THE SOUTH AFRICAN VETERINARY MEDICAL ASSOCIATION

THE PAPERS

To be read before

THE FIFTY-SEVENTH ANNUAL CONGRESS
SEPTEMBER 25th — 28th, 1962

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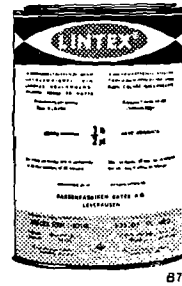

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PROGRAMME

SCIENTIFIC CONGRESS AND FIFTY-SEVENTH ANNUAL GENERAL MEETING

FACULTY BUILDINGS, ONDERSTEPOORT

25–28 SEPTEMBER, 1962

(Contributors to propose introducers and recorders to the Secretary)

TUESDAY, 25 SEPTEMBER, 1962

8.00 a.m.	Registration.
9.00 a.m.	Convocation of the Meeting.
9.05 a.m.	WELCOME TO GUESTS, DELEGATES AND MEMBERS BY HIS WORSHIP THE MAYOR OF PRETORIA, MR. ERNEST SMIT.
9.15 a.m.	OPENING OF THE CONGRESS BY THE HON. MR. P. M. K. LE ROUX, MINISTER OF AGRICULTURAL TECHNICAL SERVICES.
9.30 a.m.	EXPRESSION OF THANKS AND APPRECIATION TO THE MINISTER BY THE CHIEF OF THE VETERINARY RESEARCH INSTITUTE, ONDERSTEPOORT, DR. B. C. JANSEN
9.45 a.m.	PRESIDENTIAL ADDRESS.
10.00 a.m.	OPENING OF THE TRADE EXHIBITION BY THE VICE-PRESIDENT.

TEA

11.15 a.m. (with break for lunch)	Symposium on Rabies in Animals in South Africa. (*a*) An Introduction to the Subject — R. A. ALEXANDER. (*b*) The Epizootiology and Diagnosis of the disease — C. J. MARE. (*c*) An Analysis of Histological Examinations — R. C. TUSTIN and J. D. SMIT (*d*) Field Control of Rabies — P. R. MANSVELT. (*e*) Rabies and the Private Practitioner — A. F. TARR, J. M. O'GRADY and W. L. JENKINS. (*f*) Rabies from the Point of View of the Medical Officer of Health or the Medical Practitioner — H. NELSON.
4.00 p.m.	GROUP MEETINGS.

8.00 a.m.–4.00 p.m. **TRADE EXHIBITS.**

8.00 a.m. Registration.

9.00 a.m. (with break for tea) Fifty-Seventh Annual General Meeting of Members.

AGENDA

1. Notification of Meeting and confirmation of the Minutes of the Fifty-Sixth Annual General Meeting.
2. Matters arising from the Minutes of the previous Meeting held at Durban on 27 September, 1961.
   (*a*) Resolutions (i) and (ii) from 1962 A.G.M.
   (*b*) Use of the title of M.R.C.V.S.
   (*c*) Refresher courses at the Veterinary Faculty.
3. Report by the President of the Council's activities.
4. Amendments to the Veterinary Act (Act No. 16 of 1933).
5. Membership:
   (*a*) Deaths.
   (*b*) Resignations and removals from Membership.
   (*c*) Election of new Members.
   (*d*) Associate Membership.
6. Consideration of Reports:
   (*a*) Income and Expenditure A/c. and Balance Sheet.
   (*b*) Standing Committees.
7. Publication of the Journal of the Association — Assistance by the Department of Education, Arts and Science.
8. Veterinarians in Commerce.
9. Current Committees of Investigation.
10. Amendment to the Constitution of the Association.
11. World Veterinary Association.
12. Notification of election of Council Members.
13. General.
14. The Venues for the 1963 and 1964 Congresses.
15. Resolutions.
16. Adjournment.

2.00 p.m.	Studies on Specific Oculovascular Myiasis of domestic animals (Uitpeuloog)	P. A. Basson
3.00 p.m.	Bluetongue — recent advances in research	P. G. Howell R. M. du Toit
4.00 p.m.	Group Meetings.	

8.00 a.m.–4.00 p.m. TRADE EXHIBITS.

THURSDAY, 27 SEPTEMBER

8.00 a.m.	Registration.	
8.30 a.m.	Skin lesions in South African Domestic Animals with special reference to the incidence and prognosis of various skin tumours.....	J. D. Smit
9.15 a.m.	Etiological Factors in Geeldikkop and Enzootic icterus.....	J. M. M. Brown
10.00 a.m.	Treatment of Heartwater in Sheep and large-scale Immunization against Heartwater in Small Stock.....	J. D. Poole

TEA

11.15 a.m.	Film — **Inyamazane** (Control of Game in the Kruger National Park).	
11.45 a.m.	Foot and Mouth disease in Game Animals	M. J. N. Meeser

AFTERNOON SPORT

FRIDAY, 28 SEPTEMBER

8.30 a.m.	Demonstrations by The Reproduction Section, Veterinary Research Institute, Onderstepoort:	
	(*a*) Production, recovery, preservation and transport of Ova	S. J. van Rensburg I. S. McFarlane
	(*b*) Pudendal block in the bull and ram	I. S. McFarlane
	(*c*) Collection of bovine semen by massage of the Ampullae.....	I. S. McFarlane

TEA

11.15 a.m.	Preliminary communication on the cultivation of Besnoitia besnoiti (Marotel 1912) in tissue culture.....	R. D. Bigalke
12.00 noon	The Health of the Weanling Pig.....	R. K. Loveday
2.00 p.m.	Films.	
3.00 p.m.	Resolutions.	
3.30 p.m.	Adjournment.	

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RABIES IN SOUTH AFRICA
THE EPIZOOTIOLOGY AND DIAGNOSIS OF THE DISEASE

C. J. MARÉ — Section of Virology, Veterinary Research Institute, Onderstepoort

(Received for publication, July 1962)

SUMMARY

A brief review of the growth of the rabies problem in South Africa is given. The change in the geographical distribution of the disease which has come about during the past decade is discussed, with particular reference to the increasingly important role of the dog as a carrier of the disease. The two great rabies epizootics of recent years are discussed, and their influence on the overall rabies picture evaluated. Rabies was shown to be subject to seasonal fluctuations and hypotheses as to the reason for these fluctuations have been put forward. In conclusion a short resume of the diagnostic procedures adopted in South Africa is given.

INTRODUCTION

The existence of rabies in South Africa was first referred to by Thunberg;[8] (1780), when writing of his travels through Africa. In 1857 however, David Livingstone recorded his surprise at the absence of the disease in this country. This observation was soon proved erroneous when in 1893 Duncan Hutcheon diagnosed rabies in a dog and confirmed his diagnosis by sub-inoculation into rabbits.

