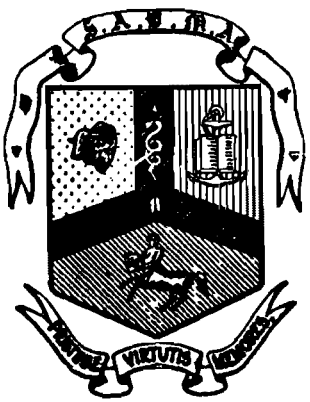
JOURNAL

OF

THE SOUTH AFRICAN VETERINARY MEDICAL ASSOCIATION

VOLUME 38 NUMBER 2
JAARGANG 38 NOMMER 2

JUNIE 1967
JUNE 1967



TYDSKRIF

VAN

DIE SUID-AFRIKAANSE VETERINÊR-MEDIESE VERENIGING

just published

Animal Health and Housing

DAVID SAINSBURY, M.A., Ph.D., B.Sc., M.R.C.V.S., Lecturer in Animal Health, Department of Veterinary Clinical Studies, University of Cambridge.

This book is the first to make a detailed study of the factors (other than genetics and nutrition) that can influence housed animals of the principal domestic species. The first chapters summarise the environmental needs of livestock with emphasis on temperature, humidity and air requirements. Next, practical and economical methods of housing to ensure these requirements are given, with particular attention to the basis of satisfactory intensive management, ventilation and thermal insulation. Housing systems are reviewed in some detail to try to place them in perspective, emphasising their limitations from the livestock and hygienic points of view. In matters of health, emphasis is placed throughout the book on the prevention of disease.

336 pages 123 illustrations 50s.

recent publications

Lameness in Horses

O. R. ADAMS, D.V.M., M.S., Professor and Head of Veterinary Clinics and Surgery, College of Veterinary Medicine, Colorado State University, U.S.A.

This popular book continues to be the only one of its kind, not only in its authoritative guidance in the field of lameness, but also the broad coverage given to equine medicine and surgery. This edition has been brought up-to-date, and two new chapters on methods of therapy and radiology are included.

2nd edition 563 pages 362 illustrations 100s.

Veterinary Pathology

H. A. SMITH, D.V.M., M.S., Ph.D., Formerly Research Associate, Baylor University College of Medicine, Houston, and T. C. JONES, B.S., D.V.M., Director of Pathology, Angell Memorial Animal Hospital.

The third, revised edition of this standard veterinary text. Particular attention is paid to domestic animals, laboratory animals and to certain captive wild animals. There are two new chapters. The first covers genetically determined diseases and the new techniques of cytogenetics for effective studies of disease processes in man and animals. The second is a sound consideration of disease processes in man and animals.

3rd edition 1192 pages 839 illustrations 2 colour plates 160s.

Baillière, Tindall & Cassell

7 & 8 Henrietta St. London WC 2

THE MASTITIS PROBLEM IN SOUTH AFRICA—SOME OBSERVATIONS

L.W. VAN DEN HEEVER* AND W. H. GIESECKE**.

SUMMARY

General observations regarding the significance of bovine mastitis to the economy of milk production and public health are followed by remarks regarding the obligations of milk producers to consumers and the dairy industry. Emphasis is laid on the limitations of field diagnostic aids and the hazards of uncontrolled therapy. Data relevant to the mastitis situation in dairy herds and based on bacterio-cytological and CMT examinations on udder and quarter samples are provided in seven tables.

Staphylococcus aureus and *Streptococcus agalactiae* caused most cases of infectious mastitis in four herds supplying certified raw milk; 29% of quarters were quite normal whereas 20.8% were severely diseased. Only 33.2% of 1089 udder samples, and only 38.0% of 2489 quarter milk samples from 41 herds were completely normal on laboratory examination. CMT reactions were misleading in that 8.9% of healthy udders and 10.7% of healthy quarters gave positive results, whereas 16% and 18.5% diseased udders and quarters gave negative CMT reactions.

Four hundred and fifty isolates of *S. aureus* from mastitic quarters were tested for drug sensitivity *in vitro;* 57.5% and 69.9% were sensitive to penicillin and streptomycin respectively. Seventy-two strains from a problem herd showed only 13.9% and 23.7% respectively to be sensitive to these drugs.

The necessity for proper and continuous udder health control is clearly indicated by these results. Literature is quoted.

INTRODUCTION

In addition to the obvious necessity of eradicating tuberculosis and brucellosis, the problem of combating inflammation of the mammary gland in cows in South Africa has long been well-known. To date little has been done to achieve this realization in spite of the knowledge which exists throughout the world[1-31] and also in South Africa[32-35], regarding the importance of the healthy milk gland for the economical production of milk which complies with the Public Health[36] and the Food Drugs and Disinfectants Acts[37].

To producers, the economic importance of bovine mastitis stems mainly from its effect on yield and quality of milk[38-47] and the shortening of the cow's productive life. The importance to the dairy industry exists in the inferior quality of milk and the correspondingly poor quality or complete failure of manufactured milk products. To the consumer it is of economic importance in that the increasing appearance of various forms of mastitis renders the distribution of wholesome milk and milk products at minimal prices impossible; mastitis undoubtedly raises the cost of milk production.

As far as public health is concerned the importance of mastitis centres around the fact that large quantities of raw milk and milk products are still distributed to the consumer. Of further importance to public health is the as yet unpredictable effect of more or less continuous ingestion of dairy products containing antibiotics resulting from their indiscriminate use for mastitis therapy. With regard to milk hygiene it has to be accepted that milk from cows with secretional disturbances cannot be made normal by subsequent technological dairy procedures. Milk hygiene not only consists of clarification and pasteurization in the dairy plant or depot but must inevitably start on the producer's farm. Healthy milk can only be produced by healthy cows and the quality of milk for liquid consumption as well as manufacturing purposes is dependant on the hygienic production and handling of farm milk.

Despite the efforts of the various official milk control agencies and advisory services, the appearance of evidence of mastitic secretions in farm milk[48,58], repeated reports concerning abnormal composition of such milk[48-50,59], and the results of recently performed examinations of a small number of dairy farms producing milk for raw consumption (table 1) casts serious doubts regarding the general knowledge of producers about the aetiology, symptoms, effect and treatment of mastitis. Without a sound understanding of these matters the development of an adequate conscience

*Faculty of Veterinary Science, University Pretoria, Onderstepoort.
**Veterinary Research Institute, Dept. A.T.S., Onderstepoort.

by milk producers towards milk consumers cannot be expected. Every dairyman who is producing food for human consumption should be aware of his responsibilities and display a real desire to solve the mastitis problem in his herd and to maintain continuous control with the assistance of control officers.

In contrast, many producers do not seem to be at all concerned about the presence and extent of mastitis in their dairy herds. All too often, they only consider the obvious clinical forms of the disease. To them these cases seem to present the entire mastitis problem in the herd until notified to the contrary by official authorities. Most producers fail to realize that the most important form of mastitis is chronic, subclinical, and of hidden character, and that recession of a clinically diseased udder into the subclinical stage does not necessarily indicate complete recovery. Such a misconception is of particular significance in the case of producers of milk for raw consumption as shown in table 1.

Thus health risks and production losses are certainly increased, if not actually caused, by ignorance and neglect of important factors concerning udder health in dairy herds and by the hazardous "do-it-yourself" attitude of most producers. The adequacy of consumer protection against pathogenic micro-organisms and their toxins, inferior chemical and poor keeping quality of milk and milk products, residues of antibiotics, disinfectants, pesticides and insecticides remains questionable until such time as all possible steps for removal and curtailment of these risks and losses have been exhausted.

It cannot for a moment be doubted that the consumer has the right to assume that milk is the thoroughly mixed secretion of the regularly and completely milked udder of one or several healthy, sufficiently fed cows kept under hygienic conditions, to which nothing is added nor extracted, which contains not less than 8.5% SNF and 3% fat, and no colostrum or late lactation secretion. Similarly only such milk should be used for the manufacture of milk products to justify the pretention of high nutritional and hygienic quality.

The following is an attempt to summarise certain basic principles concerning mastitis, its diagnosis and therapy in the light of the results of examination of some problem herds.

The term "mastitis" means inflammation of the mammary gland[59]. Although characterised by pathological changes in the glandular tissue and duct system of the mammary gland, and by physical, chemical, cytological, and bacteriological changes in the milk, the term "mastitis" permits no conclusions concerning cause or degree of the pathological changes: the cause may be either of a purely traumatic or an infectious nature; inflammation of the mammary gland manifests itself in a variety of forms, ranging from obviously acute to hidden and chronic. The diagnosis has to cover these points so as to give evidence of or exclude inflammation or infection and to identify the cause of the latter[60]. Because the presence of inflammatory cells detected in milk by laboratory procedures does not provide evidence of an infection, and the presence of micro-organisms or clinical evidence of discrete changes in the consistency of a quarter does not prove inflammation, satisfactory mastitis diagnosis can only be obtained by a combination of methods. These include the clinical examination of the mammary gland and the secretion, the chemical and physical nature of the secretion, and its cellular and bacterial content. The necessity for combining several methods is less essential in the more obviously acute cases, but more important in herd examinations undertaken to detect chronic inflammatory secretional disturbances.

Methods

In the course of investigations into the situation in herds with known mastitis problems, considerable data have been accumulated by microbiological, cytological and antibiotic sensitivity tests. Microbiological examination consisted of plating whole milk sediment onto tryptose blood agar plates and picking off colonies for further identification as necessary. Leucocyte counts were made on Prescott-Breed smears of whole milk. Antibiotic sensitivity was assessed by high level sensitivity discs (OXOID and DIFCO) on blood tryptose agar. The present report is intended also to deal with some observations on the CMT — discussed in greater detail in a previous paper[61] — and its application in practice.

For the purpose of herd sanitation work, the udders or quarters were classified according to the bacteriological-cytological results as healthy, suspicious and diseased as follows:

a) *Healthy udders/quarters*: Secretion free of mastitis organisms and containing up to 250,000 leucocytes/ml.

b) *Aseptic diseased udders/quarters*, with possibly hidden infections not readily detected: Secretion free of mastitis organisms and containing over 300,000 leucocytes/ml.

c) *Suspicious udders/quarters* (chronic cases, sample possibly contaminated): Secretion with mastitis organisms and containing up to 250,000 leucocytes/ml.

TABLE 1.—DISTRIBUTION OF RESULTS OF MICROBIOLOGICAL-CYTOLOGICAL EXAMINATION OF QUARTER MILK SAMPLES FROM CERTIFIED RAW MILK HERDS.

Method		Examined			Diagnosis									
					(a) healthy		(b) aseptic diseased		(c) suspicious		(d) slightly diseased		(e) severely diseased	
		herds	udders	quarters	udders	quarters	udders	quarters	udders	quarters	udders	quarters	udders	quarters
Laboratory bacterio-cytological		4	209	822	22 10.5%	239 29.1%	24 11.5%	230 27.9%	34 16.3%	147 13.9%	25 12.0%	36 4.3%	103 49.2%	171 20.8%
Isolations	*S. aureus*.....	—	—	—	—	—	—	—	23 67.6%	88 59.8%	14 56.0%	21 58.3%	63 61.2%	105 61.4%
	Streptococci.....	—	—	—	—	—	—	—	9 26.5%	52 35.3%	10 40.0%	9 25.0%	31 30.1%	57 33.3%
	Streptococcus agalactiae*	—	—	—	—	—	—	—	8 99.9%	46 88.5%	6 60.0%	5 55.6%	29 93.6%	52 82.2%
	Coliforms and others.....	—	—	—	—	—	—	—	2 5.9%	7 4.8%	1 4.0%	6 16.7%	9 8.7%	9 5.3%

* The no. and percentage of *Streptococcus agalactiae* isolates in this table is again included under the total no. and percentage of Streptococci isolates.

TABLE 2.—DISTRIBUTION OF RESULTS OF LABORATORY DIAGNOSIS AND CMT REACTIONS OF UDDER MILK SAMPLES.

Herds examined	Diagnostic method	Udders examined		Diagnosis									
				(a) healthy udders		(b) aseptic diseased udders		(c) suspicious udders		(d) slightly diseased udders		(e) severely diseased udders	
27	Laboratory bacteriological, cytological	1989		661 33.2%		663 33.3%		128 6.4%		83 4.2%		454 22.8%	
	CMT reaction	+	—	+	—	+	—	+	—	+	—	+	—
		895 44.9%	1094 55.1%	59 8.9%	602 91.1%	348 57.0%	285 43.0%	10 7.8%	118 92.2%	45 54.2%	38 45.8%	403 84.4%	51 15.6%

TABLE 3.—DISTRIBUTION OF RESULTS OF LABORATORY DIAGNOSIS AND CMT REACTIONS OF QUARTER MILK SAMPLES.

Herds examined	Diagnostic method	quarters examined		Diagnosis (a) healthy quarters		(b) aseptic diseased quarters		(c) suspicious quarters		(d) slightly diseased quarters		(e) severely diseased quarters	
14	Laboratory bacteriological, cytological	2489		947 38.0%		855 34.3%		155 6.2%		90 3.6%		442 17.8%	
	CMT reaction	+ 1112 44.6%	— 1377 55.6%	+ 101 10.7%	— 846 89.3%	+ 558 65.2%	— 297 34.8%	+ 79 12.3%	— 136 87.7%	+ 52 57.8%	— 38 42.2%	+ 382 86.4%	— 60 13.6%

TABLE 5.—ANALYSIS OF MICROBIOLOGICAL-CYTOLOGICAL RESULTS OF 2801 SINGLE MILK SAMPLES FROM LACTATING UDDERS [TABLE 4(c), (d), (e)].

	Leucocyte counts/ml.	Up to 250,000 = suspicious udders			Between 300,000—500,000 = slightly diseased udders			Over 500,000 = severely diseased udders		
	Microbiological examinations	Primary cultures pure	Primary cultures mixed	Total	Primary cultures pure	Primary cultures mixed	Total	Primary cultures pure	Primary cultures mixed	Total
Isolations	*S. aureus*	141 73%	10 37%	151 69.2%	75 58.5%	13 24%	88 48.1%	345 59.4%	135 48.2%	480 55.7%
	Streptococci	44 22.8%	15 55.5%	59 27%	48 37.4%	31 57.3%	79 43%	196 33.4%	135 48.1%	331 38.3%
	Streptococcus agalactiae*	23 52.3%	8 53.3%	31 52.6%	31 64.6%	26 83.9%	57 72.2%	136 69.4%	105 77.8%	241 72.8%
	Coliforms	3 1.5%	1 3.7%	4 1.9%	4 3.1%	—	4 2.1%	33 5.6%	5 2.3%	38 4.3%
	Corynebacteria	3 1.5%	1 3.7%	4 1.8%	2 1.6%	10 18.4%	12 6.5%	8 1.3%	5 1.8%	13 1.4%

* The no. and percentage of *Streptococcus agalactiae* isolates in this table is again included under the total no. and percentage of Streptococcus isolates.

d) *Slightly diseased udders/quarters* (chronic cases, possibly aseptic and samples contaminated): Secretion with mastitis organisms and 300,000 - 500,000 leucocytes/ml.

e) *Severely diseased udders/quarters*: Secretion with mastitis organisms and over 500,000 leucocytes/ml.

The results of investigations concerning the CMT are summarised in tables 2 and 3.

Conclusions

From these tables it may be concluded that it is possible to obtain a general impression of the udder health situation in a herd by means of the CMT procedure. Arbitrary application of antibacterial therapy to all CMT positive cows would have resulted in 57% and 65% of aseptically inflamed udders and quarters respectively being unnecessarily treated. Similarly, 45% and 42% of infected udders and quarters with slight inflammatory reactions respectively would have been overlooked as requiring therapy if the CMT had been used as the only diagnostic method. It appears that it is impossible to use the CMT to select udders or quarters for mastitis therapy or bacteriological-cytological examination. We conclude therefore that the CMT is of little value in an organised mastitis control programme aimed at therapy and prophylaxis.

To obtain an indication of the situation in herds in which mastitis constitutes a problem, data obtained from the bacteriological-cytological examination of single secretion samples from 2801 lactating udders in 40 herds were analysed. The summary is presented in table 4.

as the bacterial cause of mastitis in these herds. Staphylococci were isolated from a relatively larger proportion of milk samples with a low leucocyte count than in more obviously altered secretion samples. The ability of streptococci to stimulate a greater leucocyte response than is the case with staphylococci is well known. Coliforms and corynebacteria did not appear to constitute a major problem.

Concerning the sensitivity *in vitro* of the strains of *S. aureus* isolated from the herds under consideration, the results of antibiogram tests, using the disc method, have been summarised in table 6 and 7.

Table 6 represents the sensitivity pattern in herds with a mastitis problem. The data in table 7 reflect the situation in a herd of 110 lactating cows with a severe and particular problem, where the owner lost three cows and 20% of his milk yield during the four months prior to seeking veterinary advice, a period during which he also spent some R1,200.00 on antibiotics and suffered a total loss of about R1,850.00. This occurrence is not unique.

Unfortunately, mastitis therapy is usually oversimplified and frequently performed with unwarranted faith in the so-called wonder drugs in shotgun or cocktail remedies. These are commercially available in numerous more or less suitable and effective forms to anybody regardless of his ability to use his discretion. Often enough therapy is instituted on wrong advice by unscrupulous salesmen, misinformed pharmacists or the staff of agricultural co-operatives and mostly based on an indirect mastitis test but without any

TABLE 4.—DISTRIBUTION OF RESULTS OF DIAGNOSIS BASED ON MICROBIOLOGICAL-CYTOLOGICAL EXAMINATION OF SINGLE COW (UDDER) SAMPLES.

Total examined		Diagnosis				
Herds	Udders	(a) healthy udders	(b) aseptic diseased udders	(c) suspicious udders	(d) slightly diseased udders	(e) severely diseased udders
40	2801	790 28.2%	901 32.2%	206 7.3%	151 5.4%	752 26.1%

Using the same system of classification as previously, it is concluded that only a rather small number (28.2%) of udders could be classed as normal, with 26.1% being severely diseased, 32.7% showing evidence of aseptic inflammation and 12.7% being suspicious or only slightly diseased.

Table 5 indicates that *S. aureus* and mastitis streptococci, notably *Sc. agalactiae*, predominate

confirmative, differentiating and controlling diagnosis. This tendency, activated by the urge that something must be done, unfortunately often results in a variety of antibiotic-resistant strains of microbes and particularly staphylococci.

Whereas the antibiotics, sulphonamides and nitrofurans available today have such a wide effective spectrum that they are able to cope ef-

TABLE 6.—DRUG SENSITIVITY OF HAEMOLYTIC, COAGULASE POSITIVE STAPHYLOCOCCI ISOLATED FROM QUARTER MILK SAMPLES WITH A LEUCOCYTE CONTENT OVER 300,000/ML.

Strains tested	Sensitivity	Chlortetracycline 50 mcg	Chloramphenicol 50 mcg	Furazolidone 100 mcg	Penicillin 5 U	Streptomycin 25 mcg	Oxytetracycline 50 mcg	Erythromycin 50 mcg	Nitrofurazone 100 mcg	Kanamycin 30 mcg	Neomycin 30 mcg	Novobiocin 30 mcg	Tetracycline 30 mcg
450	Sensitive.....	91.3%	96.7%	91.8%	57.5%	69.9%	84.7 %	98.4%	98.8%	91.3%	95.1%	93.2%	94.2%
	Partly sensitive.....	4.5%	2.4%	5.7%	4.5%	4.5%	6.8%	0.4%	—	8.7%	3.9%	3.9%	—
	Resistant.....	4.2%	0.9%	2.5%	38.0%	25.6%	8.5%	1.2%	1.2%	—	1.0%	2.9%	5.8%

TABLE 7.—DRUG SENSITIVITY OF HAEMOLYTIC, COAGULASE POSITIVE STAPHYLOCOCCI ISOLATED FROM QUARTER MILK SAMPLES WITH A LEUCOCYTE CONTENT OVER 300,000/ML OBTAINED FROM A MASTITIS PROBLEM HERD.

Strains tested	Sensitivity	Chlortetracycline 50 mcg	Chloramphenicol 50 mcg	Furazolidone 100 mcg	Penicillin 5 U	Streptomycin 25 mcg	Oxytetracycline 50 mcg	Erythromycin 50 mcg	Nitrofurazone 100 mcg	Kanamycin 30 mcg	Neomycin 30 mcg	Novobiocin 30 mcg
72	Sensitive.....	86.1%	90.3%	86.1%	13.9%	23.7%	100.0%	100.0%	94.4%	97.2%	100.0%	100.0%
	Partly sensitive.....	13.9%	9.7%	13.9%	8.3%	5.6%	—	—	5.6%	5.6%	—	—
	Resistant.....	—	—	—	77.8%	70.7%	—	—	—	—	—	—

fectively with most of the common gram positive and gram negative udder pathogenic microbes *in vitro*, the results in practice show too often that none of the therapeutic agents can be regarded as "ultima ratio", even less so when factors such as adequate therapeutic level in the mammary gland, the importance of the vehicle, the loss of concentration and the pathological changes in the mammary gland are ignored.

Discussion and Conclusions

From table 4 it appears remarkable that apart from the low percentage of healthy udders (28.2%) and healthy quarters (38.3%), there exists a relatively high percentage of udders (32.2%) and quarters (29.8%) with symptoms of aseptic or nonspecific forms of mastitis. This is an important factor, as glands showing this form of inflammation have little resistance to infectious pathogenic micro-organisms[9]. Furthermore, they are difficult to dry off[62], and the secretion is hygienically objectionable due to its increased content of blood components, whilst being of lower quality for dairy processing because of poorer curdling capacity[62]. For the time being it cannot be excluded that some of these glands, classed as aseptically diseased, may be found to contain obscure pathogens when subjected to special examination or that the increased cell content resulted from particular physiological or pathological reactions[63] of the animals such as allergies[64], ovarian dysfunction[9], or hidden infections with streptococci and staphylococci[62,63-67]. However, they appear more likely to be associated with wide spread incorrect hand as well as machine milking techniques[68] and by the uncontrolled extensive use of a variety of mastitis therapeutics. On the other hand, examinations in other countries have shown these same factors to be responsible for the high incidence of staphylococcus infections[47,68-71].

In this connection it should be pointed out that in particular the unlimited and uncontrolled administration of potentially dangerous drugs, such as antibiotics, has been proved to be hazardous when, through excessive zeal and lack of thought, entire herds are treated prophylactically, the treatments are carried out without regard to an aseptic technique, and for reasons of economy, suitable and perhaps normally effective drugs are administered in far too small doses, or else drugs are used which do not counteract the infection or may even be contra-indicated. It can be stated that in spite of a multitude of effective mastitis therapeutics being available and a steady stream of new ones being introduced, the mastitis situation in South Africa is alarming and shows no prospects of improving in the near future unless concerted efforts are made to cope with this problem.

To achieve this, it must be regarded as completely inadequate to concentrate the limited and moreover divided efforts of the various interested or official bodies dealing with industrial and fresh milk, only on the so-called problem herds or to examining, controlling and treating the odd herd. Mastitis in South Africa is not a problem of some herds but must unfortunately be regarded as an overall menace and be dealt with as such, not only by the official control authorities and veterinary practitioners, but on an organised basis by dairy farmer co-operatives and dairy plants. Furthermore it would be basically wrong to centre one's hopes on the discovery of new therapeutic substances. Generally, it has to be realised that the real answer to the problem of mastitis lies in herd management, prevention of predisposing factors and spontaneous infections rather than in therapy.

ACKNOWLEDGEMENTS

The Chief, National Veterinary Research Institute, Onderstepoort, is thanked for facilities and the permission to publish. Mr. J. J. van Staden and Miss Denise Hope ably assisted in the laboratory.

REFERENCES

1. ALTERAUGE, W. 1963 *Der prakt. Tierarzt* 5: 184.
2. ANKE, H. 1964 *Mh. Vet.-Med.* 6: 19.
3. BECKER, W. 1963 *Dtsch. tierärztl. Wschr.* 70: 117.
4. CERNEA, J., BUTURA, J., BANGAU, S., SIRBU, Z. & SUTEU, E. 1963 *Mh. Vet. Med.* 23: 883.
5. DIERNHOFER, K. 1937 *Wien tierärztl. Mschr.* 24: 386.
6. FODSTAD, F. H. 1963 *Dairy Sci. Abstracts* 25: 333.
7. FRANCIS, J. 1962 *Austr. J. Dairy Techn.* 17: 144.

8. GLÄTTLI, H. R., 1961 *Schweiz, Arch. Tierheilkunde* 103: 140.
9. HEIDRICH, H. J. & RENK, W. 1963 *Krankheiten der Milchdrüse bei Haustieren* Berlin und Hamburg, Paul Parey.
10. HERHHOLDT, U. & STEINHARDT, M. 1964 *Mh. Vet.-Med.* 4: 121.
11. HOWELL, D., WILSON, C. D. & VESSEY, M. P. 1964 *Vet. Rec.* 76: 1107.
12. KLEINSCHROTH, E. 1965 *Deutsche Molkerei Zeitung* 83: 1288.
13. KRÜGER, W. 1962 *Dtsch. Milchwirtschaft*, 8: 235.
14. LAING, C. M. & MALCOLM, J. F. 1965 *Vet. Rec.* 68: 447.
15. LEIDL, W. 1963 *Berl. Münch. tierärztl. Wschr.* 76: 201.
16. MAQBOOLUR, R. M. D. 1964 *Dairy Sci. Abstracts* 26: 597.
17. METZGER, R. W. 1962 *J. Amer. vet. med. Ass.* 141: 1173.
18. MILOJEVIC, Z. & MILENKOVIC, D. 1962 *Dairy Sci. Abstr.* 25: 161.
19. MUNCH-PETERSON, E. 1938 Bovine Mastitis. Weybridge, Surrey, England. Imp. Bur. An. Health. *Rev. Ser. I.*
20. OBIGER, G. 1962 *Kieler Milchw. Forsch.-Ber.* 14: 307.
21. OBIGER, G. 1964 *Tierärztl. Umschau*, 19: 117.
22. RENK, W. 1957 *Zbl. Vet.-Med.* 4: 325.
23. RENK, W. 1958 *Dtsch. tierärztl. Wschr.* 65: 497.
24. SCHALM, O. W. 1962 *Canad. Vet. J.* 3: 90.
25. SCHMAHLSTIEG, R. 1956 *Dtsch. tierärztl. Wschr.* 63: 474.
26. SCHMAHLSTIEG, R. 1961 *Dtsch. tierärztl. Wschr.* 63: 474.
27. SEELEMANN, M. 1962 *Mh. Tierhk.* 14: 233, 287.
28. UDALL, D. H. & JOHNSON, S. D. 1931 *Cornell Vet.* 21: 190.
29. ULLNER, W. 1959 *Tierärztl. Umschau*, 14: 286.
30. WENDT, K. & LESKE, U. 1964 *Mh. Vet.-Med.* 19: 49.
31. ZIEGLER, H. & MOSIMANN, W. 1960 *Anatomie und Physiologie der Rindermilchdrüse* Berlin und Hamburg, Paul Parey.
32. RENSBURG, S. W. J. VAN 1939 *Farming in S. Afr.* 14: 334.
33. RENSBURG, S. W. J. VAN 1942 *Farming in S. Afr.* 17: 588.
34. CREWE, G. 1964 *Bladskrif No.* 11, *Dept. Landbou Tegn. Dienste.*
35. LANDREY, J. S. A. 1965 *Jl. S. Afr. vet. med. Ass.* 36: 515.
36. Public Health Act No. 36, 1919, Article 113, South Africa.
37. Regulations under the Food, Drugs and Disinfectants Act, No. 13, 1929, Article 7, South Africa.
38. SEELEMAN, M. 1932 *Die Streptokokkeninfektionen des Euters insbesondere der gelbe Galt.* Hannover, M. and H. Schaper.
39. SHAW, A. O. & BEAM, A. L. 1935 *J. Dairy Sci.* 18: 353.
40. HASTINGS, E. G. & BEACH, B. A. 1937 *J. Agric. Res.* 54: 199.
41. HASTINGS, E. G. & BEACH, B. A. 1939 *J. Agric. Res.* 58: 543.
42. HORWOOD, R. E., CLARK, C. F. & BRYAN, C. S. 1943 *Quart. Bull. Mich. agric. exper. Sta.* 26: 43.
43. MCDONALD, F. H. 1945 *N.Z.J. Sci. Tech.* A27: 258.
44. WARD, A. H. & CASTLE, O. M. 1945 *21st Ann .Rep. N.Z. Dairy Bd.* 46.
45. EDWARDS, S. J. & BROWNLEE, A. 1946 *Vet. Rec.* 58: 335.
46. CROSSMAN, J. V., DODD, F. H., LEE, J. M. & NEAVE, F. K. 1950 *J. Dairy Res.* 2: 128.
47. SCHALM, O. W. 1962 *A Syllabus on the bovine mammary glands in health and disease.* Davis, Calif, University of Calif.
48. PULLINGER, E. J. 1935 *J. Dairy Res.* 6: 369.
49. PULLINGER, E. J. 1942 *J. S. Afr. vet. med. Ass.* 13: 116.
50. PULLINGER, E. J. 1944 *J. S. Afr. vet. med. Ass.* 15: 39.
51. MEARA, P. J. & MOWAT, J. 1947 *J. S. Afr. med. vet. med. Ass.* 18: 129.
52. MEARA, P. J. 1950 *S. A. Med. J.* 24: 593.
53. MEARA, P. J. 1951 *J. S. Afr. vet. med. Ass.* 22: 49.
54. MEARA, P. J., GREATHEAD, M. M., & HUYSER, J. H. 1957 *J. S. Afr. vet. med. Ass.* 28: 353.
55. VAN DEN HEEVER, L. W. 1959 *J. S. Afr. vet. med. Ass.* 30: 271.
56. CREWE, G. 1965 *J. S. Afr. vet. med. Ass.* 36: 509.
57. HERMANN, M. N. 1965 *J. S. Afr. vet. med. Ass.* 36: 521.
58. VAN DEN HEEVER, L. W. 1965 *J. S. Afr. vet. med. Ass.* 36: 527.
59. BAKALOR, S. & DE KOCK, A. A. 1946 *Farming in S.A.* 21: 453.
60. HEIDRICH, H. J. 1965 *Der prakt. Tierarzt.* Sonderdruck aus 46/3 1.3.1965.
61. GIESECKE, W. H. & VAN DEN HEEVER L. W. 1967 *J. S. Afr. vet. med. Ass.* 38:6
62. SEELEMANN, M. 1928 *Arch. Tierhk.* 58: 1.
63. RENSBURG, S. W. J. VAN 1947 *Onderstepoort J. vet. Res.* 22: 91.
64. PETERSON, E. H., HASTINGS, E. G. & HADLEY, F. B. 1939 *Cornell Vet.* 29: 11.
65. RUDOLF, J. 1930 *Münch. tierärztl. Wschr.* 81: 321.
66. LITTLE, R. B. & PLASTRIDGE, W. N. 1946 *Bovine Mastitis.* New York and London, McGraw-Hill Book Co.
67. DIERNHOFER, K. 1950 *Wien .tierärztl. Mschr.* 37: 809.
68. KÄSTLI, P. 1963 *Tierärztl. Umschau*, 18: 527.
69. THÖRNE, H. 1958 *Nord. Vet.-Med.* 10: 309.
70. DERBYSHIRE, J. B., DAVIDSON, J. & WILSON, C. D. 1961 *Vet. Rec.* 73: 1011.
71. BRODAUF, H. 1964 *Dtsch. tierärztl. Wschr.* 71: 85.

BETSOLAN

rapid effective relief in allergic and inflammatory conditions

A very high proportion of dogs suffering from allergic and inflammatory skin conditions can be treated rapidly, effectively and economically with Betsolan. Betsolan is the most potent anti-inflammatory corticosteroid at present available; it is forty times more active than cortisone, its parent compound. While it produces all the anti-allergic and anti-inflammatory effects of cortisone in lower dosage, systemically, it exercises less influence on electrolyte balance.

OTHER USES FOR BETSOLAN INJECTION Corticosteroids have far-reaching effects on many functions and tissues and are being used in an increasing number and diversity of conditions in veterinary practice. Betsolan offers the advantages of safety and economy in the treatment of: Shock, Burns, Toxaemias, Dermatoses, Allergies, Inflammations of joints and tendons.

Dosage for small animals: In trauma or shock 0.25 to 2 ml.
Presentation: Vial of 20 ml.

GLAXO-ALLENBURYS (S.A.) (PTY.) LTD.

P.O. Box 485, Germiston, Transvaal

4333/2

THE ANTHELMINTIC EFFICACY OF TWO PHENOTHIAZINE PREPARATIONS OF DIFFERENT PARTICLE SIZE SYNTHESIZED AND PROCESSED IN SOUTH AFRICA

D. K. SHONE AND J. R. PHILIP(*)

SUMMARY

The anthelmintic efficacy of two types of phenothiazine synthesized and processed in South Africa have been investigated. Phenothiazine with a thiodiphenylamine content of 87.3 per cent and specific surface area of 9,000 sq cm per gram brought about a 99.8 per cent reduction of adult *Haemonchus contortus* at a dose rate of 300 mgm thiodiphenylamine per kg. At a dose rate of 600 mgm thiodiphenylamine per kg there was a 51.9 per cent reduction of adult *Oesophagostomum columbianum.*

A phenothiazine with a thiodiphenylamine content of 87.5 per cent and a specific surface area of 17,000 sq cm per gram resulted in a reduction of 85.2 per cent of adult *Oesophagostomum columbianum* and 99.9 per cent reduction of *Ostertagia circumcincta* adults at a dose rate of 600 mgm per kg. The activity against adult *O. columbianum* tended to be erratic.

The phenothiazine in both trials was administered by stomach tube into the rumen.

(1) Research and Development Division,
A.S. Ruffel (Pty.) Limited,
P.O. Box 38,
ISANDO, TRANSVAAL.

INTRODUCTION

The anthelmintic activity of phenothiazine has been known for over 25 years and constitutes more than 50 per cent of the estimated 100 million doses of anthelmintics used for the treatment of sheep in South Africa. Until recently all phenothiazine used in South Africa was imported from Western Germany, Great Britain, or France.

This work was undertaken in order to evaluate the anthelmintic efficacy of two wettable powder preparations manufactured from phenothiazine synthesized in South Africa.

MATERIALS AND METHODS

This work was undertaken in two parts. In the first part sheep with natural helminth infestations were used and the phenothiazine wettable powder was designed to match imported phenothiazines and is designated "standard" phenothiazine. In the second part of the trial, artificially infested sheep were used and the phenothiazine wettable powder contained particles of approximately half the size of the standard phenothiazine and has been designated "superfine" phenothiazine.

Phenothiazine

Both phenothiazine preparations used in this work were in the form of a wettable powder and were synthesized and processed by Messrs. Synchem (Pty.) Ltd.*

The thiodiphenylamine content was determined by the chromatographic column separation method. The particle size, expressed as the specific surface area, was determined by the air permeability method on a Fisher sub-sieve sizer.

	Standard phenothiazine	Superfine phenothiazine
Thiodiphenylamine content as percentage w/w	87.3	87.5
Specific surface area as cm^2 per gram	9,000	17,000

The dose of the standard phenothiazine was calculated on the basis of 90% w/w thiodiphenylamine.

Sheep

Standard phenothiazine trial. Sixty Merino sheep were purchased in the Bethlehem District of the Orange Free State. A *post mortem* examination, conducted on one sheep on the farm, established the presence of *Haemonchus contortus* and *Oesophagostomum columbianum.* The sheep were transported to our laboratories by road, immediately individually identified by ear-tags and weighed. The 18 lambs and 42 adults were randomly allocated to 3 groups. Individual doses were calculated and 12 hours after arrival the phenothiazine was administered by stomach tube. One group was dosed at the rate of 300 mgm thiodiphenylamine per kg bodyweight and slaughtered 144 hours after treatment. The other group was dosed at the rate of 600 mgm thiodiphenyla-

*Alrode, Alberton, Transvaal.

mine per kg bodyweight and slaughtered 96 hours after treatment. The untreated control group was slaughtered 72 hours after treatment.

Superfine phenothiazine trial. Thirteen lambs reared under worm-free conditions were artificially infested with *Ostertagia circumcincta* and *O. columbianum* as scheduled below. Six of the sheep were dosed by stomach tube at the rate of 600 mgm thiodiphenylamine per kg bodyweight.

Day minus 67 *O. columbianum* — 300 larvae
Day minus 62 *O. columbianum* — 300 larvae
Day minus 57 *O. columbianum* — 300 larvae
Day minus 52 *O. circumcincta* — 500 larvae
Day minus 49 *O. circumcincta* — 250 larvae
Day minus 46 *O. circumcincta* — 300 larvae
Day O Treated

All the sheep were slaughtered 120 hours after treatment.

Collection of faeces:

Faecal collecting bags were attached to the 40 naturally infested sheep treated with standard phenothiazine 6 hours after treatment. All faeces passed during the following 90 hours were collected. The daily faecal collections of each sheep were broken down in water, washed onto B.S.S. 100 mesh sieves and all helminths recovered were identified and counted.

Post Mortem Examinations:

The gastro-intestinal tract was tied off between the omasum and reticulum, at the duodenum 12 inches distal to the pylorus and at the ileo-caecal junction.

The contents of the abomasum and of the 12 inches of duodenum distal to the pylorus were washed onto B.S.S. 200 mesh sieves. All helminths recovered were identified and counted.

The contents of the caecum and colon were washed onto B.S.S. 100 mesh sieves and all helminths recovered were identified and counted.

Results

Standard Phenothiazine:

The results of the controlled trial against *H. contortus* and the critical trial against *O. columbianum* are presented in Tables 1 and 2 respectively.

From Table 1 it will be noted that the percentage reduction of adult *H. contortus* at a dose of 300 mgm/kg was 99.8%. From Table 2 it will be noted that the percentage reduction of adult *O. columbianum* at a dose rate of 300 mgm/kg was 44.8 and at a dose rate of 600 mgm/kg reduction was 51.9.

Superfine Phenothiazine:

The results of this part of the trial are presented in Table 3.

TABLE 1.—THE RESULTS OF A CONTROLLED TEST TO DETERMINE THE PERCENTAGE REDUCTION *Haemonchus contortus* ADULTS FOLLOWING TREATMENT WITH STANDARD PHENOTHIAZINE AT A RATE OF 300 MGM THIODIPHENYLAMINE PER KILOGRAM BODYWEIGHT.

	Control Group	Treated Group
Number of sheep.....	20	20
Mean number of adult *Haemonchus contortus* recovered at *post mortem*..	1,474	2
Range.....	30 to 5,811	0 to 21
Percentage reduction 99.8		

TABLE 2.—THE RESULTS OF A CRITICAL TEST TO DETERMINE THE PERCENTAGE REDUCTION OF ADULT *Oesophagostomum columbianum* FOLLOWING TREATMENT WITH "STANDARD" PHENOTHIAZINE AT A RATE OF 300 AND 500 MGM THIODIPHENYLAMINE PER KILOGRAM BODYWEIGHT.

	Group treated with 300 mgm/kg	Group treated with 600 mgm/kg
Number of sheep.....	20	20
Total number of *Oesophagostomum columbianum* recovered from faeces....	142	170
Range.....	0 to 39	0 to 44
Total number of adult *Oesophagostomum columbianum* recoverd at autopsy..	174	157
Range.....	0 to 65	0 to 43
Percentage reduction.....	44.8	51.9

It will be noted that a small number of *H. contortus* were present in the lambs, presumably due to a contaminated larval culture. The percentage reduction achieved for all three species was high but the action against the adult *O. columbianum* tends to be erratic.

Haemonchus contortus	100% reduction
Ostertagia circumcincta	99.9% reduction
Oesophagostomum columbianum	85.2% reduction

Discussion

Four factors are known to influence the anthelmintic efficacy of phenothiazine. The influence of particle size is demonstrated in this trial by the increase of activity against *O. columbianum* adults from 51.9 per cent for phenothiazine with a specific surface area of 9,000 sq cm per gram to 85.2 per cent with phenothiazine of a specific surface area of 17,000 sq cm per gram. Both phenothiazine preparations used were from com-

TABLE 3.—THE RESULTS OF A CONTROLLED TEST TO DETERMINE THE PERCENTAGE REDUCTION OF *Ostertagia circumcincta* AND *Oesophagostomum columbianum* ADULTS FOLLOWING TREATMENT WITH A "SUPERFINE" PHENOTHIAZINE AT A DOSE RATE OF 600 MGM THIODIPHENYLAMINE PER KG BODYWEIGHT.

	Control Group	Treated Group
Number of sheep.....	8	7
Haemonchus contortus Mean number recovered at autopsy.....	17	0
Range.....	10 to 29	—
Percentage reduction.....	—	100
Ostertagia circumcincta Mean number recovered at autopsy.....	341	1
Range.....	105 to 458	0 to 3
Percentage reduction.....	—	99.6
Oesophagostomum columbianum Mean number recovered at autopsy.....	49	8
Range.....	22 to 116	1 to 18
Percentage reduction.....	—	85.2

mercially produced batches and contained wetting agents. The effect of particle size on phenothiazine activity has been reviewed by Gibson[2].

Particle sizes may be determined by various methods but unfortunately comparisons cannot be drawn between results obtained by different methods even when the results are expressed in the same terms. For example, the specific surface area determined by the sedimentation method is consistently smaller than that obtained by the air permeability method.

The thiodiphenylamine content is important for the calculation of the dose rate. The chromatographic separation method described in 1965 edition of the British Veterinary Codex appears to give a valid reflection of the thiodiphenylamine content, which is not done by other methods. This method is unfortunately very time consuming.

The thiodiphenylamine content is important, not only because the dose rate is calculated from it, but because the presence of large quantities of impurities interfere with the anthelmintic action of the phenothiazine even when the dose is corrected for the thiodiphenylamine content. Forsyth, *et al*[3] found that if the thiodiphenylamine content falls below 85 per cent, the anthelmintic action becomes very erratic.

