKOEKOENILAND

Brucellose besmetting onder naturelle in Sekoekoeniland is reeds aangetoon deur Schrire (1962). *Brucella* kulture verkry vanaf 'n hospitaal in die gebied, is hier tipeer en het hoofsaaklik *Br. melitensis* stamme opgelewer.

Die vraag ontstaan hoe die besmetting oorgedra word na hierdie mense, aangesien *Br. melitensis* besmette skaap- en bokkuddes, meesal tot Suidwes-Afrika beperk is. Slegs by twee geleenthede is *Br. melitensis* in diere van die Republiek van Suid-Afrika aangetref. (Van Drimmelen, 1963, Ermelo.) Geen omvattende toetse is nog onder vee in Sekoekoeniland uitgevoer nie. Die omvang van die siekte aldaar is nie bekend nie.

Dit is egter onrusbarend dat soveel as agt (8) uit twaalf (12) verdagte gevalle van maltakoors *Brucella* kulture gelewer het, en ses (6) van die positiewe gevalle was te wyte aan *Br. melitensis* besmetting. (Ongepubliseerde data).

Behalwe vir die gevaar wat besmette kuddes vir alle mense inhou, bestaan daar 'n moontlikheid dat mense soos veeopsigters wat entstowwe inspuit dalk die besmetting van dier tot dier kan oordra.

'n Toetsprogram vir diere, en moontlik ook mense, sal 'n duideliker beeld van die omvang van die siekte lewer. Daadwerklike optrede om die siekte te bestry, en sover moontlik uit te roei, word in die vooruitsig gestel.

G. C. VAN DRIMMELEN
Onderstepoort

WORLD ASSOCIATION OF VETERINARY FOOD HYGIENISTS

The 4th Symposium of the *World Association of Veterinary Food-Hygienists* will be held in *Lincoln (Nebraska, U.S.A.) from 25–30 July*, 1965. The scientific programme is as follows:

1. Tasks and training of veterinary food-hygienists.
2. Salmonella problems.
3. Problems in poultry inspection.
4. Measures to guarantee wholesomeness of food of animal origin:
   (*a*) Fresh meat and processed meat.
   (*b*) Milk and milk products.
   (*c*) Eggs and egg products.
   (*d*) Fish and other sea-foods.
5. Prevention of food poisoning and contamination of food.
6. Biological residues in food of animal origin.
7. Free communications.

This is the first meeting of its kind ever to be held in the U.S.A.

Speakers have been invited by the Board for the main lectures. Those desiring to present short communications concerning the different themes and the free communications should contact Dr. L. W. van den Heever, Onderstepoort, before 15th November, 1964.

OBITUARIES

A MEMOIR

Dr. ISAAC PIETER MARAIS

Dr. Isaac Pieter Marais was born in Pretoria on February 12th, 1900.

He attended school at Malmesbury, Pretoria, and later at Stellenbosch, where he obtained a First Class Matriculation Certificate in 1917. He completed the first year B.Sc. Agric. at Stellenbosch University and the last three years at the Transvaal University College, Pretoria, where he qualified at the end of 1921.

He then completed the course of B.V.Sc. at the University of Pretoria and graduated in 1925, as one of the second batch of South African students to train as Veterinarians. He joined the Staff of the Division of Veterinary Services in 1926 and served the Onderstepoort Veterinary Research Institute until 1938 when he resigned to take up the post of Abattoir Superintendant with the City Council of Pretoria.

He married Miss T. N. Ehrlich in 1927 and out of this marriage two sons and one daughter were born.

He relieved at Allerton Laboratory in 1928 and was transferred there as officer in charge from 1930 to 1931. On his return to Onderstepoort he moved to the Department of Surgery and Gynaecology as Senior lecturer under Professor J. B. Quinlan. While in this Department he conducted research into the sex physiology of sheep, using the data which he collected to present a thesis for the degree of Dr. Med. Vet. at the University of Leipsig, where he undertook post-graduate studies during 1935–1936.

In June, 1940, he was granted leave of absence by the Municipality of Pretoria to enlist with the South African Veterinary Corps, in which unit he served with the rank of Major for the duration of the Second World War.

In 1953 he entered private practice at Alberton, where he built up a large equine practice.

In his student days he played rugby, tennis and hockey.

He was a keen follower of the turf, owning and racing several race-horses.

He was an official Judge of thoroughbred horses.

In 1961 he suffered an attack of cardiac thrombosis and was eventually forced to retire from Veterinary practice and lived with his family in Pretoria where he worked in a temporary capacity as translator in the Bureau of Standards.

He died in hospital on 22nd October, 1964, after a short illness.

He married a second time in 1950 and leaves a widow. To her and to the other members of his family we extend our deepest sympathy.