Further investigations revealed that the disease was more widespread than was formerly believed, and by 1934 the disease had been diagnosed in twenty-five magisterial districts mainly in the Northern Cape Province, the Western Free State, and the South Western Transvaal.

Numerous reviews of the rabies position in South Africa have been written during the past fifty years, the most notable being those of du Toit[2] (1929), Neitz and Thomas[6] (1934), Snyman[7] (1940) and Alexander[1] (1952). These authors have in turn described the problem of rabies in South Africa, the distribution and spread of the disease, the animals affected, and in some instances the control measures which were applied. It is in an effort to describe the rabies situation in South Africa at the end of 1961 that this article is being written.

THE DISTRIBUTION OF THE DISEASE AND THE SPECIES AFFECTED

Rabies is now present in every province of South Africa. On perusal of the distribution map (Fig. I) it will clearly be seen that practically the entire Orange Free State, the Eastern, Northern and Western Transvaal, the Northern and North-Western Cape, and almost half of Natal are now rabies infected areas.

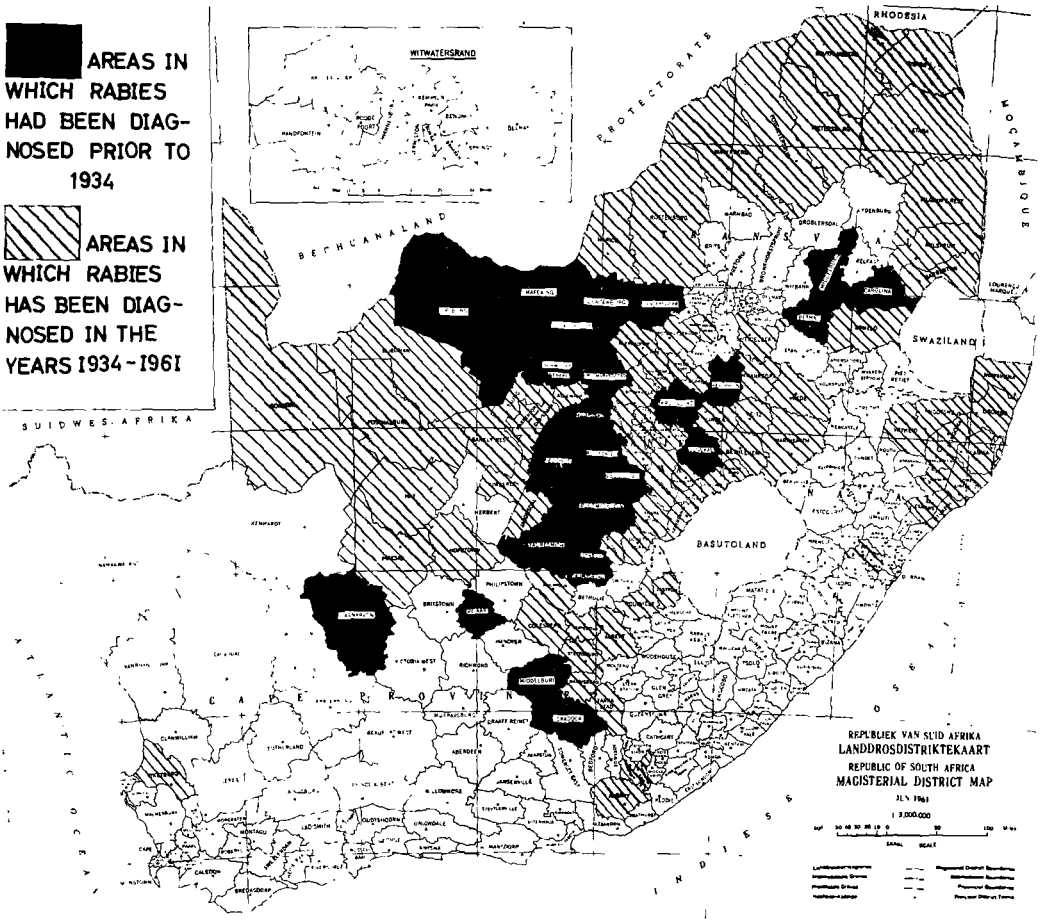

FIG. I.—"Map of South Africa showing districts in which rabies has been diagnosed"

How did this state of affairs come about? All the earlier reviews on rabies in this country have stressed that the disease occurred mainly in the Orange Free State, the Western Transvaal and the Northern Cape (see black areas on map) and have in addition observed that the incidence of the disease is related to the regional population density of various species of the family viverridae (meercat, suricate, etc.). The chief species affected were cattle and meercat, with dogs being of relatively minor importance. The disease was thus a disease of the viverridae and of cattle, rather than of dogs as is the case in Europe. Even in its clinical manifestations the South African disease differed from the classical "mad dog" syndrome observed in other countries. Alexander[1] in 1952 described experiments which were performed in an attempt to transmit the disease from rabid meercat to dogs. Although these dogs were

severely bitten by the rabid animals they did not contract the disease. This phenomenon Alexander puts down to viral loss of infectivity for the dog through its long association with the viverridae.

Today, rabies in South Africa has taken on a somewhat more complex form. In the old "meercat-rabies" areas the position is much as it was in the past, but in the other important areas, for example in the Northern and Eastern Transvaal and in Natal, an entirely new situation has arisen. Rabies in these areas can be regarded as being essentially "dog rabies", the vast majority of cases being in dogs, with a few jackal and cattle succumbing to the disease.

This state of affairs resulted from the two great rabies epizootics which South Africa has experienced during the past decade.

The Northern Transvaal Epizootic

In 1952, after a few sporadic cases of rabies had been reported from the Northern Transvaal, Alexander predicted that this area was in danger of becoming a rabies infected area. This supposition was soon proved accurate and within a few years the districts of Sibasa, Soutpansberg, Letaba, Pietersburg and Potgietersrust were experiencing an epizootic of a type hitherto unknown in South Africa. The disease was initially confined to dogs, but has subsequently become enzootic in the wild animal population in these areas. Vigorous control measures (mass vaccination and destruction of stray dogs) have markedly reduced the incidence of the disease, but the wild animals remain a reservoir for the infection.