The wetting agents may also influence the anthelmintic efficacy and in this regard the wetting agents used in the locally synthesized material are the same as those used in one of the imported phenothiazines.

It is clear from an examination of the literature that the anthelmintic efficacy of phenothiazine has in recent years been re-examined in a number of countries. In South Africa, Reinecke[4] has reported on the efficacy of several "standard" phenothiazines and one very small particle size phenothiazine. The use of modern air attrition mills permit the production of "superfine" phenothiazine of predictable efficacy on a commercial scale.

ACKNOWLEDGEMENT

We wish to thank Mr. P. Bloom who undertook the assays and particle size determinations, and Messrs. P. Alderton and R. Cain for their very able assistance.

REFERENCES

1. British Veterinary Codex. 1965 London. The Pharmaceutical Press.
2. GIBSON, T. E. 1965 *Veterinary Anthelmintic Medication.* Farnham Royal, England. Commonwealth Agricultural Bureau.
3. FORSYTH, B. A., SCOTT, M. T. and BAINBRIDGE, J. R. 1961 Vet. Rec. 73: 67.
4. REINECKE, R. K. 1966 *Jl.* S. Afr. vet med. Ass. In Press.

Synalar

Fluocinolone Acetonide

Trade Mark

For topical application in local inflammatory, pruritic and allergic conditions of the skin and mucosa

- ✱ A powerful and dramatically swift corticoid.
- ✱ Forty times more potent than Hydrocortisone.
- ✱ Achieves success in many cases previously refractory to other corticoids.
- ✱ Free from systemic effects.
- ✱ Effective in low concentrations and easily applied.

Synalar the economical corticoid

Available as

"Synalar"	Cream/Ointment	0.025%	Fluocinolone	Acetonide	5 mg.	R0.90
					15 mg.	R2.25
					30 mg.	R4.00
"Synalar"	Lotion	0.025%	Fluocinolone	Acetonide	20 c.c.	R2.40

Synalar-N is also available with 0.5% Neomycin

when concurrent topical antibacterial therapy is required for an inflammatory dermatosis complicated by local infection.

"Synalar"-N Cream/Ointment 5 gm. R0.95
"Synalar"-N Cream/Ointment 15 gm. R2.28

ICI

A Product of Syntex Corporation

I.C.I. SOUTH AFRICA (PHARMACEUTICALS) LIMITED

P.O. Box 11270, Johannesburg; P.O. Box 948, Durban;
P.O. Box 1519, Cape Town; P.O. Box 273, Port Elizabeth.

PV 2997

FOCAL NECROTIC PNEUMONIA AND RUMENO-ENTERITIS IN AFRIKANER CALVES.

J. L. DU PLESSIS*, C. M. CAMERON** & E. LANGEN***

SUMMARY

The characteristic pathological feature in a disease of young calves, manifested clinically by severe diarrhoea, marked dehydration, dyspnoea and nervous symptoms, was well-defined, coagulative, focal necrosis in the lungs, spleen, rumen, small and large intestines. The possibility that these lesions were associated with *Actinobacillus equuli* is discussed. Infection by this organism was experimentally produced in a calf with some histological lesions resembling those in natural cases. Mucormycosis was observed as a secondary infection in the brain of one and the rumen of another case.

INTRODUCTION

Members of the genus *Actinobacillus* have occasionally been associated with pneumonia in calves. *Actinobacillus actinoides* was first isolated from pneumonic lungs of calves by Smith[1], who considered it the sole aetiologic agent in an outbreak of broncho-pneumonia. An organism, which in many respects resembled *A. actinoides,* was recovered from two outbreaks of bovine bronchopneumonia in 1945[2]. Although enzootic pneumonia of calves is generally assumed to be primarily a virus disease, *A. actinoides* is one of the bacterial species associated with this disease[3], and notably the only one that could produce a bronchopneumonia after intranasal instillation.

The purpose of this report is to record the pathological lesions of a disease in one to three months old Afrikaner calves during the late summer and autumn months on a Northern Free State farm.

SYMPTOMS

There was a morbidity rate of 10 to 20 percent and a mortality rate of five per cent. Consistent symptoms were dyspnoea, severe diarrhoea, hyperthermia (103°F) and marked dehydration, evidenced by sunken eyes and loss of skin elasticity. Erosions of the nasal and buccal mucosa were occasionally seen. In the majority of cases the central nervous system was involved, as evidenced by symptoms of paralysis and opisthotonus.

MATERIALS AND METHODS

Five calves, aged two to three months, were autopsied; two calves were destroyed while *in extremis*. These two calves and one other had been treated with broad spectrum antibiotics at high levels. Portions of lung, liver, spleen and brain were collected in sterile containers for bacteriological examination.

Specimens for histopathological examination were fixed in 10% formalin, blocks were cut, embedded in paraffin wax, sectioned at 3μ thickness with a sliding microtome and stained with haematoxylin and eosin, and by Gram's method in some cases. Gomori's methanimine silver method[4] was employed as a fungal stain on sections of the rumen, lung and intestinal tract in all the cases.

RESULTS

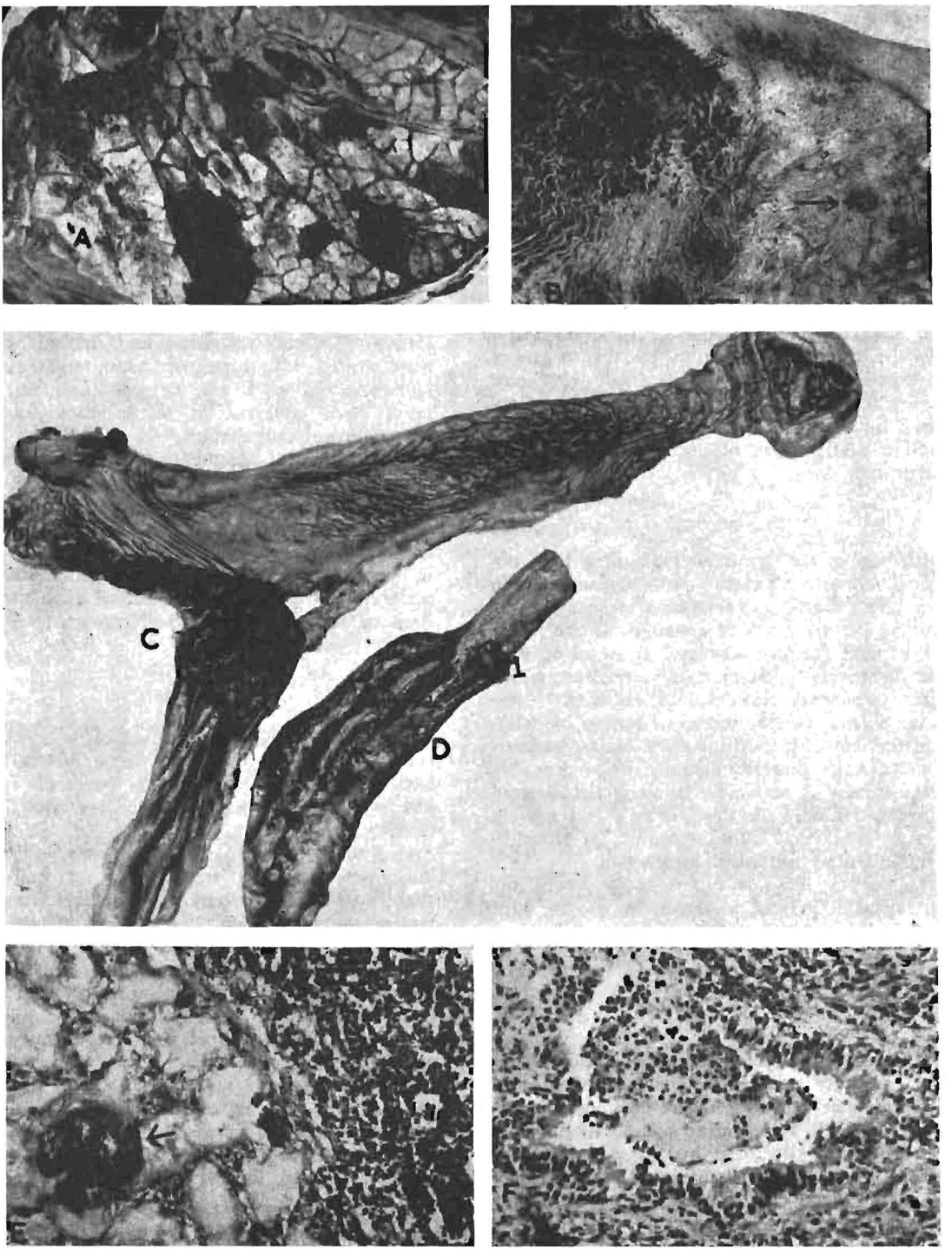
Macroscopic lesions. In all five cases the lungs, rumen and large intestine were consistently affected. There was considerable variation in the distribution and severity of lesions in treated and untreated cases.

One or a number of adjacent lobules in the lung were necrotic, reddish-grey to dull-grey, slightly sunken and sharply demarcated from the rest of the tissue (Fig. 1 A). These lesions were disseminated in all the lobes and in both lungs and on section were dry and firm. Interlobular septa in most cases were prominently distended by oedema. In two cases fibrinous adhesions between the pneumonic foci and the parietal pleura were present.

A focal necrotic rumenitis was seen regularly in the lower portions of the ventral sac and occasionally in other areas of the rumen. Sharply demarcated areas of necrosis, from one half to two cm in diameter, were raised above the surface.

*Section Pathology, Onderstepoort.
**Section Bacteriology, Onderstepoort.
***Private Practitioner, Koppies, O.F.S.

Figure 1.



The papillae on these foci were thickened, velvety and dull-grey. (Fig. 1B). On section the dry appearance, clear demarcation and, occasional, extension through the entire wall of the rumen, were characteristic. Confluence of these necrotic areas at times resulted in the formation of pseudomembranes, detached fragments of which were mixed with the ingesta. Viewed from the serosal surface these focal lesions were dark, reddish-grey and surrounded by subserosal haemorrhages.

The blind end of the caecum and the ileocaecal junction were other favourite sites of clearly demarcated necrosis. The ileocaecal orifice was often observed to have undergone necrosis (Fig. 1 C). Focal, coagulative necrosis of the ileum, extending from the mucosa to the serosa, was frequently found, resulting in fibrinous adhesions between loops of small intestine. Extensive necrosis of Peyer's patches was a striking feature in some cases (Fig. 1D).

In the spleen, firm pea-sized nodules were visible and palpable through its capsule. On section these lesions were well circumscribed, reddish-grey foci of necrosis. Swelling of this organ seldom occurred.

The brain was noticeably flaccid, the meninges tense and the sulci of the cerebral hemispheres widened and filled by clear oedematous fluid. In one case an area of encephalomalacia and haemorrhage was present in the frontal pole of the right cerebral hemisphere, immediately adjacent to the olfactory bulb. Thrombosis of meningeal vessels was in evidence over this area. Notable in this animal were several necrotic foci in the mucosa of the dorsal concha and septum nasi. In addition, there was coagulative necrosis of the tonsillar sinus and tonsil and a marked necrotic gingivitis of both the upper and lower molar regions, separated by a sharp hyperaemic line from the healthy oral mucosa.

Consistent general features were marked dehydration of internal organs, musculature and the subcutis and petechial to ecchymotic submucosal and subserosal haemorrhages.

Histopathology. A focal fibrino-necrotic pneumonia, affecting alveolar walls, bronchioli and bloodvessels and bordered by a zone of leucocytes, was the basic pulmonary lesion. The leucocytes were chiefly round cells, with macrophages and small lymphocytes in the majority (Fig. 1E). Alveoli and bronchioli in these pneumonic foci were plugged to varying degree by a mixture of fibrin, leucocytes and cellular debris (Figs. 1F and 2E). Markedly widened interlobular septa contained loosely arranged strands of fibrin in a pale staining serous ground substance. Not infrequently thrombosis of interlobular vessels was present (Fig. 2C).

The rumen showed coagulative necrosis, some times confined to the mucosa (Fig. 2A), but usually extending into and through the muscular layers. In early lesions the cellular reaction on the margin of the necrosis as well as between the fibres of the muscular layers, was dominated by round cells, particularly macrophages, whereas neutrophils were more abundant during later stages. Thrombosis of submucosal and subserosal vessels occurred in several cases. In one case large, branching, nonseptate fungal hyphae were observed in all the layers of the necrotic ruminal wall (Fig. 2B), as well as in the sub-mucosal thrombi. According to their morphology in histological sections these fungi were assumed to belong to one of the genera *Mucor, Rhizopus* or *Absidia.* As these fungi cannot be differentiated histologically, lesions in which they are present in this report are referred to as mucormycosis.

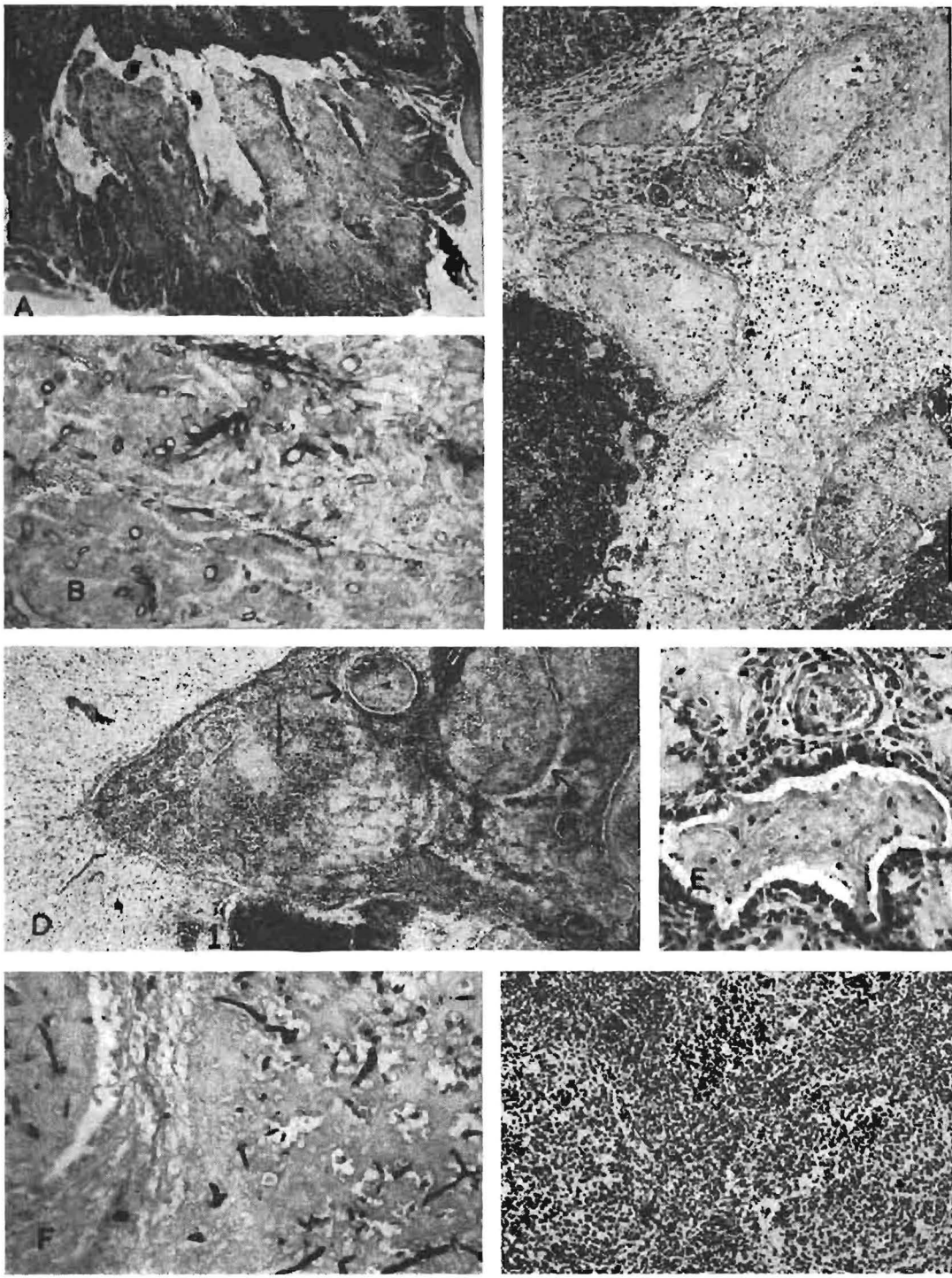
The coagulative, focal necrosis in the caecum and small intestine histologically closely resembled that observed in the rumen. In one protracted case there was a focal ulcerative typhlitis and colitis.

The early lesion in the spleen was characterised by well-circumscribed foci of necrosis in and immediately surrounding the Malpighian corpuscles (Fig. 2G). In advanced, untreated cases, through confluence of these foci, large areas of coagulative necrosis were sharply demarcated by a leucocytic zone from the rest of the splenic tissue. Here, Malpighian corpuscles were small and depleted of lymphoid elements and the trabeculae prominent. Thrombosed vessels in and immediately adjacent to the necrotic areas were occasionally seen.

In the brain, excessively widened perivascular and perineuronal spaces (Fig. 3A and B) suggested the possible presence of oedema and imparted a moth-eaten appearance to certain areas in the brain, particularly the cerebral hemispheres. A striking feature was the presence in all arterial vessels, from medium-sized arteries to capillaries, of intensely staining, basophilic corpuscles, proteinaceous in nature and of various sizes and shapes (Fig. 3A and B). This material, also present in the bloodvessels of other organs and particularly noticeable in the myocardium, adrenal gland and liver, appeared to be more abundant in the cerebral capillaries. These, in many instances, appeared to be entirely filled by sausage-shaped aggregations of the proteinaceous globules (Fig. 3B).

Histological examination of the encephalomalaceous area near the olfactory bulb of one case, already mentioned in the previous section, showed

Figure 2.



haemorrhages, necrosis, perivascular infiltration by predominantly round cells, and extensive thrombosis of submeningeal vessels (Fig. 2D). Gitter cells were in abundance. Numerous mucormycotic hyphae were demonstrable in the thrombi (Fig. 2F), in the walls of thrombosed vessels and in the encephalomalacic brain substance.

In the case from which *Actinobacillus equuli* was isolated, numerous gram negative organisms were seen in the necrotic and peri-necrotic tissue in all organs with microscopic lesions. These organisms were present in small clumps, (Fig. 3C) or singly and in large cloudlike colonies, (Fig. 1E), particularly in bloodvessels. They were extremely pleomorphic and varied from ringlike and granular structures to bi-polar, coccoid bacilli and short, slightly bent clubs. Organisms resembling these were demonstrable in some of the organs of the other cases, but due to their marked pleomorphism, no deductions as to their identity with the organism isolated could be made.

Bacteriological results. A. equuli was isolated in pure culture from the liver, spleen, lungs and brain of one calf autopsied four hours after death. This organism was identified according to its biochemical and biological characteristics[5]. *Pseudomonas aeruginosa* was recovered from the lung, spleen and rumen of another and the cultures from a third calf were overgrown by *Proteus vulgaris*. No bacterial growth could be obtained from the organs of the remaining two calves. It is noteworthy that both these calves, and the one whose cultures were overgrown by *P. vulgaris,* had been treated.

Experimental Transmission (i.v.)

In an initial attempt at artificial transmission, three Afrikaner-cross calves, 2 to 3 months old, received respectively 4, 7 and 10 ml of a blood agar culture of the *A. equuli* isolated from the one case, suspended in saline and the density adjusted to correspond to Brown's opacity tube No. 4. The calves showed a moderate rise in temperature over two to four days, a serous to muco-catarrhal nasal discharge and slightly bloodstained, mucous-covered faeces of decreased consistency. All three calves made an uneventful recovery.

A fourth calf, into which 6 ml of the same culture of a Brown's 10 density was injected, developed a profuse watery diarrhoea within 12 hours after the injection and died 20 hours after the administration of the culture.

Post-mortem examination within two hours of death revealed severe oedema and congestion of the lungs as the cause of death. In addition severe hydrothorax, oedema of bronchial, mediastinal and mesenteric lymphnodes and superficial necrosis of the mucosa in the blind end of the caecum was noted.

Histological changes of significance were marked widening of interlobular septa in the lung by oedema (Fig. 3D), engorgement of alveolar capillaries and thickening of alveolar walls by a fibrinoid substance. Alveoli were filled by an oedematous substance centrally and fibrinoid strands peripherally (Fig. 3 E). In the spleen, circumscribed foci in the Malpighian corpuscles or adjacent interfollicular tissue showed karyorrhexis of lymphocytes and macrophages (Fig. 3 F). Karyorrhexis was also present in the lymphoid follicles at the ileo-coecal junction where patchy necrosis of the superficial parts of the ileal mucosa was also in evidence. Similar karyorrhectic changes were observed in the mesenteric lymph nodes. Finally, necrosis of the mucosa of blind end of the caecum, varying in depth from the epithelium to the muscularis mucosa, was present (Fig. 3 G).

Bacteriologically an *Actinobacillus* sp. and *Streptococcus bovis* was isolated from the lungs and *Streptococcus durans* from the liver and spleen of this calf.

Three months later, five additional calves were infected artificially with the same culture of a Brown's 20 density by intravenous, sub-cutaneous and oral routes, but without success. When further attempts at experimental reproduction were planned three months later, the culture could not be reactivated from the frozen state.

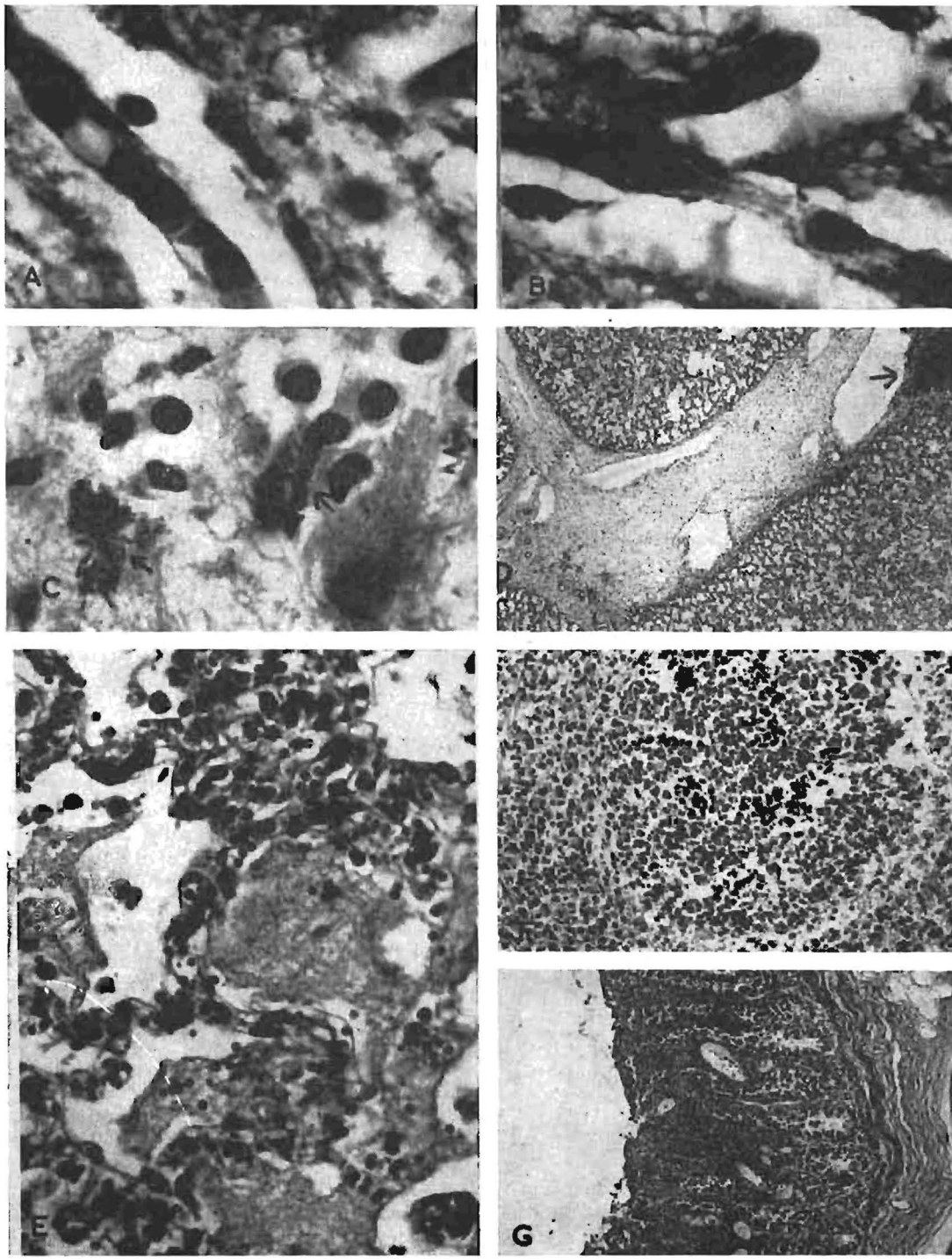
Discussion

A significant feature of both the macro- and microscopic pathology in these cases was the consistency in the character of the lesions, the organs affected and the distribution of the lesions in the organs. Well-circumscribed areas of necrosis were regularly present in the lungs, rumen and the ileo-caecal region of the intestinal tract. Although the lesions were less pronounced in the treated cases, the findings indicated a common aetiologic agent.

The focal, disseminate distribution of necrosis, the leucocytic cellular reaction bordering the necrotic areas and the submucous and subserosal haemorrhages point to an infectious aetiologic agent in these cases. Another histological finding in support of this indication was the frequent thrombosis of vessels encountered in and adjacent to the necrotic foci.

Because *A. equuli* was isolated in pure culture from one untreated calf, the possibility that it was the causal agent in this syndrome was considered

Figure 3.



and subsequently investigated. However, the justification to associate the lesions observed with this organism is questioned by the difficulty and limited success with which artificial transmission could be effected and by the fact that it was isolated from only one out of five natural cases. Although Blakemore[2] successfully reproduced a broncho-pneumonia by intranasal injection of early cultures of an organism which closely resembled *A. actinoides,* he found that the organism had lost its pathogenicity after further subcultivation and suggested that such a loss of pathogenicity may be the result of some modification of the organism when grown under artificial conditions. Smith[1] was unable to produce any lesions by intravenous injection of cultures of *A. actinoides* into two animals, but was successful in eliciting small necrotic foci in the lungs of three out of five calves by the intratracheal injection of the same culture. These experiences point to the difficulty with which infections by *Actinobacillus* species are reproduced. In this study transmission via the intranasal or intratracheal route was not attempted.

The resemblance of the histopathology of the lung, spleen and intestine in the one case produced experimentally to that encountered in the natural cases, produces some evidence in favour of *A. equuli* as the causal agent in these mortalities.

Additional evidence of limited value pointing to *A. equuli* as the aetiologic agent in these cases are certain features of resemblance to infection by this organism in the new-born foal[6]. They are the septicaemic nature of the disease, the distinctly necrotizing effect of the causal agent and its affinity for the lung. In this organ the well-demarcated focal necrosis in both animal species is characteristic.

Possible reasons why *A. equuli* was isolated from one out of five cases only are firstly, that three of the four cases from which this organism could not be cultured had been submitted to antibiotic treatment. Secondly, it is known that cultivation of *Actinobacillus* species from infected tissue is carried out with difficulty, as they require enriched media and do not grow in the absence of serum or blood[1,2].

The intravascular proteinaceous corpuscles observed and described in various organs, and particularly the brain capillaries, may be explained by the haemoconcentration which presumably resulted from the dehydration. This in turn was chiefly caused by the diarrhoea. Such a haemoconcentration and a concurrent relative hyperproteinaemia may have resulted in concentration and aggregation into globules of proteinaceous substances. Alternately, the aggregations observed histologically may only have been formed post-mortally through coagulation of a protein-rich serum by the formalin fixation.

Although it would be mere speculation to relate the plugging of cerebral capillaries by these proteinaceous aggregations to the oedema of the brain and the consequent nervous symptoms, the possibility should be mentioned. Admittedly, the mechanism for the control of the water balance in the body in the case of dehydration would tend to withdraw fluid from the tissues into the blood and thus counteract an oedema of the brain, but the possibility that the proteinaceous plugs may in these cases have interfered with the nutrition and permeability of the capillaries, should be borne in mind. In addition, capillary permeability may have been enhanced by the action of the causal agent or a toxin produced by it.

The presence of the fungal mycelia, presumably mucormycosis, in the rumen of one and the brain of another case, is regarded as a secondary infection, facilitated by an initial necrotic lesion of the ruminal and nasal mucous membranes, possibly due to the actinobacillosis in these cases. The route of infection in the case of the mycotic encephalitis was presumably through the nasal cavity. It was noteworthy in this case that no fungi could be demonstrated at the sites of the necrotic rumenitis. In domestic animals mucormycotic encephalitis has only once before been recorded and that in a dog[7].

The necessity in this study to ascertain the role played by mucormycosis was realised, but the GMS fungus stain employed on sections of the rumen, lung and intestinal tract produced evidence of this infection only in the two organs cited.

The possible role of a primary viral or related infectious agent not detectable by histological examination was not investigated in these cases. Specific attention should be paid to such a possibility in future cases.

ACKNOWLEDGEMENTS

The authors wish to thank the Chief, Veterinary Research Institute, for his permission to publish this article. Appreciation is expressed to Mr. A. M. du Bruyn and his staff for the photographs and to the technical staff in the Department of Pathology for the preparation of the sections.

REFERENCES

1. SMITH, T. 1921. *J. Exp. Med.*, 33: 441.
2. BLAKEMORE, T. 1945. *J. comp. Path.*, 55: 132.
3. JUBB, K. V. F. and KENNEDY, P. C. 1963. *Pathology of domestic Animals*, Vol. 1, p. 168. *New York and London.* Academic Press.
4. GROCOTT, R. G. 1955. *Am. J. clin. Path.* 25: 975.
5. BREED, R. S., MURRAY, E. G. D. and SMITH, N. R. *Bergey's manual of determinative bacteriology.* 7th ed. London. Bailliére, Tindall and Cassel.
6. DU PLESSIS, J. L. 1963. *Jl. S. Afr. vet. med. Ass.* 34: 25.
7. GLEISER, C. A. 1953. J. Am. vet. med. Ass. 123: 441.

BOOK REVIEW

MYXOMATOSIS

FENNER, F. and RATCLIFFE, F. N. Cambridge University Press, London. 1965. 379 pages, 15 plates, 30 figures, 59 tables.

This most excellent book, is divided into 18 chapters, and covers a tremendously wide field on the subject of myxomatosis, as indicated by the title. In fact this book serves as a model how to study and present all the aspects of an infectious agent. The book describes the history and distribution of myxomatosis, the introduction of the European rabbit (*Oryctolagus cuniculus*) into Australia, its colonizing spread and its eventual economic importance as an agricultural pest. Rabbit biology and behaviour are described in detail. Three further chapters are devoted to the classification, structure and properties of myxoma virus, interactions between myxoma virus and the host cell and the host range of myxoma and fibroma viruses. The disease in the European rabbit is described from the point of view of pathogenesis, histopathology, immunilogical response and effects of environmental factors.

The last eight chapters deal with the mechanisms of transmission of myxomatosis, field observations on transmission in Australia, genetic changes of myxoma virus and of the European rabbit, and the occurrence of disease in the Americas, Australia and Europe. In conclusion there is an outstanding chapter in which the observations described in the previous chapters are collated and integrated and the continuing evolution of myxomatosis is speculated on.

It is generally accepted that infectious diseases must have played an important part in the evolution of animals and plants. Occasions to study this process in detail are extremely rare, but fortunately the opportunities for study offered by the outbreaks of myxomatosis among rabbits in Australia in 1950 and in Europe in 1952 were immediately recognised, in particular by the Australian scientists.

According to the authors, myxoma virus has apparently, through ages of contact with American rabbits (*Sylvilagus*), attained the ideal host-parasite relationship. This is characterised by the production of localised skin tumours from which virus can be mechanically transmitted by biting arthropods for long periods. This virus, however, causes a very severe, generalized disease with almost 100 per cent mortality in European rabbits. The introduction of this highly lethal virus into vast populations of highly susceptible European rabbits produced dramatic results. The initial high mortality rate served as a strict selective factor and soon the average survival time and survival percentage of rabbits increased, partly due to enhanced genetic resistance to myxoma virus. Other factors, such as vector transmission also markedly influenced the evolution of viral virulence. Mosquito transmission favoured the spread of less virulent or attenuated strains, whereas fleas favoured transmission of highly virulent or lethal strains. This great experiment in nature which was observed and recorded by the authors proved beyond doubt that an infectious agent could exercise a profound selective influence upon a given population.

This comprehensive book is written in a lucid style and the text is supplemented by numerous photographs (some in colour), graphs, maps and figures, as well as a most complete bibliography and citation index. It should be of great value to ecologists, virologists, parasitologists, zoologists, geneticists and public health workers, but particularly to those interested in the intricate interaction between parasite and host and the resultant genetic changes which might occur in both.

Those interested in certain aspects of myxomatosis only, can read the book selectively and will still maintain the thread of the account for most chapters are followed by a clear and concise summary.

B. J. E.

THE EPIDEMIOLOGICAL PATTERN OF VIRAL, PROTOPHYTAL AND PROTOZOAL ZOONOSES IN RELATION TO GAME PRESERVATION IN SOUTH AFRICA.

by W. O. Neitz,

Veterinary Research Institute, Onderstepoort.

Introduction

The importance of wild animals serving as reservoirs of diseases, transmissible to domestic animals and man, is well known to all engaged in the study and control of these infections, commonly referred to as zoonoses. It seems that the first account of such occurrence in South Africa appeared about 200 years ago. Thunberg in his "Travels" refers to an outbreak of rabies in 1772 and Theophilus Shepstone to another one in 1828[1]. Further records are those of Cumming[2], who described bovine malignant catarrhal fever (snotsiekte) in his oxen which grazed in close contact with gnus (black wildebeest) in the Orange Free State, and of Livingstone, who drew attention to a fatal disease in man and animals, now known to have been anthrax[3], on the border of the Western Transvaal. Although nagana was also known to be a zoonosis by the indigenous people and early European pioneers, systematic studies on the host-vector cycle were first undertaken by Bruce[4] at the request of the governor of Natal. At his field laboratory in Zululand he demonstrated one of the causal agents, *Trypanosoma brucei* Plimmer and Bradford, 1899, and that tsetse flies were responsible for the transmission. This important achievement was followed by fruitful investigations which disclosed the existence of other zoonoses in various parts of Africa.

The available records on the zoonoses harboured by wild animals have been assembled and published by Thomas and Neitz[5], Martinaglia[6], Lobry[7] and Neitz[8]. From the references cited by these writers it will be seen that the investigations have been a combined effort of veterinary and medical biologists and zoologists. Consideration of the observations, described in more than 200 publications, makes it apparent that great advances have been made in recognizing the characteristic features of a large number of diseases transmissible to man and his domestic stock. From epidemiological studies it has become evident that pathogens which are spread by contact such as rabies, rinderpest, foot and mouth disease, African swine fever, anthrax, tuberculosis, etc., can be expected to establish themselves wherever susceptible animals occur. Those which require arthropod vectors (mosquitoes, midges, fleas and ticks) for their transmission (horsesickness, Rift Valley fever, nagana, East Coast fever, bubonic plague, heartwater, etc.) are restricted to certain regions, and are popularly referred to as diseases of place. Their incidence is seasonal, and is dependent upon the climatic conditions obtaining within endemic regions. Rain and water conservation promote the development of arthropod vectors. The dissemination of pathogens to other regions is thus dependent upon the presence of potential vectors and susceptible vertebrate hosts. This has been observed by the behaviour of East Coast fever and bubonic plague after their introduction into South Africa at the beginning of this century.

It is also interesting that the epidemiological pattern of a disease may become modified, when it is introduced into a potential endemic region, depending upon the availability of vectors and susceptible animals. In regions where wild fauna formerly served as reservoirs of pathogens and as hosts for blood sucking arthropods, domestic animals have to a very large extent taken their place concomitantly with the dwindling to a very low level of the large type of wild animal. The area of the Republic of South Africa is 472,359 square miles, and that of South West Africa 317,887 square miles. The total area of game reserves in both territories is approximately 55,000 square miles, which exceeds that of the Orange Free State. The total human population in the two territories is approximately 18 million, and that of domestic animals 13 million head of cattle, 36 million sheep, 5 million goats, 600,000 solipeds, 700,000 pigs, 1 million dogs and about 20 million birds (fowls, turkeys, ducks, etc.). No figures are available for the number of wild animals. Consideration of the very much smaller area reserved for game — particularly with regard to carrying capacity for herbivorous animals — makes it clear that their number, with the exception of that of wild rodents and birds, is very much smaller than

that of domestic animals. Consequently the epidemiological pattern of the zoonoses has become markedly modified as compared to that many decades ago, when game was very prevalent in Southern Africa. Despite this new host system, the danger of zoonoses has not been reduced. The application of prophylactic measures still remains an important function of the veterinary and medical professions.

A modification in the epidemiological pattern is not only seen when wild animal disease carriers are replaced by domestic stock. A similar phenomenon can also occur when the role of one group of wild animals is taken over by another one. Bubonic plague, which is flea-borne, is an excellent example. This disease was introduced by infected rats at several seaports in South Africa at the beginning of this century. Rodents mainly concerned in the spread of this disease in the coastal towns were the brown and black rats. The latter species carried by rail laden with forage and other materials, continued its role in the inland centres[9]. A new chapter in the history of plague was opened when this disease was established in veld rodents in the Tarkastad district (Eastern Cape Province). The disease had shifted from the urban to rural districts[10]. Human plague outbreaks that occurred since 1914 became localized almost entirely to farms and native areas in the rural regions, while the urban areas remained almost free from infection[9]. Subsequent investigations revealed that numerous wild rodent species served as the source of infection, and that a large number of flea species were involved in the transmission of plague to rodents and man. Evidence was also brought forward that transmission followed cannibalism. Additional interesting observations showed that in urban areas domesticated carnivores (cat, ferret and dog) were sometimes affected, while in rural regions wild carnivores (suricate and yellow mongoose) became victims of the disease. Readers interested in the epidemiology of plague in South Africa are referred to the very instructive publication by De Meillon, Davis and Hardy[11]. For obscure reasons no cases of plague have been reported in man and rodents during recent years[12].

The discussion on the modification of epidemiological patterns of diseases after their introduction into a new environment, naturally raises the question about their origin. Field surveys, supported by laboratory investigations, have shown that many diseases of place, such as horsesickness, bluetongue of sheep, Rift Valley fever, nagana, Corridor disease and heartwater, are true African diseases, although not all of them, e.g. plague, murine typhus, anaplasmosis and benign bovine theileriosis. By means of their respective vectors, they can all maintain themselves in wild hosts in the complete absence of man and/or domestic animals. With the exception of foot and mouth disease caused by African strains (SAT_1, SAT_2, SAT_3), African swine fever and rabies due to the viverrid strain, some of the diseases spread by contact (rinderpest, tuberculosis, anthrax and European strain of rabies), are of exotic origin, while the primary source of several other infections with a ubiquitous distribution (rat-bite fever, listeriosis, necrobacillosis, toxoplasmosis, etc.) is obscure. Their introduction into South Africa could have been brought about by the indigenous people with their cattle, sheep and goats after their migration through the central African disease barrier many centuries ago or by Europeans who started to import domestic animals 300 years ago. Domestic solipeds and pigs never traversed the disease barrier. It is believed that they became victims of horsesickness and African swine fever respectively, as well as nagana[13], upon introduction by European settlers. Solipeds and pigs were imported chiefly from Europe and America. By contrast, there has been no evidence so far that any of their specific pathogens have established themselves in wild equine or porcine hosts.

Knowledge about the origin of pathogens is very important. It makes it possible to gain information about the significance of diseases in their previous habitat, and to what extent available control measures can be applied in a newly created endemic region. Furthermore, it also serves as a warning about risks of other infections likely to be brought in. Veterinary and medical authorities are fully aware of these dangers and apply adequate precautions. Louping ill, a virus disease which occurs in Scotland, is transmitted to sheep, cattle and horses by one of the European ticks, *Ixodes ricinus* (Linn.). Accidental laboratory infections have occurred also in human beings. Alexander and Neitz[14 15] determined that the African brown tick (*Rhipicephalus appendiculatus* Neum.) is capable of transmitting this disease. It is thus obvious that in the event of an accidental introduction into brown tick infested regions, louping ill would constitute an animal and a public health problem. The introduction into South Africa of melioidosis (*Pseudomonas pseudomallei* (Whitmore, 1913) infection), harboured by rodents and man in India and Malaya, was interrupted by a timely diagnosis, isolation and treatment of a patient, who had been on active service in these countries[16]. Transmission experiments revealed that several indigenous rodents (gerbille, multimammate mouse, Cape striped mouse, white-tailed and vlei rats) are susceptible[17]. This inter-

vention prevented the possible spread of this disease into a potential endemic region.

Necessary precautions are being taken in South Africa to prevent the introduction of diseases in order to safeguard the health of man and domestic and wild animals. This is even more important at present than in the past when one considers how rapidly disease carriers can be transferred by air from one continent to another. Regulations governing the importation of animals are not only directed against internal pathogens (bacteria, protozoa and viruses) but also against endoparasites (helminths) and ectoparasites (ticks, mites and other arthropods).

The various zoonoses harboured by wild animals are produced by viruses, Protophyta (bacteria, *Borrelia* spp., *Rickettsia* spp., etc.), Protozoa (*Trypanosoma* spp., *Babesia* spp., *Toxoplasma* spp., etc.), Arthropoda (nasal fly, mites, tampans, ticks, etc.), Platyhelminthes (flukes, tapeworms, *Bilharzia* worms, etc.) and Nemathelminthes (sandworm, wireworm, nodularworm, etc.). In this paper attention will be paid to most of the pathogens belonging to the first three groups mentioned.