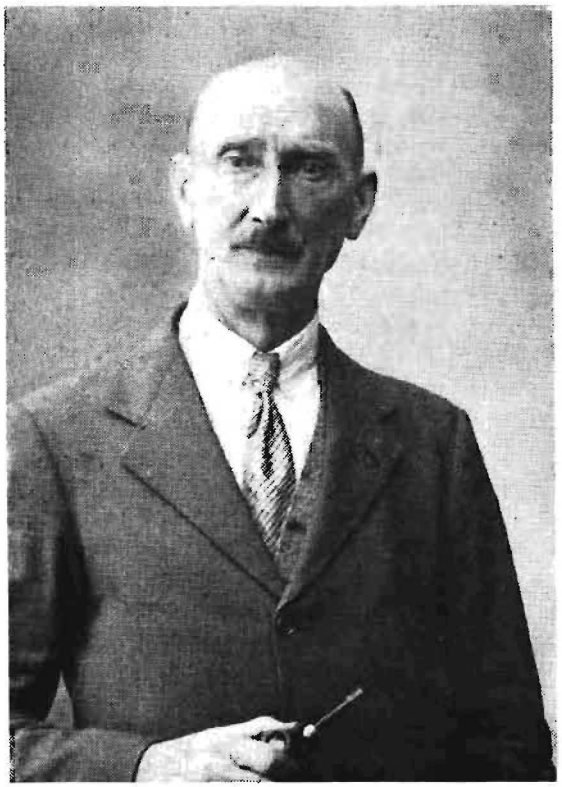
A MEMOIR

ROGER SUTTON GARRAWAY

It is with great regret that the sudden and unexpected death of Roger Sutton Garraway is recorded.

Dr. Garraway died at his home at 332 Prinsloo Street, Pretoria on 29th September, 1964.

He was born at Waterford in Southern Ireland in 1876, and graduated from the Royal (Dick) Veterinary College, Edinburg in 1899.



He came to South Africa as a volunteer with the British Forces during the South African War, and received an appointment as Government Veterinary Officer at the conclusion of hostilities.

He was stationed at Pretoria and during the severe and extensive outbreak of East Coast Fever in the Pretoria district in 1919, transferred to Premier Mine and placed in charge of the campaign against this disease. This outbreak was so extensive that at one stage there was a farm-to-farm infection for over 100 miles.

Through his organizing ability and strict application of the control measures against the disease, it was eradicated by 1922 and never recurred again in those parts.

He married Miss Olive Sharp of Bloemfontein in 1914 whose companionship he enjoyed up to the time of his death.

Dr. Garraway was promoted to the rank of Senior Veterinary Officer in 1926 and stationed at Windhoek, South West Africa. He was later transferred to Bloemfontein and served as Senior Veterinary Officer for the Orange Free State until his retirement in 1936.

He carried on a private practice in Pretoria for a number of years after his retirement and was well known for his unselfish attention to sick animals.

He was a brother to Sir Edward Garraway, a former Resident Commissioner of Bechuanaland and Basutoland.

Roger Garraway was a very keen sportsman. He was very fond of game shooting particularly bird shooting and was an excellent shot.

He was a great lover of horses — particularly of Thoroughbreds, and owned some of his own in the early days when Pretoria still boasted of a race-course.

Our sincere sympathy goes to his wife in her very sad bereavement.

A.M.D.

A MEMOIR

JOHN MACKINNON, M.R.C.V.S.

The sudden death of Mr. John MacKinnon, ex-Director of Veterinary Services of the Federation of Rhodesia and Nyasaland, on 9th August, 1964, came as a rude shock to his many friends.

With the break-up of the Federation, Mr. MacKinnon had taken abolition of office in December, 1963, but rejoined the Southern Rhodesian Veterinary Department in June, 1964, as a Veterinary Officer in the Salisbury District. Having shed the endless cares of high office, he was thoroughly enjoying his return to every-day field duties and his tragic demise at the early age of 53 was totally unexpected.

After a few years in practice in the North of England, he joined the Southern Rhodesian service in 1934, of which he became Assistant Director in 1950, and Director of the Federal Veterinary Services in 1959.

In the last decade, the service was built up to an extremely high level of efficiency, largely due to his untiring efforts, and although he characterised himself as a "policeman" rather than as a "scientist", his penetrating and shrewd analysis of the scope and character of disease control measures was always soundly based and of a highly effectual and practical nature.

The profitable position enjoyed by Southern Rhodesia in the beef export market to-day is largely due to his determined representations of the efficiency of its veterinary services and controls at International Conferences.

Many will remember his dry and pawky sense of humour at the Inter-Territorial Foot and Mouth Disease Conferences which often was employed to sooth the ruffled tempers, but his active participation in debate had unfortunately been limited of late by his growing deafness.