The Natal Epizootic

The second half of 1961 was to see the next great change in the rabies picture in South Africa. In July of that year rabies was diagnosed in the hitherto rabies-free province of Natal. This diagnosis precipitated an intensive investigation by the Field Service of the Division of Veterinary Services, and by the end of the year sixty-six cases of rabies had been diagnosed in this province, thirteen magisterial districts being affected. At this stage rabies had been diagnosed in the districts of Ingwavuma, Ubombo, Hlabisa, Nongoma, Vryheid, Lower Umfolozi, Ngotshe, Entonjaneni, Pietermaritzburg, Mtunzini, Eshowe, Durban and Mahlabatini (see map).

An analysis of the species affected in this outbreak reveals that in Natal typical dog rabies is encountered. Of the 66 cases diagnosed, 50 have been in dogs, 14 in cattle and 2 in domestic cats. Not one case of rabies in a wild animal has been observed in Natal.

As a result of these two outbreaks, the disease has become established over a very much greater area than was previously the case. What now of the actual number of cases recorded?

In Table I a comparison is made of the figures for the 33 year period 1916 to 1949[5], with those for the 9 year period 1953 to 1961.

TABLE I

Known cases of rabies for the periods 1916 to 1949, and 1953 to 1961

	1916 to 1949	1953 to 1961
Cattle	150	299
Meercat	142	171
Dogs	50	321
Domestic cat	31	54
Genet	19	6
Jackal	3	35
Wild-cat	8	9
Domestic pig	6	4
Horses	4	1
Donkeys	3	1
Other species	1	7
TOTAL	417	908

Several interesting observations can be made from the figures given in this table. During the period 1916 to 1949, 417 cases of rabies were recorded, while in the 9 year period 1953 to 1961, 908 diagnoses were made Admittedly better knowledge of the disease, public awareness and improved diagnostic methods all contributed to the higher number of cases diagnosed in the latter period, but the increase is still so spectacular that one must conclude that rabies is on the increase in South Africa.

Rabies in the dog has obviously become a major problem in South Africa as the tremendous increase of cases in this species indicates. While dog rabies has been successfully controlled in many civilised countries, it must be remembered that in South Africa the areas in which this form of rabies is encountered embrace some of the largest native reserves, making effective control virtually impossible.

THE SEASONAL OCCURRENCE OF RABIES IN SOUTH AFRICA

Rabies is transmitted through the bite of a rabid animal. If the disease was insect-borne, one would expect a seasonal variation in the incidence, but where contact with an infected animal is the essential factor in transmission, such variation would not be expected. In an effort to determine whether the incidence of rabies was in fact influenced by season, the results of positive tests performed during the eight-year period 1953 to 1960 were analysed, and it was found that a fairly consistent seasonal pattern did in fact emerge.

TABLE II

Peak months, and months of lowest incidence of rabies for the years 1953/1960

	Highest incidence		Lowest incidence	
	Month	No. of cases	Month	No. of cases
1953	October	10	March	3
1954	August	15	February	6
1955	June	11	November	4
1956	November	13	April	1
1957	July	18	February	4
1958	July	16	March	1
1959	August	17	December	3
1960	June	12	April	1

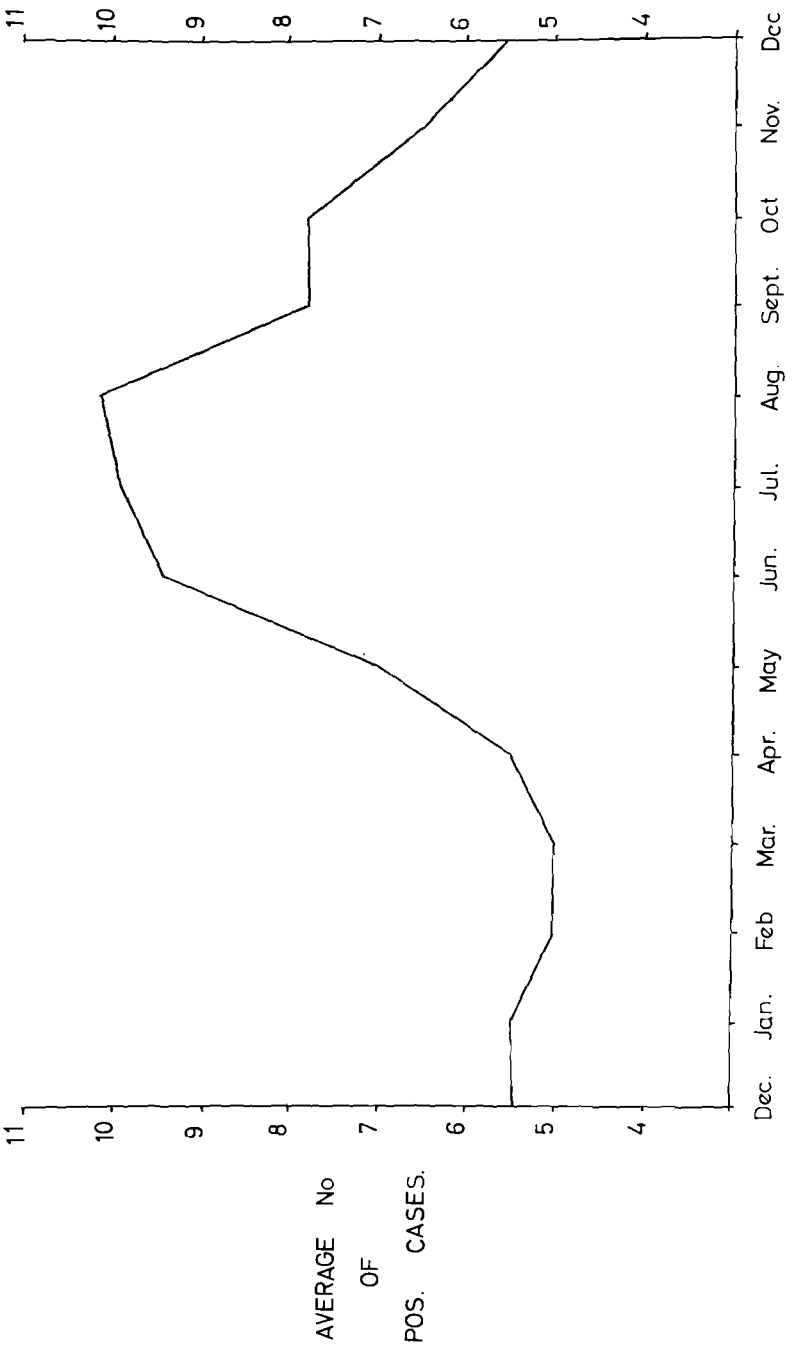

FIG. II.—"Graphic illustration indicating the average monthly incidence of rabies for the years 1953 to 1960."