A. *Virus Diseases*

Of the 13 viral zoonoses in which wild animals may be involved as carriers all, with the exception of the form of rabies caused by what appears to be a European strain, foot and mouth disease due to the A virus type recorded in South West Africa, canine distemper and rinderpest, are typical African diseases. They are all of domestic animal health importance. Rabies, Rift Valley fever, Wesselsbron and Middelburg virus diseases, foot and mouth disease and Newcastle disease are communicable to human beings. Up to the present there is no evidence that either of the latter two diseases have affected man in South Africa.

Two distinct strains of rabies virus are recognized in South Africa. The indigenous viverrid strain occurs on the Highveld of Transvaal, the Orange Free State and Western Cape Province. Most of the human victims have been bitten either around or in their homesteads or huts by affected members of the family Viverridae (yellow mongoose, genet cat, etc.). The second strain behaves like the European strain, and is disseminated mainly by dogs and jackals. Its distinct behaviour was first recognized in South West Africa in 1947. From there it spread through Bechuanaland into Zambia and Rhodesia and Transvaal in 1949. It continued its spread eastward and southward through Moçambique into Natal, Eastern and Western Cape Province. Immunization of dogs is practised for its control.

When canine distemper was introduced is unknown. It is widely distributed in dogs, and has been encountered on several occasions in wild members of the family Canidae. Martinaglia[8] reported its presence in the silver jackal in the Johannesburg Zoological Garden, and Hofmeyr[18] observed it also in this species, the long-eared fox and Cape hunting dog in the Pretoria Zoological Garden. Immunization of susceptible wild carnivores, kept in captivity, has proved to be very effective for its control.

African horsesickness was first recorded in the Cape Province in 1719. It occurs enzootically in the summer rainfall areas. Severe epizootics have occurred at irregular intervals during summer. The determination of the susceptibility of the zebra by Rickmann[19] in South West Africa, and the fact that domestic solipeds were not present in Southern Africa prior to their introduction from horsesickness-free countries, permits the conclusion that zebras served as reservoirs. According to Du Toit[20 21], the transmission is effected by midges (*Culicoides* spp.). With the exception of a few records of its periodic occurrence in Yemen (West Coast of Arabia), horsesickness appeared to be confined to Africa. Its spread into Palestine, Syria, Lebanon and Jordan during 1944[22] and subsequently into Iraq, Iran, Turkey, Afghanistan, West Pakistan and India[23], is evidence how widely potential vectors must have been distributed before they served as transmitters. Immunization of solipeds with polyvalent vaccines is an effective prophylactic measure.

Although bluetongue virus may have been harboured by wild ruminants before the indigenous people and their stock reached South Africa, there is sufficient reason to believe that their domestic ruminants acquired infection en route. It is interesting to note that in contradistinction to the European sheep breeds, which are highly susceptible, the indigenous breeds are highly resistant. The demonstration of the susceptibility of the blesbok[24], and the isolation of bluetongue virus from a wild caught Cape striped mouse and a vlei otomys rat by Du Toit and Goosen[25] are evidence that wild animals can serve as reservoirs. As in the case of horsesickness, bluetongue has been responsible for severe epizootics in Merino and other European breeds. Bluetongue has also crossed the African boundary. It has been described from the United States of America, the Iberian Peninsula, Cyprus, Turkey, Syria, Jordan and West Pakistan[26]. Du Toit[20 21] has shown that midges (*Culicoides* spp.) are involved in the transmission. Immunization of European breeds of sheep with polyvalent vaccines is practised on a large scale.

Several outbreaks of foot and mouth disease have occurred in South Africa up to 1903. The disease-free period that followed was interrupted in 1933, when this disease spread from Rhodesia into Transvaal. Investigations, conducted at that time, revealed that not only domestic but also wild ruminants were affected. Since 1933, virus strains from outbreaks which occurred at irregular intermittent intervals until 1964, have been submitted to Pirbright, England, for typing. They were identified as SAT_1, SAT_2, and SAT_3 types, and are distinct from the A, O and C strains commonly found in Europe. It also transpired that during the interepizootic periods the African virus types were maintained by antelopes occurring in the large game reserves of Rhodesia, Bechuanaland, Moçambique and Transvaal. Surveys revealed that 12 antelope species (African buffalo, bluewildebeest, duiker, eland, gemsbok, hartebees, impala, kudu, sable antelope, steenbok, springbok and waterbok) and the warthog showed typical clinical symptoms[27 28 29 30 31 32]. Contact between game and cattle at drinking sites, particularly during periods of drought, resulted in transmission; dissemination then followed along the routes of commerce. Outbreaks have occurred in Transvaal, Northern Natal and North Western Cape Province along the Bechuanaland border. Not only cattle but also sheep, goats and pigs became involved in some of the periodic epizootics. Although the mortality was extremely low, indirect losses due to emaciation, very costly prophylactic measures and trade restrictions resulted in a severe drain on the finances of the country. The erection of a substantial fence around the Kruger National Park has prevented close contact between domestic and wild animals. Since 1961 there has been no spread from this source. The recent outbreaks in 1964 in the South Eastern Transvaal originated from neighbouring territories[33]. Strict quarantine measures together with immunization have proved to be of great value.

Rinderpest was introduced into Transvaal in 1896. It spread right through Southern Africa, and was responsible for a high mortality, not only in cattle but also among antelopes. Conditions prevailing at that time did not permit systematic surveys. Available records have shown that the African buffalo, bushbuck, duiker, eland and kudu were the chief victims. The application of prophylactic measures resulted in its eradication by the end of 1903. There has been no recurrence of rinderpest since then in South Africa.

Bovine malignant catarrhal fever (snotsiekte) has occurred sporadically in cattle. Experimental observations have shown that clinically healthy black and blue wildebeest can serve as reservoirs[34]. In years that followed it was determined that sheep can also be carriers of this malady. Observations at Onderstepoort have shown that two African buffalo heifers, derived from the Hluhluwe Game Reserve (Natal), developed a disease clinically indistinguishable from snotsiekte. Both buffaloes had been kept in close contact with cattle and at some distance away from sheep. The relationship between the bovine and syncerine infections is still obscure.

Rift Valley fever appeared quite unexpectedly in South Africa during the summer of 1950-51[35]. It is a disease of sheep, goats and cattle, causing many abortions and high mortality in pregnant animals and new born lambs. Antelopes have been reported to have died or aborted[36]. In man it is chiefly an occupational disease of a relatively mild nature unless complicated by retinal damage. Kaschula[37] recorded 20,000 human infections following the handling of affected animals and infected meat. Wesselsbron disease, which possesses clinical features resembling those of Rift Valley fever, was recognized as a distinct infection several years later[38]. It also causes abortion in cows. Man is susceptible. Middelburg virus disease was isolated in the Western Cape Province from wild caught *Aedes* spp.[39]. The virus is transmissible to lambs but its importance as a pathogen was masked, at the time of its isolation, by a concurrent outbreak of Wesselsbron disease. Man is susceptible and develops a relatively mild infection.

These three diseases are mosquito-borne (*Aedes* spp.). It appears that they have always been present in South Africa as evidenced by the existence of a silent focus of Rift Valley fever in the Addo Forest in the Cape Province, and that wild animals served as reservoirs for the infection of mosquitoes. This assumption is supported by the fact that in the Semliki Forest at Mengiro in Uganda, Rift Valley fever exists under natural conditions in the complete absence of man and domestic animals. In this locality the virus was isolated from wild caught mosquitoes and from a dead buffalo (*Syncerus* sp.). Soil and water conservation during the last four decades in South Africa undoubtedly promoted the development of mosquitoes. The explosive nature of the epizootics of Rift Valley fever and Wesselsbron disease is an excellent example of what can happen when the wild host-vector cycle is shifted across to a domestic host-vector cycle. Fortunately it has been possible to develop reliable vaccines at Onderstepoort for the protection of domestic ruminants. This prophylactic measure has also reduced both infections as occupational diseases in man handling animals and their products.

African swine fever occurs enzootically in Northern and Eastern Transvaal and South West Africa but not in Natal and Eastern Cape Province despite the presence of wild pigs which serve as reservoirs of the causal agent in enzootic regions. The epidemiology of this disease should be considered from two aspects: — its mute existence in primary foci where wild pig carriers exist, and its fulminating appearance in secondary foci where domestic pigs are being raised. The source of infection has been traced in nearly all instances to wart-hogs. The existence of the infectious agent in wild pigs would probably never have been disclosed, had domestic pig breeding not been started in the primary foci. Wart-hogs and bush pigs from Africa have been introduced on many occasions into zoological gardens in many parts of the world without any complications. Evidence of its occurrence in the Northern Transvaal dates back to 1926, when a large number of domestic pigs died from a highly fatal virus disease which was identified as East African swine fever. Biological tests conducted subsequently on domestic pigs revealed that six out of fourteen wart-hogs and one out of four bush pigs harboured the infectious agent. With exception of a single dead wart-hog all the remaining wild pigs, shot for the survey, appeared perfectly normal[40].

Transmission of African swine fever occurs by contact: the feeding of garbage, contaminated with infected wart-hog offal, to domestic pigs readily results in infection of the latter, as several farmers have experienced after wart-hog hunts, but, remarkably enough, cohabitation between known wart-hog virus carriers and susceptible domestic pigs in the same sty does not do so. Once the disease has established itself in domestic pigs, it spreads from animal to animal by the ingestion of food contaminated with excreta of affected swine. The virus is highly resistant, so that uncontrolled distribution of pork could result in disseminating the disease in remote places. From 1926 to 1961 no less than 142 outbreaks of African swine fever have been recorded: 32 in Transvaal, 88 in Western Cape Province and 22 in South West Africa. Approximately 30,000 domestic pigs were involved, and of these at least 10 percent died before control measures were employed. The slaughter of affected and in contact pigs and the destruction of all carcasses, accompanied by stringent quarantine measures, have resulted in the eradication of the disease in domestic pigs. In enzootic regions maintenance of domestic pigs in paddocks, preferably double fenced, has proved to be a highly efficient prophylactic measure. Since its application in 1956 until the end of 1964 only one outbreak has been recorded. This followed the accidental feeding of contaminated swill. The danger of African swine fever is not only confined to Africa. The unforeseen introduction of infected pork products into Portugal in 1957 resulted in its spread into Spain in 1960, and from thence into France in 1964. The financial losses in Spain have amounted to more than US $10,000,000.

There is no evidence that outbreaks of Newcastle disease of fowls and turkeys have been initiated by wild birds. Laboratory tests have nevertheless shown that the Cape francolin, laughing dove and Cape sparrow are susceptible. Periodic epizootics of this scourge, wellknown to poultry farmers since 1946, have resulted from the introduction of infected fowls and poultry products[41]. Adequate prophylactic measures (quarantine, slaughter of affected and in contact birds and immunization of healthy birds on adjacent premises) have been applied with satisfactory results.

Tern virus infection was identified for the first time during April and May of 1961 by Becker and Uys[42]. It has been responsible for the death of many hundreds of common terns (*Sterna hirundo* Linn.) along the coast line of the Cape Province. There is no evidence that fowls became affected, even though this species is susceptible according to laboratory tests.

B. *Protophytal Diseases.*

In contradistinction to the virus infections, of which at least nine are true African diseases, only one of the 25 protophytal zoonoses, namely heartwater of ruminants, is a true indigenous disease. Relapsing fever of man, caused by *Borrelia duttoni* (Novy and Knapp, 1906) need not necessarily be an indigenous disease even though the vector the eyeless tampan (*Ornithodoros moubata* (Murray)), is confined to Africa. This statement is based upon the fact that the vector specificity has not yet been determined satisfactorily, so that the possibility exists that one or other *Borrelia* sp., introduced by a carrier from Europe or Asia, could have established itself in the eyeless tampan. It serves as an excellent reservoir in that the pathogen is transmitted hereditarily to the ensuing generation. It appears that tuberculosis and anthrax are exotic diseases while the remaining 21 infections have a ubiquitous distribution.

The protophytal zoonoses are caused either by members of the class Schizomycetes (bacteria, bacilli, *Borrelia* spp. etc.) or by those of the class Microtatobiotes (*Rickettsia* spp., *Cowdria* sp., *Coxiella* sp., *Anaplasma* spp., etc.). Their role as pathogens is given below.

(a) *Schizomycetes.*

Spirillum minus Carter, 1899 is responsible for one type of human rat-bite fever. This pathogen has been isolated from a black rat in Transvaal but up to the present has not been responsible for a public health problem in South Africa. A second form of rat-bite fever is caused by *Streptobacillus moniliformis* (Levaditi *et al.*, 1925). Its local occurrence was revealed when a human being developed an infection after having been bitten while handling a yellow-footed squirrel, derived from Transvaal, immediately after its arrival at the harbour of Hamburg in Germany[43]. Nothing is known about its prevalence in South Africa. It is nevertheless strongly recommended that, in the event of being bitten by rodents, the victims should seek medical advice.

Salmonella spp. have a world-wide distribution, and are responsible for a variety of diseases, such as food poisoning and enteric fever in man, paratyphoid in calves, abortion in mares and ewes and fowl typhoid. Their occurrence in wild mammals and birds has also been recorded in a few instances.

Salmonella typhimurium (Loeffler, 1907) has a wide host range in South Africa. It has been responsible for food poisoning in man. Fatal infections have been recorded in naturally infected calves, chickens, ducklings, adult pigeons and squabs, canaries and wild finches[44]. Recently Cameron, Tustin and Meeser[45] have determined that this pathogen produced mortality in several blue wildebeest calves in the Kruger National Park.

Salmonella braenderup Kauffmann and Juel Henningsen, 1937 has been responsible for food poisoning in human beings after they had a meal in a restaurant. Investigations revealed that a black rat, caught on the premises, harboured this contagion[46].

Brucelloses in man and domestic animals are caused by several *Brucella* spp. The infection in man, of caprine origin, is commonly referred to as Malta fever and that of bovine origin as undulant fever. In cattle the disease is referred to as contagious abortion, in ewes as ovine abortion and in rams as infectious infertility. In a review of the world literature, Rementsova[47] assembled data which show that among wild animals, 24 species (rodents, ungulates, carnivores, insectivores and birds) have been proved to be carriers of *Brucella* spp. commonly encountered in man and domestic animals. These organisms have also been isolated from several ixodid ticks in which they persist for periods of up to 150 days, and in argasid ticks in which they survived for 2 years. At present there is no evidence that the fauna and ectoparasites harbour *Brucella* spp. in South Africa, even though they are widely distributed in domestic animals. A local survey in future may yet reveal their presence in game.

Necrobacillosis is a common infection of the respiratory and alimentary tracts, claws or hoofs as well as the internal organs of cattle, sheep, pigs and solipeds. It is commonly encountered under unhygienic conditions in stables or even on pastures, but it may also manifest itself where the sanitation is beyond reproach. The contagion *Sphaerophorus necrophorus* (Flügge, 1886) has been encountered in a duiker, steenbok, lechwe and red brocket held in the Johannesburg Zoological Garden[6]. Field infections have been encountered in a duiker in Natal[5], and in a black wildebeest from the Summerville Game Reserve in the Orange Free State. The disease can be cured readily with sulphonamides and antibiotics.

Listeriosis, also known as "Tiger River disease" is caused by *Listeria monocytogenes* (Murray *et al.*, 1926). This disease has been recorded for the first time by Pirie[48] when it appeared as an epizootic in Lobengula's gerbilles along the Tiger river in the Orange Free State. Epizootics have also occurred subsequently in this rodent species in Transvaal and Natal. Laboratory tests have shown that at least eleven other indigenous rodent species are susceptible[48 49]. Sporadic outbreaks in chinchillas have also been recorded recently in Transvaal and Eastern and Western Cape Province[50]. There is no evidence of its occurrence in other species of animals in South Africa even though it is known that in Europe man, domestic animals and birds are susceptible.

Anthrax is widely distributed in South Africa. It has been responsible for serious losses in domestic ruminants, solipeds and swine but only to a limited extent in ostriches reared in the Eastern Cape Province. Man has been affected quite frequently after handling infected carcasses, hides and wool. Sporadic outbreaks in the zebra, hartebeest, springbok, blackwildebeest and kudu have been recorded up to 1943. During the period from September, 1950 to October, 1960 more than 1100 fatal cases have been diagnosed from various areas in the Kruger National Park in the Eastern Transvaal by Pienaar[51 52]. Animals involved were a baboon, wild carnivores, elephant, hippopotamus, wild pigs, fourteen antelope species and a vulture. The highest incidence was in kudus of which 837 cases were proved to have been victims of anthrax.

Human tuberculosis is a rather common disease and under normal conditions man is the source of infection. *Mycobacterium tuberculosis* Zopf, 1883 has been diagnosed in a dog[53] and domestic pigs[54] and on one occasion in a wild animal, the giraffe[55].

A natural infection has also been diagnosed in a parrot[56].

Mycobacterium bovis Bergey *et al.*, 1934 is a common infection in cattle, and is a serious animal health problem. In man approximately 30 cases of bovine tuberculosis have been diagnosed[57 58]. Natural infections have been encountered in a goat[59], a large number of pigs[54 60] a duiker and many kudus in their natural habitat in the Eastern Cape Province[62] and in a few springboks maintained in a zoological garden[18 61].

Fowl cholera, due to *Pasteurella avisepticum* (= *Pasteurella multocida* (Lehmann and Neumann, 1899)), has occurred at irregular intervals since its first appearance in the Eastern Cape Province in 1909. The source of infection for the sporadic outbreaks, which occurred mainly along the coastal regions of Natal and Eastern Cape Province, was a puzzle. During the last epizootic in the Western Cape Province in 1948, it was found that seagulls (*Larus dominicanus* Lichtenheld) can serve as carriers of the contagion. From this observation it was deduced that this bird species was, in all probability, responsible for the previous introductions[63].

Relapsing fever of man, due to *Borrelia duttoni*, and its vector, the eyeless tampan, are fairly widely distributed in South Africa. Up to the present, natural infections have been encountered only in man. Zumpt[64], who determined that the multimammate mouse is susceptible, suggests that this and possibly other indigenous rodent species may serve as a source of infection in nature. The destruction of tampans in human habitations has reduced the incidence in man.

Borrelia anserina (Sakhareff, 1891) is responsible for a highly fatal disease of fowls, ducks and geese. If is often referred to as fowl spirochaetosis. The vector is the fowl tampan (*Argas persicus* Oken). Coles[65] described a *Borrelia* sp. in the Jackass penguin on Dassen Island where the penguin tampan (*Argas talaje capensis* (Neum.)) appears to be the vector. The relationship between the fowl and penguin parasites still needs to be determined. The incidence of this disease in poultry has been reduced considerably since synthetic acaricides became available.

A benign form of borreliasis, caused by *Borrelia theileri* (Laveran, 1903), is a common infection of domestic solipeds and ruminants in regions where the blue tick (*Boophilus decoloratus* (Koch)) and red-legged tick (*Rhipicephalus evertsi* Neum.) occur. It may sometimes cause alarming symptoms of a temporary nature in horses and cattle. The blesbok has been found to be susceptible[66]. It is thus possible that game may serve as reservoirs in nature.

(b) *Microtatobiotes.*

Murine typhus, which is harboured in nature by black and brown rats, is caused by *Rickettsia typhi* Wolbach and Todd, 1923 and transmitted by the rat flea (*Xenopsylla cheopis* (Rotsch.)). It has a world-wide distribution. The permanent close association between the mammalian host and arthropod vector is the reason for its ubiquitous dissemination. Man contracts the disease when exposed to infection in rat-infested premises.

Tick-bite fever, frequently contracted by man in tick-infested regions of South Africa, is caused by *Rickettsia conorii* Brumpt, 1932, It has been determined that the black rat, Cape striped mouse and vlei rat are natural reservoirs[67]. The dog is susceptible and therefore, can be included in the host-vector cycle. Transmission to man is effected by seed ticks (larvae) belonging to the genera *Amblyomma, Boophilus, Haemophysalis, Hyalomma* and *Rhipicephalus*. The infectious agent is transmitted hereditarily from one tick generation to another.

Studies on the pathogenicity have shown that murine typhus and tick-bite fever can be transmitted to gerbilles (*Tatera* spp.). These rodents are being reared in captivity and serve as excellent laboratory animals[68]. They have also been used for vaccine production in the past at the South African Institute for Medical Research, Johannesburg[69]. Tetracyclines are effective remedies.

Q-fever has a world wide distribution, and is produced by *Coxiella burnetii* (Derrick, 1939). It is tick-borne but the vectors in South Africa have not yet been determined. Experiments in Australia and the United States of America have shown that transmission results not only from tick bites but also by inhaling dust and drinking milk contaminated by tick excreta which contain the highly resistant infectious agent. Affected cows may also pass this agent into milk. *C. burnetii* can remain viable in tick faeces for periods of up to 1000 days. Man and domestic animals are susceptible. Local observations have shown that cattle, black rats, Cape striped mice and vlei rats can serve as reservoirs[70 71]. In man Q-fever is usually an occupational disease and occurs in people handling stock.

Malignant canine rickettsiosis was first diagnosed in several privately owned dogs suffering from a highly fatal disease in the Kruger National Park in 1938. The conspicuous reduction in the number of Cape hunting dogs in this park during previous years has been attributed to this malady. This assumption is supported by a fatal case of rickettsiosis in a black-backed jackal which followed the exposure to ticks in a kennel at the Onderstepoort Veterinary Research Institute. The causal

agent (*Rickettsia canis* = *Ehrlichia canis* (Donation and Lestoquard, 1935)) is transmitted by the dog tick (*Rhipicephalus sanguineus* (Latreille)). In common with other tick-borne rickettsias, this parasite is also transmitted transovarially to the ensuing generation. A milder form of canine rickettsiosis has been observed in several regions of Transvaal. This indicates that strains occurring in nature need not necessarily be of the same virulence. Treatment with tetracyclines is very effective.

Heartwater has been and still is a serious hazard to domestic ruminants in extensive regions of Africa. It was suspected to be a tick-borne disease in the Eastern Cape Province where its presence was associated with the bont tick (*Amblyomma hebraeum* Koch) as far back as 1838. It is prevalent in Northern and Eastern Transvaal, Natal and Eastern Cape Province despite systematic tick control by dipping. In contradistinction to other tick-borne rickettsias, the causal agent of heartwater ((*Rickettsia ruminantium* = *Cowdria ruminantium* (Cowdry, 1925)) is not transmitted transovarially in bont ticks. Had this been the case, heartwater would have established itself in Mauritius, Reunion, West Indies and Guatemala, where another African vector (*Amblyomma variegatum* (Fabricius)) was introduced many decades ago.

The absence of a transovarial transmission, and the high incidence of infected ticks in the presence of immune domestic ruminants, suggested the existence of an unexplored source of infection for vectors. Attention was naturally paid to wild ruminants as possible reservoirs. Investigations revealed that the blesbok and black wildebeest are susceptible, and that natural fatal heartwater infections occurred in indigenous springboks on the Springbok Flats in the Northern Transvaal. The very low incidence or complete absence of antelopes in many parts of the enzootic regions in relation to the high prevalence of infected ticks, as evidenced by the very high morbidity and mortality rates in domestic ruminants introduced from a non-enzootic into an enzootic heartwater region, failed to offer an acceptable explanation about the source of infection for ticks. This puzzle was solved finally when it was determined that immune domestic ruminants fulfilled the requirements of an adequate source. These animals, on reinfection with infected bont ticks, develop a clinically imperceptible infection, thus permitting vectors to acquire the pathogen. This phenomenon is yet another example how the wild host-vector cycle has shifted effectively across to the domestic host-vector cycle. Advances have been made in combating heartwater. Treatment with certain sulphonamides and recently with the more effective tetracyclenes has proved to be very satisfactory. Immunization of domestic ruminants and, on a very limited scale, of antelopes and European deer has given gratifying results.

The three forms of anaplasmosis (gallsickness) of domestic ruminants have a ubiquitous distribution. The virulent parasite, *Anaplasma marginale* Theiler, 1911 has been responsible for high mortality in cattle in South Africa. The benign parasite *A. centrale* Theiler, 1911 is used as an immunizing agent against the former infectious agent. Both parasites are transmitted mainly by the blue tick (*Boophilus decoloratus* Koch). *A. ovis* Lestoquard, 1924 produces a mild disease in sheep and goats. The tick vector has not yet been determined in South Africa. Recovered animals usually remain carriers for life. It is not surprising therefore, that anaplasmoses established themselves in numerous countries where potential vectors occurred. Although the three parasites have not yet been isolated from antelopes, laboratory tests have shown that several of them are susceptible. *A. marginale* has been transmitted to the black wildebeest, blesbok and duiker, *A. centrale* to the blesbok and *A. ovis* to the blesbok and eland. The parasitaemia was very low and reactions very mild in all the recipients. The blood, derived from cattle, also contained *Babesia bigemina* and *Theileria mutans*. Since the recipients failed to react to these parasites it became clear that pure strains of either *A. marginale* and *A. centrale* can be obtained by passage through the blesbok, duiker and black wildebeest. Immunization of cattle and timely treatment with tetracyclines are effective in controlling infections due to *A. marginale*.

Eperythrozoon ovis Neitz, Alexander and Du Toit 1934 has a world-wide distribution. It produces a transitory anaemia and loss in condition in sheep and goats. Experimental evidence is available that the blesbok is susceptible. As the mode of transmission is unknown, it is impossible to state what role, if any, the blesbok could play in the host-vector cycle. Affected sheep respond well when treated with certain arsenical compounds and antibiotics.

C. *Protozoal Diseases*

Protozoa responsible for the zoonoses in which wild animals are involved as reservoirs are all, with the exception of *Toxoplasma gondii* (Nicolle and Manceaux, 1909), arthropod-borne parasites. The responsible pathogens, members of the classes Mastigophora and Sporozoa, have caused severe stock losses, especially among cattle, in South Africa. The inability to develop reliable vaccines

directed the attention of biologists and chemists to the production of effective chemotherapeutic agents and acaricides. Their application, generally speaking, has been successful. In the case of nagana and East Coast fever, vector control eventually terminated in the extermination of both diseases in South Africa.

(a) *Mastigophora*

Only members of the genus *Trypanosoma,* transmissible to domestic and wild animals, have been encountered in Transvaal and Natal. There is no evidence of sleeping sickness ever having occurred in man in South Africa, even though this disease occurs in neighbouring states. History relates that there was a decline in the incidence of tsetse flies (*Glossina morsitans* Westwood) in Transvaal before rinderpest made its appearance in 1896. The high mortality in cattle and antelopes due to this virus infection deprived the remaining flies of their source of food and thus contributed to their disappearance. Since then nagana remained confined to Northern Natal, where the tsetse flies, *Glossina austeni* Newst., *G. brevipalpis* Newst. and *G. pallidipes* Aust., served as transmitters. The latter species was the chief vector, and responsible for the periodic epizootics until its extermination in recent years. Four pathogenic *Trypanosoma* spp. have been encountered in the enzootic area. Field and laboratory observations have shown that these species have a wide host range.

The South African records in this respect are listed in Table 1.

Fatal infections of nagana in domestic animals maintained in the enzootic area were found to be due to *T. brucei* Plimmer and Bradford, 1899 in solipeds and dogs, *T. congolense* Broden, 1904 in cattle and dogs, *T. vivax* Ziemann, 1905 in cattle and *T. simiae* Bruce *et al.,* 1911 in pigs. Within the enzootic region only a relatively small number of sheep and goats was kept around the residential areas, and thus escaped frequent attacks by tsetse flies. Although specific remedies were available, years of experience proved that treatment was only palliative. Recovered animals sooner or later became reinfected. Chemoprophylaxis applied to horses used for tsetse fly surveys proved effective against *T. brucei.*

The total number of cattle that died from nagana in Natal is unknown. Judging from the records of the last epizootic during 1942 to 1945,

TABLE 1.—KNOWN HOST RANGE OF TRYPANOSOMA SPP. FROM NATAL.

Host	*T. brucei*	*T. congolense*	*T. vivax*	*T. simiae*
Horse	N	N	—	—
Mule	N	—	—	—
Donkey	N	N	—	—
Zebra	—	N	—	—
Ox	N	N	N	—
Sheep	E	E	E	E
Goat	E	N	E	—
African buffalo	N	—	—	—
Black wildebeest	—	E	—	—
Blue wildebeest	N	—	—	—
Bushbuck	N	N	N	—
Duiker	—	—	N	—
Eland	—	N	—	—
Kudu	N	N	N	—
Reedbuck	N	—	—	—
Steenbok	N	—	—	—
Domestic pig	N	N	—	N
Wart hog	—	—	N	—
Dog	N	N	—	—
Black-backed jackal	E	E	—	—
Long-eared fox	—	E	—	—
Spotted hyaena	N	—	—	—
Domestic cat	E	E	—	—
Rock rabbit	E	E	—	—
Albino mouse	E	E	—	—
Albino rat	E	E	—	—
Multimammate mouse	E	—	—	—
Guinea pig	E	E	—	—
Rabbit	E	E	—	E

N = Natural infections, E = Experimental infections.

when 60,000 head of cattle died, the mortality since the beginning of the century must have been far more than 100,000. As neither the treatment of affected cattle, nor the destruction of far more than 100,000 game animals in the enzootic region proved to be effective, attention was directed to the efficacy of insecticides that were being utilized at the time for the control of malaria mosquitoes and epidemic typhus lice. The successful control of both diseases suggested that this form of prophylaxis would be equally effective against nagana. A detailed description of the campaign which commenced in 1945 has been given by Du Toit[72]. The results were remarkable in that the scourge was finally exterminated, to the great relief of the farming community.

(b) *Sporozoa*

Babesioses. Of the eight *Babesia* spp., which have a world distribution, South Africa also has its share as shown by their presence in domestic animals except sheep and goats. When these tick-borne parasites were described originally they were regarded to be host specific. Since then it has been determined at several veterinary research centres that at least eight species can be harboured by wild animals. Observations on the host-system, as determined in Southern Africa appear in Table 2.

to serve as natural hosts of *B. felis* Davis, 1929 in East and North Africa. "Elsa" the lioness, described by Joy Adamson[70] in the widely read book entitled "Living free", died from feline biliary fever[74]. Its sporadic occurrence in domestic cats in South Africa suggests that wild cats are carriers. With the exception of *B. trautmanni,* which has been encountered only in domestic pigs in the Soutpansberg and Piet Retief districts of Transvaal, the remaining *Babesia* spp. of cattle, solipeds, dogs, and cats are widely distributed in tick infested regions. They may cause serious losses unless timely treatment is applied.

Theilerioses. Two forms of bovine theileriosis, East Coast fever caused by *Theileria parva* (Theiler, 1904) and Corridor disease due to *T. lawrencei* Neitz, 1955 are typical African diseases, while benign bovine theileriosis produced by *T. mutans* (Theiler, 1906) has a world-wide distribution. These diseases have two features in common:— the brown tick (*Rhipicephalus appendiculatus* Neum.) is the chief vector, and African buffaloes are their wild hosts. In its original habitat in East Africa, *T. parva* established itself in cattle. Consignments of cattle imported from Tanzania into Moçambique and Rhodesia at the beginning of this century, resulted in the introduction of East Coast fever. In the absence of tick control measures, the disease spread within the brown tick infested regions of Transvaal, Natal, Transkei and

TABLE 2.—HOST RANGE OF BABESIA SPP. IN SOUTH AFRICA.

Babesia spp. and vectors	Domestic hosts	Wild hosts
B. equi (Laveran, 1901). *Rhipicephalus evertsi* Neum.	Horse, mule donkey	Burchell's zebra (Kruger National Park, Zululand)
B. caballi (Nuttall, (1910)	Horse, mule donkey	Burchell's zebra (Kruger National Park)
B. canis (Piana and Galli-Valerio, 1895). *Rhipicephalus sanguineus* (Latreille), *Haemaphysalis leachi* (Audouin).	Dog	Black-backed jackal, Cape hunting dog (Tick transmission in kennels, Onderstepoort)
B. trautmanni (Kuth and Du Toit, 1921)	Domestic pig	Bush pig (Artificial infection, Veterinary Laboratory, Rhodesia).

There is no evidence that *B. bigemina* (Smith and Kilborne, 1893) is harboured by antelope in South Africa even though Enigk and Friedhoff[73] determined that the Sudanese gazelle (*Gazella soemmeringi* Cretzschmar) is susceptible. The latter observation suggests that the relationship between *B. irvinsmithi* Martinaglia, 1936 of the sable antelope, which morphologically resembles *B. bovis* (Babes, 1888), needs to be determined. Several wild members of the family Felidae have been found

Eastern Cape Province. Before dipping in arsenical formulations was applied as a prophylactic measure in 1914, more than one million head of cattle died in the enzootic region. This serious menace to the cattle industry followed upon the heavy toll exacted by rinderpest a few years earlier. Quarantine measures, systematic dipping and slaughter of cattle (in isolated outbreaks) over a period of 40 years resulted in the eradication of East Coast fever in South Africa in 1954.

Towards the end of the East Coast fever campaign an unexpected epizootic occurred in cattle in the Corridor, a stretch of country 100 square miles in size lying between the Hluhluwe and the Umfolozi Game Reserves in Northern Natal. Since the danger of nagana no longer existed, two farmers allowed their cattle (585 in number) to graze along the border of the Hluhluwe Game Reserve. Buffaloes and other antelopes were known to stray for a variable distance into the Corridor. Tick life was active at the time (April and May, 1953) and during the ensuing four weeks 300 animals died. The mortality would have been even greater had the animals not been evacuated. Subsequent investigations revealed that brown ticks had acquired the infection from African buffalo *T. lawrencei* carriers, and then had transmitted the pathogen to cattle. It was also determined that buffalo calves had contracted the disease soon after birth and that some had died. A second outbreak of Corridor disease occurred on a farm adjoining the southern boundary of the Kruger National Park in 1960. The pattern of this epizootic was similar to that of the previous one. Of the 700 oxen exposed on veld where buffaloes had grazed several months previously, 300 sickened and 250 died. The evacuation of cattle to buffalo-free localities terminated the epizootics.

An important feature of Corridor disease in South Africa is that affected and recovered cattle do not serve as reservoirs for the infection of ticks. Affected and recovered buffaloes, on the other hand, exhibit a microscopically visible parasitaemia in the red blood ceells, and thus form a readily available source for the infection of vectors. These distinctive features serve as the basis for prophylaxis of Corridor disease in cattle. Prevention of contact between game and cattle by the erection of a substantial fence around the Kruger National Park is not only effective for the control of foot and mouth disease but is equally efficient for checking the dissemination of Corridor disease-infected ticks by buffaloes.

With the exception of turning sickness (cerebral theileriosis) and brown tick toxicosis, both fatal diseases, a pure infection of benign bovine theileriosis ((*T. mutans* (Theiler, 1906) infection)) does not constitute an important animal health problem.

Aegyptianellosis can be expected to occur in domestic fowls, ducks and geese when they are exposed to *Aegyptianella pullorum* Carpano, 1929 infected tampans (*Argas persicus* Oken) in infested premises. The mortality rate is high as determined in Africa, Europe and Asia. The host-range, which is comprised of unrelated domestic birds, suggests that the relationship between *A. pullorum* and the *Aegyptianella* sp. of the jackass penguin, described on Dassen Island[65], be established even though there is no danger that poultry would acquire the disease from sea birds. The eradication of the tampan by means of synthetic acaricides has proved to be an effective prophylactic measure.

Toxoplasmosis has a ubiquitous distribution. In South Africa its presence has been diagnosed in man[75], domestic pigs and solipeds[75], dogs[77], black-backed jackal[78] and Cape hunting dog[18]. The disease is of great public and animal health importance. No specific drug is at present available for its treatment.

Summary and Conclusions

An account has been given of the zoonoses, due to viral, protophytal and protozoal diseases, in which wild and domestic animals and man are involved. In doing so, attention was paid to the origin of the pathogens. Surveys have shown that some were either of indigenous or exogenous origin, while in the case of diseases with a ubiquitous distribution it was impossible to trace the primary source. From these observations it has become evident that wild animals served as reservoirs of indigenous and ubiquitous diseases. As time progressed they also acquired exogenous diseases which reached South Africa by either the transoceanic (plague, anthrax, tuberculosis) or transcontinental route (rinderpest). There is no evidence that poultry diseases were introduced from the north by any of the migratory birds.

With the exception of murine listeriosis, indigenous diseases, as judged from the extremely large game population in the past, appear to have had little detrimental influence on wild animal health. The migratory habits of game together with the elimination of sick animals by carnivores undoubtedly contributed to reduction in incidence of active disease reservoirs, while the colostral milk induced passive immunity and so offered protection to young animals for several months after birth.

Disastrous manifestations of various diseases resulted when a shift-over from the wild to the domestic host-system occurred, as seen in the fulminating epizootics of nagana, Corridor disease, African swine fever, foot and mouth disease, Rift Valley fever and Wesselsbron disease. A similar manifestation was observed when the reverse process took place, as shown by rinderpest and anthrax introduced by cattle, and by bubonic plague that entered at sea ports with house rats. The number of wild animals, including rodents, that succumbed, is unknown, because sick animals and carcasses soon become devoured by beasts of prey.

The manifestations of epizootics are well known to many stockowners who suffered severe losses, to veterinarians and medical officers engaged in their control, and to those keenly interested in wild life. It is, therefore, not surprising that stockowners agitated for the destruction of game at all costs. This in its turn resulted in counterpropaganda by game conservationists and even hunters that wild life should be preserved for posterity. This lead to heated discussions which terminated in a state resolution that game destruction should be carried out in the enzootic nagana region of Northern Natal. This was done in a campaign which commenced in 1928. The results were disappointing, for it transpired that cattle in secondary tsetse fly foci served as a food supply for the vectors, and as reservoirs for the responsible pathogens. The wild host-vector cycle had shifted across to the domestic host-vector cycle.

The campaign was abandoned when it became evident that scientific research had reached a level at which it could offer far more effective prophylactic measures which would make co-existence of domestic stock and wild fauna possible. Chemical agents became available for the destruction of insects and ticks, and new vaccines were developed at the Onderstepoort Veterinary Research Laboratory for the control of heartwater, Rift Valley fever and Wesselsbron disease and at the Pirbright Veterinary Research Institute (England) for the control of foot and mouth disease. Funds also became available for the erection of a substantial fence around the Kruger National Park for the control of foot and mouth disease and Corridor disease. Farmers in enzootic African swine fever areas maintained their domestic pigs in double fenced paddocks to avoid contact with wild pigs.

The application of improved or new prophylactic measures has not only made stock raising in many regions profitable but it has also reduced the danger to man in contracting certain zoonoses harboured by domestic and wild animals. Had veterinary authorities not applied these prophylactic measures in South Africa, the fate of wild animals undoubtedly would have been a tragic one. Peaceful co-existence between wild and domestic animals and man is a scientific victory.

REFERENCES

1. CLUVER, E., 1927. *J. med. Ass. S. Afr.*, 1, 247.
2. CUMMING, R. G., 1850. Five years of a hunter's life in the interior of South Africa. Vol. 2. London: John Murray.
3. VILJOEN, P. R., CURSON, H. H. and FOURIE, P. J. J., 1928. *13th and 14th Rep. vet. Res. Un. S. Afr.*, p. 431.
4. BRUCE, D., 1895. Preliminary report on the tsetse fly disease or nagana in Zululand. Durban: Bennet & Davis.
5. THOMAS, A. D. and NEITZ, W. O., 1933. *S. Afr. J. Sci.*, 30, 419.
6. MARTINAGLIA, G., 1937. *S. Afr. J. Sci.*, 33, 833.
7. LOBRY, M. A., 1964. *Bull. epiz. Dis. Afr.*, 12, 43.
8. NEITZ, W. O., 1965. *Onderstepoort J. vet. Res.*, 32, 189.
9. FOURIE, L., 1938. *S. Afr. med. J.*, 12, 352.
10. MITCHELL, J. A., 1927. *Publ. S. Afr. Inst. med. Res.*, 3, 89.
11. DE MEILLON, N., DAVIS, D. H. S. and HARDY, F., 1961. Plague in Southern Africa. The Siphonaptera. Vol. 1. Pretoria: Govt. Printer.
12. DAVIS, D. H. S., 1964. Johannesburg: Personal communication.
13. NEITZ, W. O., 1963. *Jaarboek S. Afr. Akad. Wetensk. & Kuns*, p. 167.
14. ALEXANDER, R. A. & NEITZ, W. O., 1935. *Onderstepoort J. vet. Sci.*, 5, 15.
15. ALEXANDER, R. A. & NEITZ, W. O., 1933. *Vet. J.*, 89, 320.
16. MAYER, J. H. & FINLAYSON, M. H., 1944. *S. Afr. med. J.*, 18, 109.
17. FINLAYSON, M. H., 1944. *S. Afr. med. J.*, 18, 113.
18. HOFMEYER, C. F. B., 1956. *Jl. S. Afr. vet. med. Ass.*, 27, 263.
19. RICKMANN, W., 1908. Cited by Knuth, P. & Du Toit, P. J., 1921. Tropenkrankheiten der Haustiere. Leipzig: Verlag Joh. Ambrosius Barth.
20. DU TOIT, R. M., 1944. *Onderstepoort J. vet. Res.*, 19, 7.
21. DU TOIT, R. M., 1955. *Jl. S. Afr. vet. med. Ass.*, 26, 263,
22. ALEXANDER, R. A., 1948. *Onderstepoort J. vet. Sci.*, 23, 77.
23. HOWELL, P. G., 1963. *F.A.O. Agric. Series, Rome*, No. 61, p. 73.
24. NEITZ, W. O., 1933. *Jl. S. Afr. vet. med. Ass.*, 4, 24.
25. DU TOIT, R. M. and GOOSEN, J., 1949. Onderstepoort: Personal communication.
26. HOWELL, P. G., 1963. *F.A.O. Agric. Series, Rome*, 61, p. 111.
27. ROSSITER, L. W. and ALBERTYN, A. A. L., 1947. *Jl. S. Afr. vet. med. Ass.*, 18, 16.
28. LAMBRECHTS, M. C., BUHR, W. H. B. and VAN DER MERWE, J. P., 1956. *Jl. S. Afr. vet. med. Ass.*, 27, 133.
29. BASSON, P. A., 1961. Windhoek: Personal communication.
30. BASSON, P. A., 1962. *Jl. S. Afr. vet. med. Ass.*, 33, 519.
31. VILJOEN, J. H., 1964. Windhoek: Personal communication.