His passing removes another of the "Old Brigade" who so assidously laboured in the early days to build up the efficiency of the cattle industry on a sound basis of prophylaxis and disease control but the memory of his loyalty and devotion to duty will ensure his remembrance in the minds of the many friends and colleagues who mourn his death.

L.M.

A MEMOIR

JURGENS JOHANNES ADAMS

The death occurred on 20th July 1964, of Jurgens Johannes (Jimmy) Adams, retired Under Secretary for Agriculture and Chairman of the Tobacco Industry Control Board.

Jimmy Adams was born at Porterville, Cape on 23rd November 1897.

He took the degree of B. Comm. at the University of Pretoria. He received an appointment in the Department of Agriculture in 1917 and in 1926 was promoted to the post of Inspector of Co-operative Societies.

From 1931 he was closely associated with the Co-operative movement in South Africa and from 1945 to 1958 was the Registrar of Co-operative Societies. He became the Chief Accountant of the Department of Agriculture in 1939, and was largely responsible for the preparation of the enlarged Co-operative Societies Act No. 29 of 1939.

He was promoted to the post of under Secretary for Agriculture in 1942 and held this post until his retirement in 1958. During this time he was also the Registrar of Co-operative Societies

and Chairman of the Tobacco Industry Control Board.

After his retirement he became Manager of the Tobacco Board, which post he held until his death.

The Veterinary profession owes a great deal to the sense of appreciation which "Jimmy" had for Veterinary Services in South Africa.

He was chairman of the Adams Committee which during 1945 investigated and reported on certain aspects of Veterinary Serivces and Veterinary Education. The other members of the Committee were, Dr. P. J. du Toit, Mr. G. Lotz, Col. H. Nelson, Dr. W. M. Power, Prof. A. S. Strachan and Prof. R. W. Wilcocks.

This Committee made far-reaching recommendations regarding the Veterinary Services of the Department of Agriculture; and now twenty years later, the report is still being referred to.

Of him it can, truly be said that he was a wise man, whose decisions and advice were always sound and fair.

He leaves a widow, two married daughters and three grandchildren to mourn his loss. To them this Association extends its heartfelt sympathy.

A.M.D.

UNIVERSITEIT VAN PRETORIA

BEURSE EN LENINGS

VOORWAARDES VIR SKENKING VAN FEDERASIE VAN LEWENDEHAWE-AFSLAERSBEURS

1. *Naam van beurs*: Federasie van Lewendehawe-Afslaersbeurs.

2. *Skenker*: Die Federasie van Lewendehawe-Afslaers van S.A.,
   Posbus 683, Johannesburg.

3. *Waarde*: R400 per jaar of R1,200 elke derde jaar.

4. *Wie vir beurs kwalifiseer*: Veeartse wat die B.V.Sc.-graad te Onderstepoort verwerf het vir navorsing of verdere studie in 'n spesiale rigting. Die beurs word verkieslik toegeken aan 'n veearts wat 'n spesiale studie of nagraadse studie of navorsing wil doen in verband met *slagveeproduksie* — veral pogings wat daarop gemik is om die slagveeproduksie in Suid-Afrika, met klem op beesrasse, te verbeter.

5. *Duur van beurs*: Een jaar.

6. *Basis van toekenning*: Meriete.

7. *Wie beurs toeken*: Die Beurs- en Leningskomitee op aanbeveling van die Dekaan van die Fakulteit Veeartsenykunde.

8. *Besondere voorwaardes*: Die beurshouer moet by voltooiing van sy studie/navorsing 'n verslag of publikasie in verband met sy bevindinge aan die skenker voorlê sodat die resultate tot die verryking van algemene kennis, veral tot voordeel van die boeregemeenskap (blank en nie-blank) aangewend kan word.

9. *Applikasies*: Aan die Dekaan, Fakulteit van Veeartsenykunde Onderstepoort so speodig moontlik.

REPORT ON THE III. INTERNATIONAL MEETING ON DISEASES OF CATTLE (20—23.8.64) AND THE SYMPOSIUM ON LEUKOSIS OF CATTLE (19.8.64) AT COPENHAGEN

Thanks to the circumspect preparation by the organizing committee under the presidence of Prof. Dr. H. C. Bendixen and the secretariat of Prof. Dr. N. O. Christensen, this meeting with its abundant scientific and social program took a splendid and successful course.