From the figures in Table II it is immediately evident that in every case the peak month fell in the winter or spring, in fact in six out of the eight years the peak fell in the 3 month period June to August. The lowest incidence in every case occurred in the summer or autumn ,and in six out of the eight years, in the three month period February to April.

This graph, compiled from the results of tests performed during the mentioned period, illustrates clearly the seasonal trend in the incidence of rabies. The question now arises, why is there a seasonal variation?

The months of highest incidence are the months when the vegetation in the rabies areas is at its lowest. Consequently, sick meercats, which in the ordinary course of events would not be easily found, are conspicuous, and thus more specimens are found and submitted for examination. Furthermore during these months meercats are inclined to wander further afield in search of food than is the case during the summer. In this way an actual spread of the disease may be accomplished.

Rabid meercats would be more easily seen by cattle during these months, and the natural curiosity of the bovine together with the abnormal boldness of the rabid meercat, would result in contact. This could be the reason for the increase in bovine rabies in the winter and spring months.

Dog rabies is less affected by seasonal fluctuation, and it is probable that now that this form of rabies is on the increase, the overall fluctuation will be considerably reduced.

The Diagnosis of Rabies in South Africa

The history of the case and the clinical symptoms observed are very important in the diagnosis of the disease. There are however, numerous other disease conditions which produce symptoms indistinguishable from those seen in rabies. In the dog, distemper, strychnine poisoning and even verminosis can be confused with rabies. In cattle, heartwater, tetanus, sporadic bovine encephamyelitis, plant and mineral poisons, may cause confusion, while viral or bacterial encephalitis in any species of animal could result in a clinical diagnosis of rabies. The importance of confirming a diagnosis of rabies cannot be over-stressed. It is important that the diagnostic procedures be fully understood by all persons liable to submit specimens for diagnosis, and for this reason a short description of the diagnostic tests performed is given.

The Laboratory Diagnosis of Rabies

It is essential that specimens be submitted for histological and for biological examination if a rapid and accurate diagnosis is to be made.

For histological examination hippocampus in 10% formalin must be submitted. In large animals this portion of the brain is easy to identify, but in small animals, where difficulty may be experienced, it is advisable to submit half the brain in formalin. On arrival, the formalised specimens are processed and examined for *Negri* bodies.

A very rapid, on the spot, diagnosis may be performed by the microscopic examination of hippocampus impression smears stained with

Seller's stain. The staining process is very simple, but the examination and evaluation of the smears requires some experience. Smears are prepared and stained in the following way:—

The hippocampus is removed, a small piece thereof placed on a piece of filter or blotting paper, and a clean slide firmly pressed down on the piece. Blot the smear, thus removing excess brain material, open the stain, dip the wet unfixed smear, into the stain for about 3 seconds, wash off in tap water, and air-dry.

If the stain is correctly balanced, the erythrocytes will stain a reddish-orange colour, nuclei of the brain cells bright blue, the background pinkish, cytoplasm of the cells light blue, and the *Negri* bodies a magenta colour (reddish-purple) with dark granules showing up on careful examination. In the brain, *Negri* bodies are intracytoplasmic, but in impression smears they are often found lying loose, due to the technique of smear preparation.

In dogs and cats, confusion may be caused by the presence of reddish inclusions very similar to the *Negri* body. The inclusions of Distemper and Feline panleucopaenia are hyaline in appearance and no granular structure can be seen. It is always advisable, when any doubt exists, to regard the case as "highly suspicious" pending the result of the biological test.

The preparation of the stain involves titration of the two components to obtain the correct balance between blue and red, known positive smears being used in this procedure. It is advisable to obtain the stain from Onderstepoort where it can be prepared on a large scale.

For the biological test, brain (not necessarily hippocampus) in 50% glycerin should be submitted. On receipt of this brain material, a 10% suspension in saline is made, and inoculated intra-cerebrally into 3-week-old Swiss Albino mice. These mice are observed for a period of 35 days after which, if no mortality has occurred, a negative result is given out. If the case is positive however, the mice will show symptoms of paralytic rabies from the 7th day onwards, usually dying within 24 hours of exhibiting the first signs. Hippocampus impression smears are then made, stained with Seller's stain and examined for the presence of *Negri* bodies. The authorities concerned are then immediately informed of the result.

Specimens submitted for rabies diagnosis must be securely packed and clearly marked "SUSPECTED RABIES" on the specimen bottles and on the outside container. A covering letter should be included with the specimens, giving details of the name of the owner, human contacts, species affected, symptoms observed, history of previous vaccination, time of death and collection of specimens. If specimens are taken and despatched as requested, the diagnosis is greatly facilitated. On receipt of the specimens a histological diagnosis can be made in from 2 to 24 hours, while the biological confirmation will usually take from 7 to 16 days.

ACKNOWLEDGEMENTS

The author wishes to thank the Chief, Veterinary Research Institute, for permission to publish this article, Drs. Weiss and Howell who were responsible for the collection of the earlier data, and the technical staff who have assisted in the compilation of the figures on which the observations were based: also Mr. de Bruyn for the preparation of the photographs.

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RABIES IN SOUTH AFRICA

AN ANALYSIS OF HISTOLOGICAL EXAMINATIONS

R. C. TUSTIN, J. D. SMIT
Section of Pathology Veterinary Research Institute, Onderstepoort

Received for publication, July 1962

SUMMARY

1. The results of the examinations of histological sections for the diagnosis of rabies are analysed.

2. One thousand and seventy four of 2,455 suspect cases proved biologically and/or histologically positive.

3. The overall efficiency of the histological test was 66 per cent.

4. No statistical difference is shown between the histological diagnosis of animals that died, and those that were killed while suffering from rabies, when the hippocampus alone was examined.

5. The reliability of the histological test for different species of animals varied considerably.

6. In biologically positive cases 71 per cent and 46 per cent proved histologically positive where the hippocampus alone, and other parts of the brain were examined respectively.

7. Two staining methods are compared.

BRIEF HISTORICAL REVIEW

In 1903 *Negri*[1, 2] described inclusion bodies with characteristic properties in the neurons of the brain and ganglia of rabid animals. He found that while the inclusion bodies could be detected in sections of the brain of animals which had been killed in the early stages of the disease, the longer the duration of the disease, the more numerous and bigger they became. He considered that they could be demonstrated more constantly and in the greatest numbers in Ammon's horn; in 50 out of 52 cases of rabies in dogs he demonstrated inclusion bodies in Ammon's horn, and in another eight cases of rabies where other parts of the brain, but not Ammon's horn, were examined inclusion bodies could only be detected in three. These inclusion bodies, later designated "*Negri*" bodies are regarded as being specific for rabies.