32. MEESER, M. J. M., 1962. *Jl. S. Afr. vet. med. Ass.*, 33, 351.
33. LAMBRECHTS, M. C. and EDWARDS, L. T., 1964. Pretoria: Personal communication.
34. METTAM, R. W. M., 1923. *9th and 10th Rep. vet. Res. Un. S. Afr.*, p. 393.
35. ALEXANDER, R. A. and DICKSON, J., 1951. *Jl. S. Afr. vet. med. Ass.*,, 22, 105.
36. GEAR, J., DE MEILLON, B., LE ROUX, A. F., ROFSKRY, R., ROSE-INNES, R., STEYN, J. J., CLIFF, W. D. and SCHULZ, K. H., 1955. *S. Afr. med. J.*, 29, 54.
37. KASCHULA, V. R., 1961. D.V.Sc. Thesis, University of Pretoria, 102 pp.
38. WEISS, K. E., HAIG, D. A. and ALEXANDER, R. A., 1956. *Onderstepoort J. vet. Res.*, 27, 183.
39. KOKERNOT, R. H., DE MEILLON, B., PATERSON, H. E., HEYMAN, C. S. and SMITHBURN, K. C., 1957. *S. Afr. med. J.*, 22, 145.
40. NEITZ, W. O., 1963. *F.A.O. Agric Series, Rome*, No. 61 p. 3.
41. KASCHULA, V. R., CANHAM, A. S., DIESEL, A. M. and COLES, J. D. W. A., 1946. *Jl. S. Afr. vet. med. Ass.*, 17, 1.
42. BECKER, L. B. and UYS, C. J., 1963. *S. Afr. med. J.*, 37, 1095.
43. SCHOTTMÜLLER, H., 1914. *Derm. Wschr.* Supplement to Vol. 58, p. 77.
44. HENNING, M. W., 1939. *Onderstepoort J. vet. Res.*, 23, 79.
45. CAMERON, C. M., TUSTIN R. C. and MEESER, M. J. N., 1963. *Jl. S. Afr. vet. med. Ass.*, 34, 53.
46. GEAR, J., ROUX, P., and BEVAN, C. de V., 1942. *S. Afr. med. J.*, 16, 125.
47. REMENTSOVA, M. M., 1962. Brucellosis in wild animals. Alma Ata: Akademia Nauk Kazakhskoi's S.S.R.
48. PIRIE, J. H. H., 1927. *Publ. S. Afr. Inst. med. Res.*, p. 16.
49. WINTER, P. A. D., 1946. *Ann. Rep. S. Afr. Inst. med. Res.*, p. 15.
50. DU PLESSIS, J. L., 1964. Onderstepoort: Personal communication.
51. PIENAAR, U. DE V., 1960. *Koedoe*, No. 3, 238.
52. PIENAAR, U. DE V., 1961. *Kudu*, No. 4, 4.
53. ROBINSON, E. M., 1942. *Jl. S. Afr. vet. med. Ass.*, 13, 76.
54. ROBINSON, E. M., 1958. *Jl. S. Afr. vet. med. Ass.*, 29, 129.
55. MARTINAGLIA, G., 1930. *16th Rep. Div. vet. Res. Un. S. Afr.*, p. 143.
56. MARTINAGLIA, G., 1929. *Jl. S. Afr. vet. med. Ass.*, 1, 37.
57. MARTINAGLIA, G., HOBBS, W. B. and BLAINE, M. G., 1957. *S. Afr. med. J.*, 31, 339.
58. WORTHINGRON, R. W., 1964. *Jl. S. Afr. vet. med. Ass.*, 35, 390.
59. FOURIE, P. J. J., 1928. *13th & 14th Rep. vet. Res. Un. S. Africa*, p. 623.
60. ROBINSON, E. M., 1955. *Jl. S. Afr. med. Ass.*, 26, 259.
61. ROBINSON, E. M., 1953. *Jl. S. Afr. vet. med. Ass.*, 24, 87.
62. PAINE, R., and MARTINAGLIA, G., 1928. *Jl. S. Afr. vet. med. Ass.*, 1, 97.
63. KASCHULA, V. R. and TRUTER, D. E., 1951. *Jl. S. Afr. vet. med. Ass.*, 22, 191.
64. ZUMPT, F., 1959. *Nature (London)*, 184, 793.
65. COLES, J. D. W. A., 1941. *Jl. S. Afr. vet. med. Ass.*, 12, 23.
66. NEITZ, W. O., 1935. *Onderstepoort J. vet. Sci.*, 5, 7.
67. WOLSTENHOLME, B. and HARWIN, 1951. *Ann. Rep. S. Afr. Inst. med. Res.*, p. 36.
68. GEAR, J. and DAVIS, D. H. S., 1942. *Ann. Rep. S. Afr. Inst. med. Res.*, p. 22.
69. BEVAN, C. DE V., 1944. *S. Afr. J. med. Sci.*, 9. 1.
70. GEAR, J., 1949. *Ann. Rep. S. Afr. Inst. med. Res.*, p. 29.
71' WOLSTENHOLME, B., 1952. *Ann. Rep. S. Afr. Inst. med. Res.*, p. 34.
72. DU TOIT, R., 1954. *Onderstepoort J. vet. Res.*, 26, 317.
73. ENIGK., K. and FRIEDHOFF, K., 1963. *Z. Tropmed. Parasit.*, 14, 503.
74. BARNETT, S., 1963. Kenya: Personal communication.
75. KLENERMAN, P., 1951. *S. Afr. med. J.*, 25, 273.
76. GEAR, J. and WOLSTENHOLME, B., 1959. *Ann. Rep. S. Afr. Inst. med. Res.*, p. 117.
77. SPENCER, I. W. F., 1957. *Ann. Rep. S. Afr. Inst. med. Res.*, p. 65.
78. NEITZ, W. O., 1953. Cited by Van der Merwe, N. J., 1953. *Fauna and Flora, Transvaal*, No. 5, p. 33.
79. ADAMSON, J., 1961. Living free — The story of Elsa and her cubs. London: Collins & Harvill Press.

QUALITY EQUIPMENT FOR THE MEAT INDUSTRY

ANCO®

Division of CHEMETRON

"ON - THE - RAIL" CATTLE DRESSING

Complete mechanization from slaughter to by-products

The Allbright-Nell system is saving time, labour and money in a number of South Africa's large abattoirs where it has been installed but the system is also adaptable and economic for smaller plant, down to 10 cattle per hour.

As well as the complete supply and installation of abattoirs we can also supply a full range of abattoir and by-products plant and equipment including stunners, dehorners, power skinning tools, electric saws and a large range of South African made abattoir trolleys, tables and hardware.

A MEMBER OF THE HUDACO GROUP

Hudamech

BRANCHES: Johannesburg, Durban, East London, Port Elizabeth, Cape Town, Welkom, Bloemfontein, Windhoek, Klerksdorp, Witbank, Dundee, Pretoria, Phalaborwa, Bulawayo, Salisbury, Gwelo, Lusaka, Ndola.

TREATMENT OF INFECTIOUS OPHTHALMIA WITH SOLCOSERYL* AND CHLORAMPHENICOL.

J. J. OBERHOLSTER, P.O. Box 128, Bethlehem. O.F.S.

In the farming area round Bethlehem the incidence of infectious ophthalmia is high. Mainly sheep and calves are affected but the disease also frequently occurs in adult cattle. Treatment with antibiotics eventually controls the infection but residual opacity of the cornea often leads to permanent blindness.

Menna[1] reported very good results obtained in eye disease of man with the topical application of the drug solcoseryl. I was impressed by Menna's observation that in suppurative keratitis and following injuries to the cornea, regeneration of the corneal epithelium and healing of the wounds proceeded rapidly thereby preventing permanent damage to the cornea. It appeared particularly interesting, with regard to infectious keratitis in ruminants, that in cases of burn injuries involving the deep structures, and where the cornea presented the typical chalky appearance, repeated application of solcoseryl achieved partial clearing of the cornea in 48-72 hours.

A review of the vast literature, both experimental and clinical (human) on solcoseryl, is beyond the scope of this short preliminary report. I can only briefly summarise the action of the drug : it increases the utilisation of oxygen by the cells which becomes strikingly evident after oxygen supply is reduced by pathological conditions in or around the lesions. Enhanced oxygen utilisation results in the rapid formation of healthy granulation tissue and regeneration of epithelium.

This information induced me to try solcoseryl in the treatment of infectious keratitis of ruminants. For the purpose of the trial Saphar Laboratories prepared a formulation containing solcoseryl (20%, later 10%) and chloramphenicol (1%) in a jelly base. For controls an identical jelly was prepared which contained chloramphenicol only. The two kinds of jelly were filled into identical tubes and I did not know the code of the marking until the first results were compared.

For the first tentative trial I selected six calves on one farm and about ten sheep on another. Half of the animals were treated with solcoseryl/chloramphenicol, the other half with chloramphenicol only. After three days the difference in the conditions of the two sets of animals was so striking that I could not refuse the owners' request for tubes with the marking that caused the rapid improvement, i.e. solcoseryl/chloramphenicol.

When I was called to a dairy farm where four cows suffered from severe ophthalmia refractory to conventional treatment for more than a week, I instituted treatment with solcoseryl/chloramphenicol in all four animals. It would have been more correct to take two cows for control but considering the condition of the animals, I could not reconcile this with my conscience and risk permanent blindness for the sake of having controls. In all four animals healing was complete in six days with corneas clear.

In a flock at Brakfontein more than 180 sheep were affected with infectious ophthalmia. Twenty had extensive corneal opacity and were blind. I selected the worst 16 for treatment with Solcoseryl-chloramphenicol. On the 5th day five sheep had almost completely recovered with partial clearing of the cornea. On the 7th day eight sheep had completely recovered and were returned to the flock. Six had greatly improved and healing was complete after a further two days. Two animals had hypopyon before beginning of treatment and I expected perforation of the cornea. By the 7th day all signs of inflammation had subsided, the cornea showing partial clearing without perforation. The aqueous humour was still turbid, yet the animals recovered without permanent corneal damage. The condition in these animals was refractory to previous treatment and, according to my experience, I considered the corneal damage in the sixteen animals to be irreversible.

The four sheep that had chloramphenicol treatment only, also eventually recovered but there was no doubt as to the advantages offered by the combined treatment.

*Solcoseryl—Respiratory co-enxyme (protein free extract of calf's blood). Prepared by Solco Basle Ltd., Switzerland.
‡Chloramphenicol-p-nitrophenyl-z-dichlor-acetamidopropane — 1:3 — diol.
†Translations of part of the European literature were made available to me by Saphar Laboratories, Johannesburg.

One hundred and twenty-two sheep of the flock were treated with solcoseryl-chloramphenicol and thirty-six with the antibiotic only. Again, the most severe cases were selected for the combined treatment. After six treatments sixty-five sheep had completely recovered, the others had improved greatly and healing was complete within a further two to three days.

Of the thirty-seven mild cases treated with antibiotic only, twenty-seven gradually recovered and ten improved. The degree of recovery and rate of improvement in the animals treated with solcoseryl and chloramphenicol were vastly superior to the improvement seen during and after treatment with antibiotics only.

I am fully aware of the shortcomings of this report. It is very difficult to carry out adequately controlled trials in a busy country practice. I decided to publish my experiences in order to draw the attention of the profession to this method of treatment which is far more effective than all forms of therapy hitherto employed.

ACKNOWLEDGEMENT:

I wish to express my sincerest thanks to Dr. A. Janovics of Saphar Laboratories for supplying the information on solcoseryl and making up the material.

REFERENCE

1. MENNA, F., 1965. Arch. Ottalmol. 68: 11.

BOOK REVIEW

HAGAN'S INFECTIOUS DISEASES OF DOMESTIC ANIMALS

With special reference to Etiology, Diagnosis and Biologic Therapy by

D. W. BRUNER AND J. H. GILLESPIE.

5th Ed. 1966. pp. 1103. Baillière, Tindall & Cassell, London.

Originally "The Infectious Diseases of Domestic Animals" this book now bears the name of the first author, who died in 1963, and serves, as was intended by Hagan, as a general reference and discussion on the broad principles of immunology and the various infectious agents. It covers a very wide field and is well set out and presented; starting with mechanisms of infection and resistance and a chapter on chemotherapeutics, it progresses through the bacteria, fungi, and protozoa to the section on viruses and rickettsiae. The variety of organisms described in the section on pathogenic fungi will come as an eye-opener to the average S.A. veterinarian.

It is a pity that in a book of this wide scope more use could not have been made of colour photography and more imagination in the portrayal of bacteria. We have been subjected for so long to these poorly reproduced rod forms and lack of contrast in protozoal forms.

The book will be of considerable value to veterinarians as well as those engaged in agricultural pursuits as a most informative, general reference book with an adequate bibliography leading to more detailed reading. Initially the book developed from a series of lectures for students on bacteriology and immunity but has now reached the stage of becoming a text book on infectious diseases for students and can certainly be recommended as such.

P. W. T.

THE EFFECT OF PROLONGED RAIL TRANSPORT ON SLAUGHTER CATTLE

Comparison of broken and continuous journeys.

L. W. v.d. Heever*, G. D. Sutton†, J. F. W. Grosskopf¶ and P. D. Fourie‡

Summary

Steers were transported by rail during summer for four days without interruption for feed and water, and for five days with two four hour rest periods with feed and water. Controls were kept in pens. Live weights and slaughter data of groups were compared.

All groups showed loss of live weight, about half due to loss of ingesta. The loss of weight was attributed mainly to the differences in management, feeding and watering; rail travel resulted in but a slight additional loss.

Provided rest, feed and water were given for 24-48 hours before slaughter carcase yields showed no significant difference.

Steers kept without food and water for four days showed dehydration and mobilization of energy reserves; the latter were not depleted. Recovery was rapid when rest feed and water were provided before slaughter.

When cattle are handled carefully, bruising is slight, regardless of whether they have been detrained *en route* or not. Tables of data are provided.

Introduction

The present system of slaughtering food animals in abattoirs situated in consumer areas involves *protracted rail transport for most slaughter stock.* Cattle from South West Africa travel some 1500 miles for five days to abattoirs in Cape Town and Johannesburg. Present Railway regulations require two interruptions of four hours for rest, food and water. It has been contended that these breaks in the journey have little value and prolong travel time, and that repeated entraining and detraining add to the amount of bruising entailed in transport[1]. Starke[2] has summarised the available knowledge *concerning weight losses of livestock in* transit, and although information on lambs and pigs is available there is little on cattle.

The S.A. Railways determined that unbroken rail journeys from S.W.A. would take up to 96 hours. A preliminary study was therefore undertaken to compare the two systems of in-transit management and establish the time necessary for animals to regain physiological normality after transport and before slaughter is undertaken. Control groups were kept in pens with and without food and water for the same duration as the rail journeys, which took place during hot summer weather in the North Eastern Transvaal.

Experimental Procedure.

Sixty similar grade Afrikaner oxen aged from four tooth to full mouth were used in the experiment. The majority were dehorned, some had horn scurs and others horns of various types. Initially they were ear-tagged, branded and placed on veld grazing. Allocation to five groups ensured that dehorned, scurred and horned animals were equally represented in each group. The groups were treated as shown in table 1.

Table 1.—Details of grouping and treatment of animals.

Group	No. of oxen	Experimental Procedure
IA	12	Railed for unbroken rail transport for 4 days without food or water.
IB	12	Control for Group IA—Kept in abattoir pen for 4 days without food or water.
IIA	12	Railed for 5 days. Offloaded twice for 4 hour periods of rest, feeding and watering after 24 and 72 hours on rail.
IIB	12	Control for IIA. Kept in abattoir pen for 5 days. Fed and watered twice for 4 hour periods after 24 and 72 hours.
III	12	General control. Kept in abattoir pen with free access to feed and water for 5 days.

*Faculty Veterinary Science, Univ. Pretoria.
†Veterinary Res. Inst., Onderstepoort.
¶Faculty Agriculture, Univ. Pretoria.
‡Animal Husb. & Dairy Res. Inst., Irene.

The oxen were weighed and bled immediately before being subjected to any experimental procedures and again immediately before slaughter. Red cell packed volume, total plasma proteins, blood sugar and blood urea nitrogen were determined.

During all operations the animals were handled quietly without the use of sticks, whips or prodders.

On the afternoon preceding commencement of transport the oxen were mustered, driven about 12 miles at a steady walk from their grazing to Onderstepoort and there penned in groups. Feed and water were available *ad lib*. The travelling groups were entrained the next morning. Thereafter the procedure shown in Table 1 was followed.

The railed groups travelled in one half of a GZ-type railway truck. During transport the animals' behaviour was observed and subsequently described by Sutton *et al*[3]. The control animals received identical treatment except for the railway journey. General controls in the pens had free access to feed and water.

The oxen were slaughtered according to the scheme outlined in Table 2.

TABLE 2.—DETAILS OF PRE-SLAUGHTER AND SLAUGHTER PROCEDURES.

Group	No.	Slaughter:
IA	4	*On arrival. No feed or water.*
	4	24 Hours after arrival with feed and water ad lib.
	4	48 Hours after arrival with feed and water ad lib.
IB	4	From pen. No feed or water.
	4	From pen after 24 hours with feed and water ad lib.
	4	From pen after 48 hours with feed and water ad lib.
IIA	4	On arrival. No feed or water.
	4	24 hours after arrival with feed and water ad lib.
	4	48 hours after arrival with feed and water ad lib.
IIB	4	From pen. No feed or water.
	4	From pen after 24 hours with feed and water ad lib.
	4	From pen after 48 hours with feed and water ad lib.
III	12	From pen. General control.

Except for the animals slaughtered without being given feed and water, the oxen were penned in their groups with feed and water available *ad lib* until 6 p.m. on the day prior to slaughter.

At slaughter liveweight and the weight of blood, warm liver, digestive tract and carcase was recorded. The weight of ingesta was calculated as the difference between the weight of the full and empty digestive tracts. The degree of carcase bruising was scored. Carcase muscle samples were taken from *M. triceps brachii* for assessment of pH, using a glass electrode pH meter, and of the moisture content by drying at 100°C to constant weight.

RESULTS

1. *Blood analyses* results are given in table 3.

An increase in red cell packed volume is indicative either of an increase in circulating red cells or of a decrease in plasma volume. The circulating red cells may be increased temporarily by contraction of the spleen as in excercise or fear, or more permanently by increased haemopoesis. Plasma volume may be decreased by dehydration in which case there is an increase in plasma protein concentration. The figures for these blood factors do not indicate any severe reactions. The increases in packed cell volume are apparently partly due to haemoconcentration and partly to splenic contraction. The duration of the experiment was too short for haemopoesis to have played any significant role.

The increases in blood glucose are probably due to adrenergic effects.

The blood urea figures remained within normal limits but showed a general tendency to rise. This may have been due to increased gluconeogenesis in the stressed animals and increase protein intake in the controls.

In general the figures do not indicate any severe dehydration or functional collapse.

2. *Live Weight Loss and Recovery after Rest, Feed and Water.*

The live weight losses were calculated as the difference between live weight prior to the journey or holding period in the pens and live weight before slaughter; details are provided in Table 4.

In groups IA and IB the live weight losses of animals slaughtered immediately were almost the same and 1% higher than in groups IIA and IIB respectively. All groups showed a live weight loss varying from 11.6% to 13.1%.

After 24 hours with rest, feed and water, groups IA and IB regained slightly more of their original live weight than groups IIA and IIB.

TABLE 3.—MEAN INDEX FIGURES OF BLOOD ANALYSES BASED ON 100 FOR VALUES OBTAINED FROM BLOOD TAKEN WHEN OXEN WERE ON GRAZING PRIOR TO EXPERIMENT.

	Time of sampling.	Experimental Group.				
		IA	IB	IIA	IIB	III
Packed Cell Volume	(*a*) Start of experiment	100	—	103	97	
	(*b*) After experiment	122	120	118	120	94
	(*c*) After 24 hours rest	116	115	115	106	
	(*d*) After 48 hours rest	104	94	111	104	
Total Plasma. Proteins	(*a*) As above	101	—	100	100	
	(*b*) ,,	111	107	103	102	100
	(*c*) ,,	102	106	96	97	
	(*d*) ,,	97	97	98	98	
Blood Glucose	(*a*) As above	97	—	109	104	
	(*b*) ,,	139	113	109	105	110
	(*c*) ,,	149	141	104	94	
	(*d*) ,,	136	150	110	105	
Blood Urea Nitrogen	(*a*) As above	118	—	120	120	
	(*b*) ,,	183	166	121	149	165
	(*c*) ,,	135	119	110	113	
	(*d*) ,,	117	107	119	118	

TABLE 4.—MEAN LOSS IN LIVE WEIGHT AFTER RAIL TRANSPORT OR HOLDING PERIOD IN PENS, IN LBS. AND AS A PERCENTAGE OF THE ORIGINAL LIVE WEIGHT.

Group	Time given for feed rest & water (hours)	Mean original live weight (lb)	Mean live weight at slaughter (lb)	Loss of live live weight. (lb—%)
IA	None	787	684	103—13.1
	24	734	700	34— 4.6
	48	847	789	58— 6.8
IB	None	804	701	103—12.8
	24	798	759	39— 4.9
	48	831	782	49— 5.9
IIA	None	809	711	98—12.1
	24	780	714	66— 8.5
	48	823	760	63— 7.7
IIB	None	862	762	100—11.6
	24	819	753	66— 8.1
	48	789	727	62— 7.9

After 48 hours with rest, feed and water groups IIA and IIB had regained more of their original live weight than groups IA and IB.

It appears that the live weight loss is affected mainly by the feeding and watering regimen and that rail travel had only a minimal additional effect.

3. *Loss of Ingesta.*

To determine to what extent live weight loss was due to emptying of the digestive tract, the ingesta of each group was weighed at the time of slaughter and compared with the weight of the ingesta in the relevant control group. Details are provided in Table 5.

The quantity of ingesta in groups IA, IB, IIA & IIB varied from 4 to 5% less than in control group III. There was very little difference between the other groups. Loss of live weight due to elimination of ingesta did not exceed 5% in any group. Total live weight loss being between 11.6 and 13.1% in the various groups (see Table 4), the difference of 6 to 8 per cent must have been due to other causes.

TABLE 5.—MEAN WEIGHT OF INGESTA IN LBS., EXPRESSED AS A PERCENTAGE OF THE ORIGINAL LIVE WEIGHT AT THE START OF THE EXPERIMENT AND OF THE LIVE WEIGHT AT SLAUGHTER.

Group	Time given for rest feed & water (hours)	Mean live weight: (lb)		Mean Ingesta weight. (lb)	Percentage of mean live weight:	
		at start of experiment.	before Slaughter.		at start of experiment.	before Slaughter.
IA	None	789	684	86.1	10.9	12.5
	24	734	700	92.3	12.6	13.2
	48	847	789	88.5	10.4	11.2
IB	None	804	701	80.2	10.0	11.4
	24	798	759	102.9	12.9	15.6
	48	831	782	89.6	10.8	11.5
IIA	None	809	711	82.9	10.2	11.6
	24	780	714	89.4	11.5	12.5
	48	823	760	105.9	12.9	13.9
IIB	None	862	762	91.5	10.6	12.0
	24	819	753	121.0	14.8	16.1
	48	789	727	93.5	11.9	12.9
III	Control	808.8	838.7	137.35	17.0	16.4

4. *Dehydration of muscle* was assessed by the moisture content of a portion of *M. triceps brachii* obtained at slaughter; details are provided in Table 6.

TABLE 6.—MEAN PERCENTAGE OF MOISTURE CONTENT OF M. TRICEPS BRACHII AT SLAUGHTER

Group	Period of rest before slaughter:		
	None	24 hours	48 hours
1A	74.8	77.5	77.4
IB	75.3	77.2	77.9
IIA	77.3	77.9	78.0
IIB	77.3	77.4	77.8
III	77.4	(Control)—	—

It is clear that there was some decrease of moisture content of the musculature of groups IA and IB (4 days without food and water) whereas groups IIA and IIB (access to feed and water) showed no less muscle moisture than the control group.

After only 24 hours with rest, feed and water the animals in groups 1A and 1B had completely regained their normal tissue moisture.

5. *Liver Weights*

Liver weight relative to body weight is considered a good indication of depletion of glycogen and fat reserves. Decrease of relative weight may be caused by stress or starvation. Particulars are given in Table 7.

TABLE 7.—MEAN LIVER WEIGHTS IN LB OF SUB GROUPS SLAUGHTERED AFTER VARIOUS PERIODS OF PRE-SLAUGHTER REST.

Group	No rest	24 hours	48 hours
IA	10.7 (2.89)	8.9 (2.41)	10.7 (2.43)
IB	9.7 (2.42)	9.0 (2.28)	10.3 (2.40)
IIA	9.6 (2.33)	10.8 (2.72)	10.9 (2.62)
IIB	9.8 (2.26)	9.4 (2.39)	11.9 (2.79)
Mean of total	9.75 (2.48)	9.25 (2.40)	10.95 (2.56)
III	11.4 (2.67)	—	—

(The figures in parentheses indicate liver weights as percentage of warm carcase weights.)

These data indicate a significant decrease of mean liver weights of the experimental groups when compared with the "normal" control steers in group III. There is a clear tendency for weights to return to "normal" as the period of rest and exposure to feed and water is extended. When viewed against blood values (Table 3) and ultimate muscle pH values (Table 10), there is no indication of depletion of energy reserves.

6. *Percentage Carcase Yield*

The percentage carcase yield was calculated by expressing the warm carcase weight as a percentage of the live weight of the oxen at the start of the experiment, i.e. before rail travel or experimental holding.

The general controls (Group III) were used to establish a norm. They had been kept in pens and had gained in live weight since the start of the experiment. It was thus necessary to base their

slaughter percentage on live weight at slaughter. Particulars are given in Table 8.

TABLE 8.—INDIVIDUAL AND MEAN CARCASE YIELD PERCENTAGES OF STEERS IN THE EXPERIMENTAL GROUPS WARM CARCASE WEIGHT EXPRESSED AS A PERCENTAGE OF LIVE WEIGHT AT THE START OF THE EXPERIMENTAL PROCEDURES.

Group.	Slaughter groups according to the duration of pre-slaughter rest etc.		
	None.	24 hours.	48 hours.
IA	47.2	52.9	53.0
	49.7	50.1	51.1
	47.2	48.3	51.7
	43.9	51.7	51.6
mean 49.9	47.0	50.7	51.9
IB	49.3	48.2	51.7
	49.2	50.0	51.8
	46.2	49.5	51.4
	48.2	49.4	51.5
mean 49.7	48.2	49.3	51.6
IIA	48.4	51.4	51.5
	49.4	51.6	49.0
	53.0	48.5	49.1
	49.8	44.9	51.0
mean 49.8	50.2	49.1	50.2
IIB	48.8	48.7	52.4
	51.0	48.3	50.1
	49.5	48.6	52.1
	52.1	46.3	48.4
mean 49.7	50.4	48.0	50.8
III	50.8	(Based on live weight at slaughter).	

These figures were subjected to statistical analysis. The small number of animals in each group and the variations between slaughter groups on specific days cast doubt on the reliability of the results. No significant difference existed in the carcase yield percentages of the batches of four animals from groups IA, IIA and IIB slaughtered on successive days. The difference in the carcase yield percentages of the batches of four animals each slaughtered on successive days in group IB are significant at the 1% level. This indicates that cattle become dehydrated during long periods without water and that normality is restored when animals are given rest, feed and water.

Had larger numbers of animals been used in the experiment the tendency in group IB would possibly also have been seen in the other groups. Statistical analysis indicated such a tendency in group IA.

Further analysis of the data showed that there was no statistically significant difference between the carcase yield percentages of group IA and IB, between group IIA and IIB, between group IA and IIA or between group IB and IIB.

7. *Bruising*

The amount of bruising was assessed on five defined areas of the carcase, i.e. the pin bones, hip, back, side and shoulder. Each area was given a score according to the following scale:—

1. — Very slight
2. — Slight
3. — Medium
4. — Severe
5. — Very severe

The total score for each carcase was an index of the degree of bruising, and details are provided in table 9.

TABLE 9.—MEAN TOTAL BRUISING SCORE FOR EACH GROUP. (Maximum 600)

Group.	Index of Amount of Bruising.
IA	39.5
IB	36.0
IIA	60.0
IIB	48.5
III	56.0

In all cases the degree of bruising was slight and there was no significant difference between the groups.

8. *Meat pH*

The ultimate pH of the meat was determined by direct application of the glass electrode of an electric pH meter to a fresh incision in the *M. triceps brachii* of a side kept at chill temperature for 24 hours. The results are detailed in table 10.

TABLE 10.—pH OF MEAT AFTER STORAGE FOR 24 HOURS IN COLD STORE AT —1°C.

Group.	Period of rest before slaughter:		
	None.	24 hours.	48 hours.
IA	5.92	5.86	5.74
IB	5.69	5.81	5.64
IIA	5.53	5.59	5.55
IIB	5.53	5.60	5.58
III	5.68	(Control)	—

The variations fall within the normal range and show availability of adequate reserves of glycogen. Where these reserves are depleted the ultimate pH may be expected to exceed 6.2 - 6.3.

At first sight it appears that oxen subjected to a train journey of four days without feed, water or rest have a slightly higher pH than that of other groups. The small number of animals in the experiment does not justify such a conclusion.

GENERAL CONCLUSION:

1. Some loss of live weight occurred in all the experimental groups, the loss in those given no food and water for four days not exceeding that seen in other groups by more than 1%. Losses were mainly caused by the differences in management and the feeding and watering routine, with very little additional effect attributable to rail travel.
2. Only about half of the live weight loss could be explained by loss of ingesta, the remainder being possibly due to tissue dehydration, *inter alia*.
3. The oxen transported over four days by rail without food and water suffered some stress. There was mobilization of energy reserves but these were not exhausted. Stress was measurably greater than in the oxen railed for five days with two interruptions for rest with access to feed and water.
4. Some dehydration occurred in oxen transported by rail or kept in pens for four days without food and water as demonstrated by loss of moisture from musculature and by blood analysis. These conclusions do not apply to other groups. Normal moisture content was restored within 48 hours of being given feed and water.
5. There was no significant difference in the carcase yield of steers transported for 4-5 days with or without food and water or kept in pens under a similar regimen, provided a pre-slaughter rest period of 24 to 48 hours with feed and water available was allowed. There was no difference in the ultimate pH value of the meat.
6. When handled carefully and considerately, steers show practically no increase in the amount of bruising due to being detrained twice *en route* for rest, feed and water.
7. With rest, feed and water the oxen transported for four days by rail without feed and water returned to normal as rapidly as these on an interrupted five-day journey. After 48 hours there was no significant difference.
8. Rest periods of 24 hours with feed and water but preferably 48 hours is necessary after prolonged rail transport.
9. Uninterrupted long distance transport presupposes feeding and watering prior to loading and immediate detraining at destination; without such guarantees such transportation cannot be considered.
10. This trial was conducted without consideration of humane factor; these should not be overlooked.

ACKNOWLEDGEMENTS

1. The Livestock and Meat Industry Control Board made the experimental animals available, and their technical officers, Messrs. R. Hirzel, J. H. Lombard and J. S. Retief assisted and advised on the details of the trial.
2. The South African Railways and Messrs. E. Daff, J. J. Swart and H. J. van Wyk provided a special train and technical assistance.
3. Messrs. J. J. van Staden, T. Beuster, J. J. P. Smit, R. J. J. Briel, P. J. de Wet and R. Gray, of the Veterinary Research Institute, Onderstepoort assisted in the study.

The Chief, Veterinary Research Institute, Onderstepoort, is thanked for facilities and permission to publish. Prof. J. H. R. Bisschop, Prof. F. N. Bonsma, Dr. G. B. Laurence and Dr. W. A. Verbeek, of the Dept. Agricultural Technical Services, assisted in planning the trial.

REFERENCES

1. BISSCHOP, J. H. R. *et al* 1962 *Report on the effect of padding of railway trucks on the bruising of cattle in transit.* Dept. Agric. T.S., Rep. S. Africa.
2. STARKE, J. S. 1948 Agric. Bull, No. 288, U. of S.A.
3. SUTTON, G., FOURIE, P. D., & RETIEF, J. S. elsewhere, this journal.

BOOK NEWS

The third edition of **GARNER'S VETERINARY TOXICOLOGY** carefully revised by Dr. E. G. C. Clarke and his wife, Mrs Myra Clarke F.R.C.V.S. is now available.

Among the many useful additions are the tables of organochlorine and organophoshorus pesticides, which show the common, chemical and proprietary names together with a brief summary of the nature of each drug.

The section dealing with poisonous plants has been considerably enlarged and now includes not only plants indigenous to Great Britain but also the toxic species from other countries.

New developments in the field of radioactivity have necessitated the complete rewriting of the section on radioactive materials, and this has been contributed by Dr. Garner. Finally, entirely new sections on venomous bites and stings and on animal doping have been added to the text. R6.45.

In the new 10th edition of **POULTRY PRODUCTION** Dr. L. Card has been joined by Dr. M. C. Nesheim, who brings to the text an outstanding experience especially in the field of nutrition. The rapid and revolutionary changes that have taken place in recent years are recorded fully. Special attention is given to the recommended procedures of business management as it applies to poultry and egg production, and to the broiler business. The importance of automation in feeding, egg collection, and poultry and egg processing is recognized throughout. R7.15.

ANIMAL HEALTH AND HOUSING by Davis Sainsbury is a new book in which due regard is given to the new problems created for veterinarians and farmers by the rapid growth of intensive farming and the resultant revolution in methods of livestock husbandry. It is the first book to make a detailed study of the factors that can influence housed animals, including poultry. 336 pages, 123 illustrations; R5.35.

DISEASES OF THE MAMMARY GLANDS OF DOMESTIC ANIMALS by Heidrich and Renk is an excellent translation of a highly regarded German book by two internationally known specialists. It is designed to give the practising veterinarian comprehensive up to date help in diagnosis and control of mammary diseases. It covers cattle, sheep, goats, swine, equines, dogs & cats, and deals with the inflammatory and non-inflammatory conditions plus congenital and acquired malformations. About 500 pages illustrated. R14.15.

A book that should be most valuable and interesting to veterinarians in South Africa is **COMMON NAMES OF SOUTH AFRICAN PLANTS.** by C. A. Smith, edited by Dr. E. P. Phillips, and published by the Department of Agricultural Technical Services. Its 642 pages list over 2,000 plants, and it is far more than a list of common names, as it also gives the derivation and any subsequent modification of the names as the history of Southern Africa unfolded from the time of Governor van Riebeeck. The price in the Republic of South Africa is R7.25 and elsewhere R9.10 postage paid.

The Transvaal Agricultural Union Building,
279, Struben Street,
Pretoria.
Phone: 2-1289.

from a single injection—intrasynovial or intramuscular

prolonged anti-inflammatory effects

Depo-Medrol

Depo-Medrol, long-acting, multipurpose, injectable methylprednisolone, is recommended for intramuscular and intrasynovial use in dogs and horses, and for intramuscular use in cats. It is of value when prolonged anti-inflammatory effects are needed to alleviate the pain and stiffness associated with acute localised or generalised arthritic conditions. Depo-Medrol is also highly beneficial in treating allergic dermatitis, moist and dry eczema, urticaria, and bronchial asthma. As supportive or adjunctive therapy, Depo-Medrol is indicated in inflammatory ocular conditions and in overwhelming infections with severe toxicity.

Supplied: Sterile Aqueous Suspension of Depo-Medrol, 20 mg. or 40 mg. methylprednisolone acetate per cc., in 5 cc. vials.

REGISTERED TRADEMARKS DEPO. MEDROL SA 3558 2

Upjohn *where science turns to healing*

VETERINARY DIVISION • TUCO (PTY) LTD. • JOHANNESBURG

THE BEHAVIOUR OF CATTLE IN TRANSIT BY RAIL

G. D. SUTTON*, P. D. FOURIE**, J. S. RETIEF***.

SUMMARY

Observations during the transport of two truckloads of experimental slaughter cattle have revealed a distinct pattern of behaviour in transit and whilst the train was stationary. Their reactions to short periods of rest in pens, with food and water available, were also recorded. These are considered of significance in reviewing existing regulations under which animals are transported over long distance by rail.

INTRODUCTION

As part of an investigation on the transport of cattle by rail, the behaviour of two consignments was continuously observed during transit. Each consignment consisted of twelve Afrikaner-type oxen all of about 800 lbs live weight. Each group contained animals with or without horns. The oxen were veld-reared but had been handled for dipping and inoculation. They resented handling during bleeding or weighing prior to loading. They had experienced a short railway journey before the commencement of the present study. The oxen were from one herd and their positions in the herd had been established.

The first truckload was transported without off-loading for four days without food and water. The second truckload was in transit for five days but detrained after 24 and 72 hours on rail for two rest periods of four hours each in railway resting kraals where lucerne hay and water were supplied. The journeys took place at different times over the same route. Thus the conditions were approximately similar.

The normal schedule typical of goods trains, with many halts at sidings, was adhered to. The first group of oxen was in the truck for 96 hours; 44 hours were occupied by actual travel to cover 1116 miles. The second journey took 110 hours to cover 1252 miles. The oxen spent 8 hours in resting kraals; actual travelling time was 51 hours.

On the first journey the temperatures recorded inside the truck on the first day varied from 100°F to 103°F, dropping to 70°F after midnight. Later the temperature inside the truck varied from 80°F to 95°F for most of the time only dropping to 70°F after midnight. On the second journey the temperature inside the truck usually varied from 80°F to 95°F but dropped to 70°F in the early morning.

The observations were on lines similar to those of Bisschop[1].

Behaviour in the trucks

The behaviour of both groups of cattle in transit was similar.

Initially they were restless and anxious. They moved around in the truck, interfered with one another and held their heads high, well above the level of the back. The eyes were prominent, staring and showed the white. Some animals gave a soft, low moan. This stage lasted about 30 minutes.

Once they had settled down they stood still with the weight evenly distributed on all four feet and the head held either with the poll at the same height as the back or lowered with the muzzle about 6 inches above the floor of the truck. The animals only raised their heads if they were disturbed.

The position in which the animals stood in the truck was governed by the motion of the train. Whilst the train was stationary, the cattle stood in hapharzard positions. As soon as the train started to move, the oxen invariably changed their positions so that they stood parallel to each other across the length of the truck, i.e. at right angles to the direction of travel of the train. The heads would face one or the other side of the truck. This position appeared to be the best to counteract the sideways sway of the truck when the train was moving. When the train stopped the oxen tended to move out of this position but immediately re-assumed it when the train started to move.

Change of position took place mainly while the train was stationary or just after it started or stopped. In doing so they usually raised their heads and pushed in forwards between other ani-

*Veterinary Research Institute, Onderstepoort.
**Animal Husbandry and Dairy Research Institute, Irene.
***Livestock and Meat Industries Control Board, Pretoria.

1. Typical arrangement in truck when the train is moving.

mals or backed out with the head held low until they found a place into which they could go forwards. Positions were frequently changed and an animal would find itself at one or other end of the truck, or in the middle at various times. It was exceptional for an animal to change its position while the train was moving once the typical parallel pattern across the truck had been formed.

Horned animals influenced movement. If three horned animals stood parallel to each other with their heads together, the one in the middle would show signs of discomfort by raising or lowering its head in an attempt to avoid the horns of the other two. Soon it would seek a new position. Avoiding movements to escape from horns took place. When horned animals changed their position they raised their heads and the horns tended to rest on the rump, loin or back of other animals during change of position. Although these animals were accustomed to one another and no deliberate attack on another was seen, one sometimes took a short sideways swing of the head at another

2. Effect of horns. Avoiding action during stationary period.

animal which either moved away slightly or else looked for a new position.

The oxen which were not offloaded *en route* all lay down in the truck at one time or another. The first one was seen to lie down 20 hours after the journey had started. After 26 hours others also started to lie down. Sometimes the period spent lying down was short — about 15 minutes at a time — and at others long: up to five hours at a stretch. As many as four were seen lying down at the same time, almost always at the end of the truck and always across it. They assumed sternal position, usually with the head held forwards but sometimes turned back to rest on the flank. Oxen were recumbent while the train was stationary and when it was moving. When animals were lying down and the train was moving the standing animals usually maintained the typical parallel pattern across the truck. Occasionally the animals nearest the one lying down stood lengthwise in the truck with their heads over it. Standing animals were careful not to tramp on recumbent ones. They removed the hoof as soon as they felt the yielding body underfoot and put their hoof down elsewhere. It appears that trampling would be unlikely if the animals were calm and not thrown off balance.

The oxen which were given rest periods in the kraals en route also lay down on the trip but not during the first 24 hours of travel. The first one lay down in the truck 15 hours after the first rest period, after which others also started to lie down. After the second rest period 17 hours elapsed before the oxen started lying down. As many as three were seen lying simultaneously, invariably at one end of the truck and across it. Some never lay down in the truck at all.

At no time did any of the animals bellow. Some gave low moans and others were heard grinding their teeth.

Behaviour in Kraals.

After four days of travel without food or water cattle showed signs of thirst and went straight to water when offloaded. Lucerne hay in clear view was ignored until thirst was quenched.

Animals offloaded into rest kraals during the journey showed no marked preference for food or water, and no eagerness for either. They had a marked urge to walk around slowly before settling down to eat and drink, and soon thereafter all lay down in the sternal position.

During the first rest period oxen began to lie down 40 minutes after having been offloaded and after two hours all of them were recumbent. At the second rest behaviour was practically identical.

In the first rest period the oxen consumed an average of four gallons of water and five lb of lucerne hay. During the second rest they used an average of six gallons of water and 3¾ lb of hay per animal. (Unfortunately the water trough leaked so the consumption figure is not quite accurate).