A considerable number of participants especially interested in reaserch on leukaemia were already present at the *Symposium on Leukosis* which was held in the Royal Veterinary and Agricultural College before the general Meeting. Besides several papers concerning occurrence, epizoozology, and eradication of this disease, the Latex-Test, presented in 1963 by Lehnert, Stockholm for the proof of specific antibodies in bovine serum was thoroughly discussed. According to critical trials performed in the meantime by Leynert, Winquist and Hugoson in Stockholm, by Seelemann and Heeschen in Kiel and by Tolle in Göttingen, a certain specifity of this reaction seems to exist; anyhow its general use, alone or in combination with the control of the white blood picture, may not be recommended to the actual stage of knowledge. The results of this Symposium were summarized and reported to all patricipants of the Congress on Diseases of Cattle by Prof. Dr. Hansen, Stockholm. This informatory summary was highly appreciated by all members. The papers given at the Symposium and the report of Prof. Dr. Hansen will be contained in the Proceedings of the general Congress.

III. International Meeting on Diseases of Cattle

When Prof. Dr. H. C. Bendixen inaugurated in the great hall of the Institute for Anatomy of the Medical Faculty of the University in attendance of numerous honorary representatives, he welcomed 474 participants from 22 countries. The Danish Minister of Agriculture and the Rector of the Copenhagen University, emphasized in their introducing speeches the importance of research work and of international meetings combining scientists and practitioners, for the progress of animal production and health. They stressed the role of cattle for the provision of man with the more important food. The program contained three main subjects:

1. Virus Diseases of the Air Passages and the Digestive Organs (with 13 papers),
2. Hypomagnesaemia (with 11 papers), and
3. Lameness (with 13 papers).

Besides, 18 other papers were read on "free subjects" dealing with new results and experiences in different fields of Buiatrics. All lectures were simultaneously translated into English, French and German. The 55 reports given by collegues from the European countries (48), from the United States (6), and from Australia (1).

Owing to the abundance of the reports and their results, it is impossible to mention all the speakers or even to give summaries of their papers. Interested collegues, therefore, are informed, that the Proceedings of the Congress including the discussion remarks will appear as a supplement to the journal "Nordisk Veterinaer-Medicin". The report, containing 640 pages, will also include the papers which could not be held by their authors, because of travelling difficulties. The report will be send free to the participants of the Congress. Non-participants, Institutes and Libraries may acquire the report at the price of about 85 Danish crowns. Order should be adressed to the Redaction of Nordisk Veterinaer-Medicin, Bülowsvej 13, Copenhagen V.

The social events during the Congress, as well as excursions to the Royal Castles in North-Seeland, gave opportunity for personal contacts. The thanks, given by Prof. Dr. H. C. Diernhofer to the organizers of the Congress expressed the hearty feelings of all participants. The next Meeting, the fourth of this kind, will be held in Zürich in 1966 under the direction of Prof. Dr. Andres.

BOOK REVIEW

THE ANATOMY OF THE SHEEP
by NEIL D. S. MAY

University of Queensland Press, St. Lucia, Queensland. 1964. Second Edition. 6¼ in x 9½ in.: 16 mo. pp. xiii 369. Price A£4-4-0.

The sheep, humble but economically important provider of human food and clothing and pre-eminent as subject in reminant research work, has been treated in most niggardly fashion in existing anatomical texts. It is thus fitting that, of the three "Sisters of the South" whose respective economies have benefited so largely from this animal, it is Australia that has at long last taken the lead in providing an anatomical text specifically concerning the sheep.

Considering the cost of the ox and the extra time taken in dissecting such a clumsy carcass, many veterinary schools will doubtlessly avail themselves of this opportunity to utilise the sheep more extensively as dissection subject.

As the author clearly states in his preface to the second edition, this book is "primarily intended as a teaching manual" and a "by no means complete anatomical treatise". In fact, it is purely and simply a students' dissecting guide. It would have been prudent to have the words "A Dissection Guide" added as a subtitle. There is no didactic exposition of basic anatomical principles; this is left to the teacher and to existing texts on veterinary anatomy. Neither is there any comment on practical applications of any of the anatomical features, barring a passing reference to the occurrence of larvae of oestrus ovis (the term here printed in the form in which it occurs in the book) in the major compartment of the frontal sinus.

The author's style is succinct and he has taken pains to condense as much detail as one could reasonably expect into 267 pages, in order to keep the price of the book within the average student's reach. He has added, however, 44 pages on osteology in comparative style — a feature not commonly encountered in dissection guides — as well as 28 pages on arthrology: the only condescensions to the systematic approach. Furthermore, a list of 36 references is given with the clear warning that it is not complete and the references are meant for further study. Sixteen of these have specific bearing on the sheep. The last eighteen pages of text are devoted to a tabulation of species differences, compiled from various sources, of the horse, ox, sheep, pig, dog and cat: most useful to students and all those concerned with meat inspection.

There is an adequate index, which gives page references for both text and illustration, the latter in bold type. The occasional error is there: see for instance the indications for "Pulvinar". The typography is neat; no printing errors have been spotted. Instructions for dissection are given in bold type, as are the names of important structures when these are mentioned in the text for the first time. Paper and binding are reasonably in keeping with the dictates of economy.