Thomas *et al*[3] emphasise the importance of the histological examination of the *nucleus oculomotorius* of rabid rabbits. They were able to demonstrate *Negri* bodies in this site in almost every instance in which they were present in the hippocampus, and also in a considerable number of cases in which they could not be found in the latter. This finding was supported by Nicolau *et al*[4] who reported that *Negri* bodies are more numerous in the basal optic nucleus than in the hippocampus of rabid rabbits. Muratowa[5] found that the first appearance of *Negri* bodies in

the brain of mice is not in the horn of Ammon, but in the mesencephalon in the neighbourhood of the central canal, and stated that even when the disease is fully developed Negri bodies may be absent in the horn of Ammon and in the cerebellum, though present in other parts of the brain.

In 1937 Webster[6] showed that the intracerebral inoculation of Swiss white mice with suspected brain material is a much more reliable method of diagnosing rabies than the demonstration of *Negri* bodies in nervous tissue from the suspected animal. Of 200 specimens submitted, that were found to be negative for *Negri* bodies, 15 to 20 per cent proved to be positive for the mouse test. Leach[7] and Sellers[7] reported that approximately 11 per cent of specimens that were found to be negative for *Negri* bodies proved to be infected when tested in mice.

The effect of autolysis on *Negri* bodies was studied by Jonescu[8] who found that at 37°C they may maintain their characteristic appearance for ten days, whereas the nuclei and cytoplasm of the neurons had completely disappeared by then.

Hagan[9] stated that at the New York State Veterinary College when specimens, which on original examination had been diagnosed as negative for *Negri* bodies, but positive on animal inoculation, were re-examined, *Negri* bodies were found in practically all. Herzog[10] in 1945 reported that in almost 50 per cent of 52 cases of rabies he could not find *Negri* bodies in sections of the *Cornu Ammonis*. However, simultaneous histological examination of the *ganglion nodosum* from the same cases always disclosed the alterations that he associated with rabies, and which had been described by van Gehuchten and Nelis in 1900. He suggested that if the examination of the Ammon's horn and *ganglion nodosum* yields negative results, the lesions of a "rabies encephalitis" may be sought in the mesencephalon and medulla.

Lapi *et al*[11] found that histologic examination of the *gasserian ganglion* is a valuable adjunct in the diagnosis of rabies, and that the changes are sufficiently characteristic to permit a presumptive diagnosis of rabies when *Negri* bodies are not demonstrated. The value of this procedure is enhanced when traumatic destruction or putrefactive changes of the brain substance make examination for *Negri* bodies unsatisfactory or impossible.

According to Lepine[12], if smears or impressions of various parts of the brain are negative, a regular histopathological examination should be made of sections stained after embedding by a rapid method; samples from at least six sites should be examined:— Ammon's horn (both sides), the cerebral cortex of the motor area, the cerebellum, the medulla and a ganglion (gasserian ganglion or upper cervical ganglion). The *Negri* bodies are found to be mainly in the central pyramidal layer of Ammon's horn and of the hippocampus, and in the lower loop and in the middle layer of the neuroganglia of the Ammon's horn, and, less frequently, in the neurons of the cerebellum, of the motor areas, and of the medullar nuclei. They may be present in very large numbers in the ganglia but are generally small in size. The sections should be examined for two possible manifestations: signs of a meningo-encephalomyelitis (these are described) and for specific lesions (*Negri* bodies). Lepine considered that if the animal has died from rabies, it is normally easy to detect the specific

lesions; whereas if it has been killed, death might have occurred before the appearance of the specific lesions.

The importance of animal inoculation tests for the isolation of virus from suspected brain tissue in *Negri*-negative specimens cannot be overemphasised (W.H.O. Technical Report[13]). Exhaustive surveys of large numbers of routine specimens submitted for diagnosis have shown that 10 to 15 per cent of those cases proved positive by mouse inoculation, had been missed by direct microscopic examination for *Negri* bodies.

In 1960 McQueen[14] reported that the fluorescent rabies antibody test for the detection of antigen in brain tissue is as efficient as is mouse inoculation for the detection of virus. He found that impression smears of fresh or frozen material stained by Seller's method for the detection of *Negri* bodies, is 90·5 per cent efficient.

Recently, the Expert Committee on Rabies of the World Health Organisation [15] stated that microscopic examination of brain for *Negri* bodies, isolation of rabies virus from tissue specimens and, where necessary, the confimatory serum-virus neutralisation test are still the most important techniques in the laboratory diagnosis of rabies. However, new advances have been made in the application of other methods, examples of which are the fluorescent antibody and complement-fixation tests. The Committee stresses that the latter methods are valuable tools to be added to the rabies diagnostic armamentarium and are not meant, at this time, to supplant existing techniques.

The present analysis was undertaken to test the reliability of the histological techniques followed at Onderstepoort.

Material

The results of the histological examinations presented in this analysis are taken from the records of the Onderstepoort Veterinary Research Institute covering the ten year period, March 1952 to March 1962. This laboratory is responsible for the diagnosis of rabies in animals originating from the Republic of South Africa, and South West Africa.

Specimens are collected in the field by Veterinarians, Stock Inspectors and Medical Health Officers and are submitted for examination. The delay which occurs between the collection of the specimens and their receipt necessitates their preservation. The brain of suspected rabid animals is removed; a part is preserved in 50% aqueous glycerine solution for biological testing, and part in 10% formalin for histological examination. Emphasis is placed on the importance of the hippocampus for the latter examination.

The nomenclature of the wild animals proved a difficulty. In the majority of cases the common name was given. As the vernacular name of many animals varies from district to district, zoological identification was not attempted. The wild animals are grouped as follows:—

(*a*) *Meercats.*—In this group fall the various species described colloquially as meercats (meerkatte) or mongooses (muishonde), the majority of which belong to the family *Viverridae*. The yellow meercat (rooimeerkat) and suricate (stokstertmeerkat) for example are included, but the genets and the ground squirrels are, however, excluded.

(*b*) *Jackals.*—Four kinds of jackal or fox, *Canis mesomelas* (black-backed jackal, rooijakkals), *Otocyon megalotis* (bat-eared fox, bakoor-jakkals), *Vulpes* (*Cyalopex*) *chama* (silver fox, silwerjakkals) and *Canis adustus* (side-striped jackal, witkwasjakkals) occur in this country. The species representation in this analysis is unknown. The commonest jackal in South Africa is the black-backed jackal[18].