Loading and Offloading

The oxen gave remarkably little difficulty in loading and offloading. They walked through the loading crush into the truck without any trouble. All that was necessary was to urge them on with the voice, show a raised arm behind them or give them a slap with the flat hand on the rump, allowing them enough time to walk into the truck at their own pace. When offloading, they walked quietly out of the truck as soon as they realized the door was open. The door of the railway truck was in the middle of its side. This caused confusion to the animals during loading because they were not sure whether they should turn left or right once inside the truck. Some would turn round to face the door and make it more difficult for the others to enter.

Natural Functions

Both groups of oxen were bled and weighed before being loaded. During loading they defaecated and urinated freely. This probably had its effect on elimination during the journeys.

The group on the through journey defaecated and urinated very little. Small quantities of hard dry pellets of dung, which were soon trampled fine, were voided even up to the fourth day. The truck floor remained dry with little dung on it. Urine patches were occasionally seen on the truck floor even on the fourth day.

The group given rest, food and water during the journey produced far more dung and urine which accumulated on the truck floor until a layer of wet, pasty material was present underfoot. Urination was profuse for an hour after reloading subsequent to consumption of food and water in the rest kraals.

There was a smell of ammonia in the truck with both lots of animals but it was much stronger and more marked in the case of the animals given food and water during the journey.

There was practically no defaecation and urination by the cattle in the rest kraals while they were feeding, drinking and resting.

Rumination was infrequent in both groups of animals either in the truck or resting kraals. No unusual respiration was noticed.

External appearance

The group which travelled without rest, food and water had beads of moisture on their muzzles during the first day but by the second day the muzzles were dry with a little dirty mucus in the nostrils. Those given rest, food and water on the journey showed beads of moisture on their muzzles during the whole journey as well as a little dirty mucus in their nostrils.

Salivation was seen in some of the animals in both groups only up to the second days of the respective journeys.

The eyes of the animals not given food and water were deeply sunken in their sockets by the fourth day. The others did not show this.

By the end of the journeys the ribs of all animals were prominent and the flanks hollow but it was far more marked in those not given food and water.

Those not given food and water were listless at the end of the journey. On offloading they were stiff, especially in the hocks and walked unsteadily with a swaying gait. They raised their tails, arched their backs, strained and attempted to defaecate but were unsuccessful. They did not urinate. The others did not appear stiff and walked slowly and steadily. They voided some hard dry dung but did not urinate.

Conclusions

1. The general pattern of behaviour was very similar to that seen by Bisschop[1].
2. Cattle soon settle down to travel restfully in a railway truck.
3. The position taken up in the truck is governed mainly by the movement of the train.
4. Change of position takes place mainly wh the train is stationary and on stopping a starting.
5. Horns cause discomfort both to the horn animal as well as its companions.
6. Cattle cannot travel for much longer th 24 hours on rail before they would be forc to lie down in the truck. Given suitable re periods in kraals they might not find th necessary.
7. Cattle which are lying down in the truck a not trampled if their companions can avoid i
8. Cattle on a long train journey without res food and water develop thirst and go direct to water on being offloaded. After their thir is quenched they feed.
9. Cattle given rest periods in kraals during long train journey show no definite preferen for food or water. They exercise themselve eat and drink as they feel inclined and whe satisfied, lie down to rest. The food and wat intake is small.
10. Loading and offloading are facilitated b giving the cattle plenty of time to move a their own pace and by working quietly.
11. A rail journey of four days without food an water causes excessive thirst, fatigue and stiff ness and also depresses functions such as de faecation, urination and sweating.
12. Many factors can affect the behaviour c cattle in trucks so that although a simila pattern would be expected with most con signments it need not necessarily be so.

ACKNOWLEDGEMENTS

The authors wish to thank the Chiefs of the Institutes concerned for permission to publish this article and the S.A Railways for providing the transport facilities and arranging the journeys.

Mr. T. W. P. Beuster and the Photography section of the Onderstepoort Veterinary Research Institute are thanke for their assistance.

The co-operation of Mr. E. F. Daff, Mr. J. J. Swart and Mr. H. J. van Wyk of the South African Railways wa invaluable and the work could not have been done without their help.

REFERENCES

1. Bisschop, J. H. R. 1961 J.S. Afr. vet. med. Ass. 32: 235.

THE ANTHELMINTIC ACTIVITY OF TETRAMISOLE* AGAINST GASTROINTESTINAL WORMS AND LUNGWORMS IN SHEEP

J. L. PRETORIUS, P.O. Box 11260, Johannesburg.

SUMMARY

The activity of tetramisole has been demonstrated against *Haemonchus contortus, Ostertagia circumcincta, Trichostrongylus* spp., *Oesophagostomum columbianum, Gaigeria pachyschelis, Nematodirus* spp., *Trichuris* spp., and *Chabertia ovina* in three series of experiments involving 70 sheep. Tests against *Dictyocaulus filaria* were carried out on 25 young sheep.

Doses were varied from 5-15 mg/kg to establish the optimum oral dose.

At the lower levels (at 5 and 7½ mg/kg) excellent results were obtained against the mature stages of *Haemonchus, Trichostrongylus, Nematodirus, Chabertia, Oesophagostomum,* and *Gaigeria,* while against *Ostertagia* these doses eliminated approximately 66% of both adult and immature forms. Of four sheep given 5 mg/kg 28 days after infection with *Dictyocaulus,* three contained no worms and one only three worms.

At 10 mg/kg tetramisole gave excellent results against both mature and immature forms of all the above mentioned parasites except that in immature *Osteragia,* the results were erratic, against *Trichuris,* there was no effect, and against the third stage larvae of *Oesophagostomum* the effect was not worthwhile.

At 15 mg/kg the drug proved highly effective against all immature and adult stages of the parasites listed, including *Dictyocaulus* from the seventh day after infestation.

INTRODUCTION

The first publication on tetramisole by Thienpont *et al*[1] in 1966 was followed by a paper by Walley[2] comprising the results of both laboratory and field studies and some toxicological data. Results of work in Australia are being compiled by Forsyth[3], and by other workers elsewhere.

It was considered advantageous to test this drug under South African conditions.

MATERIALS AND METHODS

Tetramisole (dl, 2, 3, 5, 6- tetrahydro-6-phenyl-imidazo (2, 1-b) thiazole hydrochloride) (I.C.I. 50,627) is a white odourless amorphous powder with a slightly bitter taste. It is 19% soluble in water at 60°F, forming a clear solution. For ease of handling and measurement a 1% solution was used for most of this work. Animals were dosed individually by means of a drenching gun fitted with an oesophageal tube. The following animals were used in the experiment:—

Group I — Merino and Merino X German Merino

Day	*O. circumcincta*	*T. colubriformis*	*O. columbianum*
—41	—	—	100
—37	—	—	50
—34	—	—	65
—30	—	—	52
—27	—	—	50
—23	410	380	45
—20	180	450	60
—16	175	450	55
—13	155	270	65
— 8	210	225	60
— 5	220	400	85
— 2	300	550	150
Total	1,650	2,725	837

Group II — Merino and Merino X Hampshire Down

Day	*H. contortus*	*T. colubriformis*	*O. columbianum*	*G. pachyschelis*
—49	—	—	—	y0
—42	—	—	—	55
—24	310	—	—	—
—21	330	—	—	—
—18	305	—	—	—
—15	280	—	—	—
—10	300	—	—	—
— 6	500	500	280	—
— 5	340	580	140	—
— 4	500	650	150	—
Total....	2,865	1,730	570	115

*"Tramisol"—(I.C.I. South Africa) Ltd. (d, 1-2, 3, 5, 6-tetrahydro-6-phenyl-imidazo- (2, 1-b)-thiazole hydrochloride).

Group III — Merino X Dorper (Dorset Horn X Blackhead Persian)

Day	*C. ovina*	*H. contortus*	*O. columbianum*
—54	10	—	—
—50	9	—	—
—46	11	—	—
—41	9	—	—
—36	7	—	—
—31	18	—	—
—25	32	—	—
—22	22	—	225
— 2	30	2,000	—
Total..	144	2,000	225

Group IV — Merino lambs.

Day 0 — 1,500 *Dictyocaulus* larvae
Day + 7 — 15 mg/kg tetramisole to 3 sheep.
Day + 14 — 15 mg/kg ,, ,, 3 ,,
Day + 21 — 15 mg/kg ,, ,, 3 ,,
5 mg/kg ,, ,, 2 ,,
Day + 28 — 15 mg/kg ,, ,, 4 ,,
5 mg/kg ,, ,, 3 ,,
Day + 35 — All experimental and 6 control sheep slaughtered.

Groups I-III — varied in age from 6 months to adult, and Group IV consisted of lambs aged about 4 months and in very poor condition.

The larval anthelmintic test of Reinecke[4] was used throughout the work on stomach and bowel worms, although sheep reared under worm-free conditions were unobtainable. All animals were therefore treated with 100 mg/kg of thiabendazole and 100 mg/kg of "Lintex", some 14 days before the artificial infestation, and kept under worm-free conditions thereafter. Because the "take" under these conditions might be somewhat lower than in animals raised worm-free from birth, doses of larvae given were slightly in excess of the numbers suggested by Reinecke. Infective larvae were administered orally twice weekly, except for *G. pachyschelis* which were placed on the skin of the neck on the first day and on the skin of the hindquarters on the second day.

Trichuris spp. infestation occurred naturally in Groups I and II, and *Nematodirus* spp. infestation in Group III. It is presumed that these species survived the preliminary anthelmintic treatment.

The animals were infested as follows:—

The total worm burdens of *H. Contortus, O. circumcincta* and *T. colubriformis* were estimated by the dilution technique described by Reinecke[5]. Total worm counts were made of the immature and mature forms of *O. columbianum, G. pachyschelis, C. ovina* and *Trichuris* spp. The lambs in Group IV were heavily infested with gastro-intestinal parasites. When purchased, they were dosed with 30 mg/kg of tetramisole and moved to concrete floors 14 days before infestation with a single dose of 1,500 *D. filaria* larvae. (It was interesting to note that these animals showed marked clinical improvement after 7 days).

The lungs were carefully dissected and all visible worms collected and counted. The lungs were then cut up into small pieces measuring about ¼" x ½" and then washed under pressure through a 100 mesh sieve. This washed material together with the sliced lungs was then kept above room temperature in normal saline solution, each pair of lungs being made up with saline to approximately 1 litre. After about 6 hours, this material was again passed through a 100 mesh sieve and the washings collected. Worm tails were then counted under a stereo-microscope after staining with iodine and decolourising with sodium thiosulphate solution.

Results

The results are summarized in tables 1-4.

Discussion and Conclusions

Although the numbers of larvae given were in excess of the numbers suggested by Reinecke[4], the numbers recovered post mortem from the control animals were rather small. However, the variation was not great, and the overall results are in line with those obtained by other workers.

Tetramisole proved to be highly efficient and at a dosage of 15 mg/kg it removed practically every worm of every species. It is suggested that this dose should be the one to be generally recommended.

The following paragraphs discuss the effect of the drug on particular parasites.

H. contortus

At 5 mg/kg practically all adult worms were removed; against the fifth stage the efficacy was 88%. It must be remembered that *post mortem* examination was carried out 7 days following treatment, i.e. these parasites were probably fourth stage larvae at the time of treatment. It is quite evident that the drug is highly effective against the young adults. The fact that 210 worms were recovered from one sheep, suggests that a dose of 5 mg/kg has rather an erratic effect against the fourth stage of the parasite. On the other hand, a dose of 7½ mg/kg gave a uniformly good effect against all stages. Where specific outbreaks of this parasite occur, tetramisole could presumably be

TABLE 1.—NUMBER OF WORMS RECOVERED. GROUP I.

Dosage	Sheep No.	*O. circumcincta*				*T. colubriformis*				*O. columbianum*				*Trichuris* spp.
		*4th	*5th	Adult	Total	*4th	*5th	Adult	Total	*4th	*5th	Adult	Total	Total
Controls.....	B8	170	160	130	556	117	95	245	447	60	50	45	155	15
	B12	198	290	430	928	87	715	770	1572	12	75	90	177	20
	B7	10	20	30	50		Less than ten			10	—	—	10	—
	B13	70	125	155	350	0	125	360	485	43	25	70	138	25
	B21	123	135	248	506	153	269	525	947	17	35	60	112	20
	B25	20	50	40	110	74	311	466	851	10	25	25	60	10
	B27	40	307	140	487	10	288	762	1060	38	45	50	133	10
Average.....		90	156	167	426	64	259	446	767	27	36	48	112	14
5mg/kg.....	B5	27	83	85	195		Less than ten		10	25	22	10	57	10
	B6	73	125	100	298		Less than ten		10	12	12	0	24	0
	B11	40	37	10	87		Less than ten		10	20	0	0	20	0
Average.....		46	82	65	193				10	19	11	3	101	
Efficiency.....		49%	47%	61%	53%	99.5%	99.5%	99.5%	98%	N.S.E.	N.S.E.	N.S.E.	N.S.E.	
7½mg/kg.....	B4	0	20	0	20		Less than ten			10	0	0	10	0
	B2	101	123	87	311		Less than ten			10	0	0	10	10
	B17	40	120	175	235		Less than ten			10	0	0	10	0
Average.....		47	88	87	172	10	10	10		10	0	0	10	
Efficiency.....		47%	43%	50%	60%	99.8%	99.8%	99.8%	99.8%	N.S.E.	N.S.E.	N.S.E.	N.S.E.	
10mg/kg.....	B9	43	120	130	193		Less than five		5	10	10	10	30	12
	B10	4	10	12	26	0	0	0	0	0	0	0	0	0
	B19	32	32	40	104	0	0	0	0	0	55	0	55	45
	B20	20	—	—	20	0	0	0	0	0	10	0	10	20
	B23	—	20	—	20	0	0	0	0	0	0	0	0	20
Average.....		25	36	36	72	0	0	0	0	2	15	2	19	
Efficiency.....		77%	76%	78%	83%	99.5%	99.5%	99.5%	99.5%	90%	42%	95%	83%	N.S.E.
12½mg/kg.....	B3	20	0	0	20	0	0	1	1	10	0	0	10	10
	B14	10	0	0	10	0	0	1	1	10	0	0	10	—
	B18	65	80	15	160	0	0	0	0	0	0	0	—	10
	B22	20	15	10	45	0	0	0	0	8	0	0	8	—
	B24	0	0	0	0	0	0	0	0	10	0	0	10	—
Average.....		23	19	5	47	0	0	0	0	8	0	0	8	
Efficiency.....		74%	87%	97%	88%	100%	100%	100%	100%	70%	100%	100%	93%	N.S.E.
15mg/kg.....	B1	0	20	10	30	0	0	0	0	10	0	0	10	10
	B16	0	0	0	0	0	0	0	0	10	0	0	10	15
	B26	0	60	35	95	0	0	0	0	20	0	0	20	7
Average.....		0	27	15	42	0	0	0	0	13	0	0	13	
Efficiency.....		100%	82%	91%	90%	100%	100%	100%	100%	48%	100%	100%	88%	N.S.E.

*Larval Stage.
N.S.E. = No significant effect.

used at half the standard dose (i.e. 7½ mg/kg) with complete confidence. This is of particular interest following Reinecke's[5] demonstration that phenothiazine may be less effective against this parasite than hitherto believed.

O. circumcincta

This parasite proved to be the most resistant to treatment with tetramisole, and it is for this reason that suggested dosage for general use was set at 15 mg/kg. From 12½ mg/kg upwards, its efficacy was over 80%, except against fourth stage larvae.

As there was considerable variation in the numbers of parasites recovered from animals treated at the same dose level, one suspects that under field conditions results might be erratic if less than the full dose were used.

T. colubriformis

This parasite was used in both Group I and Group II, and was completely eliminated by doses as low as 5 mg/kg.

O. columbianum

All the experimental sheep were infested with this parasite when purchased, and thus presumably had acquired a varying degree of immunity which would account for the big variation in "take".

Tetramisole removed all the more mature stages at 10 mg/kg and over. Against fourth stage larvae,

TABLE 2.—NUMBER OF WORMS RECOVERED. GROUP II.

Dosage	Sheep No.	*H. contortus*				*T. colubriformis*				*O. columbianum*				*G. pachyschelis*	*Trichuris* spp.
		*4th	*5th	Adult	Total	*4th	*5th	Adult	Total	*4th	*5th	Adult	Total	Adult	Adult
Control.....	A7	219	146	106	471	0	0	0	0	27	21	24	72	18	4
	C7	365	215	440	1020	0	0	645	645	96	—	5	101	44	10
	A1	335	195	290	820	0	2	0	2	26	—	7	33	11	—
	A9	480	360	578	1418	0	187	325	512	70	—	—	70	26	1
	A12	540	340	750	1630	0	0	735	735	67	—	4	71	26	2
	A15	745	490	711	1946	0	0	397	397	126	—	3	129	36	7
Average.....		447	291	479		0	31	350	382	68	315	7	78	27	4
5mg/kg.....	C4	10	23	3	36	0	0	0	—						
	C8	27	—	—	27		Contents of intestines lost.				Contents of intestines lost			—	—
	C10	210	78	—	288	—	31	—	31	119	—	3	122	—	9
Average.....		82	33	1	117	No statistical value				No statistical value					—
Efficiency.....		81%	88%	99%	—										0
7½mg/kg.....	C3	10	10	0	20	0	0	0	0	2	0	0	2	1	7
	C12	0	0	0	0	0	0	0	0	23	0	0	23	0	3
	C15	0	0	0	0	0	0	0	0	26	0	0	26	0	1
Average.....		3	3	0	7	0	0	0	0	17	0	0		03	—
Efficiency.....		99%	98%	100%	—	100%	100%	100%			100%	100%		98%	0
10mg/kg	C1	0	0	0	0	0	0	1	1	17	0	0	17	1	0
	C2	0	0	0	0	0	0	0	0	18	0	0	18	0	1
	C5	0	0	0	0	0	0	0	0	6	0	0	6	1	0
	C6	10	10	10	30	0	0	0	0	24	0	0	24	0	20
	C9	10	0	0	10	0	0	0	0	2	0	0	2	1	17
Average.....		4	2	2	8	0	0	1	1	13			13	1	—
Efficiency.....		99%	99%	99%	—	100%	100%	99%	99%	80%	100%	100%	80%	85%	N.S.E.
12½mg/kg.....	C11	0	0	0	0	0	1	0	1	17	0	0	17	0	20
	C13	0	0	0	0	0	0	0	0	8	0	0	8	0	12
	C14	0	0	0	0	0	0	0	0	30	0	0	30	0	15
	C16	0	0	0	0	0	0	0	0	7	0	0	7	0	25
	A3	0	0	0	0	0	0	0	0	27	0	0	27	0	4
Average.....		0	0	0	0	0	0	0	1	18	0	0		0	—
Efficiency.....		100%	100%	10%	100%	100%	99%	100%	99%	73%	100%	100%	73%	100%	N.S.E.
15mg/kg.....	A5	0	0	0	0	0	0	0	0	1	0	0	1	0	0
	A10	0	0	0	0	0	0	0	0	25	0	0	25	0	20
	A11	0	0	0	0	0	0	0	0	4	0	0	4	0	31
	A13	0	0	0	0	0	0	0	0	53	0	0	53	0	14
	A16	0	0	0	0	0	0	0	0	1	0	0	1	0	0
Average.....		0			0				0	17	0	0	17	0	—
Efficiency.....		100%	100%	100%	100%	100%	100%	100%	—	75%	100%	100%	78%	100%	N.S.E.

* = Larval stage.

results were somewhat erratic, lower doses being apparently more effective than higher ones. They differ in this respect from those of Reinecke[4], who found a dose of 15 mg/kg to be 89 to 100% effective against all stages.

G. pachyschelis

Uniform numbers of parasites were present in the control animals, and doses of 7½ mg/kg and over proved highly effective, thus confirming the results of Reinecke[4].

N. spathiger

This group of sheep received 100 mg/kg of thiabendazole about 6 weeks before the beginning of the experiment, and were kept on cement floors thereafter. Pens were cleaned out daily. As infective larvae of *N. spathiger* were not obtainable at the time, the sheep could not be infested experimentally. The experiment started a few weeks before the holiday season, and effective control of the cleaning of the pens was not assured. Probably some of the animals were carrying a few parasites, and infestation built up over that period. However, the fact that all controls carried the parasite at all stages of development indicates that the whole group was infested.

Very few *N. spathiger* indeed were recovered from treated animals, indicating an anthelmintic efficiency similar to that reported by Walley[2].

TABLE 3.—NUMBER OF WORMS RECOVERED. GROUP III.

Sheep No.	Dose mg/kg	*H. contortus* *4th stage	*C. ovina* *4th	*C. ovina* *5th	*C. ovina* Adult	*O. columbianum* *4th stage	*N. spathiger* *4th	*N. spathiger* *5th	*N. spathiger* Adult
E6	0	248	5	1	8	4	97	3	18
E16	0	140	24	30	60	36	63	5	7
D13	0	120	2	4	9	12	113	26	30
D5	0	200	25	5	7	52	185	14	110
Average		177	14	10	21	26	114	12	41
D15	7.5	120	10	0	0	29	75	0	0
D16	7.5	0	10	3	6	10	0	0	0
Average		60	5	1	3	20	37	0	0
Efficiency		60%	64%	90%	85%	23%	67.5%	100%	100%
E18	10	0	0	0	0	12	—	—	—
D21	10	0	0	0	0	5	—	—	—
E8	10	0	—	—	—	—	—	—	—
Average		0	0	0	0	8	—	—	—
Efficiency		100%	100%	100%	100%		69%	—	—
D10	12.5	10	3	0	0	0	15	0	0
E19	12.5	0	0	0	0	0	0	0	0
D19	12.5	0	0	0	0	0	0	0	0
Average		3	1	0	0	0	5	0	0
Efficiency		98%	92%	100%	100%	100%	95%	100%	100%
A8	15	0	0	0	0	4	0	0	0
D6	15	0	0	0	0	5	7	0	0
E20	15	0	0	0	0	7	0	0	0
D14	15	0	0	0	0	0	0	0	0
Average		0	0	0	0	5	2	0	0
Efficiency		100%	100%	100%	100%	80%	98%	100%	100%

Trichuris spp.

No significant effect was shown against this parasite, which was found in small numbers in all the experimental animals.

C. ovina

In order to avoid the purgation which is associated with heavy infestations of this parasite and which results in the expulsion of many worms, very small numbers of infective larvae of this species were dosed. One sheep with a total of 114 *Chabertia* did in fact purge severely on the day of treatment, and this condition improved only slightly in the week before slaughter. Overall results against this parasite were very good.

D. filaria

Although the method used for recovering the parasite was somewhat primitive, it confirmed Walley's[2] finding that tetramisole is a most effective drug against all stages of this lungworm. Even at 5 mg/kg, it remained highly effective against the mature stage.

TABLE IV—NUMBER OF D. FILARIA RECOVERED. GROUP IV.

Dose	Sheep No.	Time of treatment Post-infestation	No of parasites at 35 days (slaughter)	Average	Efficiency
Nil	C8	Untreated	43		
,,	C9	,,	93		
,,	C18	,,	242		
,,	C19	,,	235		
,,	C7	,,	266		
,,	C15	,,	290	195	
15 mg/kg	C23	7 days	1		
,,	C1	,,	28		
,,	C2	,,	106	45	77
15 mg/kg	C3	14 days	5		
,,	C4	,,	2		
,,	A14	,,	1	3	98
15 mg/kg	C6	21 days	0		
,,	C12	,,	2		
,,	E16	,,	3	1.6	99
15 mg/kg	C22	28 days	1		
,,	C16	,,	0		
,,	A4	,,	0		
,,	C5	,,	0	0.25	99
5 mg/kg	C10	21 days	46		
,,	C17	,,	0	23	88
5 mg/kg	C13	28 days	3		
,,	C11	,,	0		
,,	C20	,,	0	1	99

REFERENCES

1. Thienpont, D.C.L. *et al* (10 authors) 1966 *Nature* 209:104.
2. Walley, J. K. 1966 *Vet. Rec.* 78:406.
3. Forsyth, B.A. In press.
4. Reinecke, R. K. 1966 *J.S. Afr. vet. med. Ass.* 37:27.
5. Reinecke, R. K. 1966 *J.S. Afr. vet. med. Ass.* 37:133.

Hibitane

Foremost skin antiseptic

HIBITANE is bactericidal and bacteriostatic . . . broad spectrum in action . . . stable and active in presence of blood and pus.

"HIBITANE" EFFERVESCENT PESSERIES

Ideal for routine treatment as well as for purulent metritis. Not inactivated by pus or body fluids. Each pessary contains 1 gm Hibitane.

"HIBITANE" OBSTETRIC CREAM

A bland non-irritating cream. Ideally suited to protect hands and arms against bacterial infection in rectal examinations. Contains 1% Hibitane.

"HIBITANE" INDUSTRIAL CREAM

A non-greasy cream to prevent cross infection in surgery in the field. Contains 1% Hibitane.

ICI UDDER WASH

Ideal for "pre-op" skin prepping.
Economical to use.

Dilution: 1 pint ICI udder wash
1½ pints distilled water
7½ pints spirit

Contains: 7½% Hibitane

Dairy Hygiene . . . Eliminates all bacterial organisms causing mastitis.
For prevention, use 6 c.c. to 2 gallons water.
For treatment, use 24 c.c. to 2 gallons water.

HIBITANE IS A PRODUCT OF ICI RESEARCH

ICI SOUTH AFRICA (PHARMACEUTICALS) LTD.

P.O. Box 11270, Johannesburg.

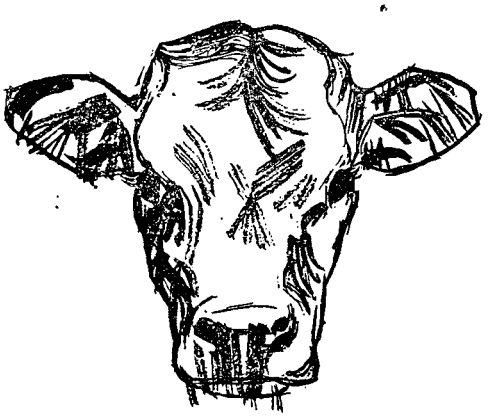
Branches at : Cape Town, Durban, Port Elizabeth and Salisbury.

TYLAN 200

Tylosin

A new and more effective antibiotic for Cattle Diseases

pneumonia
foot rot
leptospirosis
wound infections



metritis
shipping fever
contagious calf pneumonia
pneumoenteritis of calves

bacterial infections associated with virus diseases

Stockists :
Goldfields Veterinary Supplies
A. S. Ruffel (Pty.) Ltd.
S.A. Cyanamid (Pty.) Ltd.

THE ELANCO DIVISION
LILLY LABORATORIES (S.A.) (Pty.) Ltd.
Short Street - Isando - Transvaal

ANTHELMINTIC AND TOXICITY STUDIES TETRAMISOLE: I. ANTHELMINTIC EFFICACY

D. K. SHONE, J. R. PHILIP*

SUMMARY

(1) Tetramisole has been demonstrated to be highly effective against the immature and adult forms of *Ostertagia circumcincta, Haemonchus contortus, Trichostrongylus colubriformis, Gaigeria pachyscelis, Oesophagostomum columbianum* and *Chabertia ovina.*

(2) The importance of slaughtering the control sheep on the day of treatment when investigating efficacy against the immature stages is discussed.

(3) A method using a water bath for the recovery of immature and adult helminths from the gastro-intestinal tract is described.

(4) It was found that the 4th ecdysis of *G. pachyscelis* was completed by the 31st day in contrast to the previously reported 49 to 56 days.

(5) Following the administration of tetramisole into the rumen, the expulsion of helminths from the caecum and colon commences within 2 hours.

INTRODUCTION

Tetramisole is the generic name of dl 2, 3, 5, 6 - tetrahydro - 6 - phenyl - imidazo (2, 1-b) thiazole, a new anthelmintic compound discovered by Janssen Pharmaceutica (Beerse, Belgium)[1]. In South Africa, Reinecke[2] found that tetramisole was highly effective against the helminth species used in the development of his larval anthelmintic test. The work reported here was undertaken to investigate the anthelmintic efficacy of tetramisole against the immature and adult forms of the major nematode species occurring in South Africa.

MATERIALS AND METHODS

Tetramisole: The hydrochloride salt is a white amorphous powder, up to 20 per cent soluble in water at room temperature. The powder is extremely stable and solutions may be boiled for several hours with only a minor breakdown of tetramisole.

As the commercial product (Ripercol**) was to be marketed as a soluble powder, freshly prepared aqueous solutions were used.

Administration and calculation of doses: All doses were based upon the hydrochloride salt and were calculated from exact bodyweights. The solution of tetramisole was administered by stomach tube directly into the rumen.

Sheep: The sheep used in these trials were chiefly Merino and German Merino lambs but some crossbred lambs (Dorper types) were also used. The lambs were from 4 to 10 months of age.

Infestation of lambs: Except in the case of *D. filaria,* infective larvae were obtained by culturing the faeces from lambs harbouring pure infestations of a single helminth species. The faeces and infective larvae of *D. filaria* were supplied by the Helminthology Section of the Veterinary Research Institute, Onderstepoort.

Post mortem procedures: Methods used to recover the helminths varied, depending upon the nature of the trial and the helminth species.

Adult helminths from the small intestine were collected on 200 mesh to the inch British Standard Specification (B.S.S.) sieves, while those from the abomasum and caecum and colon were collected on 100 mesh to the inch B.S.S. sieves. In trials involving immature stages, a 300 mesh to the inch sieve was used throughout. Before sieving the suspensions, the helminths were killed by adding a strong aqueous solution of iodine.

Total counts were made of all adult helminths with the exception of *Trichostrongylus colubriformis* where the number present in three one-tenth aliquots were counted and the total number calculated. Total counts of immature forms were made or the total calculated from three one-tenth aliquots.

Identification of larvae: Larval stages were identified microscopically according to the descriptions of Veglia[3,4], Ortlepp[5], Douvres[6,7,8], and Threlkeld[9],

*Terenure Research Station, A.S. Ruffel (Pty) Limited, P.O. Box 38, Isando, Transvaal.
**Registered trademark of Janssen Pharmaceutica, Belgium and Johnson and Johnson, South Africa.

and classified according to Reinecke[10] except that sexually immature and mature adults were not differentiated into 5th stage and adults respectively.

Design of Anthelmintic Trials: The controlled anthelmintic trials were in general patterned after Reinecke[2].

Trial No. 1

The efficacy of tetramisole against a natural infestation of *Haemonchus contortus* at a dose rate of 7.5 mgm/kg bodyweight was investigated.

Materials and Methods: Nineteen sheep were purchased in the eastern Free State and transported by road to the research station. The following day 5 sheep were treated and the remaining 14 served as untreated controls. All the sheep were slaughtered on the 4th day after treatment.

Results: The results are presented in Table 1 from which it was calculated that the antelmintic efficacy was 99.9 per cent.

Table 1.—*Haemonchus contortus* adults recovered from untreated sheep and sheep treated with 7.5 mgm of the hydrochloride salt of tetramisole per kg bodyweight.

	Fourteen untreated sheep	Five treated sheep
Total	14,592	5
Range	30 — 5,811	0 — 5
Mean	1,042	1

Percentage reduction—99.9

Trial No. 2

The anthelmintic efficacy of tetramisole against a combined artificially induced infestation of adult *Ostertagia circumcincta* and adult *Oesophagostomum columbianum* at a dose rate of 15 mgm/kg bodyweight was investigated.

Materials and Methods: Fifteen nine month old lambs, reared at the research station, were infested according to the programme set out in Table 2.

Table 2.—Programme of infestation with infective larvae of *O. circumcincta* and *O. columbianum*. (Trial No. 2).

Day.			
Minus 68	O. columbianum	300	Larvae
,, 63	,, ,,	300	,,
,, 58	,, ,,	300	,,
,, 53	O. circumcincta	500	,,
,, 50	,, ,,	250	,,
,, 47	,, ,,	300	,,

Day 0 = day of treatment.

The lambs were randomly allocated to 2 groups; the group of 7 lambs was treated and slaughtered on the 6th day after treatment.

Results: The results are presented in Table 3. The anthelmintic efficacy for *O. circumcincta* adults was 99.9 per cent and for *O. columbianum* adults 99.9 per cent.

Table 3.—Ostertagia circumcincta, Oesophagostomum columbianum and Haemonchus contortus adults recovered from untreated lambs, and lambs treated with 15 mgm of tetramisole per kg bodyweight.

	Seven treated lambs.	Eight untreated lambs.
H. contortus		
Total number recovered	0	137
Mean number recovered	0	19.6
Range	0	10—29
Percentage reduction	100	—
O. circumcincta		
Total number recovered	1	2,724
Mean number recovered	0.14	340.5
Range	0—1	105—458
Percentage reduction	99.9	—
O. columbianum		
Total number recovered	1	392
Mean number recovered	0.14	49
Range	0—1	22—116
Percentage reduction	99.9	—

The *H. contortus* adults which were present were probably naturally acquired, for, while every endeavour was made to rear the lambs free of worms, a periodic shortage of water precluded thorough cleansing of the pens.

Trial No. 3

The anthelmintic efficacy of tetramisole at a dose rate of 15 mgm/kg bodyweight was investigated against a combined artificially induced infestation of adult and immature forms of *Ostertagia circumcincta, Trichostrongylus colubriformis* and *Oesophagostomum columbianum.*

Materials and Methods: Seventeen lambs reared under identical conditions to those used in Trial No. 2 were infested with larvae according to the programme set out in Table 4.

The nine untreated sheep were slaughtered on days 3, 4 and 5 post treatment and the eight treated sheep were slaughtered on days 5, 6 and 7.

Recovery of Helminths: The helminths were recovered by a method based upon a principle suggested by Anderson and Reinecke[10].

A box, 78 inches long by 54 inches wide and 24 inches high with an open top was constructed. The sides were lined with 1 inch polystyrene and the floor with 2 inch polystyrene. A water bath 8 inches deep and constructed of half an inch thick perspex sheets rested upon slotted angle iron,

TABLE 4.—PROGRAMME OF INFESTATION OF WORM-FREE LAMBS WITH INFECTIVE LARVAE OF OSTERTAGIA CIRCUMCINCTA, TRICHOSTRONGYLUS COLUBRIFORMIS AND OESOPHAGOSTOMUM COLUMBIANUM (TRIAL NO. 3).

Day	Number of larvae administered		
	Ostertagia circumcincta.	*Trichostrongylus colubriformis.*	*Oesophagostomum columbianum.*
Minus 60	—	—	300
„ 55	—	—	300
„ 50	—	—	300
„ 45	500	1,000	—
„ 42	250	250	—
„ 39	300	300	—
„ 30	500	500	40
„ 25	500	500	200
„ 20	500	500	200
„ 15	500	500	200
„ 10	500	500	200
„ 7	500	500	200
„ 4	500	500	200
„ 3	500	500	200
„ 2	500	500	200
„ 1	500	500	200
Day 0	Day of treatment.		

fixed 8 inches from the top. Four 6 foot fluorescent light tubes were fitted into the space below.

A frame within the water bath, held 2 inches above the floor, carried 18 transparent trays constructed by lining wire document baskets with clear polythene sheets. Into each tray a second basket lined with 250 micron aperture nylon gauze was fitted so as to allow a 1 inch space between the nylon gauze and polythene. Sufficient normal saline was added to cover the nylon gauze. The sides of the polythene lined trays were covered to a depth of 2 inches with the water in the water bath which was maintained at 42°C with a Thermomix Model 2. The bath was closed with 2 inch thick polystyrene sheets covered with black polythene sheets.

Immediately after slaughter the gastro-intestinal tract was removed, the abomasum and the first 12 inches of the duodenum were opened up and washed with warm normal saline. This suspension was poured onto the nylon gauze of one tray. The rugi and the wall were cut up into small portions and placed on the nylon gauze of a second tray.

The remaining portion of the small intestine was stripped of its mesentery and a plastic insemination pipette with a hole at one end was inserted into the lumen. The intestine was anchored to the pipette and by drawing the small intestine over the point of the pipette, the intestine was completely everted. The exposed mucous membrane surface was washed with normal saline and the suspension placed into one tray. The intestinal wall was placed into two trays.

The caecum and colon were treated as one and similarly to the abomasum. Two trays were used for the suspension of ingesta and one for the walls. The migration of helminths was allowed to proceed for approximately 3 hours.

The material which remained on the upper surface of the nylon mesh is termed the residue and that remaining in the lower tray, after removal of the nylon mesh, is termed the filtrate. The intestinal walls were digested in a solution of pepsin and hydrochloric acid, and is termed the digesta. Thus components obtained from the treatment of the gastro-intestinal tract of each sheep are as follow:—

Abomasum	Small Intestine	Caecum and Colon
ingesta residue	ingesta residue	ingesta residue
ingesta filtrate	ingesta filtrate	ingesta filtrate
wall filtrate	wall filtrate	wall filtrate
wall digesta	wall digesta	wall digesta

Results: The results are presented in Table 5. In calculating the anthelmintic efficacy of tetramisole in this trial, the 3rd and 4th stages have been grouped together as immatures. In view of the dynamic nature of a helminth population in the host, it was not possible in a trial of this design to evaluate the anthelmintic efficacy against each larval stage.

The anthelmintic efficacy, as calculated from Table 5, is:—

Species	Immatures	Adults
O. circumcincta	99.71 per cent	97.96 per cent
T. colubriformis	99.91 per cent	99.75 per cent
O. columbianum	71.69 per cent	99.75 per cent

The results of the illuminated water bath method for the recovery of helminths is dealt with later in this paper.

TABLE 5.—DETAILS OF OSTERTAGIA CIRCUMCINCTA, TRICHOSTRONGYLUS COLUBRIFORMIS AND OESOPHAGOSTOMUM COLUMBIANUM RECOVERED FROM UNTREATED LAMBS AND LAMBS TREATED WITH 15 MGM OF TETRAMISOLE PER KG BODYWEIGHT.

	Ostertagia circumcincta			*Trichostrongylus colubriformis*			*Oesophagostomum columbianum*		
9 Untreated lambs.	3rd	4th	Adult	3rd	4th	Adult	3rd	4th	Adult
370	76	334	440	133	320	889	63	77	115
379	36	404	472	56	432	726	23	119	40
388	0	382	596	0	544	1044	0	115	38
390	0	339	537	0	675	1823	0	257	55
397	15	186	417	0	389	912	0	66	44
402	4	274	304	7	675	851	0	54	19
403	100	294	668	82	428	1053	0	91	35
405	7	344	649	88	593	1079	0	98	59
413	0	290	729	0	743	1399	0	114	62
Total	246	2847	4812	366	4799	8377	86	991	467
Mean	27.44	316.33	534.67	40.67	533.22	930.78	9.56	110.11	51.89
8 Treated lambs.									
385	0	0	8	0	0	0	3	26	0
391	0	0	2	0	0	0	1	38	0
393	0	0	1	0	0	0	0	17	0
395	0	5	67	0	4	6	0	109	0
396	0	0	0	0	0	1	0	4	0
404	0	0	0	0	0	3	0	51	0
406	0	0	8	0	0	0	0	16	0
408	3	0	1	0	0	9	0	6	1
Total	3	5	87	0	4	19	4	267	1
Mean	0.38	0.63	10.9	—	0.5	2.38	0.5	33.38	0.13
Percentage reduction	99.71		97.96	99.91		99.75	71.69		99.75

While processing the gastro-intestinal tract of the untreated lambs, it was noted that a large number of nodules caused by immature stages of *O. columbianum* was present in the walls. As the wall kept tearing at the site of these nodules, the stripping of the mesentery from the intestine was made difficult. In marked contrast, few or no nodules were found in the intestinal walls of the treated sheep. Counts of the nodules present were not made, as the difference was only observed when the treated sheep were processed after the untreated sheep had been dealt with. The interval lapsing between treatment and slaughter was either 5, 6 or 7 days.

TRIAL NO. 4

The anthelmintic efficacy of tetramisole at a dose rate of 15 mgm/kg bodyweight against *Dictyocaulus filaria* of various ages was investigated by the treatment of artificially infested sheep at intervals after infestation.

Materials and Methods: In view of the difficulty of establishing repeated infestations of *D. filaria*, it was decided to infest all the sheep simultaneously and to treat at various intervals after infestation.

Twenty one lambs purchased in the Pretoria area were each given 1,000 infective larvae *per os*.

On each of days 4, 13 and 35 post infestation, 5 lambs were treated. The remaining 6 lambs served as untreated controls and were slaughtered on day 36. The lambs treated on days 4 and 13 were slaughtered on day 37, and the lambs treated on day 35 were slaughtered on day 39.

The bronchi and bronchioles were meticulously opened and all helminths observed were removed and placed into normal saline. The saline solution was heated to 60°C to kill the helminths and to

separate the worms. The lung tissue was then cut up into ½ to ¾ inch portions which were held immersed in normal saline at 42°C over nylon gauze in an illuminated water bath for 5 hours. The helminths which passed through or were trapped in the nylon gauze, were concentrated on a 100 mesh to the inch B.S.S. sieve. Undamaged helminths were removed and counted. The remaining material was examined microscopically and all tail ends of *D. filaria* were identified and total counts obtained.

Results: The results are presented in Table 6.

TABLE 6.—DICTYOCAULUS FILARIA RECOVERED FROM UNTREATED LAMBS AND LAMBS TREATED WITH 15 MGM OF TETRAMISOLE PER KG BODYWEIGHT.

Sheep No.	Age in worms in days		*D. filaria* recovered from lungs at autopsy		
	at treatment	at slaughter	direct	water bath	total
619			171	27	198
620			365	1	366
623	Control	36	154	12	166
625			341	8	349
627			507	10	517
632			619	0	619
			Mean		369
614			83	14	97
618			0	116	116
622	4	37	64	15	79
626			224	13	237
629			93	14	107
	Percentage reduction	65.6%			127
613			2	7	9
616			2	0	2
617	13	37	11	1	12
624			13	10	23
630			0	2	2
	Percentage reduction	97.4%			10
615			0	0	0
621			8	7	15
628	35	39	2	0	2
631			8	7	15
633			0	2	2
	Percentage reduction	98.5%			7

The anthelmintic efficacy was calculated for each age of *D. filaria*.