There are 77 illustrations, all in black and white line drawing. Although the draughtsmanship is amateurish, the illustrations are infinitely superior to the smudgy photographs of the first edition.

The brain is dealt with in as much detail as one can study by gross dissection and unspecialised techniques: probably that is why the author did not bother to include Wilkie's Dissection of the Sheep's Brain in his list of references. The action of each muscle is mentioned briefly. Eruption of the teeth is described in three quarters of a page; this contains some useful information. The sequence in which the various structures is dealt with can only be criticised by completing a dissection strictly according to instructions. This the reviewer has not done and he bows to the superior experience of the author and his students, despite a suspicion that there may be unnecessary shuttling back and forth: *vide* the following sequence:

vertebral canal and contents, the frontal sinus, the cranial meninges (actually dura mater and sinuses only), nasal cartilages, nasal cavity, vomero-nasal organ, paranasal sinuses, arachnoid membrane and pia mater (no subheading in case of the latter two).

Not so much in deference to reviewing custom but rather from a sense of cooperation a few minor criticisms are made:

1. The nervus transversarius (not mentioned by name) is said to be *formed by* branches *from* the second to seventh cervical vertebrae and to *pass down* the neck to the first thoracic ganglion (p. 63).
2. The inguinal canal "is formed by the abdominal tunic and the aponeurosis of the external oblique muscle" (p 76).
3. The embryology of the ruminant stomach by now is well established enough that it is unnecessary to regard the fore-stomachs as "said to be" developed from the "stomach region".
4. The homology of the muscle described as the M. piriformis is doubtful.
5. The origin of the umbilical artery is not given (or your reviewer could not find it in the book).
6. The terminology, although purporting to be in Latin "throughout the text" and based on the *N.A.P. et B.N.A.*, is purely Sissonian.
7. English, despite its universality, is a fickle tool: "A number of haemal nodes are also present", or, "the rectovesicle pouch".

As the book is the only one of its kind, it can and probably will be used as a source of reference. The individual who has little anatomical background will encounter grave difficulties in finding his way about and in obtaining the required information in proper systematic and relevant order. This is not a criticism but a warning to over-optimistic blithe spirits. And he who has an adequate background might be disappointed in the lack of detail (e.g. sacral plexus). One can only express the fervent hope that this work will be the forerunner of an extensive and systematic treatise on the sheep, however formidable and economically unprofitable the task, in the same way that Miller's Guide to the Dissection of the Dog was the forerunner of Evans and co-worker's tome. Surely a country that reaps millions annually from its sheep industry owes that much to its veterinary profession, and its veterinary profession owes that much to the world of learning.

H. P. A. DE BOOM.

THE YEAR BOOK OF VETERINARY MEDICINE. VOLUME 1. 1963.

Year Book Medical Publishers Incorporated, 35 East Wacker Drive, Chicago 1. Price $10.

In the preface the Editors state: "The object of the Year Book is to give the reader better access to the large volume of world-wide veterinary and allied scientific journal literature. We have made a painstaking effort to assemble a good representation, in the form of detailed abstracts, of the most worthwhile articles that appeared in the recent past. In making selections, special attention was given to new ideas and the latest scientific discoveries that may have application to the practice of veterinary medicine. Editorial comments and illustrations are included whenever they are considered pertinent".

The contents of the book are divided into three sections: Large Animal Medicine, Small Animal Medicine, and Laboratory Animal Medicine. Each section is divided into chapters; that on Large Animal Medicine contains chapters on Infectious Diseases, Non-Infectious Diseases, Diagnosis and Therapy, Surgery and Nutrition, Neoplasms and Economics. The Small Animal Medicine section is set out on similar lines, while that on Laboratory Animal Medicine includes chapters on Genetics and Breeding, Specific-Pathogen-Free Animals, Germ-Free Animals, Infectious Diseases, Non-Infectious Diseases, Zoonoses, Zootechnics and Facilities (housing).

This is the first volume of the Year Book of Veterinary Medicine to appear. It is planned by the publishers to produce one volume annually. The book is well indexed and also contains an author index.

As the literature on Veterinary Science becomes more and more extensive books of this nature will become increasingly popular especially with practising veterinarians and others who have neither the time nor libraries to fall back on. The articles abstracted cover a very wide range, and, for this reason, all cannot, of course, appeal or be of interest to all, but every veterinarian will find articles which are of interest and of importance to him.