(*c*) *Genet cats.*—The genet cats (muskeljaatkatte) are tabulated seperately. They, too, belong to the family *Viverridae.*

(*d*) *Wild cats.*—Any animal described as a wild cat was included in this group. It is possible that a number of domestic cats which had reverted to the wild state, appear in the tables under this heading.

(*e*) *Miscellaneous.*—In this group are listed wild animals such as the honey badger (ratel), caracal (rooikat), ground squirrel (waaierstert-meerkat), kudu, duiker and rock rabbits (dassies — klipdas) as well as pigs, goats and man.

Routine Procedures

Elsewhere in this journal the methods for the biological examination of specimens which is performed by the intracerebral inoculation of brain suspensions in young mice, and the preparation and staining of histological sections are described in detail[16, 17]. Histological sections are cut from the hippocampus; should, however, the hippocampus not be present, other regions of the brain* are sectioned, the parts used being dictated by the nature of the material.

Before 1957 all sections for rabies diagnosis were stained by Mann's Methyl Blue — Eosin method and were checked against or confirmed by the examination of duplicate sections stained a Methyl Blue-Phloxin method. Subsequently an Acid Fuchsin—Methylene Blue staining method replaced the former methods, both for paraffin and for frozen sections. Prior to 1959 the brains of mice which died in the biological test were sectioned and examined. Subsequently brain impression smears stained by Seller's method have been employed instead.

An histologically positive diagnosis is made where there is no doubt but that *Negri* bodies are present. If intracytoplasmic inclusions, which could conceivably be *Negri* bodies, are observed, the diagnosis is recorded as histologically suspicious.

The histological examinations during the ten year period were performed by three veterinary pathologists.

Results

The results of the histological examination are summarised in Tables 1 to 10 and are followed by a statistical analysis.

* In this article wherever "brain" is mentioned it must be assumed that the hippocompus is excluded.

TABLE 1

Results of the examinations for rabies

March, 1952 to March, 1962

	Cattle	Dog	Meer-cat	Do-mestic cat	Jackal	Horse and donkey	Sheep	Genet cat	Wild cat	Mis-cella-neous	Total
Number of suspect specimens.....	604	1001	324	214	67	16	25	36	23	145	2455
Total number of positive; either biologically, histologically or both	352	374	184	52	33	4	10	20	17	28	1074
% Positive.....	58	37	57	24	49	25	40	56	74	19	44
Species % Positive.....	33	35	17	5	3	0·4	1	2	2	3	

TABLE 2

The manner in which the diagnosis was made

	Cattle	Dog	Meer-cat	Do-mestic cat	Jackal	Horse and donkey	Sheep	Genet cat	Wild cat	Mis-cella-neous	Total
1. Total number of positive.....	352	374	184	52	33	4	10	20	17	28	1074
2. Histologically positive; biologically positive.....	250	184	125	19	21	0	6	11	13	11	640
2. (*a*) As a % of total.....	71	49	68	36	64	0	60	55	76	39	60
3. Histologically suspicious; biologically positive.....	8	33	3	8	0	0	0	0	1	2	55
3. (*a*) As a % of total.....	2	9	2	15	0	0	0	0	6	7	5
4. Histologically negative; biologically positive.....	76	112	30	17	4	4	3	6	1	6	259
4. (*a*) As a % of total.....	22	30	16	33	12	100	30	30	6	21	24
5. Histologically positive; biologically negative.....	3	5	1	0	0	0	0	0	0	1	10
5. (*a*) As a % of total.....	1	1	—	0	0	0	0	0	0	3	1
6. No histological test; biologically positive.....	10	31	20	7	8	0	0	2	1	6	85
6. (*a*) As a % of total.....	3	8	11	13	24	0	0	10	6	21	8
7. Histologically positive; no biological test.....	5	9	5	1	0	0	1	1	1	2	25
7. (*a*) As a % of total.....	1	2	3	2	0	0	10	5	6	7	2
8. Histologically suspicious; biologically negative.....	0	11	0	11	0	0	0	0	0	6	28
9. Total of histologically suspicious [i.e. (3) + (8)].....	8	44	3	19	0	0	0	0	1	8	83
10. Histologically suspicious; biologically positive as a % of total number of histologically suspicious [i.e. (3) as a % of (9)]....	100	75	100	42	0	0	0	0	100	25	66

TABLE 3*
Positive cases where both histological and biological examinations were performed

	Cattle	Dog	Meer-cat	Do-mestic cat	Jackal	Horse and donkey	Sheep	Genet cat	Wild cat	Mis-cella-neous	Total
1. Total positive	337	334	159	44	25	4	9	17	15	20	964
2. Histologically positive; biologically positive	250	184	125	19	21	0	6	11	13	11	640
2. (*a*) As a % of total	74	55	79	43	84	0	67	65	87	55	66
3. Histologically suspicious; biologically positive	8	33	3	8	0	0	0	0	1	2	55
3. (*a*) As a % of total	2	10	2	18	0	0	0	0	7	10	6
4. Histologically negative; biologically positive	76	112	30	17	4	4	3	6	1	6	259
4. (*a*) As a % of total	23	34	19	39	16	100	33	35	7	30	27
5. Histologically positive; biologically negative	3	5	1	0	0	0	0	0	0	1	10
6. Total of histologically suspicious and positive [i.e. (2) + (3) + (5)]	261	222	129	27	21	0	6	11	14	14	705
6. (*a*) As a % of total	77	66	81	61	84	0	67	65	93	70	73
7. Histologically suspicious; biologically negative	0	11	0	11	0	0	0	0	0	6	28
8. Total of histologically suspicious [i.e. (3) + (7)]	8	44	3	19	0	0	0	0	1	8	83
9. Histologically suspicious; biologically positive as a % of total number of histologically suspicious [i.e. (3) as a % of (8)]	100	75	100	42	—	—	—	—	—	—	66

* Table 3 is the sum of the results appearing in tables (4) and (5), and of tables (6), (7) and (8).