Age	Anthelmintic efficacy
4 day old *D. filaria*	65.6 per cent
13 day old *D. filaria*	97.4 per cent
35 day old *D. filaria*	98.5 per cent

Numerous gravid females and first stage larvae were recovered in the untreated lambs (36 days post infestation) while the helminths recovered from the treated lambs were smaller and no gravid females were present.

TRIAL NO. 7

The anthelmintic efficacy of tetramisole at a dose rate of 15 mgm/kg bodyweight against immature and adult forms of *Haemonchus contortus* was investigated.

Materials and Methods: Ten Merino lambs purchased in the eastern Free State were used. Faecal samples examined by the McMaster method were negative for worm eggs.

The lambs were infested with infective larvae produced from cultures of faeces collected from sheep with pure infestations of *H. contortus*. In three of the sheep, filter paper onto which the larvae had been taken, was later recovered from the abomasum.

Details of the programme of infestation is presented in Table 7. Five lambs were treated and five served as untreated controls. The control lambs were slaughtered on the day the remaining lambs were treated. The latter were slaughtered 2 days later.

The helminths were recovered using the illuminated water bath method described previously.

TABLE 7. THE PROGRAMME OF INFESTATION OF LAMBS WITH INFECTIVE LARVAE OF HAEMONCHUS CONTORTUS (TRIAL NO. 7).

Day	No. of larvae
Minus 20	360
„ 18	750
„ 15	750
„ 12	750
„ 8	750
„ 5	750
„ 3	750
„ 2	1,000
„ 1	1,000
„ 0	Day of treatment and the controls slaughtered
Plus 2	Treated lambs slaughtered

Numerous adults were found caught up in the nylon mesh. No attempt was made to remove them but the nylon was spread out on trays and flooded with water. The helminths were then counted *in situ*.

Results: The results are presented in Table 8. The anthelmintic efficacy was calculated for each helminth stage. None of the adults was sexually mature.

Stage	Anthelmintic efficacy
H. contortus — adults	99.83 per cent
H. contortus — 4th stage	99.75 per cent
H. contortus — 3rd stage	72.55 per cent

TRIAL NO. 8

The anthelmintic efficacy of tetramisole at a dose rate of 15 mgm/kg bodyweight against immature and adult forms of *Gaigeria pachyscelis* and *Chabertia ovina* was investigated.

Materials and Methods: Ten Merino lambs purchased in the eastern Free State were used. Faeces samples examined by the McMaster method were negative for worm eggs.

The lambs were infested with infective larvae obtained from the culture of faeces collected from sheep with pure infestations of *G. pachyscelis* and *C. ovina*. The programme of infestation is presented in Table 9. It should be noted that the infective larvae of *G. pachyscelis* were applied percutaneously to different sites in an attempt to obtain repeated infestations in the same sheep. The *C. ovina* larvae were taken up on to filter paper, placed in gelatine capsules and administered *per os*.

The immature and adult forms were recovered by the method described in Trial No. 3 and 4, except that the intestine walls were thoroughly shaken in water after removal from the bath and before digestion. The untreated lambs were slaughtered on the day the treated group were dosed. The treated lambs were slaughtered 3 days later.

Results: The results are presented in Table 10. All the adult *G. pachyscelis* and *C. ovina* recovered were sexually immature.

TABLE 8.—IMMATURE AND ADULT HAEMONCHUS CONTORTUS RECOVERED FROM UNTREATED LAMBS AND LAMBS TREATED WITH 15MGM OF TETRAMISOLE PER KG BODYWEIGHT.

Untreated lambs				Treated lambs			
Lamb No.	*3rd*	*4th*	*Adult*	*Lamb No.*	*3rd*	*4th*	*Adult*
788	106	374	593	698	55	2	0
790	171	520	930	793	27	2	3
792	132	583	794	796	72	2	1
800	112	395	1017	803	6	0	0
806	91	545	876	807	8	0	3
Total	612	2417	4210	Total	168	6	7
Mean	122.4	483.4	842	Mean	33.6	1.2	1.4
Percentage reduction					72.55	99.75	99.83

The anthelmintic efficacy of tetramisole against the immature and adult forms was calculated and is presented below.

Gaigeria pachyscelis	— adults	96.2 per cent
Chabertia ovina	— adult	97.4 per cent
	— 4th stage	99.5 per cent
	— 3rd stage	96.6 per cent

A few *Trichostrongylus* spp. were recovered in the untreated sheep but the numbers were so small that they have been disregarded.

It will be noted that adult *Gaigeria pachyscelis* were recovered from the lambs within 31 days of the time the first larvae were applied percutaneously.

TABLE 9.—PROGRAMME OF INFESTATION WITH CHABERTIA OVINA INFECTIVE LARVAE ADMINISTERED ORALLY AND GAIGERIA PACHYSCELIS INFECTIVE LARVAE APPLIED PERCUTANEOUSLY (TRIAL NO. 8).

Day	*C. ovina* larvae number	C. pacyscelis larvae number	C. pacyscelis larvae site*
Minus 31	150	135	neck
„ 28	150	150	abdomen (anterior)
„ 25	150	—	—
„ 21	150	—	—
„ 18	160	163	abdomen (posterior)
„ 16	150	—	—
„ 13	150	320	right hind leg
„ 10	150	160	left hind leg (upper).
„ 7	150	150	left hind leg (lower)
„ 4	150	—	—
„ 3	150	—	—
„ 2	300	—	—
0	Day of treatment and slaughter of untreated lambs		
Plus 3	Treated lambs slaughtered.		

* percutaneous application.

TABLE 10.—IMMATURE AND ADULT GAIGERIA PACHYSCELIS AND CHABERTIA OVINA RECOVERED FROM UNTREATED LAMBS AND LAMBS TREATED WITH 15 MGM OF TETRAMISOLE PER KG BODYWEIGHT

	Gaigeria pachyscelis		*Chabertia ovina*		
	4th	Adult	3rd	4th	Adult
Untreated lambs No.					
699	2	35	48	376	141
791	4	32	33	469	204
801	6	32	94	586	179
804	17	26	48	562	144
808	0	31	67	238	160
Total	29	156	290	2,231	828
Mean	5.8	31.2	58.0	446.2	165.6
Treated lambs No.					
789	0	1	5	9	22
794	0	1	1	1	0
802	0	0	1	0	0
805	0	1	1	1	0
809	0	3	2	1	0
Total	0	6	10	12	22
Mean	0	1.2	2	2.4	4.4
Percentage reduction	100.0	96.2	96.6	99.5	97.4

TRIAL NO. 9

The anthelmintic efficacy of tetramisole at a dose rate of 15 mgm/kg against immature and adult forms of *Chabertia ovina* was investigated. *Materials and Methods*: Five Merino lambs purchased in the eastern Free State were used. Faeces samples examined by the McMaster method were negative for worm eggs.

Pure cultures of infective larvae of *C. ovina* and *G. pachyscelis* were taken up onto filter paper, placed in gelatine capsules and administered *per os*. The programme of infestation is presented in Table 11.

Faecal collection bags were attached to the lambs immediately after treatment with tetramisole, renewed after 2, 4, 6, 8, 22 and 32 hours and finally removed after 48 hours when the lambs were slaughtered.

The faeces were emulsified in water, washed onto 300 mesh to the inch B.S.S. sieves and all

TABLE 11.—PROGRAMME OF ORAL INFESTATION WITH THE INFECTIVE LARVAE OF CHABERTIA OVINA AND GAIGERIA PACHYSCELIS (TRIAL NO. 9).

Day	Number of infective larvae	
	Chabertia ovina	*Gaigeria pachyscelis*
— 58	150	950
— 55	150	—
— 52	150	—
— 48	150	—
— 45	160	163
— 43	150	—
— 41	150	320
— 38	150	160
— 35	150	150
— 32	150	—
— 31	150	—
— 30	300	—
0	Day of treatment	
+ 2	Day of slaughter	

adult and immature helminths counted and identified. The contents of the small and large intestines were similarly treated *post mortem*.

Results: The results are presented in Table 12. It will be noted from this table that the percentage efficacy of tetramisole against *Chabertia ovina* adults is 99.9 per cent and against 4th stage, 99.8 per cent.

No *G. pachyscelis* were recovered from any of the lambs. This confirms the findings of Ortlepp[4] that oral infestation of *G. pachyscelis* does not take place.

The rate of expulsion of the helminths is discussed later in this paper.

TRIAL NO. 10

The rate of expulsion of helminths following treatment with tetramisole was investigated in Trial No. 2 and Trial No. 9.

Materials and Methods: In Trial No. 2 faecal collecting bags were attached to the lambs immediately after treatment, renewed at 2 hours and removed after 4 hours.

In trial No. 9 the faecal collecting bags were also attached to the lambs immediately after treatment and renewed at 2, 4, 6, 8, 22 and 32 hours and finally removed after 48 hours.

In both trials the faeces were emulsified and washed onto 300 mesh to the inch B.S.S. sieves and the helminths counted and identified.

Results: The results obtained in trial No. 2 are presented in Table 13, while those obtained in trial No. 9 are presented in Table 12.

It will be observed from Table 14 that the physiological condition of the intestine as reflected by the physical nature of the faeces influenced

TABLE 12.—NUMBER OF CHABERTIA OVINA RECOVERED IN THE FAECES OF SHEEP AND AT THE POST MORTEM EXAMINATIONS FOLLOWING TREATMENT WITH 15 MGM TETRAMISOLE PER KG LIVEWEIGHT.

No. of helminths	700		795		797		798		799	
recovered	4th	Adult	4th	Adult	4th	Adult	4th	Adult	4th	Adult
0 — 2 hours.....	33	98	3	386	23	65	3	194	0	366
2 — 4 hours.....	107	1	43	76	160	10	30	368	0	101
4 — 6 hours.....	23	18	10	16	40	6	43	63	10	41
6 — 8 hours.....	0	12	30	27	0	2	13	22	10	25
8 — 22 hours.....	0	4	17	4	0	1	0	21	3	16
22 — 32 hours.....	0	0	0	1	0	0	0	6	0	0
32 — 48 hours.....	0	0	0	0	0	0	0	3	0	0
Total.....	163	133	103	510	223	84	89	677	23	549
No. recovered at *post mortem*.....	0	0	3	2	0	1	0	0	3	0
Percentage expelled by 2 hours.....	20.2	73.7	2.9	75.7	10.3	77.4	3.4	28.6	0	66.7
Percentage expelled by 4 hours.....	85.9	74.4	44.7	90.6	82.1	89.3	37.1	83.0	0	84.2
Percentage expelled by 6 hours.....	100.0	88.0	54.4	93.7	100.0	96.4	85.4	92.3	43.5	92.5
Percentage expelled by 8 hours.....	—	97.0	83.5	99.0	—	98.8	100.0	95.6	87.0	97.0

Anthelmintic efficacy.

C. ovina	Adults	99.9 per cent
,,	4th stage	99.8 per cent

TABLE 13.—OESOPHAGOSTOMUM COLUMBIANUM ADULTS RECOVERED FROM FAECES AT 2 HOUR INTERVALS FOLLOWING TREATMENT WITH TETRAMISOLE.

Sheep No.	*O. columbianum* recovered		Physical nature of faeces
	0—2 hours	2—4 hours	
385	0	4	soft
468	43	17	soft
404	0	0	hard dry pellets
395	0	0	hard dry pellets
393	0	11	soft.

the rate of expulsion. *O. columbianum* adults were recovered in soft faeces within 4 hours of treatment but not in hard dry pellets.

In trial No. 9 the faeces were collected over a period of 48 hours and from Table 12 it will be noted that in 4 of the lambs more than two thirds of the total number of adult *C. ovina* had been expelled in 2 hours and that after 4 hours, over 83 per cent had been expelled from all 5 lambs. At the end of 8 hours, over 95 per cent of *C. ovina* adults had been expelled from all the lambs.

The expulsion of 4th stage *C. ovina* did not always proceed as rapidly as the adults.

The results of using the illuminated water bath are presented in detail in Table 14. The percentage of every stage of each helminth species recovered from each portion of the gastro-intestinal tract is also presented in Table 14.

Adult helminths: It will be noted that the method of washing the adults from the wall of the abomasum could be improved. A number of those helminths, which had attached themselves to the wall, were still recovered from the wall digesta and wall filtrate of *O. circumcincta* 9.5% remained; of *H. contortus* 3.3% of *T. colubriformis* 8.6%; and of *G. pachyscelis* 11.5% remained. This was not the case with *O. columbianum* and *C. ovina*, though the reduced number of the latter was probably due to improved techniques.

If it had been possible to detach all adults from the walls, then the percentage recovered in the filtrates would have been:—

O. circumcincta	93.6 per cent
H. contortus	88.1 per cent
T. colubriformis	89.8 per cent
G. pachyscelis	15.6 per cent
O. columbianum	29.6 per cent
C. ovina	6.8 per cent

The higher percentage of *O. columbianum* recovered in the filtrate in comparison to *C. ovina* is due to fact that many of the adult *O. columbianum* were small as they had only just completed the 4th ecdysis.

4th Stage Helminths: The pattern of recovery of the 4th stage helminths was similar to that observed with the adults. A higher percentage of the 4th stages of the more active helminths was recovered from the filtrates.

O. circumcincta	89.6 per cent
H. contortus	87.2 per cent
T. colubriformis	91.7 per cent
G. pachyscelis	66.7 per cent
O. columbianum	66.2 per cent
C. ovina	62.9 per cent

The filtrates plus wall digesta yielded 97.6 per cent of 4th stage larvae of *O. columbianum*.

3rd Stage Helminths: With the exception of *C. ovina*, the pattern of recovery of 3rd stage immatures was similar to that obtained with the adults and 4th stages, the numbers recovered from the filtrates being in the region of 90 per cent or more.

O. circumcincta	97.6 per cent
H. contortus	95.8 per cent
T. colubriformis	100.0 per cent
G. pachyscelis	—
O. columbianum	88.4 per cent
C. ovina	65.7 per cent

DISCUSSION

Anthelmintic Efficacy: Tetramisole hydrochloride has been shown to possess outstanding efficacy against the helminth parasites of major importance occurring in South Africa.

The percentage efficacy achieved against the adult forms of *Ostertagia circumcincta, Haemonchus contortus, Trichostrongylus colubriformis, Gaigeria pachyscelis, Oesophagostomum columbianum, Chabertia ovina* and *Dictyocaulus filaria* at a dose rate of 15 mgm per kg bodyweight exceeded 97.9 per cent.

The efficacy of tetramisole against the 4th stage larvae of the above species, with the exception of *O. columbianum*, was equally good. The design of the trial in which the activity of tetramisole against the 3rd and 4th stage larvae of *O. co-*

TABLE 14.—HELMINTHS RECOVERED FROM THE VARIOUS FRACTIONS OF THE ILLUMINATED WATER BATH.

	Ostertagia circumcincta			*Haemonchus contortus*			*Trichostrongylus colubriformis*			*Galgeria pachyscelis*			*Oesophagostomum columbianum*			*Chabertia ovina*		
Number of sheep	9			5			9			4			9			4		
	Range	Total	%	Range	Total	%	Range	Total	%	Range	Total	%	Range	Total	%	Range	Total	%
Adult Helminths																		
Ingesta residue	12–106	301	6.4	75–134	499	11.9	9–360	983	10.1	23–30	103	84.4	9–102	329	70.4	120–200	640	93.2
Wall digesta	0–18	86	1.8	6–29	69	1.6	2–47	136	1.4	—	0	0.0	0–3	6	1.3	0–1	1	0.1
Ingesta filtrate	240–622	3,982	84.1	503–874	3,569	84.8	528–1,683	7,856	80.3	0–2	5	4.1	3–31	122	26.1	0–6	7	5.7
Wall filtrate	9–146	365	7.7	2–32	73	1.7	26–300	800	8.2	1–6	14	11.5	0–7	10	2.2	3–22	39	1.0
4th Stage Helminths																		
Ingesta residue	0–10	301	0.8	7–40	107	4.3	7–90	328	6.8	2–4	9	33.3	0–10	25	2.4	67–238	691	37.1
Wall digesta	12–63	275	9.6	17–56	202	8.4	0–31	72	1.5	—	0	0.0	10–93	333	31.4	—	0	0.0
Ingesta filtrate	116–294	1,738	61.2	253–496	1,933	80.0	127–565	3,255	67.9	0–7	12	44.5	3–56	218	20.4	169–380	1,156	62.1
Wall filtrate	17–166	809	28.4	7–59	175	7.2	29–244	1,144	23.8	0–6	6	22.2	27–150	486	45.8	1–8	14	0.8
3rd Stage Helminths																		
Ingesta residue	—	0	0.0	0–3	3	0.5	—	0	0.0	—	0	0.0	—	0	0.0	0–5	8	3.3
Wall digesta	0–4	6	2.4	0–10	23	3.7	—	0	0.0	—	0	0.0	0–7	10	11.6	3–37	75	31.0
Ingesta filtrate	0–80	154	62.6	29–99	280	45.8	0–93	218	61.1	—	0	0.0	0–43	63	73.3	2–46	76	31.4
Wall filtrate	0–34	86	35.0	31–101	306	50.0	0–55	139	38.9	—	0	0.0	0–13	13	15.1	6–27	83	34.3

lumbianum was tested, was such that it was not possible to determine whether or not this lowered activity was due to poor activity against the 3rd stage larvae or to erratic activity against the 4th stage larvae. The results of Reinecke[2] and the reduction in the number of nodules in the intestinal walls would suggest that the former is the case.

The efficacy of tetramisole against the 3rd stage larvae of *H. contortus* and the 4 day post infection larvae of *D. filaria* was not as good as that against the 4th and adult stages but the percentages reduction of 72.6 and 65.6 respectively, are nevertheless useful.

Design of anthelmintic efficacy trials: Reinecke[2] has focussed attention upon the importance of the control "indicator" sheep slaughtered on the day of treatment when using repeated infestations as an indication of the status of infestation at the time of treatment. He has rightly drawn attention to the dynamic state of the helminth population in a host and its importance in anthelmintic evaluation trials.

In order to determine the efficacy of an anthelmintic against the various helminth stages, it is of vital importance to slaughter all the control sheep on the day of treatment. This method is, in our opinion, the only valid method for evaluating the efficacy against the two immature stages.

The anthelmintic tests which are based upon the treatment of sheep harbouring larvae of a particular age and the slaughter of the sheep when the helminths reach maturity, does not allow comparisons to be drawn between results obtained in different trials. The dynamic nature of a helminth population in the host has already been mentioned and it is obviously impossible to be certain of the stage of development a helminth has reached without actual identification. Host-parasite relationships are known to affect helminth life histories and published life histories cannot be utilized to determine the exact stage of development the parasitic larvae have reached in the period elapsing between infestation and treatment. The difference we found in the period required by *G. pachyscelis* larvae to reach the 4th ecdysis as compared to that reported by Ortlepp[4], lends considerable weight to this opinion.

Rapidity of action of tetramisole: The rapidity of action of tetramisole against helminths of the caecum and colon following administration into the rumen was investigated. *C. ovina* and *O. columbianum* adults were expelled within 2 hours of treatment but the rate of expulsion was to a certain extent governed by the physical state of the faeces. Expulsion was more rapid when the faeces were soft, than when hard dry faecal pellets were present.

The recovery of nematodes from the gastro-intestinal tract: The use of an illuminated water bath and nylon mesh to separate the helminths from the ingesta was highly successful with all the parasitic stages of *H. contortus, O. circumcincta* and *T. colubriformis*. The results obtained with all the developmental stages of *O. columbianum, C. ovina* and *G. pachyscelis* were not as good, but was of considerable assistance in reducing the time and labour involved in recovering the helminths from the gastro-intestinal tract. Further investigations will probably lead to simplification of the present equipment and methods. This method should assist materially in the evaluation of the efficacy of anthelmintics against the immature stages of nematodes and also in studies on the life cycles of nematode parasites.

The life history of Gaigeria pachyscelis: Ortlepp[4] found that the 4th ecdysis of *G. pachyscelis* took place during the 7th or 8th week and that ecdysis may extend over a week. In our work we found that the majority of *G. pachyscelis* present in lambs slaughtered 31 days after the first percutaneous application of infective 3rd stage larvae were immature adults (5th stage).

Lambs drawn from the same group were not found to be harbouring a natural infestation of *G. pachyscelis*.

ACKNOWLEDGEMENT

The invaluable assistance of Messrs. P. Alderton and R. Cain, and the advice of Drs. Reinecke and Anderson, are gratefully acknowledged.

REFERENCES

1. Thienpont, D. et al (10 authors) Nature 209: 1084.
2. Reinecke, R. K. 1966 J.S. Afr. vet med. Ass. 37: 29.
3. Veglia, F. 1915 3rd & 4th Rep. Dir. Vet. Res: Dept. of Agric. Union of South Africa: 347.
4. Veglia, F. 1923. 9th & 10th Rep. Dir. Vet. Ed. & Res: Dept. of Agric. Union of South Africa: 811.
5. Ortlepp, R. J. 1937, Onderstepoort J. vet Sci & Anim Ind. 8: 183.
6. Douvres, F. W. 1956. J. Parasitol. 42:626.
7. Douvres, F. W. 1957 Proc. Helm. Soc. Wash. 24: 4
8. Douvres, F. W. 1957. Am. J. Vet. Res. 18: 81.
9. Threlkeld, W. L. 1948 Virginia Agric. Expt. Sta. Tech. Bull. 111p .27.
10. Reinecke, R. K. 1963 J.S. Afr. vet. med. Ass. 34: 233.
11. Anderson, P. and Reinecke, R. K. 1966. Personal communication.

BOOK REVIEW

STUDIES ON FOOT-AND-MOUTH DISEASE

A report of the Argentine United States Joint Commission on foot-and-mouth disease.

PUBLISHED BY NATIONAL ACADEMY OF SCIENCES, NATIONAL RESEARCH COUNCIL, WASHINGTON DC. 1966.

This monograph presents a detailed description of the design and results of two projects conducted jointly by the governments of Argentine, Chile and the United States. It is an attempt to modify on the results of further scientific investigation the existing regulations, prohibiting the importation into the United States of fresh, chilled or frozen meat, from countries in which foot-and-mouth disease or rinderpest are present. The first study concerns the effect of vaccination and the process of curing on the survival of virus in meat and particularly lymph nodes. The second study is in the form of an epizootiological survey conducted on the island of Tierra del Fuego, which it was claimed was free of the disease and in view of its isolated geographical position, deserved a less restrictive interpretation of the existing regulations.

A great deal of information has been accumulated in the past, on the distribution of foot-and-mouth disease virus in the carcass and the influence of enviromental factors on its survival. This experiment covers new ground, in which previously immunized cattle, were exposed to severe infection and slaughtered at the anticipated peak of viraemia. The carcasses were then dressed and cured. Considering the relatively small number of animals used, but bearing in mind that the most sensitive assay systems presently available were used, figures are presented which show that vaccination reduced the chances of recovering virus, from the lymph nodes after slaughter and that a further reduction in the risk was apparent, after a further 1 month period of curing. From a statistical analysis of the data, the view was expressed that the probability of recovering virus from the cured lymph nodes of vaccinated cattle, as used in the experiment, was low.

The second study provides an excellent illustration of the manner in which data can be gathered and collected and finally provide a conclusive result, provided the survey is well planned and adequate facilities are available for the handling and examination of a significant number of samples. On the basis of the results of in vitro serum neutralisation tests, conducted in tissue culture and suckling mice, no evidence of the presence of foot-and-mouth disease could be detected. Arising out of this work and as an essential prerequisite to its ultimate fulfilment, useful data was obtained on the animal husbandry, population distribution and movement of stock on the island.

Apart from the scientific data presented, this publication provides a useful bibliography of contributions in this particular field of investigation, between 1924 and 1964. It can be recommended as a particularly useful model on which the planning of similar surveys or investigations should be based. Furthermore it gives a clear directive upon which pertinent sanitary measures can be formulated.

P. G. H.

SOME CONSIDERATIONS IN THE DETERMINATION OF BLOOD AND PLASMA GLUCOSE IN SHEEP, CATTLE AND HORSES

R. CLARK

SUMMARY

1. The known difference between blood and plasma glucose levels are emphasised.
2. Results obtained by the Folin-Wu and Glucose Oxidase methods are compared. In general the Folin-Wu method gave slightly higher values for whole blood and slightly lower values for plasma.
3. The rate of decline in glucose concentration in sheep blood held at high ambient laboratory temperatures for up to six hours has been plotted as a guide to those who cannot carry out the determination immediately after bleeding.

INTRODUCTION

Methods for the determination of blood glucose can be generally divided into those based on the reducing action of glucose and the more recently introduced methods using glucose oxidase. Theoretically the latter are much more specific, as reducing agents other than glucose occur in blood, especially in the red cells. The specificity of the earlier reduction methods, however, has been greatly increased by refined methods of protein precipitation designed to prevent the release of non-glucose reducing agents from the cells.

Another important consideration is the well documented but generally ignored difference between the concentration of glucose in the plasma and in the red cells. In 1956, Goodwin[1] reported his findings on the distribution of glucose between these blood fractions in foetal, neonatal and adult blood from the horse, pig, ox, sheep, rabbit and guinea-pig. He found the red cell glucose concentration, in relation to that in the plasma, was highest in foetal blood and lowest in adult blood with considerable species variations. In general the adult red cells contained about 20% of "reducing substance" as compared to the plasma.

Recently, Kidder and Rouse[2] reported their findings on 50 dogs from which blood samples were taken after 18 hours' fasting. The mean whole blood figures were 53.7 ± 8.8 mg./100 ml, while those for plasma were 75.0 ± 8.0 mg/100 ml. These results were obtained by a glucose oxidase method.

It is well-known that the glucose concentration of a blood sample decreases after collection but no figures of the rate of such decrease in sheep blood under our summer conditions are available.

METHODS

Sampling: Blood samples were collected from apparently normal adult animals, selected at random, before they had been given their daily ration. As the object was merely to compare results obtained from identical samples, no cognizance was taken of age, or sex.

Anti-coagulants: Where the true packed cell volume (P.C.V.) was required to calculate the glucose concentration in the blood cells, oxalate and citrate were not used. These cause reduction in red cell volume[3]. (In a small comparative trial on 9 pairs of samples of sheep blood the average P.C.V. of the oxalated bloods was 83% of that of the heparinised ones.) Heparin at 1 drop (1000 units/ml) per 10 ml blood and disodium versenate (E.D.T.A.) at 20 mg per 10 ml blood, as described by Schalm[3], were used.

Determination of Glucose

(a) *Folin-Wu Method* as detailed by Hawk, Oser and Summerson[4].

(b) *Glucose Oxidase Method.*

Reagents

1. Phosphate buffer, 0.1 M, pH 7.0. (See Wootton[5]).
2. Peroxidase* 4 mg and glucose oxidase* 25 mg per 100 ml buffer. (Stable for about 10 days in refrigerator).
3. Colour reagent. O-dianisidine HCl* 66 mg to 10 ml water. (Stable).
4. Glucose Reagent, 100 ml (2) plus 1 ml (3) (Make up daily).
5. Glucose Standard. 100 mg%.
6. Protein Precipitant. Uranyl acetate 160 mg and NaCl 900 mg to 100 ml water.

Dept. of Physiology, Faculty of Veterinary Science, Onderstepoort.
*Sigma Chem. Co., St. Louis, Mo.

Method

Add 0.1 ml blood or plasma to 1 ml protein precipitatant (6). Mix and centrifuge for 10 min.

Working standard, 0.1 ml glucose standard to 1 ml protein precipitatant.

Blank, 0.2 ml water.

Standard, 0.2 ml working standard.

Test, 0.2 ml supernatant.

To each add 5 ml glucose reagent.

Mix and stand at room temperature for 35 minutes. (Do not expose to direct sunlight).

To each add 5 ml 25% sulphuric acid, mix and read within 5-10 minutes at 350 mμ.

U/S x 100 = mg % glucose.

All readings were made on a "Unicam SP. 500" spectrophotometer (520 mμ for the Folin-Wu method). A clinical type photoelectric colorimeter can be used.

Except when the effect of storage was being investigated protein free filtrates were prepared within 20 minutes of bleeding.

Determination of Red Cell Glucose Concentration.

This was calculated from blood and plasma concentrations and P.C.V. without correction for trapped plasma volume.

Decline of Glucose Concentration in Held Samples.

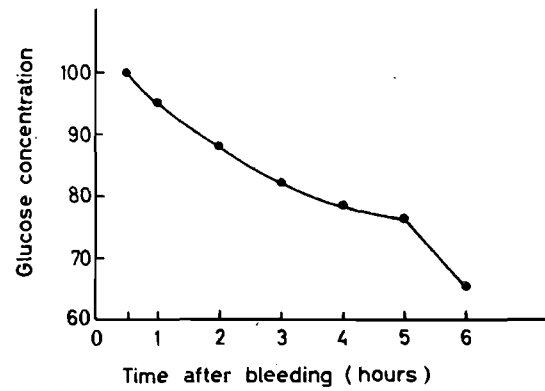
Blood samples were collected from six different sheep and held at laboratory temperature. Glucose concentration was determined by the glucose oxidase method at intervals as indicated under results.

Results

As will be seen from Table 1, the concentration of glucose was considerably higher in plasma than in blood in all three species. The concentration of glucose in the red cells would appear to be considerably lower in sheep than in cattle or horses.

A comparison of the results obtained by the two methods, carried out concurrently on identical samples, shows that the Folin-Wu method gave slightly higher figures for whole blood and slightly lower for plasma. These differences, however, were not great.

Graph 1 was compiled from the averages of the six samples, the figure obtained at 30 minutes after bleeding being expressed as 100. The maximum temperature in the laboratory that day was 28°C. The break in the exponential-like curve at 5 hours may be due to contaminating bacteria having reached the logarithmic phase of multiplication.



Graph I

Decline in Blood Glucose in Stored Samples (Concentration in sample at 30 minutes after bleeding expressed as 100.)

Discussion

Choice of Method

The glucose-oxidase method is specific and its reproducibility has been found to be better than that of the Folin-Wu method. (Approximate deviations from a limited number of triplicate determinations were G.O. ± 1.37 and F.W. ± 3.67 mg %). The differences in the results obtained, however, are not of significance in clinical work.

For a laboratory in which glucose determinations are not done continuously and where extreme accuracy is not required, the Folin-Wu method would be more practical. The reagents are more easily obtainable and are stable at room temperature. Technically there is little difference between the two methods. The Folin-Wu method

Table 1.—Glucose concentrations (mg %).

Species	Glucose Oxidase			Folin-Wu	
	Blood	Plasma	RBC	Blood	Plasma
Sheep	45.9±8.3	54.4±7.4	5.2	46.4±8.3	54.7±7.6
Cattle	53.2±3.1	72.3±6.9	22.3	58.0±6.1	70.3±8.4
Horses	61.1±6.7	83.5±4.7	22.7	64.7±8.0	83.3±8.2

Figures based on 33 sheep samples and 10 samples from cattle and horses.

is quicker only because of the incubation period required in the other.

Blood or Plasma Sugar?

Although the term "plasma sugar" is seldom encountered, it would appear to be the more logical concept. As early as 1913 Macleod (quoted by Goodwin[1]) pointed out that it is the concentration of glucose in the plasma and interstitial fluid to which the body cells respond. Tubular maximum and "renal threshold" values should obviously be calculated on plasma concentrations. For clinical work blood sugar will doubtless continue to be used. The "normal ranges" for the different species are well-known. The determination of plasma sugar necessitates centrifugation and an inevitable time lapse between sampling and protein precipitation.

Decline in Glucose Values.

The graph presented may act as some guide in the interpretation of results where there has been inevitable delay between bleeding and protein precipitation.

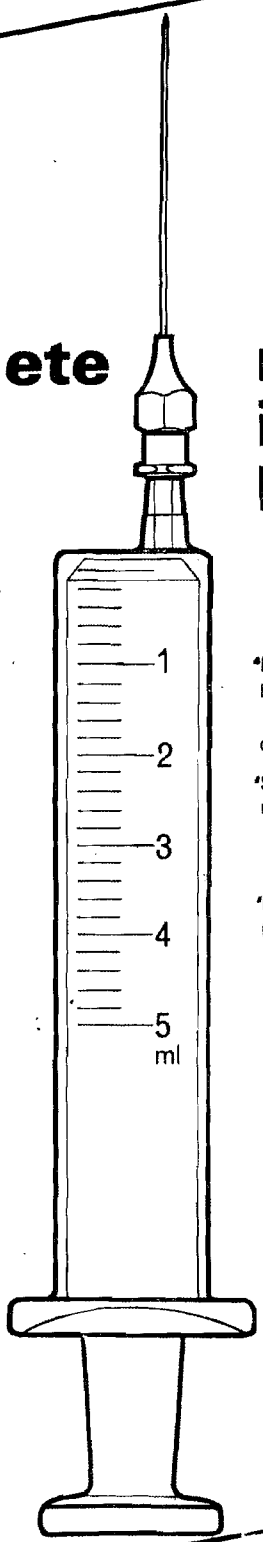
ACKNOWLEDGEMENTS

I wish to thank Miss A. M. Wagner and Mr. R. S. Gray for the meticulous manner in which they carried out the determinations.

REFERENCES

1. Goodwin, R. F. W., 1956 *J. Physiol* 134: 88
2. Kidder, D. E. & Rouse, B. T. 1966 *Vet. Rec.* 79: 87
3. Schalm, O. W., 1961 *Veterinary Hematology*. London. Baillière, Tindall and Cox.
4. Hawk, P. B., Oser, B. L. & Summerson W. H. 1954 *Practical Physiological Chemistry*. New York. McGraw-Hill.
5. Wootton, I. D. P. 1964 *Micro-Analysis in Medical Biochemistry*. London. J. & A. Churchill.

a complete range of intravenous barbiturates



'INTRAVAL'* Sodium
brand Thiopentone Sodium B. Vet. C.
for anaesthesia of short duration in dogs, cats, horses and cattle

'SAGATAL'*
brand pentobarbitone sodium
for anaesthesia of medium duration
small animals

'EUTHATAL'*
brand pentobarbitone sodium
for euthanasia in small anima

M&B brand Veterinary Products

MAYBAKER (S.A.) (PTY) LTD
Port Elizabeth P.O Box 1130
Tel: 4-5481
Branch Office.
Johannesburg P.O. Box 3926
Tel: 724-2146/7

*trade mark

THE SURVIVAL OF PASTURE INFESTATION WITH NEMATODE PARASITES OF SHEEP IN A SUMMER RAINFALL AREA

R. J. THOMAS*

SUMMARY

Faecal worm egg counts were carried out on groups of lambs born over an extended period from February to May in the Eastern Transvaal Highveld. The results indicated that the availability of infestation on pasture declined very markedly at the end of the summer rainfall season. Lambs born before this time showed 80-100% infestation rates and moderate egg counts, whereas later lambs showed a low infestation rate and low egg counts. The application of these findings to the planning of anthelmintic treatment is discussed.

INTRODUCTION

The limits of survival of the free-living stages of the common gastro-intestinal nematodes are of considerable importance in the formulation of control measures involving the use of 'clean' grazing, and have therefore received considerable attention. Kates[1] has reviewed the literature and has ranked these parasites in order of their resistance to climatic factors. Under strict summer rainfall conditions the effects of desiccation and low temperature during the winter months are severe, and studies on the seasonal egg count pattern in lambs suggest that pasture contamination dies out rapidly during the autumn[2]. The worm burden of lambs is predominantly *Haemonchus contortus,* with small proportions of *Trichostrongylus colubriformis* and *Oesophagostomum columbianum,* and these species are considered to be relatively susceptible to cold, dry conditions. The present work is concerned with observations on the pattern of infestation in lambs of different ages within a single flock, and its relation to availability of larvae on the pasture as affected by climatic conditions.

EXPERIMENTAL OBSERVATIONS

The study was undertaken on a flock of 300 merino ewes which had lambed over a relatively long period from February until June, the birth date of each lamb being recorded. During this time the flock was maintained in a single camp of natural pasture, supplemented by several hours' grazing each day on oats and rye grass, plus hay and cereals. The grazing area, therefore, received continuous contamination with nematode eggs from the ewes, and must have remained infestive until adverse climatic conditions killed off the existing larval population and prevented further development.

On the basis of earlier observations it was anticipated that conditions would become unfavourable at the end of the rainy season in late April to early May. Attention, therefore, was concentrated on assessing the rate of pick up of infestation by lambs grazing for the first time over this critical period. Six groups, each of ten lambs, were selected on an age basis, to provide a range of birth dates from February to May, and on June 26, faecal samples were taken from all the lambs and examined for nematode eggs by a modified McMaster method. At this time the younger lambs were only six to eight weeks old and this could explain a low egg count in these groups. Therefore, to allow a similar period of exposure to infestation in all age groups, a further batch of the younger lambs was examined approximately five weeks later. Egg count data for the different groups are summarized in Table 1. Meteorological records were kept throughout this period, and weekly mean figures for temperature and rainfall are presented in Table 2.

RESULTS

The results show that at the time of sampling on 26th June, Groups 1-3, comprising lambs born between 2 February and 21 April, showed 80-100% infestation, and mean egg counts in the range 140-500 e.p.g. (eggs per gram of faeces). By contrast, Groups 4-6, in which the lambs were born after 26 April, showed a markedly lower infestation rate and very low egg counts. Furthermore, on re-sampling 5 weeks later the percentage infestation in Group 7 had risen to only 28%,

*School of Agriculture, University of Newcastle upon Tyne, England.

TABLE 1A.—WORM EGG COUNTS, JUNE, 23. EGGS PER GRAMME OF FAECES (E.P.G.).

Group	No. of lambs	Date of birth	Age at sampling (weeks)	No. infested	Group mean egg count
1	10	Feb.—Mar.	12–20	9	487 e.p.g.
2	10	1—18 Apr.	9–12	10	260 e.p.g.
3	10	19—21 Apr.	9	8	143 e.p.g.
4	10	27 Apr.	8	1	7 e.p,g.
5	10	4 May	7	1	3 e.p.g.
6	10	11 May	6	0	0 e.p.g.

(b) WORM EGG COUNTS JULY, 25.

Group	No. of lambs	Date of birth	Age at sampling (weeks)	No. infested	Mean egg count
7	14	27 April—2 May	12—13	4	12 e.p.g.

TABLE 2.—WEEKLY METEOROLOGICAL DATA, MARCH-JUNE.

Week ending	Temperature °F mean max.	mean min.	Rainfall ins.
March 5	73.0	48.7	0.12
March 12	81.5	53.6	0.0
March 19	72.9	55.8	1.66
March 26	75.4	53.3	0.0
April 2	73.2	50.0	0.0
April 9	74.5	50.7	3.52
April 16	67.5	40.1	2.36
April 23	69.6	45.5	0.41
April 30	55.6	42.6	2.76
May 7	65.8	40.5	0.0
May 14	65.5	39.9	0.0
May 21	66.7	35.4	0.08
May 28	65.3	35.6	0.0
June 4	58.3	33.6	0.0
June 11	62.4	31.5	0.0
June 18	62.2	30.2	0.0
June 25	60.8	31.1	0.12

and the mean egg count to 12 e.p.g. At this time these lambs were 12-13 weeks old, and their level of infestation should have been directly comparable with that of Group 2 at the first sampling, which showed 100% infestation and a mean egg count of 260 e.p.g.

Thus generalised infestation appears to have ceased with the lambs born between 19 and 21 April, and after this date only a low level of infestation was recorded. Lambs do not normally graze to any extent until two to three weeks old, and so it may be concluded that the available infestation on the pasture declined markedly before Group 4 began to graze, which would be about the second or third week in May. Reference to Table 2 shows that this coincides with the onset of dry conditions and a fall in minimum temperature to below 40°F. Steady rainfall ended in the last week in April, only 0.08 in. being recorded in May, and this was probably the dominant factor responsible for the sudden decline in infestation during this month, as the fall in temperature was a more gradual process. The close correlation between rainfall and infestation level suggests that under these conditions the life of the infective larvae is extremely limited, pasture infestation dropping markedly within two to three weeks after the onset of dry conditions.

DISCUSSION

With the recent introduction of highly effective broad spectrum anthelmintics it is now possible to eliminate a high proportion of the worm burden with a single treatment, but to derive maximum benefit from these drugs it is essential to minimise re-infestation. The possibility of using climatic data to predict the end of the summer infestation

period is therefore of considerable practical application in designing dosing programmes for maximum efficiency and economy. Reinecke[3] has commented on the use of anthelmintics to support the natural cycle of elimination of worms in an "offensive" treatment, and has made a general recommendation for dosing against *H. contortus* in September. However, in the Ermelo area, as a result of the strictly limited rainfall season, it would appear to be advantageous to bring this treatment forward to May, in relation to the onset of the dry season.

The results obtained in this work indicate that lambs born before the end of the rainfall season are likely to pick up a considerable level of infestation, whereas those born later will remain relatively worm-free throughout the winter. In practice, lambing in the Highveld generally takes place from February to April, and therefore in most cases it can be assumed that significant autumn infestation will occur in these lambs. This is particularly important as the effect of the worm burden is likely to be severe during the winter months when the plane of nutrition tends to be low. However, treatment with a broad spectrum anthelmintic at the end of May, or more accurately in relation to the end of the summer rainfall, should reduce this burden to a low level, and subsequently little re-infestation should occur through the winter months. Observations reported elsewhere[2] in fact suggest that appreciable re-infestation is unlikely in this area until November and the effect of this single treatment may persist for up to five to six months. Similarly dosing the ewes at the same time may be expected to improve the health and performance of the flock through the winter, and indirectly to have a favourable influence on the performance of the suckling lambs. The timing of this treatment is obviously critical, for if carried out too early its value will be diminished by re-infestation. In commercial practice this tends to be the case in the Eastern Transvaal, as dosing is carried out frequently during the "haemonchosis season" from December to March, but stops shortly before lambing, and is rarely repeated afterwards.