The order of presentation of the articles appears, at first glance, to be rather haphazard; for instance, various aspects of heartworms in dogs are discussed in the chapters on Therapeutics, Surgery and Infectious Diseases. According to one Editor the arrangement of articles was determined with a purpose, so that a pattern could emerge for future editions which would enable the Editor to fit an article into an appropriate chapter without a number of subchapters, and too much refinement would become cumbersome. It is however, suggested that in the chapters on Infectious Diseases an improvement would be to group the diseases according to aetiology — viral, bacterial, protozoal, etc. Helminths should not be placed under the heading of infectious diseases.

Another feature of the book which veterinarians in South Africa would find disconcerting in the inclusion of poultry diseases in the section on Large Animal Medicine. A separate section dealing with poultry diseases along would be of in estimable value.

However, the foregoing criticisms in no way detract from its value, and the book can be recommended to all veterinarians wishing to keep abreast of the latest trends and developments in Veterinary Science.

R. C. TUSTIN.

THE VETERINARY ANNUAL 1963/64 FIFTH YEAR

Edited by W. A. Pool. John Wright and Sons Limited. Bristol. 45s.

The Veterinary Annual now in its fifth issue requires no introduction and continues in its service to the profession. The book is again divided into two main sections: Special Articles and Review of the Current Literature; and two brief chapters listing New Drugs and Appliances, and New Books have been added.

The section on Special Articles contains articles on Conditions affecting the Central Nervous System in the Dog and Cat, General Problems of Intensive Poultry Keeping, Pig Practice, 25 Year of Small Animal Practice, The Veterinary Surgeon and Doping, and The Phenothiazine Tranquillizers in Veterinary Practice.

The second section of the book contains brief reviews of the current literature by different contributors, and covers all aspects of veterinary science. It is stated that over 3,000 references are given. It is, of course, impossible to give a comprehensive account of the wealth of information given in this section which forms the main portion of the book, but, for example, the chapter on Diseases related to Viruses is divided into subchapters on: Viruses in General, Arthropod-borne Viruses, Foot-and-Mouth Disease, Pox Diseases, Rabies, Rickettsial Diseases, Virus Diseases of Birds, of Cattle, of Dogs and other Canidae, of Horses, of Cats, of Pigs, and of Sheep and Goats.

In the preface the editor states that it was felt that there was a need for an annual review organ for veterinary science. With the enormous numbers of scientific and professional articles published annually, there appeared to be merit in producing a relatively small volume summarizing the important advances in knowledge. The book serves primarily as a first source of information and it has an extensive cross-referenced index which ensures that the information sought is quickly found.

The whole purpose of this reasonably priced book is to keep the reader up to date with the latest literature, trends and developments in veterinary science and in these respects the editor has, with the help of numerous contributors, succeeded admirably. As such it can be recommended without hesitation to private practitioners, field veterinarians, teachers and some research workers, and it should be included in all veterinary libraries.

R. C. TUSTIN.

PARASITISM

An introduction to Parasitology and Immunology for students of Biology, Veterinary Science, and Medicine

by

J. F. A. SPRENT

Baillière Tindall & Cox, London. 1963.

pp. x + 145

The title of this book is misleading as it is neither an introduction to parasitology nor suitable for undergraduate students. It is an extremely well written and useful book for parasitologists, who wish to extend their field into immunology.

The writer includes not only heteroparasites (metazoa, protozoa, viruses and bacteria) but also homoparasites (tissue grafts, foetuses and tumours) in his discussion. Although many biologists will disagree with the definition of a true parasite, i.e. an associate which derives its nourishment from the host's own tissues, the inclusion of homoparasites becomes understandable, if not acceptable.

The host-relationships of heteroparasites are discussed in the first part of the book. The different types of immunity are defined, particular attention being given to acquired immunity, including the various theories of antibody formation and the union of antigens and antibodies in various serological reactions.

Part two deals with the host-relationships of homoparasites which Prof. Sprent shows elicit essentially the same host reactions as heteroparasites.

Part three deals with the host's recognition of "self" and "not self", while in part four he concludes with a hypothesis of the evolution of immunological reactions.

This book is very stimulating, guiding the reader into a broad concept of parasitism. It is open to criticism in that immunity to parasites in the generally accepted sense of the word, i.e. protozoa and metazoa, are superficially dealt with. This is surprising in view of the great progress made in this field in the last decade. It would be an improvement to include the specific names of parasites mentioned in the text, in the index.

R. K. REINECKE.

BOOKS AND JOURNALS

Publishers of scientific journals like to get subscriptions for the full calender year. Therefore we should like to know before the end of December what journals you want to subscribe to next year. To assist in your selection we give the titles, country and frequency of publication and annual subscription of some of the most popular veterinary publications in English:—

British Veterinary Journal; Britain; monthly; R7.00.

Journal of the American Veterinary Medical Association; United States; fortnightly; R15.75.

Journal of Reproduction and Fertility; Britain; bi-monthly; R14.00.

Journal of Small Animal Practice; Britain; bi-monthly; R10.00.