TABLE 4*
Positive cases where both histological and biological examinations were performed; the hippocampus alone was examined histologically

	Cattle	Dog	Meer-cat	Do-mestic cat	Jackal	Horse and donkey	Sheep	Genet cat	Wild cat	Mis-cella-neous	Total
1. Total positive	249	298	126	35	24	2	8	12	13	15	782
2. Histologically positive; biologically positive	193	179	112	18	20	0	5	9	12	8	556
2. (*a*) As a % total	78	60	89	51	83	0	63	75	92	53	71
3. Histologically suspicious; biologically positive	5	24	0	6	0	0	0	0	0	1	36
3. (*a*) As a % of total	2	8	0	17	0	0	0	0	0	7	5
4. Histologically negative; biologically positive	49	90	13	11	4	2	3	3	1	5	181
4. (*a*) As a % of total	20	30	20	31	17	100	38	25	8	33	23
5. Histologically positive; biologically negative	2	5	1	0	0	0	0	0	0	1	9
6. Total of histologically suspicious and positive [i.e. (2) + (3) + (5)]	200	208	113	24	20	0	5	9	12	10	601
6. (*a*) As a % of total	80	70	90	69	83	0	63	75	92	67	77
7. Histologically suspicious; biologically negative	0	10	0	9	0	0	0	0	0	4	23
8. Total of histologically suspicious [i.e. (3) + (7)]	5	34	0	15	0	0	0	0	0	5	59
9. Histologically suspicious; biologically positive as a % of total number of histologically suspicious [i.e. (3) as a % of (8)]	100	71	0	40	0	0	0	0	0	20	61

* Table 4 is the sum of the results appearing in tables 4 (*a*), (*b*) and (*c*).

TABLE 4 (*a*)

Positive cases where both histological and biological examinations were performed; the hippocampus was examined histologically. The animal died naturally

	Cattle	Dog	Meer-cat	Do-mestic cat	Jackal	Horse and donkey	Sheep	Genet cat	Wild cat	Mis-cella-neous	Total
1. Total positive	50	38	7	4	0	1	2	0	0	2	104
2. Histologically positive; biologically positive	39	22	7	3	0	0	1	0	0	0	72
2. (*a*) As a % of total	78	58	100	75	0	0	50	0	0	0	69
3. Histologically suspicious; biologically positive	0	1	0	0	0	0	0	0	0	0	1
3. (*a*) As a % of total	0	3	0	0	0	0	0	0	0	0	1
4. Histologically negative; biologically positive	11	15	0	1	0	1	1	0	0	2	31
4. (*a*) As a % of total	22	39	0	25	0	100	50	0	0	100	30
5. Histologically positive; biologically negative	0	0	0	0	0	0	0	0	0	0	0
6. Total of histologically suspicious and positive [i.e. (2) + (3) + (5)]	39	23	7	3	0	0	1	0	0	0	73
6. (*a*) As a % of total	78	61	100	75	0	0	50	0	0	0	70
7. Histologically suspicious; biologically negative	0	2	0	2	0	0	0	0	0	0	4
8. Total of histologically suspicious [i.e. (3) + (7)]	0	3	0	2	0	0	0	0	0	0	5
9. Histologically suspicious; biologically positive as a % of total number of histologically suspicious [i.e. (3) as a % of (8)]		33		0							20

TABLE 4 (*b*)

Positive cases where both histological and biological examinations were performed; the hippocampus was examined histologically. The animal was killed while showing symptoms of rabies

	Cattle	Dog	Meer-cat	Do-mestic cat	Jackal	Horse and donkey	Sheep	Genet eat	Wild cat	Mis-cella-neous	Total
1. Total Positive	52	142	92	16	16	1	2	7	6	4	338
2. Histologically positive; biologically positive	45	86	80	6	12	0	2	6	6	2	245
2. (*a*) As a % of total	87	61	87	38	75	0	100	86	100	50	72
3. Histologically suspicious; biologically positive	0	12	0	4	0	0	0	0	0	1	17
3. (*a*) As a % of total	0	8	0	25	0	0	0	0	0	25	5
4. Histologically negative; biologically positive	7	40	12	6	4	1	0	1	0	1	72
4. (*a*) As a % of total	13	28	13	38	25	100	0	14	0	25	21
5. Histologically positive; biologically negative	0	4	0	0	0	0	0	0	0	0	4
6. Total of histologically suspicious and positive [i.e. (2) + (3) + (5)]	45	102	80	10	12	0	2	6	6	3	266
6. (*a*) As a % of total	87	72	87	63	75	0	100	86	100	75	79
7. Histologically suspicious; biologically negative	0	5	0	2	0	0	0	0	0	1	8
8. Total of histologically suspicious [i.e. (3) + (7)]	0	17	0	6	0	0	0	0	0	2	25
9. Histologically suspicious; biologically positive as a % of total number of histologically suspicious [i.e. (3) as a % of (8)]		71		67						50	68

TABLE 4 (*c*)

Positive cases where both histological and biological examinations were performed; the hippocampus was examined histologically. The manner of death of the animal is unknown

	Cattle	Dog	Meer-cat	Do-mestic cat	Jackal	Horse and donkey	Sheep	Genet cat	Wild cat	Mis-cella-neous	Total
1. Total positive	147	118	27	15	8	0	4	5	7	9	340
2. Histologically positive; biologically positive	109	71	25	9	8	0	2	3	6	6	239
2. (*a*) As a % of total	74	60	93	60	100		50	60	86	67	70
3. Histologically suspicious; biologically positive	5	11	0	2	0	0	0	0	0	0	18
3. (*a*) As a % of total	3	9	0	13	0		0	0	0	0	5
4. Histologically negative; biologically positive	31	35	1	4	0	0	2	2	1	2	78
4. (*a*) As a % of total	21	30	4	27	0		50	40	14	22	23
5. Histologically positive; biological negative	2	1	1	0	0	0	0	0	0	1	5
6. Total of histologically suspicious and positive [i.e. (2) + (3) + (5)]	116	83	26	11	8	0	2	3	6	7	262
6. (*a*) As a % of total	79	70	96	73	100	0	50	60	86	78	77
7. Histologically suspicious; biological negative	0	3	0	5	0	0	0	0	0	3	11
8. Total of histologically suspicious [i.e. (3) + (7)]	5	14	0	7	0	0	0	0	0	3	29
9. Histologically suspicious; biologically positive as a % of total number of histologically suspicious [i.e. (3) as a % of (8)]	100	79		29						0	62

TABLE 5*

Positive cases where both histological and biological examinations were performed; the "brain" was examined histologically

	Cattle	Dog	Meer-cat	Do-mestic cat	Jackal	Horse and donkey	Sheep	Genet cat	Wild cat	Mis-cella-neous	Total
1. Total positive	88	36	33	9	1	2	1	5	2	5	182
2. Histologically positive; biologically positive	57	5	13	1	1	0	1	2	1	3	84
2. (*a*) As a % of total	65	14	39	11							46
3. Histologically suspicious; biologically positive	3	9	3	2	0	0	0	0	1	1	19
3. (*a*) As a % of total	3	25	9	22							10
4. Histologically negative; biologically positive	27	22	17	6	0	2	0	3	0	1	78
4. (*a*) As a % of total	31	61	52	67							43
5. Histologically positive; biologically negative	1	0	0	0	0	0	0	0	0	0	1
6. Total of histologically suspicious and positive [i.e. (2) + (3) + (5)]	61	14	16	3	1	0	1	2	2	4	104
6. (*a*) As a % of total	69	39	48	33							57
7. Histologically suspicious; biologically negative	0	1	0	2	0	0	0	0	0	2	5
8. Total of histologically suspicious [i.e. (3) + (7)]	3	10	3	4	0	0	0	0	1	3	24
9. Histologically suspicious; biologically positive as a % of total number of histologically suspicious [i.e. (3) as a % of (8)]	100	90	100	50							79

* Table 5 is the sum of the results appearing in tables 5 (*a*), (*b*) and (*c*).