REFERENCES

1. Kates, K. C. 1950 *Proc. Helm. Soc. Wash.* 17: 39.
2. Thomas, R. J. 1967 In Press.
3. Reinecke, R. K. 1964 *J. S. Afr. vet. med. Ass.* 35: 603.

Aan alle lede,

PUBLIKASIE VAN SEKERE BESLUITE VAN DIE VEEARTSRAAD.

Dit is ooreengekom dat die volgende besluite van die Veeartsraad wat op die vergadering van 26/8/66 geneem is aan u deurgestuur word vir publikasie in u joernaal:

1. Twee gevalle was oorweeg waar 'n veearts met 'n ander veearts onderhandel het oor 'n vennootskap of indiensneming by hom en nadat hy van hom inligting ingewin het oor die praktyk of die moontlikhede van die gebied of reeds aan kliënte voorgestel is, daarna in dieselfde gebied en as 'n mededinger van sodanige veearts begin praktiseer of wou praktiseer voor die verstryking van 'n redelike tydperk nadat sodanige onderhandelings gestaak is.
   Die besluit van die Raad was dat sodanige optrede as 'n oortreding van Artikel 15 (c) (d) van die gedragskode beskou word en versoek dat in een geval 'n tydperk van twaalf maande en in 'n ander geval 'n tydperk van agtien maande, bereken vanaf die beëindiging van die onderhandelings, moet verloop alvorens daar in die gebied 'n nuwe praktyk ge-open mag word.
2. Aandag is geskenk aan 'n geval waar 'n uitkenbord by die ingang van 'n huis aangebring is waar geen veeartseny-hospitaal is nie asook 'n uithangbord wat 'n gebou aandui waar geen veeartseny-hospitaal of diereklinick is nie. Die bevinding van die Raad was dat die borde 'n middel tot advertensie is en versoek dat hulle onmiddellik verwyder word.
3. *Registrasie van Veeartse*:
   Die volgende persone is onlangs as Veeartse in die Republiek geregistreer:
   Johannes Petrus Jordaan Joubert
   Jan Hendrik Malan
   Sarel Rens van Amstel
   Friedrich-Wilhelm Gottlob von Ludwiger

THE OXIDASE AND CATALASE ACTIVITY OF STRAINS OF BRUCELLA

G. C. VAN DRIMMELEN AND C. A. W. J. VAN NIEKERK.

SUMMARY

Strains of *Brucella* were tested for oxidase and catalase activity. Those with high oxidase activity had low catalase activity. *Br. ovis* and *Br. melitensis* which show similar oxidative metabolic patterns differ from each other in their oxidase activity.

INTRODUCTION

The oxidase test was first used by Schulze in 1910 (quoted by McLeod[1]) and the presence of the enzyme was demonstrated by using dimethyl p-phenylene diamine hydrochloride as reagent and α naphtol as indicator. Various modifications of reagents and technique were introduced subsequently[2 6]. The test was established for identifying gonococcus colonies[7], for typing *Pseudomonas* strains[4 8] and as a rapid preliminary test for identifying *Vibrio cholera*[1 6 9]. *Brucella* strains were found to be oxidase positive[2 5].

In a search for new ways of typing *Brucella* organisms the oxidase test was investigated. Special attention was given to *Br. ovis* strains because the South African organism appears to be somewhat different from the types isolated in Australia and New Zealand. The oxidative metabolic patterns of *Br. melitensis* and *Br. ovis* have been shown to be very similar. These microbes are also similar in their phage susceptibility and in H_2S production but differ in other respects such as dye inhibition, CO_2 dependence, urease pH threshold and virulence for laboratory animals.

MATERIALS AND METHODS

Brucella strains isolated in South Africa from man, cattle, goats and sheep as well as type strains obtained from Weybridge, Rhodesia and Germany were used. A total of 30 *Br. ovis*, 15 *Br. melitensis*, 8 *Br. suis* and 23 *Br. abortus* strains were used in the investigation.

Each strain was typed by the following tests:—

1. CO_2 dependence[10].
2. H_2S production[10].
3. Urease pH threshold[11].
4. Phage typing[10].
5. Monospecific agglutination[10].
6. Dye inhibition[10].
7. Oxidative metabolism tests[12] were carried out on some of the more recent isolates.

The oxidase test was carried out in the following way:—

The growth from a 48 hour serum agar slant was suspended in 0.85% NaCl solution. The suspension was standardized to have the opacity of Brown's tube No. 4. Three drops of a freshly prepared 1% ethanol solution of tetramethyl p-phenylene diamine dihydrochloride were added to the suspension. The use of metal instruments was avoided. The time for each reaction (the appearance of a purple colour) to be completed was recorded. The results were interpreted by Steel[5] in the following way:—

0 = 10 sec. = positive
11 – 60 sec. = delayed positive
60 sec. = negative.

As most strains were in the delayed positive group, the delayed positives were assumed to be positive so that either a positive or a negative result could be recorded.

Present adderss:
Dr. G. C. van Drimmelen,
Agricultural Counsellor,
Embassy of South Africa,
Washington D.C. 20008,
U.S.A.
Mr. C. A. W. J. van Niekerk,
Dept. Veterinary Services,
P.O. Skukuza,
Kruger National Park,
Transvaal.

In addition to the oxidase test a catalase test was carried out in the following way:—

To 9.5 ml of a 6% v/v H_2O_2 solution in a calibrated 15 ml centrifuge tube, 0.5 ml of the standardized cell suspension was added. The tube was closed with a rubber stopper, pierced by a thin piece of glass tubing, and inverted. The reaction was allowed to continue for 15 minutes. The volume of oxygen produced was indicated by the volume of fluid displaced.

Results

The results of general typing tests on *Brucella* organisms are given in table 1 and the results of the oxidase and catalase test in table 2.

Discussion

The results indicate that the oxidase test can be used to differentiate *Br. ovis* from other species of *Brucella*. Strains of *Br. ovis* show little oxidase activity while strains of the other three species of *Brucella* are oxidase positive and cannot be differentiated from each other.

There appeared to be an inverse relationship between oxidase and catalase activity, those strains showing high oxidase activity being low in catalase activity and vice versa.

Although the oxidative metabolic patterns of *Br. ovis* and *Br. melitensis* are similar, the oxidase and catalase tests can be used as supplementary tests to differentiate the two species.

Table 1—Typing Reactions of Typical Brucella organisms

Typing test	*Br. suis* type I	*Br. abortus* type I	*Br. melitensis* type I	*Br. ovis*
CO_2 dependence	—	+	—	—
H_2S production	++++	+	—	—
Phage	±	++	—	—
Monospecific aggl.	"abortus"	"abortus"	"melitensis"	"Rough"
Urease pH threshold	3.0	— to 5.0	7.0	—
Growth on media with:				
Thionin	+	—	+	+
Basic fuchsin	—	+	+	—
Oxidative metabolism				
Group I				
D-alanine	L	H	H	H
L-alanine	L	H	H	H
L-asparagine	L	H	H	H
L-aspartic acid	L	L	H	H
L-glutamic acid	L	H	H	H
Group II				
L-arginine	L	L	L	L
L-citrulline	H	L	L	L
L-lysine	H	L	L	L
DL-ornithine	H	L	L	L
Group III				
L (+) arabinose	H	H	L	L
D-galactose	L	H	L	L
D-ribose	H	H	L	L
D(+)xylose	H	H	L	L

+ = positive;
— = negative;
± = variable;
L = Low metabolic activity;
H = High metabolic activity.

Table 2—Summary of the results of oxidase and catalase tests

Strain type	No. of strains tested	Oxidase			Catalase		
		Min. Sec.	Max. Sec.	Result	Min. ml.	Max. ml.	Result
Br. ovis	30	>60		Neg.	6.0	9.0	High
Br. suis	8	14	30	Pos.	7.5	9.5	High
Br. melitensis	15	10	20	Pos.	5.0	7.5	Low
Br. abortus	23	8	15	Pos.	1.0	5.0	Low

ACKNOWLEDGEMENT

The Chief, Veterinary Research Institute, is thanked for his permission to publish this report.

REFERENCES

1. McLeod, J. W. 1963. *Lancet* 2:782.
2. Gordon, J. and McLeod, J. W. 1928. *J. Path. Bact.* 31:185.
3. Ellingworth, S., McLeod, J. W. and Gordon, J. 1929. *J. Path. Bact.* 32:173.
4. Kovacs, N., 1956 *Nature (London)* 178:703.
5. Steel, K. J. 1961. *J. gen. Microbiol.* 25:297.
6. Rogers, K. B. 1963. *Lancet* 2:686.
7. McLeod, J. W., Coates, J. S., Happold, F. C., Priestley, D. P. and Wheatley, B. 1934. *J. Path. Bact.* 39:221.
8. Wahba, A. H. and Darrell, J. H. 1965. *J. gen. Microbiol.* 38:329.
9. Kovacs, N. 1963. *Lancet* 2:497.
10. Alton, G. C. and Jones, Lois, 1963. *Anim. Hlth. Br. Monogr. No. 7* FAO ROME.
11. Van Drimmelen, G. C. 1962 *Onderstepoort J. vet. Res.* 29:151.
12. Meyer, M. E. and Cameron, H. S. 1961. *J. Bact.* 82:396.

SURGERY OF THE DOG AND CAT

By A. Noel Ormrod. Baillière, Tindall & Cassell, London

16 plates.

All veterinarians are aware that, quite apart from our formal training, each one of us has a store of experiences which guide our daily surgical practices. This fund of personal experience is rarely communicated by text-books, but rather by working together with a fellow veterinarian. That Mr. Ormrod has worked with many colleagues is known to readers of the advertisements in the Veterinary Record, because he made a practice of doing locum tenentes over a number of years. His book, Surgery of the Dog and Cat reflects, and, more important, imparts this wide experience. Although it does not cover the subject comprehensively, it describes in a lucid and novel manner many surgical conditions and their operative correction. The basis of the technique used in describing operative procedures is the author's gift of being able to draw clearly. These drawings are a feature of the book, and greatly simplify understanding of the steps of various operations. The text reads well and is unambiguous.

The book is divided into 17 chapters dealing with: essential equipment, surgical cleanliness, anaesthesia and haemostasis; pre- and post-operative care; laparotomy, bowel surgery, urogenital surgery and thoracotomy. Other chapters cover the surgery of the eye, ear, oesophagus, mouth, throat and nose; herniotomy, orthopaedics and electrosurgery. Each chapter has much to commend it, but those dealing with ophthalmology and orthopaedics are especially interesting and rewarding.

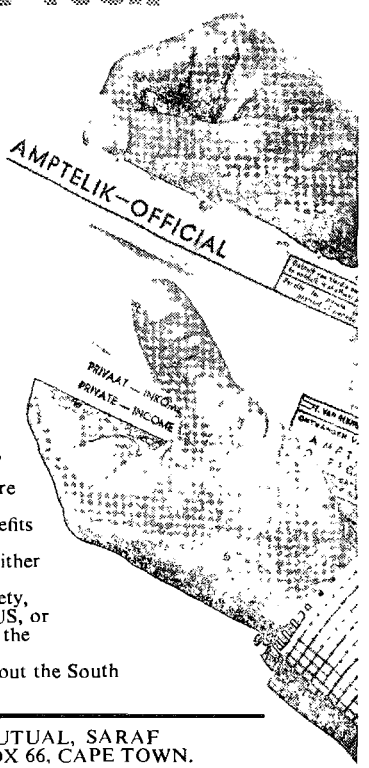
The author may mislead his readers into thinking that prostatectomy in the dog is a simple operation, whereas he warns that perineal hernia is often very difficult. The part on treatment of shock is sketchy. Apart from these and other minor differences of opinion and emphasis, this book should prove itself an aid to the success and competence of our clinicians.

D. H. G. I.

RETIREMENT PLANNING FOR THE THINKING MAN

GET BACK UP TO 50% OF YOUR CONTRIBUTIONS THROUGH TAX RELIEF

WHILE YOU SAVE FOR YOUR FUTURE RETIREMENT



What other investment presents such an offer? The South African Retirement Annuity Fund (S.A.R.A.F.) has been specially developed by the Old Mutual. It enables the professional man, self-employed businessman, the business executive, farmer and many others to enjoy the full tax relief available while contributing towards a retirement fund.

Assume your taxable income would have been R12,000 per annum. If, under the S.A.R.A.F. scheme you contribute R600 per annum, you can receive tax relief of as much as R300—half of what you paid in! As an investment proposition alone S.A.R.A.F. is proving highly attractive to businessmen, farmers, and others. However, it has many more tangible advantages from a pension fund point of view.

For instance, the inclusion of a *special disability clause*, available for a minimal increase in contribution, will ensure that should you suffer permanent disability before you reach age 60, this will be deemed to be an effective retirement and you will become eligible for the full benefits of the fund on that basis.

Again, you may arrange for your future retirement in either of the following ways:

A. A plan based fully on the profits earned by the Society, through participation in its traditionally high BONUS, or
B. A plan where the benefits are linked to the Units of the Society's "OLD MUTUAL UNIT TRUST".

It could benefit you in cold hard cash to learn more about the South African Retirement Annuity Fund.

SARAF

SOUTH AFRICAN RETIREMENT ANNUITY FUND

TO: THE OLD MUTUAL, SARAF DIVISION, P.O. BOX 66, CAPE TOWN.

I would like information regarding the scale of tax relief as it would affect me.

NAME: .....

ADDRESS: .....

.....

THE OLD MUTUAL

SOUTH AFRICAN MUTUAL LIFE ASSURANCE SOCIETY

A POLICY WITH THE OLD MUTUAL IS YOUR MOST REWARDING INVESTMENT

SAM 4315-2F

ADDITIONS TO THE HOST-LIST OF RABIES IN SOUTH AND SOUTH WEST AFRICA

B. VAN DER WESTHUIZEN*, C. D. MEREDITH**.

Neitz has produced "A check-list of the Zoonoses occurring in Mammals and Birds in South and South West Africa"[1]. Since this list was compiled (1965), additional hosts of rabies have been confirmed. These are listed in Table I.

TABLE I.

Virus	Host: Class / Order / Family / Subfamily	Genus and species	Vernacular name	Incidence	Region: O	T	N	W	E	S	Authorities
Rabies	Mammalia / Hyracoidea / Procaviidae	*Procavia capensis* (Pallas)	Rock Dassie	1 case					+		v.d. Wdsthuizen 1966
	Mammalia / Artiodactyla / Bovidae / Cephalophinae	*Sylvicapra grimmia* (Linn.)	Grey Duiker	1 case					+		v.d. Westhuizen 1965
	Mammalia / Carnivora / Felidae	*Panthera pardus* (Günter)	Leopard	1 case						+	v.d. Westhuizen 1965

O = Orange Free State, T = Transvaal, N = Natal, W = Western Cape Province, E = Eastern Cape Province, S = South West Africa.

REFERENCES

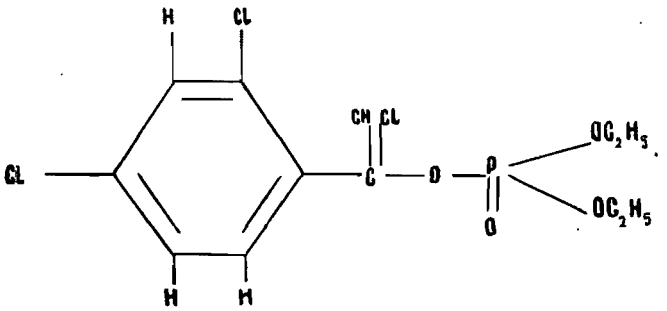
1. Neitz, W. O. 1965 Onderstepoort J. vet. Res. 32:189.

Present address:
*S.A. Cyanamid (Pty) Ltd.,
P.O. Box 7552,
Johannesburg.
**Veterinary Investigation Centre,
Onderstepoort Veterinary Research Institute,
P.O. Onderstepoort.

CHLORFENVINPHOS

IS THE COMMON NAME FOR

2-chloro-1-(2,4-dichlorphenyl) vinyl diethyl phosphate
previously referred to as GC 4072 and SD 7859.



Shell's* Registered Trade Mark for this insecticide is —

SUPONA *

Cooper workers in Britain, Africa, Australia and America, have investigated and proved this to be a stable compound and a remarkably rapid killer of ecto-parasites of domestic stock, particularly suitable for controlling all species of ticks, blowfly, keds, fleas and lice. Cattle and sheep treated with it may safely be dosed with Haloxon.

Cooper's Supadip Sheep Dip and Blowfly Remedy
Cooper's Tick and Maggot Oil
Pulvex Liquid Dog Shampoo, and
Stolex Kennel Dip
all contain this insecticide.

COOPER PRODUCT

Cooper & Nephews, S. Af. (Pty.) Ltd., P. O. Box 2963, Johannesburg.
Cooper, McDougall & Robertson (C. A.) (Pvt.) Ltd., P. O. Box 2699, Salisbury.

STUDIES ON BILHARZIA I. THE DEVELOPMENT OF AN APPARATUS TO HATCH MIRACIDIA

S.P. KRUGER & L. P. HEITMANN

SUMMARY

As result of a series of trials, which are briefly described, the authors have been able to devise a simple apparatus based on the principles of continuous turbulence and perfusion, and subsequent separation, by means of which miracidia can be obtained from faeces and urine of bilharzia sufferers, both animal and human. For experimental work the procedure yields more miracidia than could be obtained hitherto. By the same token it offers a diagnostic test of greater sensitivity and accuracy.

INTRODUCTION

Investigations are being carried out in this laboratory on *Schistosoma mattheei* Veglia & Le Roux, 1929[1]. This parasite causes bilharzia of cattle and sheep, inhabits the mesenteric blood vessels and passes its eggs out with the host's faeces. Because it is desirable to work with the natural definitive host and eggs are difficult to separate from the faeces, miracidia had to be hatched therefrom on a large scale for the infestation of snails.

McMullen & Beaver[2], described an apparatus consisting of a flask with a side arm, in which they were able to hatch and collect miracidia of various schistosomes. The faeces were placed in water and miracidia attracted by light for collection.

Various investigators have concluded that dilution of the excreta with water is essential for eggs to hatch[3,4]. This is apparently the case with both urine and faeces: Gorman, Meeser & Ross[3] worked with eggs of *Schistosoma haematobium* (Bilharz, 1852) Weinland, 1858 which occur in urine and Standen[4] with eggs of *Schistosoma mansoni* Sambon, 1907 which occur in faeces.

This paper is concerned with the application of dilution methods with various modifications in order to develop an apparatus for the efficient hatching and recovery of miracidia, thus simultaneously providing an improved method of diagnosis of bilharzia.

EXPERIMENTAL PROCEDURES AND RESULTS

1. *Plastic Container Hatching Apparatus.*

Faeces were weighed and mixed with water. The faecal suspension was placed in 24 l plastic containers which were filled with water, shaken, and exposed to sunlight. These containers were enclosed in metal holders which allowed light to penetrate the semi-transparent plastic from the top only. After half an hour a black plastic cover was used to exclude all light and a rubber stopper was placed in the opening of the plastic container. A glass tube 1 cm in diameter passed through the stopper and extended from below the surface of the fluid in the drum to a height of 10 cm above the stopper. The fluid was allowed to rise in the tube so that the meniscus was 9 cm above the stopper (Fig. 1).

Results of this experiment, summarized in Table 1, indicate that miracidia could be recovered, whereas egg counts were negative.

TABLE 1.—MIRACIDIA COLLECTED FROM PLASTIC CONTAINERS AND EGG-COUNTS IN 10G FAECES

Sheep No.	Weight of faeces in container (g)	Miracidia count	Egg counts
1	764	22	86
2	1,210	2	0
3	1,524	1	0
4	581	3	0
5	540	2	0
6	315	4	0
7	397	1	0
8	443	6	0
9	1,529	26	0

Preliminary experiments with cattle faeces indicated that miracidia had to be collected for a period of two hours and that the smaller the quantity of faeces the better the harvest of miracidia (Table 2). Experiments with different quantities of sheep faeces showed that 5 g was the optimal quantity for good miracidial recovery (Table 3).

Section Helminthology, Veterinary Research Institute, Onderstepoort

Fig. 1
Metal holder with plastic container used to hatch out miracidia.

The conclusion drawn from this experiment was that some inhibiting factor or factors prevented eggs from hatching in faeces. Therefore another experiment was planned comparing the hatching of eggs in the filtrate from different quantities of water mixed with the same amount of faeces.

2. *Demonstration of Inhibition and Retardation of Egg Hatching*

The livers of infested *Mastomys natalensis* (A. Smith, 1834) were macerated in 2% saline and *S. mattheei* eggs were harvested with a fine pipette. Five grams of filtered (egg-free) faeces from an infested sheep were added to each of the following quantities of distilled water: (i) 500 ml; (ii) 1,000 ml; (iii) 1,500 ml; (iv) 2,000 ml; (v) 2,500 ml.

Each of these serial dilutions was thoroughly mixed, filtered through filter paper and harvested eggs were added. Only 50% of the eggs hatched, the balance most probably being dead or immature. Relevant data, therefore, were derived only from those eggs capable of hatching.

The results are illustrated in Figure 2.

The five cumulative histograms illustrate the percentage miracidia that hatched in a one to five hour period for each of the five serial dilutions of faeces. A cubic parabola was fitted to the percentage of total miracidia hatched for each of the serial dilutions. The results clearly indicate that the higher the concentration of faecal filtrate the more retarded was hatching and the lower the percentage of eggs that hatched. It may thus be inferred that certain soluble factors in infested faeces prevented or retarded the hatching of eggs.

A further experiment was planned using continuous flow through an Erlenmeyer flask in attempts to wash out the soluble constituents of the faeces to enable eggs to hatch more readily.

3. *Continuous Flow Hatching Apparatus.*

A 3,000 ml Erlenmeyer flask was painted black and the opening closed with a rubber stopper (c), as illustrated in Fig. 3. Three holes in the rubber stopper (c) admitted an inlet pipe (a) which went to the bottom of the flask and an outlet pipe (b) flush with the bottom of the stopper leading to a specially constructed trap (d) having a 400 mesh to the linear inch sieve at the one end. The other

TABLE 2—CATTLE FAECES PLACED IN PLASTIC CONTAINERS AT 9 A.M. MIRACIDIA COLLECTED AT DIFFERENT PERIODS

Cattle No.	g. of faeces	Collecting times					Total
		9:40	10:10	10:40	11:10	11:40	
1	113	0	1	2	2	0	5
2	73	0	1	0	0	0	1
3	67	0	1	7	1	0	9
4	45	0	0	1	6	0	7
5	16	4	13	6	5	12	40
6	74	2	1	0	0	0	3

TABLE 3.—DIFFERENT QUANTITIES OF SHEEP FAECES IN PLASTIC CONTAINERS

Sheep No.	g faeces							Total
	5	10	15	20	25	30	40	
1	30	20	12	5	3	0	0	70
2	11	9	7	5	1	0	0	33
3	3	2	1	0	0	0	0	6
4	12	2	1	0	0	0	0	15
Total	56	33	21	10	4	0	0	124

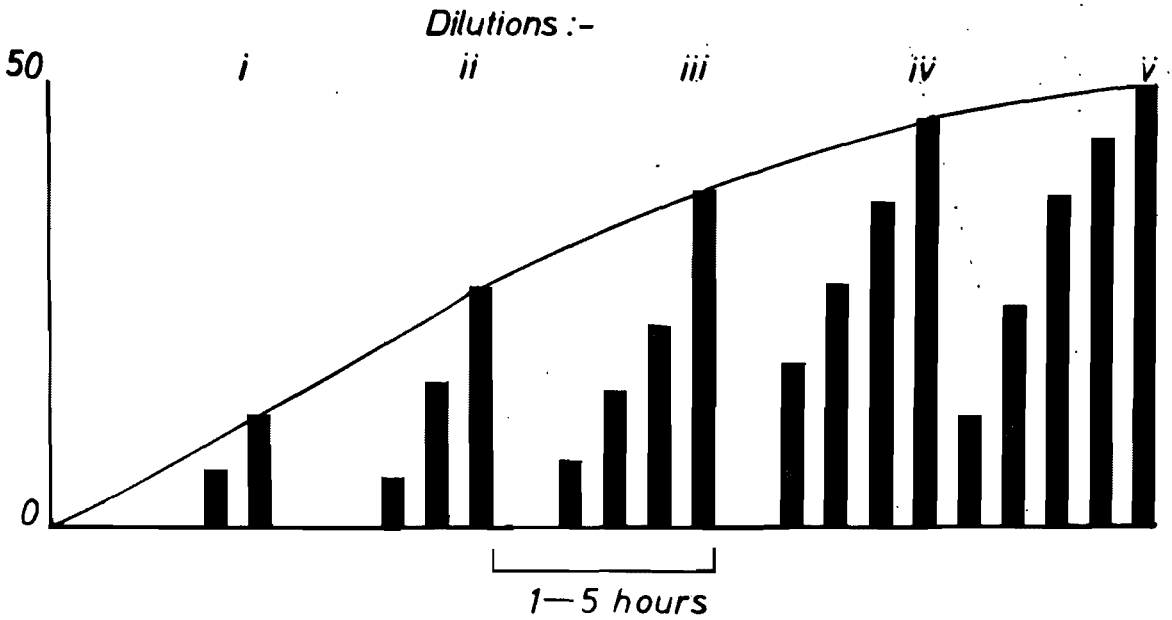


Fig. 2

Cubic parabola and cumulative histograms of miracidia hatching in serial faecal dilutions.

hole of the stopper housed a six volt light bulb, connected to the mains via a transformer. A low pressure source forced water through the apparatus at a rate of approximately 2 litres per hour.

Results of an experiment with this apparatus are summarized in Table 4. A 25 g specimen of infested sheep faeces was placed in the flask which was then filled with water, shaken well and the tube (a) connected to the water source at 9:00 a.m. From 9:30 a.m. miracidia were collected from the trap every 10 or 15 minutes. After 9:45 no more miracidia were collected, though the water continued to flow until 2:00 p.m. At that time the flask was shaken vigorously and again miracidia were collected from the trap for 20 minutes. Shaking had to be repeated at 3:00 p.m. and again at 4:00 p.m. in order to cause hatching of miracidia.

The results (Table 4) indicate that the apparatus did not fulfill the purpose for which it was designed. Miracidia were only harvested for periods of 5-15 minutes after the apparatus had been shaken. Mere flow of water did not give the desired results. It appeared that the faeces had to be mixed continually with water. A special apparatus was then constructed in an attempt to achieve this objective.

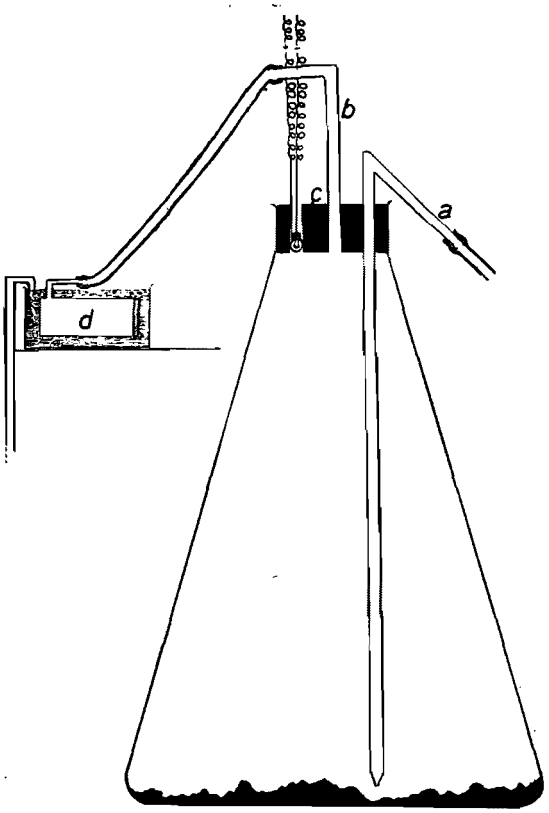


Fig. 3
Continuous flow miracidial hatching apparatus.

4. *Continuous Turbulence-Perfusion Miracidial Hatching Apparatus*

This apparatus is illustrated in Fig. 4 and consists of the following:

(a) Rubber or plastic tube connected to a high pressure water main.

(b) A B19*[1] Socket.

(c) A pipette drawn to a fine point penetrating a cork which fits into the B19 socket. (In order to vary the position of the point of the pipette (c), use can be made of a special extension tube with a male and female B19 socket.) The pipette was calibrated to deliver 20 ml per minute.

(d) Conical distilling flask.

(e) Separating funnel with a B24 socket fitted into (d).

(f) A glass tube bent at the tip.

(g) A rubber stopper with hole in the centre.

*[1] British Standards Quickfit Apparatus.

TABLE 4.—NUMBER OF MIRACIDIA OBTAINED FROM CONTINUOUS FLOW HATCHING APPARATUS

Time	No. of miracidia	Total
Started at 9:00 a.m.		
9:30 a.m.	0	
9:40	1	3
9:45	2	
No miracidia obtained up to 2:00 p.m.		
2:00 p.m.	0	
2:05	3	
2:10	4	9
2:15	1	
2:20	1	
No miracidia obtained up to 3:00 p.m.		
3:05	4	
3:10	8	14
3:15	2	
No miracidia obtained up to 4:00 p.m.		
4:05	6	
4:10	11	22
4:15	5	

(h) Plastic or rubber tubing connected to a filter side arm flask (i).

(i) Filter side arm flask of 2 litre capacity painted black.

(j) Side arm.

(k) Plastic vial. This is the only source of light to the collecting side arm flask (i).

The water main is connected to (a) and the water forced through the fine pipette (c) at high pressure. The water flows at a rate of 20 ml/min. into the distilling flask (d). Once (d) is filled, 5 g of faeces, mixed in water, are poured through a funnel into the separating flask (e), which is then allowed to fill from the source below (d) up to the neck before the tube (f) and cork (g) are put into place. The position of the tube (f) is adjusted so that the open end is below the level of light faecal material which tends to accumulate at the top of the separating funnel (e).

As this part of the apparatus fills and overflows into the side arm flask (i), it is allowed to run for a 100 minutes by which time the side arm flask is filled. This apparatus is then allowed to stand in front of a light source for an hour or longer. The curvature on the tube (f) which leaves the separating funnel is so arranged as to allow ready access to miracidia which tend to swim round the edge of this flask. The fine jet of water must not be obstructed in any way as continual mixing of faeces with clean water is essential.

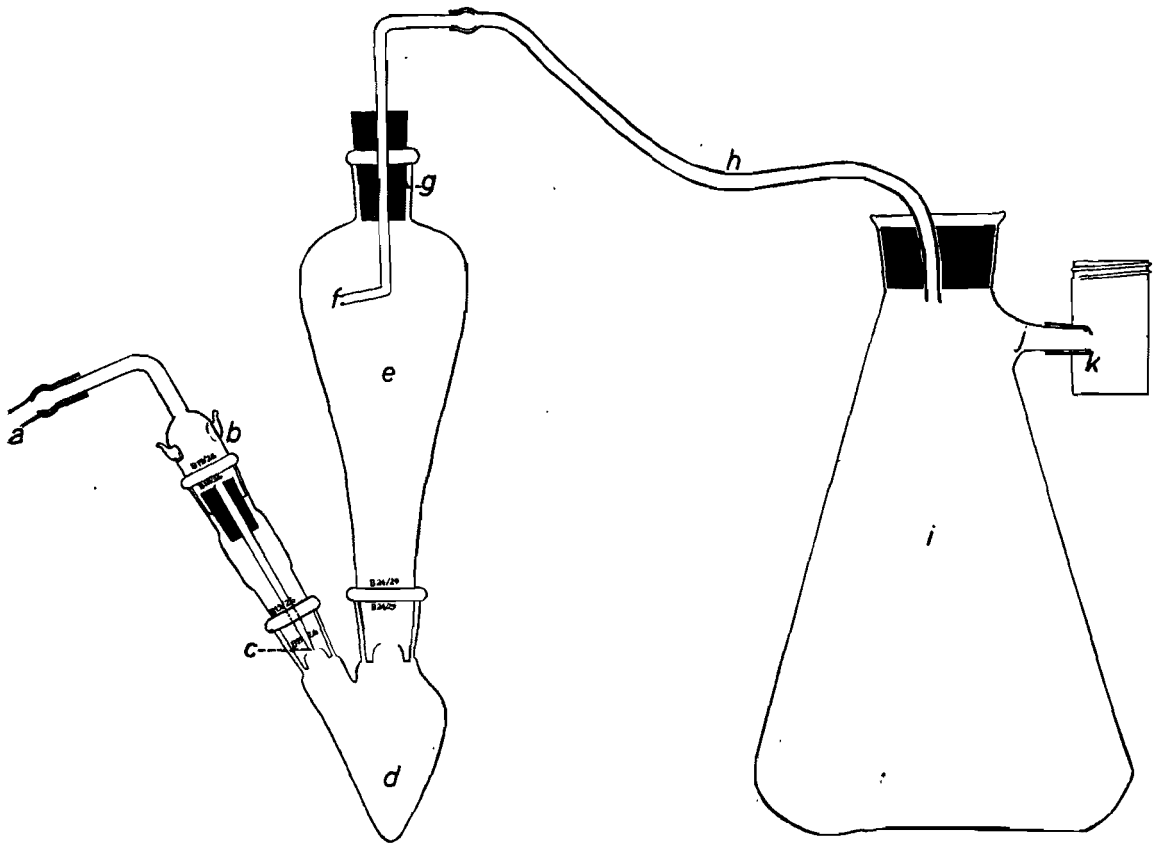


Fig. 4
Continuous turbulence-perfusion miracidial hatching apparatus.

In this apparatus miracidia start hatching within 30 minutes and continue to hatch throughout the period which the apparatus is run. They flow passively with the water into the side arm flask, and are attracted to the little plastic trap (k) through the tube (j) by virtue of the fact that it is the only light source.

Results with this apparatus indicate that miracidia reach peak hatching in 45 minutes and decrease thereafter for the period which the apparatus runs, i.e. 100 minutes. The residual faeces in the flask (d) can be removed and examined with the aid of a stereomicroscope for dead eggs or eggs containing immature miracidia.

The mass harvesting of miracidia using this apparatus has proved highly successful. A few results are summarized in Tables 5 and 6. The apparatus, moreover, hatches miracidia from a small quantity of faeces, e.g. 5 g, and there is a close correlation between the number of miracidia hatched and the worms recovered at *post-mortem* examination.

The results show that miracidia can be recovered from the faeces *ante-mortem* with the aid of this apparatus, even in mild infestations.

TABLE 5.—NUMBER OF MIRACIDIA OBTAINED FROM A SINGLE SHEEP WITH CONTINUOUS TURBULENCE-PERFUSION MIRACIDIAL HATCHING APPARATUS USING 5G OF FAECES IN EACH TEST

Date	No. of miracidia
15.8.66.....	216
17.8.66.....	205
18.8.66.....	178
24.8.66.....	322
7.9.66.....	314
7.12.66.....	247

DISCUSSION

The authors are in complete agreement with Standen (1951) who stated: "The scanty ova present in the stools could be more easily identified

TABLE 6.—NUMBER OF MIRACIDIA HATCHED FROM 5G OF FAECES COMPARED WITH WORM BURDENS

Sheep No.	No. of Miracidia	No. of worms recovered post mortem
1	0	4
2	14	88
3	8	54
4	400	2371
5	9	36

by hatching tests than by microscopic examination of considerable quantities of faecal sediment". This is confirmed by results reported in this paper for various species in both faeces and urine (Table 7).

The advantages of the miracidial hatching apparatus when compared with egg counts are twofold:

(1) it is a better diagnostic method (Table 8) and (2) provides an improved method of harvesting large numbers of miracidia for laboratory purposes.

Sheep with very light worm burdens, e.g. as few as 36 worms, are found to be positive with this apparatus (Table 6). The apparatus is cheap, easy to maintain and, apart from the blockage of tubes by faecal material, there is little which can go wrong with it during operation. The use of this simple apparatus obviates mistakes. The authors have found that lay assistants can be easily trained in a relatively short time to use the apparatus and to make a correct diagnosis of bilharzia.

TABLE 7.—NUMBER OF MIRACIDIA HATCHED COMPARED WITH EGGS COUNTED FROM 5G OF FAECES

Sheep No.	Miracidia recovered	Eggs counted
1	112	16
2	47	7
3	68	11
4	485	36
5	113	13
6	434	52
7	700	76
8	11	2
9	4	0
10	46	12
11	94	18
12	7	0
13	0	0
14	6	0
15	32	6
16	264	24
17	8	2
18	50	9
19	148	24
20	67	10

TABLE 8—MIRACIDIA RECOVERED WITH CONTINUOUS TURBULENCE-PERFUSION MIRACIDIAL HATCHING APPARATUS FROM 5G OF FAECES FROM DIFFERENT SCHISTOSOMES AND HOSTS

Host	Parasite	Miracidia recovered	Comments
Sheep.....	*S. mattheei*	3	22 worms recovered *post mortem*
Horse.....	*S. mattheei*	6	96 worms recovered *post mortem*
Sheep ram.....	*S. mattheei*	7	24 worms recovered *post mortem*, four year old infestation
Calf.....	*S. mattheei*	12	20 worms recovered *post mortem*
Human.....	*S. mansoni*	2368	Faecal examination
Human.....	*S. mansoni*	52	Faecal examination
Human.....	*S. haematobium*	62	Urine examination
Human.....	*S. haematobium*	31	Urine examination
Human.....	*S. haematobium*	6	Urine examination
Baboon.....	*S. haematobium*	12	Faecal examination

ACKNOWLEDGEMENTS

The Chief: Veterinary Research Institute, Onderstepoort, is thanked for permission to carry out the experiments and publish the results. Dr. R. K. Reinecke and Major R. M. McCully, USAF, V.C., are acknowledged for their constructive critism during the development of the apparatus and for critically revising the script. Miss C. S. Mansvelt is thanked for the neat drawings she made.

REFERENCES

1. Veglia, F. & le Roux, P. L. 1929 *15th Rep. vet. Res. Un. S. Afr.* p. 335.
2. McMullen, P. B. & Beaver, P. C. 1945 *Am. J. Hyg.* 42: 128.
3. Gorman, S., Meeser, C. V., Ross, W. F. & Blair, D. M. 1947 *S. Afr. med. J.* 21:853.
4. Standen, O. D. 1951. *Trans. R. Soc. trop. Med. Hyg.* 45: 225.

VAGINAL PROLAPSE IN THE BITCH

A. P. SCHUTTE*

SUMMARY

Clinical study involving 22 cases of various degrees of vaginal propalse, revealed that (a) the lesion begins as an eversion at a definite locus in the vagina, i.e. in the vaginal floor immediately cranial to the urethral opening; (b) the eversion may progress to an incomplete or complete prolapse, three degrees or types thus being recognisable; (c) the condition arises during oestrus, at partus or post-partum; (d) the first two types of lesion regress spontaneously during the other phases of sexual activity, but never return to complete normality and recur during subsequent oestrus or partus; (f) the third type never returns unless reduced manually and even then may recur during oestrus or partus.

A simple technique for temporarily retaining the prolapse is described. For permanent relief oöphorectomy is essential.

The condition is considered to be due to normal oestrogenic action on a locus anatomically and/or physiologically predisposed thereto. There is also a distinct breed disposition which suggests the probability of an hereditary linkage.

INTRODUCTION

Vaginal prolapse, i.e. eversion of part of the vaginal mucosa and protrusion thereof through the vulvar lips, has been identified and associated with pregnancy in most domestic animals[1 7]. In the bitch, however, this condition occurs mainly during the oestrous phase of the breeding cycle[1,8 12] and subsequently appears to revert to normal.

Different, even discrepant terms, such as hyperplasia, oestral hyperplasia, eversion, vaginal protrusion, vaginal prolapse are currently used to describe this aberration in the bitch. This reflects in part the confusion which exists.

Presentation of cases at the genesiological clinic at Onderstepoort provided an opportunity to study this condition more closely.

MATERIAL AND METHODS

Over the past two and a half years 22 canine patients — all of the brachycephalic type — with various degrees of vaginal protrusion were admitted and subjected to detailed clinical examination.

In five cases a catheter was placed into the urethra to obviate accidental snaring of the passage and a purse string suture applied as follows: The prolapsus was drawn out as far as possible and a half-curved, reverse cutting needle passed into the submucosa, ventrally, between urethral opening and base of the prolapsus. From there it was guided immediately beneath the mucosa around the whole vaginal lumen, to reappear close to the original point of entry. (In case of a wide lumen the submucosal passage of the suture was interrupted at a convenient point, the needle brought to view, drawn out and re-entered at the same point.) The prolapsus, if large, was reduced with warm water and a towel, (simulating the effect of Esmarsch's bandage) lubricated and repositioned. The suture thread was then drawn taut to close the vaginal lumen and so prevent recurrence.

All of these cases were in the oestral phase of the cycle. In a further three cases a similar procedure was followed during metoestrum. Panhysterectomy was performed on ten bitches, while four cases were left untreated.

The vaginal cytology was studied according to the method suggested by Schutte[13] in fifteen cases, while in five cases vaginal, ovarian and uterine tissue was examined histologically.

OBSERVATIONS

Clinical data pertaining to these cases are presented in table 1. As indicated, all the patients belonged to one of the brachycephalic breeds. Boxer and Boxer crosses constituted 82 per cent of the series. From the data the following clinical picture may be envisaged:

*Dept of Genesiology, P.O. Onderstepoort.

TABLE 1—BREED INCIDENCE AND THE DEGREE OF VAGINAL PROLAPSE ENCOUNTERED IN CASES SUBMITTED.