The Veterinarian; an international journal of large animal practice; quarterly; R14.00.

The Veterinary Record; Britain; weekly; R13.00.

Veterinary Medicine and Small Animal Clinician; United States; monthly; R7.25.

A new edition of Benesch & Wright's Veterinary Obstetrics has now appeared as WRIGHT'S VETERINARY OBSTETRICS by G. H. Arthur. It has been appreciably enlarged and contains several new features on various aspcets of reproduction and infertility. 560 pages; 313 illus.; R6.95.

Factory farming which has made tremendous progress overseas in recent years and all its implications are fully exposed in a provocative new book ANIMAL MACHINES by Ruth Harrison. 186 pages; 32 illus.: R2.45.

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PRETORIA.

BIOLOGICAL ASPECTS OF OCCLUSIVE VASCULAR DISEASE, 1964

Edited by D. G. Chalmers and G. A. Gresham. Cambridge University Press, Bentley House, 200 Euston Road, London, N.W. 1. £6

This book contains the papers presented at a symposium for consultants and research workers, together with edited reports of subsequent discussions. The purpose of the symposium was to consider the fundamental problems presented by atherosclerosis and thrombosis, the principle causes of cardiovascular disease in man.

The first part of the symposium contains eleven articles on some of the more recent information concerning the detailed structure, histochemistry, biochemistry and physiology of blood vessels in man and animals, together with a consideration of the abnormal with the normal. The second part deals with various aspects of the coagulation process, the blood platelet and the various mechanisms which prevent clotting, such as the physiology of blood coagulation, the structure of thrombi, experimental thrombosis, coagulation changes in thrombosis, platelet structure and function in relation to blood coagulation, the action of coumarin-type anticoagulants fibrinolytic agents, and others.

The pathology of vascular disease forms the subject matter of the third part of the book. This section is divided into three chapters concerned with the pathology of the lesions in man and animals, and experimental lesions. The lesions of coronary heart disease, and cerebral infarction and thrombosis in relation to atherosclerosis in man form the basis of the first chapter. In the chapter on animal lesions appear papers on the comparative aspects of arterial disease, cardiovascular disease in domestic animals, atherosclerosis in the turkey and some observations on degenerative vascular diseases in swine. The effect of atherogenic diet on regenerating rat arteries, tissue culture studies of arterial interia and aspects of arteriolar behaviour in arterial hypertension form the subject matter of the third chapter.

The final part of the book deals with clinical research in vascular disease and the relationship between research and clinical practice.

This symposium not only covers a wide field, but it also presents a cross section of modern thought in relation to cardiovascular disease which would be impossible to obtain elsewhere, and as such will be of great value to research workers, clinicians and teachers in human and animal medicine who are either actively involved or interested in this subject.

R. C. TUSTIN.

DISEASES OF SWINE

(*Second edition*, 1964.)

EDITED BY H. W. DUNNE. — 911 *pages*, 325 illustrations, (20 in colour). Bailliere, Tindall and Cox, London. *Price* 130s.

It is a pleasure to record the appearance of this second edition some six years after the book's original publication. The text has been lengthened by 182 new pages and 69 new illustrations, 20 of these in full colour. Nine new authors, four of them from Iowa, have joined the panel, which now comprises no less than 56 authorities.

The inclusion of six new chapters has greatly increased the scope of the work. A new chapter on the composition of body fluids provides valuable biochemical data and includes information on atherosclerosis and serum lipids. A new chapter on procine enteroviruses is contributed by Dr. Betts of Cambridge, England who is the only non-American authority on the panel. The four other new chapters are titled—"Mycoplasmosis", "Non-infectious sterility and artificial Insemination", "Abortions and Stillbirth" and "Acute Circulatory Failure and Hepatosis diaetetica" respectivley.

In addition to the new chapters described above there have been numerous additions and amplifications of the old text. The chapter on paralysis and lameness has been lengthened from 5 to 15 pages to provide a much improved account of the subject. The chapter on Teschen disease now includes reference to the various

other viral encephalomyelitides described in recent years. Entirely new information has been included on such diverse subjects as Bordetellosis, gastric ulceration, ketosis in sows and the removal of the praeputial diverticulum in boars. The text on blood groups, coli-bacillosis and swine erysipelas has been amplified and brought up to date. Each chapter is followed by an extended bibliography listing publications up to 1963.

A few minor criticisms may perhaps be permitted. The section on A.I. could be amplified to include more information. It is a pity that the new section on spirochaetosis refers only to footrot and does not describe this infection in other parts of the body. The information regarding liver damage from migrating Ascaris larvae on page 527 would be improved by reference to the immunological studies of Taffs. Finally, the text omits all reference to the pyelo-nephritis resulting from Corynebacterium suis infection and to the condition known as terminal ileitis, resembling Crohn's disease in man.

The very minor nature of these criticisms serves only to emphasise that this is a complete and up-to-date account of modern concepts concerning the causation and nature of swine disease. It will prove indispensable to all interested in these subjects.

R.K.L.

"VIRUSES OF VERTEBRATES"

by Sir Christopher Andrews—Balliére, Tindall & Cox, London, 1964.

This book is a most welcome addition to the reference library of the virologist. It is divided into four parts. Part I deals with the RNA viruses which are classified into various groups such as the Picornaviruses, Arboviruses, Myzoviruses etc. The DNA viruses are grouped according to their common characteristics in Part II. The unclassified viruses are given in Part III and Part IV is devoted to the Chlamydozoaceae.

Where possible the information concerning each virus is given under the following headings: Synonyms, Morphology and developmental cycle, chemical composition, physico-chemical characterers, haemagglutination and Antigenic properties, interference, cultivation, distribution, pathogenicity, ecology and control measures. The subject matter is easy to find and the book is well written and factual and there is adequate reference to the literature This contribution by such an eminent virologist as Sir Christopher Andrews is all the more valuable for the following reasons

(1) The realistic approach by the author in his attempted classification of the viruses. This classification will no doubt serve as a stimulus for further research and as a basis for the future classification of new viruses and those dealt with in Part III of the book.

(2) The concise and orderly manner in which the data are presented. There is no unneccessary padding and it serves as a handy quick reference to anybody who wants to know the bare facts about the morphology, physico-chemical properties, cultivation, pathogenicity and ecology of virus and the control of the disease.

(3) As intended by the author, the book is a valuable aid in the placing and identification of a newly isolated virus.

With the tremendous expansion of virus research throughout the world and the rate at which new information becomes available and new viruses are found, it is understandable that any book of this nature will not contain the latest information in respect of every virus by the time it is published, for instance, the information on lumpy skin disease virus contained in this book is by no means complete. Fortunately there appears to be very few inaccuracies in the text. Minor inaccurate statements such as the reference to the size of bluetongue virus, have been noticed, but these will no doubt be corrected in the revised edition, which must of necessity be published within a reasonably short period of time.

K.E.W.

PUBLIC RELATIONS SERVICE

Dr. J. Zwarenstein has left Onderstepoort on three months leave prior to superannuation. He will be re-employed in the Division of Veterinary Field Services and will be stationed at Allerton Laboratory, Pietermaritzburg.

Dr. W. Dorsman, helminthologist from the Centrale Diergeneeskundige Instituut, Rotterdam, has been spending a short time with Dr. Reinecke of the Section of Helminthology, Onderstepoort. Dr. Dorsman is a world authority on Chemotherapy of Fascioliasis — liver fluke disease.

Dr. P. A. Boyazoglu, after an absence of three years, returned recently from America where he obtained a Ph.D. degree at the University of Minnesota. He is attached to the section of Biochemistry and Nutrition at the Veterinary Research Institute, Onderstepoort.

Dr. M. R. Coskuner, a veterinarian from Turkey, has returned home after spending six months as a guest worker in the section of Protozoology of the Veterinary Research Institute, Onderstepoort.

Dr. Gerda Philipsborn has been appointed to the section of Pathology at Onderstepoort Veterinary Research Institute. She is a graduate of the University of Munchen but part of her time was spent at the University of Hanover.

Dr. Stottmeier of the T.B. Research Institute of Hamburg is working in the section of Bacteriology at Onderstepoort where he will be employed for about two years on combined C.S.I.R.-OP. projects.

Dr. Eberhard Munz of the University of Munchen is spending a year in the section of Virology at the Onderstepoort Veterinary Research Institute.

Dr. Trichardt of Riversdale has been appointed to the section of Medicine at the Faculty of Veterinary Science, Onderstepoort.

Dr. S. J. van Rensburg of the Section of Reproduction, Onderstepoort has recently returned after spending six months at the University of Cambridge, six months at the University of California, as a guest research worker.

Dr. M. C. Lambrechts, Chief of the Division of Veterinary Field Services, recently returned to the Republic after an official overseas tour lasting ten weeks. The objectives of the tour included a study of animal disease control and eradication, meat inspection and poultry inspection, as well as the governmental organizations concerned, in the United States of America and Canada. He also attended the Annual Conference of the Permanent Foot and Mouth Disease Commission of the O.I.E. in Paris and visited London and Madrid on official business.

Ian F. H. Purchase, B.V.Sc., Trinity Hall, has been successful in obtaining his P.H.D., Cambridge. Dr. and Mrs. Purchase and family are returning to South Africa.

LIST OF VETERINARIANS

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VOLUME 35

INDEX TO AUTHORS