	Breed	Age (Mths)	Current Oestrous cycle	Type of prolapse* (Last cycle)	Stage during breeding cycle prolapse identified
1	Bulldog.....	10	1	II	Oestrum
2	Boxer.....	18	2	I	Oestrum of 2nd cycle
3	Boxer.....	14	2	II	Oestrum of 1st & 2nd cycle
4	Boxer X.....	26	3	II	Oestrum 1st cycle, at partus, and pro-oestrum 3rd cycle.
5	Boxer.....	22	3	II	Oestrum of 2nd cycle
6	Boxer.....	22	2	II	Pro-oestrum
7	Boxer.....	15	2	II	Oestrum 1st cycle, pro-oestrum 2nd cycle.
8	Boxer X.....	9	1	I	Pro-oestrum
9	Boxer.....	36	3	II	Oestrum 1st, 2nd & 3rd cycle
10	B. Mastiff.....	18	2	II	Metoestrum 2nd cycle
11	Boxer.....	—	—	II	Metoestrum
12	Boxer.....	18	2	II	Oestrum 2nd cycle
13	Boxer.....	18	2	II	Metoestrum
14	Boxer.....	36	4	I	Oestrum 3rd & 4th cycle
15	Boxer.....	24	2	II	Oestrum 1st cycle, metoestrum of 2nd cycle
16	B.Mastiff.....	24	3	II	Oestrum 1st & 2nd cycle
17	Boxer.....	24	3	III	Oestrum of last period
18	Boxer.....	24	3	II	Late metoestrum
19	Boxer.....	40	4	II	During various phases of previous cycles, during parturition and metoestrum 4th cycle
20	Boxer/B.Terrier....	24	3	I	Oestrum
21	Boxer.....	10	1	II	Oestrum and metoestrum
22	Bulldog.....	21	3	I	Oestrum of last cycle

*See classification

During the oestrous cycle, for reasons yet unknown, a focal oedema of the central vaginal mucosa develops just cranial to the urethral opening (plates 2 and 3). This may remain localised as a small wallnut-sized eversion (plates 1A and 2). In some cases this eversion may increase in size to such an extent that it protrudes through the vulvar lips as an incomplete prolapse (plates 1B and 3). In extreme cases the prolapse becomes complete, involving the entire circumference of the vagina (plates 1C and ID).

The degree of eversion and protrusion of the vaginal mucosa encountered clinically may thus be classified into one of the following categories of successive degrees of severity of the lesion:

Type I (Eversion). A slight to moderate eversion of the vaginal floor cranial to the urethral opening. It is confined to the vestibulum and is not visible through the vulvar lips but can be seen on deeper vaginal examination.

Type II (Incomplete prolapse). Prolapse is well developed and protrudes to varying extent through the vulvar lips. On deeper vaginal examination the base of the prolapse is seen to fan out from the vaginal floor to include areas from the lateral wall as well.

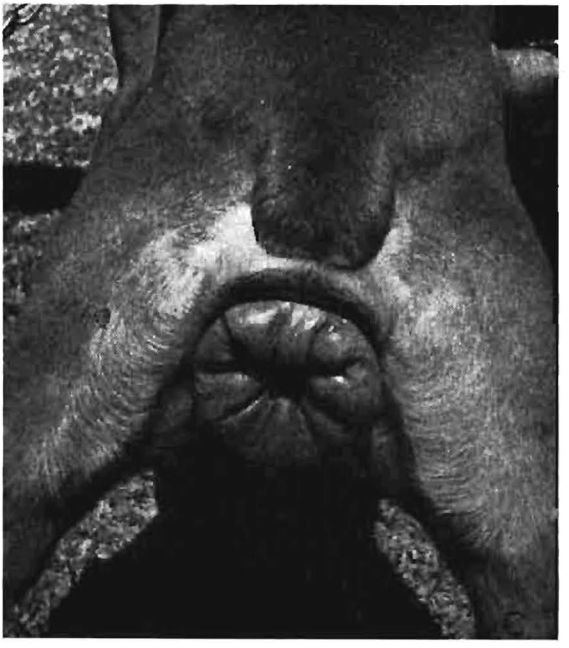
Type III. (Complete prolapse). Well developed prolaps of the entire circumference of the vagina, which protrudes through the vulva. When the ventral part of the prolapse is elevated, the urethral opening may be clearly identified.

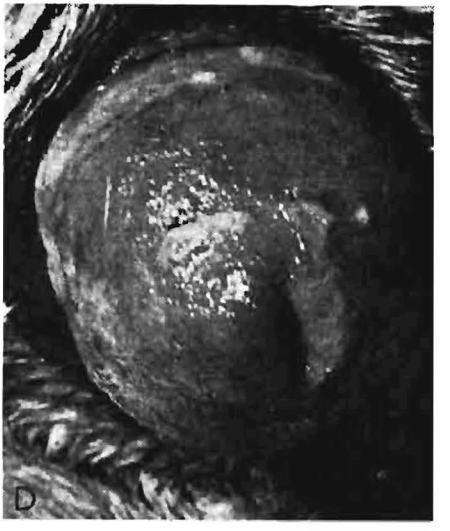
Vaginal eversion or protrusion was encountered during the various stages of the oestrus cycle. In 16 cases it was seen during oestrus, in four cases during pro-oestrum, in six during metoestrum and in two cases at partus. Of the eighteen cases which had had one or more previous cycles, 66 per cent had a recurrence of the prolapse to some degree during consecutive oestrous cycles.

In table 2 the different methods and results of treatment are given. The purse string suture method is of limited value only. When this treatment was applied during metoestrum, however, the prolapse did not recur before the following oestrus. In all the cases on which panhysterectomy was performed, total regression took place within three weeks.

The histological examination of ovarian, uterine and vaginal material collected from five cases did not appear to indicate any abnormalities which could be considered the result of abnormal hormonal influences. All the cases examined fitted the histological classification put forward by Mulligan[14].

The vaginal cytological examination performed on fifteen cases indicated a slightly higher degree of keratinization in the epithelial layers of the pro-





Degrees of vaginal prolapse.

Fig. A. Type I:
The eversion cannot be seen externally but is localised within the vestibular region. Note the marked enlargement and position of the vulva.

Fig. B. Type II:
Part of the vaginal floor everts and protrudes through the vulva.

Figs. C. and D. Type III:
A complete prolapse of the entire circumference of the vaginal wall.

TABLE 2.—METHODS OF PROLAPSE CORRECTION AND RESULTS

	Method of Treatment			Untreated group
	Intra vaginal purse string suture. (During oestral phase)	Intra vaginal purse string suture. (During metoestral phase)	Panhysterectomy	
No. of cases treated	5	3	10	4
Response to treatment	Recurrence in three cases during met oestrum of same oestrous cycle	Recurrence in all three cases during subsequent oestrous cycle	Complete regression in all cases within three weeks after panhysterectomy	—

lapsed part compared to the rest of the vaginal epithelium. This epithelium presented a cytological picture in no way different from that of a normal vagina in the corresponding phase of the cycle. The eosinophilic and superficial cell indices of the prolapsus followed the normal pattern.

DISCUSSION

Varying opinions on the aetiology of vaginal prolapse in domestic animals have been documented, but its exact nature in bitches still remains obscure[8 10]. In cattle this condition occurs more frequently in multiparous cows, while it is rarely seen in heifers. It has been postulated that weakened and relaxed uterine ligaments placed under stress by excessive straining, may play a role in causing this condition in the cow[2 6].

Although it is possible to assume that weakened ligaments may predispose to prolapse in older bitches, such an assumption, nevertheless, seems open to doubt since prolapses of type I and even of type II are very often seen in nulliparous bitches during their first or second oestrous cycle.

Several workers suggest that when incompatable copulation or forcible separation during copulation occurs, with resultant excessive stretching of the vaginal mucosa, prolapse may ensue[1 8 9]. In many of these cases it is possible that a type I prolapse may have been present within the vestibule before copulation and only became clinically apparent on separation of the male and the female.

The genital tract and its ligamentous attachments become more oedematous during oestrus due to increased oestrogen stimulation. It has been suggested that in conditions where oestrogens are produced and secreted in excessive amounts during oestrus, the vaginal mucosa may become predisposed to prolapse. None of the cases studied at this clinic, however, showed any evidence, either on histological examination of the genital tract or upon application of exfoliative vaginal cytology, to support the theory of hyperoestrogenism. The higher degree of keratinisation of the prolapsed mucosa is more likely due to mechanical causes and does not appear to reflect a localised state of hyperoestrogenism. The fact that many of these cases conceive either on natural or artificial insemination, furthermore discounts the hypothesis of any concurrent hormonal aberration.

It appears that this condition occurs mainly in brachycephalic breeds but it has also been observed in other breeds[10 11]. Eighty two per cent of the cases observed at this clinic, were of a single breed. This fact supports the contention that an hereditary breed predisposition may exist. This can only be confirmed by extensive breeding trials.

As the clinical picture readily allows classification into three types of increasing degree of severity of the condition, it enables one to gain a clear idea of the pathogenesis once visible lesions have developed. In terms of underlying mechanisms before the first type of lesion arises, the pathogenesis remains obscure.

Prolapses of the first and second types regress remarkably during the metoestral phase of the same cycle and can only be identified on deeper vaginal examination (plate 4). When a complete prolapse is present, spontaneous regression does not usually occur and manual manipulation is required to correct this condition. Even then some evidence of vaginal eversion remains on deeper vaginal examination. It would appear, therefore, that total regression never occurs, no matter what degree of prolapse may have been apparent on initial clinical examination; hence it is possible to identify the original prolapse during subsequent oestrus cycles, during subsequent pregnancy, parturition or shortly after partus.

In practice the veterinarian generally encounters only prolapses of type II and type III, mainly because owners very rarely notice prolapses of type I: in most instances these are only evident on examinations per vaginem. In ignorance an owner may permit service of a bitch that has developed a slight vaginal eversion. The condi-

tion may become clinically apparent either during the ensuing metoestrum or during subsequent oestrus cycles. Only then is veterinary advice sought.

Type I prolapse:

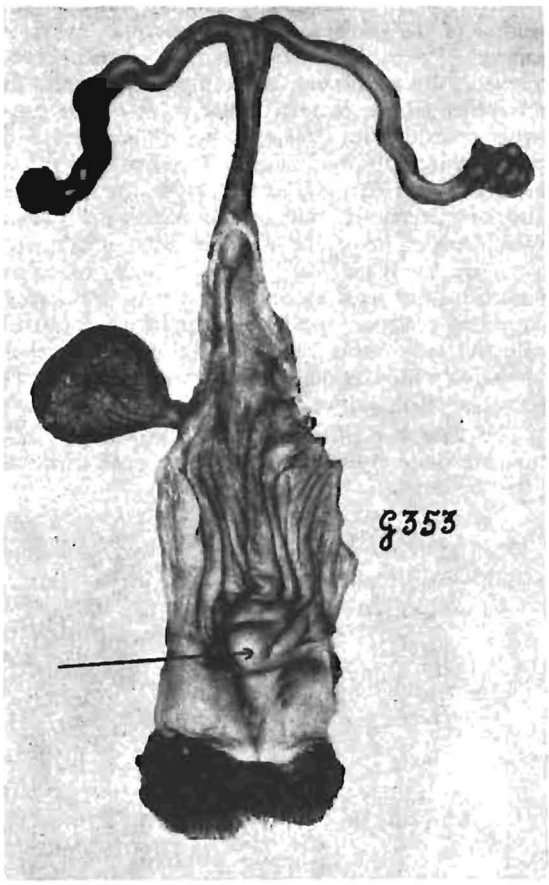
Genital organs of a bitch during the oestral phase, with slight eversion of the mucosa (see arrow) cranial to the urethral opening which partially occludes the latter. Note the well developed longitudinal folds in th vaginal mucosa.

Different methods and techniques such as resection of the prolapsed vaginal mucosa[4 15] hysteropexy[3 9 11 12] manual replacement or oöphorectomy[8 10], are advocated in treating these cases. In this series simple manual replacement of the prolapsed portion was followed by application of a purse string suture as described. When this treatment was carried out during oestrus the prolapse invariably recurred during the metoestral phase. When reduction of the prolapse was carried out when the bitch had entered metoestrum, the condition did not tend to develop until the following period of oestrus. Of all treatments carried out to date the only one which provides complete relief of the condition is oöphorectomy, or panhysterectomy for that matter.

The theory of hyperoestrogenism can be discounted since the pattern of oestrogenic stimula-

Type II prolapse:

Tipical vaginal prolapse which develops from the vaginal floor, cranial to the urethral opening. The urethral opening is indicated by the arrow. The prolapse does not involve the vestibular mucosa.



Regression of prolapse:-

Genital organs of a bitch during the metoestrum phase of the oestrous cycle, showing incomplete regression of a type II prolapse which was present during the oestral phase of the same breeding cycle.

tion of the vaginal mucosa follows the typical cyclic changes as seen in its exfoliative cytology. Many of the cases do in fact conceive to natural service or upon artificial insemination. In view of the efficacy of treatment by oöphorectomy, it is clear that oestrogens play an important part. In the case of oöphorectomy the main source of oestrogen is removed and relief is permanent. When manual replacement is carried out during oestrum, i.e. while the oestrogen level is high, relief is of short duration in contrast to the longer relief obtained by reduction during metoestrum when the oestrogen-progesterone balance is reversed. Reappearance of the prolapse during the ensuing oestrus or at parturition is again clearly associated with a rise in the oestrogen level.

Conclusion

The full pathogenesis can only become clear with a better understanding of the exact aetiology. Meanwhile it is postulated that the eversion/prolapse is due to high oestrogen levels occurring normally during certain phases of sexual function upon a locus anatomically and/or physiologically so disposed to be hypersensitive to their action.

Investigation of the aetiology of vaginal prolapse in the bitch must include a closer study of the possible effect of oestrogen on the specific zone of the vagina initially involved. Detailed morphological studies of this zone must also be undertaken in order to exclude the possibility of any structural differences in the genital tract and its ligamentous attachments in brachycephalic as against other breeds.

The distinct breed incidence of this condition suggests the probability of an hereditary susceptibility which also warrants further investigation.

ACKNOWLEDGEMENT

The Chief, Veterinary Research Institute, is thanked for permission to publish this article.

REFERENCES

1. Bloom, F. 1954 *Pathology of the Dog and Cat*. Evanston, Illinois. American Veterinary Publications Inc. p. 328.
2. Roberts, S. J. 1956 *Veterinary Obstetrics and Genital Diseases*. Arbor, Michigan, Edwards Brothers. p. 103.
3. O'Conner, J. J. 1957 *Dollar's Veterinary Surgery*. London, Baillière, Tindall and Cox. p. 765.
4. Frank, E. R. 1955 *Veterinary Surgery*. Minneapolis, Minn. Burgess Publishing Company. p. 253.
5. Williams, W. L. 1947 *The Diseases of the Genital Organs of Domestic Animals*. Worcester, Mass. E. Williams Plimpton. p. 502.

6. Rosenberger, G., and Tillmann, H. 1960 *Tiergeburtshilfe.* Berlin, Paul Parey. p. 215.
7. Arthur, G. H. 1964 *Wright's Veterinary Obstetrics.* London, Baillière, Tindall and Cox. p. 116.
8. Kirk, R. W. 1959 *Canine Medicine.* Evanston, Illinois. American Veterinary Publications Inc. p. 208.
9. Blakely, C. L. 1966 Prolapse of the vagina. *Current Veterinary Therapy.* Ed. R. W. Kirk. Philadelphia, W.B. Saunders Company. p. 421.
10. Allam, M. W. 1957 *Canine Surgery.* Evanston, Illinois. American Veterinary Publications Inc. p. 517.
11. McCunn, J. 1953 *Hobday's Surgical Diseases of the Dog and Cat.* London, Baillière, Tindall and Cox. p. 328'
12. Garbutt, R. J. 1945 *Diseases and surgery of the dog.* New York, Orange Judd Publ. Co. p. 291.
13. Schutte, A. P. 1964 *Die toepassing van sitologiese indekse soos aangetoon in die vaginale smere van tewe en die waarde hiervan ten opsigte van voortplanting.* Thesis, University of Pretoria.
14. Mulligan, R. M. 1942 *J. Morphology* 71:431.
15. Merrillat, L. A. 1943 *J. Amer. vet. med. Ass.* 103:286.

BOOK REVIEW

LAMENESS IN HORSES

By O. R. ADAMS

Lea & Febiger, Philadelphia. 1966

pp 563, Figs. 362

Second Edition

The first edition of Lameness in Horses appeared in 1962, and it was reprinted three times in as many years. Veterinarians will also appreciate the second edition which is an expanded version of this authoritative text book. The author has examined the knowledge and beliefs of the horse era and has explained many of their fallacies and truths. His wide experience is advantageously blended with the ideas from the hundreds of modern articles he lists in the bibliographies appended to each chapter.

The book is divided into twelve generously illustrated chapters. Chapter 10, Natural and Artificial Gaits, is not quite in keeping with the others: its material is better suited to a book entitled, say, Horses and Horsemanship. It could well be relegated to an appendix or omitted from the next edition. Other chapters cover the macroscopic anatomy and the physiology of the foot, and the influence of conformation on lameness. Consideration of the microscopic anatomy of, for example, bone and tendons, together with their junction, would be a welcome addition. Chapters on examination for, and diagnosis of lameness are followed by one on the pathology of musculoskeletal structures, and by another on methods for their treatment. Chapter 6, Lameness, is the longest and most important part of the book. It is divided into a section of forelimb lameness, another on hind limb lameness and a third on conditions common to both fore and hind limbs. This part could profitably be expanded by giving some case histories. Three separate chapters are devoted to shoes and shoeing, whereas their consolidation in one chapter would give better balance to the book. The chapter on radiology provides some basic information which one would be better advised to seek in a book on this speciality. Lameness in Horses is a "specialist" book in its field, and future editions would not suffer if the chapter on radiology were abridged: the sections on positioning and epiphyseal closure, amongst others, are pertinent and well done.

The book does illustrate the fact that before surgery can become a science, fundamental knowledge on tissue reactions to trauma and the influence of treatments on these reactions must be obtained. This can only be done if methods to inflict standardized lesions and their treatment with remedies of different types and grades are developed.

The book illustrates how far specialization has advanced in our profession, and it is perhaps unreasonable to expect undergraduate students to know its entire contents. Rather is it a book for specialists in equine or surgical practice, and for them it is highly recommended.

D. H. G. I.

INVESTIGATIONS INTO SWINE DYSENTERY IN NATAL

F. B. W. DUCASSE* R. C. NIXON*

SUMMARY

Field and limited laboratory investigations into four outbreaks of an enteric disease of swine in which dysentery in young pigs was the predominant symptom, are described. The economic effects of the disease in terms of weight loss are recorded and results obtained with chemotherapy are presented and discussed.

INTRODUCTION

During recent years, numerous outbreaks of dysentery in swine have been reported to the Veterinary Diagnostic Centre, Allerton, from piggeries throughout Natal. With a population of some 1,200,000 pigs in the province, including many pedigree herds, these reports became disturbing, particularly as the pig industry of this area had previously been remarkably free of the more important economic diseases of swine. Field investigations and post-mortem examinations confirmed the suspicion that vibrio's were associated with the majority of these outbreaks. Loveday[1] briefly reviewed the literature on swine dysentery, and reported his investigation of an outbreak in the Transvaal, stressing the grave economic effects of the disease.

The purpose of this report is to describe four outbreaks in this area, with particular reference to chemotherapy and the economic effects of the disease.

CLINICAL SIGNS

In general, the symptoms observed corresponded to the classical picture described by Doyle[2] and Loveday[1].

At post-mortem, the typical acute to chronic inflammation of the colon was constantly present. Examination of smears from such areas of inflammation, air dried and stained with carbol-gentian violet for 30 seconds, showed large numbers of vibrio-like organisms.

Our observations indicated that the severity of the clinical symptoms and associated pathological changes were to some extent dependent on the duration of the outbreak. On previously uninfected farms, the initial clinical picture was one of severe blood-stained scouring with marked emaciation. Generally within 6 to 12 months of the initial outbreak, further cases showed progressively less acute symptoms, intermittent scouring and failure to thrive being the main characteristics. On such chronically infected farms scouring occurred periodically; its occurrence seemed closely associated with changes of feed, sudden extreme weather changes and other stress factors. These observations appear to indicate that a degree of herd immunity develops on infected farms. The standard of hygiene and management also influence the period that acute cases, probably caused by massive infection, may occur.

Diagnosis in all cases was based on symptoms, autopsy and colon smears.

INVESTIGATIONS

Cedara outbreak. Early in 1964, the manager of a small pedigree piggery reported emaciation and chronic scouring in a litter of weaned pigs. Symptoms were first observed when the pigs were 12 weeks old and coincided with the substitution of sunflower meal for peanut cake meal in the ration. Assuming the cause to be a simple dietetic fault, he had not instituted treatment. When the scouring persisted, oxytetracycline had been added to the ration at a level of 100 g/per ton of feed for three days without success. Investigations revealed typical symptoms of swine dysentery and the disease was confirmed at autopsy. Hygiene on the farm was excellent. Records showed that previous average weights on the farm were 36.4 lb. at weaning and 74.5 lb. at 14 weeks. Pigs of the affected litter had an average weight of 28.8 lb. at weaning and 46.5 lb. at 14 weeks. Dysentery had thus reduced average weaning weights by 7.6 lb. and weights of weaners at 14 weeks by an average of 28.0 lb.

Treatment with tylosin tartrate in the drinking water was recommended but as the piggery was equipped with automatic drinking fountains, treatment had to be delayed until troughs had been

*Veterinary Investigation Centre, Allerton, Pietermaritzburg.

installed. Treatment commenced when the pigs were 16 weeks old, the drinking water being medicated at the recommended level of 250 mg tylosin tartrate per gallon for four days. At first the pigs refused the medicated water but drank it readily from the second day. The average weight of the affected pigs was 59 lb. at 16 weeks of age when treatment was commenced, and 83 lb. at 18 weeks. The average daily weight gain during the two weeks prior to treatment was thus 0.90 lb. and 1.71 lb. for the two weeks thereafter. Infection may have been brought on to the piggery by the introduction of a boar three months before the first cases occurred. Subsequently a positive diagnosis of swine dysentery was made on the farm of origin of this particular boar.

Zululand Outbreak

In April 1964 the owner of a commercial piggery reported severe scouring in piglets and weaners in a herd of some 50 breeding sows. Hygiene and management were good. Typical symptoms of dysentery were present in about 60% of all piglets from 4 to 16 weeks of age, the diarrhoea being particularly severe in the 10-14 week age group. Autopsy showed typical lesions in the colon with large numbers of vibrio-like organisms in smears.

The weaners were housed in groups of 20 in covered growing pens and fed *ad lib* on a commercial ration containing 17.5% crude protein.

In one pen 20 weaners, weighing approximately 45 lb. each at 12 weeks were divided into two groups of ten. One group was given tylosin tartrate in the drinking water at the rate of 250 mg per gallon for five consecutive days. The other group served as controls. All pigs in the two groups were weighed at the start and again 5 days later. The average weight of the pigs in the treated group was 42.3 lb. when treatment started. Diarrhoea ceased after 48 hours, and after treatment the pigs averaged 48.5 lb. Diarrhoea persisted in the control group which averaged 48.3 lb. and 52.3 lb. respectively. Weight gains per pig over the 5 day treatment period amounted thus to 6.2 lb. for the treated group and 4.0 lb. for the control group: a 2.2 lb. advantage in favour of the treated group.

As the piggery was subsequently sold no further investigation was possible.

Coastal Outbreak

In September 1964, the owner of a large commercial piggery on the Natal Coast reported severe scouring and poor weight gains in piglets and weaners in a herd consisting of some 120 breeding sows and their progeny.

Severe scouring was noted in the majority of pigs in the 4-12 week age group. The owner disclosed that the outbreak was first noted some 6 months previously after the basal crude protein in the creep ration had been increased from 18.5% to 21%. This ration was fed from creep age to sale weight (60 lb.). Assuming the diarrhoea to be of dietetic aetiology, the owner had tried various remedies without success, including vitamin and micro-nutrient fortification of the ration and antibiotic and nitrofurazone medication. Average weaning weights at 8 weeks of 38 lb. prior to the outbreak had dropped to 32 lb. Hygiene was excellent. Because breeding stock had been introduced frequently, it was impossible to establish the exact origin of the infection. As the piggery was equipped with automatic drinking troughs, the use of tylosin in the drinking water presented a problem. An attempt was made, therefore, to control the disease by reducing the crude protein in the ration to 14% and medicating this ration with arsanilic acid. This led to remission of symptoms, but poor weight gains resulted from the protein reduction. The basal protein concentration was then gradually increased but the scouring recurred at levels over 16%, so tylosin medication of the drinking water was undertaken. Suckling pigs were dosed orally with a solution of tylosin daily for 3 consecutive days. Very satisfactory control was achieved, but this method is not practicable under farming conditions. Supplies of tylosin phosphate as a feed additive were made available by the agents and field trials commenced in December 1964. The owner was not prepared to keep an untreated control group. Efficacy of treatment was assessed by the comparison of weight gains before the administration of tylosin on a ration of 21% protein and weight gains of treated pigs, on a ration of 17.5% crude protein.

Twenty four litters were fed a ration containing 17.5% crude protein, medicated at the level of 40 g. tylosin phospate per ton from 21 until 70 days of age. Twenty litters were given the identical unmedicated feed and dosed with tylosin tartrate at the level of ¼ g per piglet daily for three consecutive days as soon as evidence of scouring was noted. This treatment had to be repeated in 7 of the 20 litters after weaning, as scouring had recurred.

Details of these litters and weight gains are recorded in Table I.

At weaning the piglets fed medicated feed weighed an average of 4.4 lb. more than those dosed, and 6.7 lb. more than piglets prior to the introduction of tylosin medication. At 70 days of age the average weight of the piglets receiving me-

TABLE I.

Group	Number of litters	Number weaned	Average weaning weight	Average weight at 70 days	Details of crude protein of ration:
Pre-treatment Controls	50	434	32.0 lb.	48.0 lb.	21%
Orally dosed group	20	182	34.3 lb.	50.0 lb.	17.5%
Medicated feed group	24	227	38.7 lb.	52.5 lb.	17.5%

dicated feed was 2.5 lb. more than that of the tylosin dosed group and 4.5 lb. more than of weaners of similar age prior to the use of tylosin. This increase was obtained in spite of reduction of the crude protein content of the feed from 21.0% to 17.5%.

In order to ascertain the lowest level at which tylosin phosphate in the feed would prevent scours, the level was gradually reduced from 40 g/ton to 20 g/ton. At levels below 20 g/ton, diarrhoea consistently occurred. Odd cases occurred in litters on levels between 20 and 30 g/ton but none on levels above 35 g/ton.

Richmond Outbreak

In 1965 a large commercial piggery reported scours and poor weight gains in weaners. The piggery consisted of roughly 250 breeding sows and their progeny — a total of some 2,500 pigs. Feeding and management were excellent and hygiene was good, except that effluent from the weaner houses drained into the sties. The scouring had first been noticed about one month previously, shortly after the introduction of a group of 22 breeding sows. At the same time there was a change of brand of feed. Analysis of the latter failed to establish the cause of the scouring. After unsuccessful attempts to control the outbreak with both broad spectrum antibiotics and nitrofurazone, this centre was approached for assistance. Swine dysentery was established by autopsy on numerous pigs.

As medication of the drinking water was impractical, tylosin phosphate was recommended as a feed additive. To assess the economic and other aspects of this control method, 30 in-pig sows were placed in thoroughly disinfected sties. After farrowing, the sows and litters were divided into four groups as follows:

Group A consisted of 10 litters fed normally and kept as untreated controls.

Group B consisted of 10 litters fed medicated feed (50 g tylosin additive per ton) from creep-feeding age until the weaners weighed 110 lb; thereafter unmedicated feed was fed until they were marketed at 200 lb. liveweight.

Group C consisted of 8 litters fed tylosin medicated feed (100 g tylosin feed additive per ton) from creep-feeding age until an average liveweight of 110 lb., afterwards unmedicated feed until marketweight of 200 lb.

Group D consisted of 2 litters fed tylosin medicated feed (100 g tylosin additive per ton) from creep-feeding age until they were marketed at 200 lb. liveweight.

All groups were fed a 19.1% crude protein basal ration from creep-feeding age (approximately 3 weeks) until weaning at 8 weeks, when the protein content was reduced to 17.1%. When they had attained a live weight of 110 lb. the protein content was further reduced to 14.2% at which level they were fed until reaching market weight. Tylosin was mixed with the food by machine to ensure its even distribution.

The results are recorded in Table II.

No evidence of enteritis could be detected in group D pigs at slaughter but in 10 (71%) vibrio-like organisms were readily detected in smears from the colonic mucosa.

Since the middle of 1965 seventeen further outbreaks have been confirmed in this area and ade-

TABLE II.

Group	Number of pigs	Tylosin g/ton	Weight to which Tylosin fed	Average age to 200 lb. liveweight	Feed Conversion
Group A (controls)	96	Nil	—	189 days	3.41
Group B	100	50	110 lb.	167 days	2.91
Group C	84	100	110 lb.	163 days	2.89
Group D	14	100	200 lb.	163 days	2.89

quate control was obtained in all using tylosin medication in the feed or water. In one small piggery, tylosin tartrate in the drinking water at a level of 500 mg gallon was continued for three consecutive weeks to ascertain if this would clear an infected farm of the disease. The disease was suppressed for some 4 weeks after which time visible scouring recurred.

Results

In our investigations swine dysentery appeared to reduce weaning weight by an average of 6.5 lb. and to increase the average market age for baconers by 22 days. At present day prices medication of drinking water for 5 days at a level of 250 mg tylosin tartrate gallon costs approximately 20 cents per pig. Feed medication with tylosin phosphate, at 40 g/ton from 3 weeks of age until attainment of approximately 100 lb. liveweight, costs 60 cents per pig.

Tylosin feed medication also improved the feed conversion of swine dysentery infected pigs from 3.41 to 2.90.

While tylosin therapy had a definitely favourable influence on the course of swine dysentery and, as a feed additive, a completely preventative action, it failed to "cure" the disease or inhibit the development of those vibrionic forms in the colon, suspected of being the cause.

Tylosin tartrate at a level of 250 mg/gallon of drinking water for four or five consecutive days, or given in 5 ml. daily dosage of a 5% w/v aqueous solution for 3 consecutive days, was the cheapest control measure for infected pigs. However, this method is often impractical on farms equipped with automatic watering facilities, or in large piggeries.

Tylosin phosphate at the level of 40 g/ton of food for all piglets from 3 weeks of age until attainment of 75 or 100 lb liveweight, appeared in most cases to be the most practical and definite method of control, although slightly more expensive.

Discussion

Observations of some 20 outbreaks of swine dysentery have indicated that clinical symptoms often are accompanied by a sudden change of feed. Remission of symptoms frequently occurred spontaneously, but fresh outbreaks were prone to follow sudden climatic changes or other conditions of stress. The disease tended to occur mainly in pigs on a high plane of nutrition, especially in regard to crude protein. In most cases, outbreaks followed the introduction of mature breeding stock, thus confirming Loveday's findings and supporting the views of Terpstra, Akkermans and Ouwerkerk[3] that the appearance of the disease follows weeks or months after the introduction of clinically healthy excretors of infection. In two swill feeding piggeries scouring piglets were subjected to autopsy or faecal smear examination. Although odd cases of scours occurred on both farms, no large scale outbreaks of scours were recorded from either. Vibrio-like organisms were seen occasionally in very small numbers in colon smears from such scouring piglets. Clinical symptoms of swine dysentery were observed mainly in suckling piglets shortly after they commenced creep feeding, or in weaners shortly after weaning, when changed from creep ration to growing ration. Clinical symptoms of the condition have never been observed by us in pigs of over 100 lb. in weight on a finishing ration containing less than 15% crude protein, and the reduction of the crude protein content of rations of scouring piglets to approximately 14% has generally led to a marked reduction of scours. According to Terpstra *et al*[3], reports from practice suggest that less severe symptoms are seen in pigs on half rations. The severity of the symptoms would thus appear to be associated with both the quality and quantity of the ration.

The gradual decline in severity of clinical signs on infected farms indicates that some degree of immunity develops. Whether such immunity is due to a primary pathogen only, or whether it is associated with possible secondary infections, must await further study.

Stevens[4] states that available evidence of *Vibrio coli* being a frequent primary pathogen in swine enteritis in the United Kingdom is not convincing. He considers that haemorrhagic and necrotic enteritis are related to each other and to oedema disease, and may have the same origin, probably a shock phenomenon following earlier sensitisation to *E. coli* mucopolysaccharide.

Smith[5] mentions the close pathological association of *Bacteroides* with necrotic enteritis of swine, and suggests that the high degree of activity of tylosin tartrate against *Bacteroides* may provide an alternative or additional explanation for its beneficial effect in swine enteritis.

Sweeney[6] associates the disease with Salmonella infection. If this infection is the main basic cause of the syndrome, some beneficial effect could be expected from the use of therapeutic or prophylactic doses of the nitrofurazone drugs. We observed no such benefits. Gardner, used chloramphenicol intramuscularly and streptomycin

orally with little or no effect. Scott[8] employed a variety of drugs, including streptomycin, neomycin, tetracyclines, framomycin, nitrofurans, sulphonamides and arsenicals, both orally and parentally, with a temporary beneficial effect from all, with frequent relapses within 8 to 20 days after cessation of medication. Both these workers recorded dramatic and immediate benefits from the use of tylosin.

Although the use of arsanilic acid and oxytetracycline[1] led to remission of symptoms, relapses often occurred after the drugs had been withdrawn. Terpstra, *et al*[3] reported that oxytetracycline administered at a level of 50 g/ton of feed for 14-20 days, had definite therapeutic effect, but did not always prevent infection of new additions to the treated group, whereas tylosin tartrate at the level of 100 g/ton of feed for 18 days was very effective, scouring disappearing after 4 days. The condition of the pigs improved rapidly and subsequent additions to the treated group did not become infected.

Whatever the aetiology, it is our experience that typical swine dysentery is always associated with the presence of large numbers of vibrionic organisms in stained smears prepared from the colon i.e. mucosa. Pigs feed close to the ground, often in close contact with excreta from other swine and are thus more exposed to the hazards of oral infections than most other classes of livestock. Such infections are facilitated by the use of unsuitable troughs, lack of trough space and poor hygiene. Under these conditions swine dysentery in a herd is likely to present a recurring problem to both the veterinarian and the pigkeeper, despite the efficient available therapy.

ACKNOWLEDGEMENTS

We thank the Chief, Veterinary field Services for permission to publish, Dr. R. K. Loveday for assistance and encouragement throughout, and Messrs. Elanco S.A. (Pty.) Ltd., for the supplies of tylosin tartrate and tylosin phosphate for clinical trials.

REFERENCES

1. Loveday, R. K. 1964 *Jl. S. Afr. vet. med. Ass.* 35: 51.
2. Doyle, L. P. 1964 *Diseases of Swine* 2nd Edit. ed. Howard W. Dunne, Ames. Iowa State University Press.
3. Terpstra, J. I., Akkermans, J. P. W. M. and Ouwerkerk, H. 1965 *Tijdschr. Diergeneesk.* 90: 986.
4. Stevens, A. J. 1963 *Brit. vet. Jl.* 119 520.
5. Smith, H. Williams 1965 *Vet. Rec.* 77: 1342
6. Sweeney, E. J. 1966 *Vet. Rec.* 78: 372.
7. Gardner, P. L. 1965 *Vet. Rec.* 77: 1493.
8. Scott, W. A. 1965 *Vet. Rec.* 77: 1460.

Ter Nagedagtenis

Dr. J. H. Schoeman

Ons het met geskokte leedwese gehoor van die skielike en onverwagte afsterwe op 9.3.67 van ons ou vriend en tydgenoot, Johannes Hendrik Schoeman.

Hy was daardie aand met sy vrou en twee vriende op pad na Gravelotte vanaf Letsitele toe hy 'n hartaanval opgedoen het en is hy op Gravelotte oorlede voor hulp hom kon bereik.

Hy is op die familieplaas Schoemanshoek, Oudtshoorn gebore op die 19e Junie 1907. Hy het by die Hoërskool Outeniqua, George, gematrikuleer, daarna in 1927 sy B.Sc. op Stellenbosch behaal, en in 1933 sy B.V.Sc. op Onderstepoort voltooi.

Na voltooiing van sy opleiding het hy tot die Staatsdiens toegetree en was hy as Staatsveearts op Umatata, Armoedsvlakte en Vryheid werksaam. Daarna het hy op Johannesburg en Springs onderskeidelik 18 jaar privaat gepraktiseer. Hy het drie jaar op Potgietersrust gepraktiseer en daar was hy vir dieselfde periode voorsitter van die Vliegklub. Vir die afgelope sewe jaar het hy by Letsitele, langs die Letabarivier, met sitrus geboer en deeltyds volgehou met sy praktyk. Die jaar voor sy dood was hy Ere-President en Ere-Veearts verbonde aan die Tzaneenskou.

Vanaf 1941 tot 1943 het hy op die Voedselbeheerraad gedien.

Hy het altyd 'n groot voorliefde gehad vir enige voorwerp wat met meganiese krag aangedrewe word vandaar sy voorliefde vir vlieg en vliegtuie en deelname aan snelbootwedrenne.

Hy was altyd bekend as 'n interessante en opgeruimde persoonlikheid.

Dr Schoeman is op 3.10.36 met Mej. Sarah Vermeulen van Port Elizabeth getroud. Daar is twee kinders, Letitia en Hennie uit die huwelik gebore. Letitia is getroud en het 'n dogtertjie.

Ons dra ons innige medelye aan sy weduwee en kinders oor.

BOOK REVIEW

LEHRBUCH DER VETERINAR—PHYSIOLOGIE

Editors.

A. SCHEUNERT AND A. TRAUTMANN. 1965.

Paul Parey, Berlin and Hamburg. 848 pages including index 287 illustrations.

Published price.

Text books devoted to the physiology of domestic animals are few and far between. The general works of this nature which are available are in many respects inadequate by modern standards. Topics like the physiology of reproduction, lactation, growth and ruminant digestion are usually well covered for the purposes of pregraduate teaching and numerous books dealing solely with these topics are in existence. Neuromuscular, cardiovascular, renal and respiratory physiology are dealt with mainly by using the physiology of the human and laboratory animals as background material. Such texts are full of generalizations which are often untrue for various species of domestic animals. Few textbooks on general veterinary physiology attempt a realistic union of the physiological and biochemical processes involved in any body function and the pregraduate student has been largely left under the impression of having two unconnected disciplines to study.

This book is admirable in many respects. In the first instance it is the work of six different authors under the editorship of Scheunert and Trautmann, all of whom are specialists in various fields of veterinary physiology. This is a well-known modern trend in scientific writing. The combined result in this instance is a refreshing change from the stereotyped texts of previous years. Secondly the authors have in one volume included and interwoven most of what is contained in pregraduate veterinary physiology and biochemistry curricula. The student is left in no doubt as to the indivisibility of the two disciplines.

The approach of the authors to the compilation of their book is to be commended for teaching purposes. The opening chapters deal with, in order, the chemical composition of the animal body, chemical and nervous regulation of life processes, (with the hypothalamus placed in a dominant position) and intermediary metabolism. This is all as it should be, and is an approach which we have used successfully for some years. The student is led through the fields of general cellular physiology, to the consideration of that of the intact animal and he should in theory find the transition a gentle one, since he has found his way there by means of logical steps. The succeeding chapters embrace in order digestion, absorption, secretion and excretion, blood and the circulatory system, tissue fluids, respiration, metabolism, locomotion, neurophysiology, the special senses and finally reproduction and lactation.

The book is an outstanding one for pregraduate students and for those who wish to refresh their knowledge of physiology gleaned so painfully in years gone by. It is to be commended for use by veterinarians in general practice. Cardiovascular and neurophysiology are well discussed, particularly the former. The practitioner will find an excellent presentation of electrocardiography; in fact one of the best I have seen in general works of this nature. Special senses and locomotion also receive excellent cover as likewise do respiration and excretion. There is very little in fact which is not of value to the student and general practitioner.

The specialist in various fields of veterinary physiology will be disappointed in many of the chapters. Ruminant digestion, reproduction and mineral metabolism to name a few topics are dealt with adequately for student work but one must have recourse to more specialised textbooks for advanced and detailed information. The biochemist likewise will encounter his disappointments. Their magnitude will depend on his interest and temperament. It was, for instance, a distinct shock to the reviewer, when the structure formula given for bilirubin was seen. The subsequent discussion on bile pigments took me back nearly twenty years to those days of glorious confusion of haematoidin, haemosiderin and haematin with which we are all familiar. In view of the rapid expansion of our knowledge in this field, it was an acute disappointment to see old mistakes perpetuated and to find much of the discussion limited to irrelevant chemical reactions of bilirubin itself. The chemical

pathologist will, by the same token, be disappointed in a lack of accentuation of those particular areas of biochemistry, which find application in clinical laboratory medicine.

Notwithstanding its errors and limitations, and I have yet to see a general text book without them, I feel justified in recommending this book to all those who wish to acquire a useful background of veterinary physiology. The text is in German, but written in a most readable fashion. It should present no difficulties to those who studied German as a school subject. The liberal use of illustrations (which are in many cases outstanding), and comprehensive tables adds tremendously to the educational value of this book. It is printed on high quality glossy paper and the standard of production places it in the forefront of modern veterinary text books.

J. M. M. B.

RHODESIA

MINISTRY OF AGRICULTURE

VETERINARY SURGEONS

Applications are invited from qualified veterinary surgeons for appointment in the Department of Veterinary Services on two or three year contracts.

Duties are varied and interesting and consist mainly of investigational and extension work and the control of notifiable diseases.

Appointment may be on permanent terms on the salary scale of £1620 × £75 to £1695 × £105 to £1800 × £90 to £2250 × £125 to £2500 × £150 to £2650 p.a. with a maximum commencing salary of £2375 p.a. giving credit for 8 years previous experience.

Appointments will be considered on two or three contracts within the salary range of £1800 to £2650 per annum. Salary will in either case be assessed on qualifications and relevant previous experience.

Passages for recruit and family to Rhodesia provided, with return passages on satisfactory completion of contract.

Generous leave, medical aid, good educational facilities, low income tax, opportunity for sport and recreation in admirable climate.

Application forms and further information available from the Director of Veterinary Services, P.O. Box 8012, Causeway, Salisbury, RHODESIA.

INDEX TO ADVERTISERS