TABLE 5 (*a*)

Positive cases where both histological and biological examinations were performed; the "brain" was examined histologically. The animal died naturally

	Cattle	Dog	Meer-cat	Do-mestic cat	Jackal	Horse and donkey	Sheep	Genet cat	Wild cat	Mis-cella-neous	Total
1. Total positive	24	5	1	2	0	0	1	0	0	1	34
2. Histologically positive; biologically positive	19	0	1	1	0	0	1	0	0	0	22
2. (*a*) As a % of total	79										65
3. Histologically suspicious; biologically positive	0	3	0	0	0	0	0	0	0	1	4
3. (*a*) As a % of total	0										12
4. Histologically negative; biologically positive	5	2	0	1	0	0	0	0	0	0	8
4. (*a*) As a % of total	21										24
5. Histologically positive; biologically negative	0	0	0	0	0	0	0	0	0	0	0
6. Total of histologically suspicious and positive i.e. [(2) + (3) + (5)]	19	3	1	1	0	0	1	0	0	1	26
6. (*a*) As a % of total	79										76
7. Histologically suspicious; biologically negative	0	0	0	1	0	0	0	0	0	0	1
8. Total of histologically suspicious [i.e. (3) + (7)]	0	3	0	1	0	0	0	0	0	1	5
9. Histologically suspicious; biologically positive as a % of total number of histologically suspicious [i.e. (3) as a % of (8)]											80

TABLE 5 (*b*)

Positive cases where both histological and biological examinations were performed; the "brain" was examined histologically. The animal was killed while showing symptoms of rabies

	Cattle	Dog	Meer-cat	Do-mestic cat	Jackal	Horse and donkey	Sheep	Genet cat	Wild cat	Mis-cella-neous	Total
1. Total positive	13	18	24	3	1	1	0	2	2	1	65
2. Histologically positive; biologically positive	8	2	10	0	1	0	0	1	1	1	24
2. (*a*) As a % of total											37
3. Histologically suspicious; biologically positive	0	2	1	1	0	0	0	0	1	0	5
3. (*a*) As a % of total											8
4. Histologically negative; biologically positive	5	14	13	2	0	1	0	1	0	0	36
4. (*a*) As a % of total											55
5. Histologically positive; biologically negative	0	0	0	0	0	0	0	0	0	0	0
6. Total of histologically suspicious and positive [i.e. (2) + (3) + (5)]	8	4	11	1	1	0	0	1	2	1	29
6. (*a*) As a % of total											45
7. Histologically suspicious; biologically negative	0	0	0	1	0	0	0	0	0	1	2
8. Total of histologically suspicious [i.e. (3) + (7)]	0	2	1	2	0	0	0	0	1	1	7
9. Histologically suspicious; biologically positive as a % of total number of histologically suspicious [i.e. (3) as a % of (8)]											

TABLE 5 (*c*)

Positive cases where both histological and biological examinations were performed; the "brain" was examined histologically. The manner of death of the animal is unknown

	Cattle	Dog	Meer-cat	Do-mestic cat	Jackal	Horse and donkey	Sheep	Genet cat	Wild cat	Mis-cella-neous	Total
1. Total positive	51	13	8	4	0	1	0	3	0	3	83
2. Histologically positive; biologically positive	30	3	2	0	0	0	0	1	0	2	38
2. (*a*) As a % of total	59	23	25	0							46
3. Histologically suspicious; biologically positive	3	4	2	1	0	0	0	0	0	0	10
3. (*a*) As a % of total	6	31	25	25							12
4. Histologically negative; biologically positive	17	6	4	3	0	1	0	2	0	1	34
4. (*a*) As a % of total	33	46	50	75							41
5. Histologically positive; biologically negative	1	0	0	0	0	0	0	0	0	0	1
6. Total of histologically suspicious and positive [i.e. (2) + (3) + (5)]	34	7	4	1	0	0	0	1	0	2	49
6. (*a*) As a % of total	67	53	50	25							59
7. Histologically suspicious; biologically negative	0	1	0	0	0	0	0	0	0	1	2
8. Total of histologically suspicious [i.e. (3) + (7)]	3	5	2	1	0	0	0	0	0	1	12
9. Histologically suspicious; biologically positive as a % of total number of histologically suspicious [i.e. (3) as a % of (8)]											83

TABLE 6*

Total number of positive cases where the animal died naturally

	Cattle	Dog	Meer-cat	Do-mestic cat	Jackal	Horse and donkey	Sheep	Genet cat	Wild cat	Mis-cella-neous	Total
1. Total positive	74	43	8	6	0	1	3	0	0	3	138
2. Histologically positive; biologically positive	58	22	8	4	0	0	2	0	0	0	94
2. (*a*) As a % of total	78	51	100								68
3. Histologically suspicious; biologically positive	0	4	0	0	0	0	0	0	0	1	5
3. (*a*) As a % of total	0	9	0								4
4. Histologically negative; biologically positive	16	17	0	2	0	1	1	0	0	2	39
4. (*a*) As a % of total	22	40	0								28
5. Histologically positive; biologically negative	0	0	0	0	0	0	0	0	0	0	0
6. Total of histologically suspicious and positive [i.e. (2) + (3) + (5)]	58	26	8	4	0	0	2	0	0	1	99
6. (*a*) As a % of total	78	60	100								72
7. Histologically suspicious; biologically negative	0	2	0	3	0	0	0	0	0	0	5
8. Total of histologically suspicious [i.e. (3) + (7)]	0	6	0	3	0	0	0	0	0	1	10
9. Histologically suspicious; biologically positive as a % of total number of histologically suspicious [i.e. (3) as a % of (8)]											50

*Table 6 is the sum of the results appearing in tables 4 (*a*) and 5 (*a*).

TABLE 7*

Total number of positive cases where the animal was killed while showing symptoms of rabies

	Cattle	Dog	Meer-cat	Do-mestic cat	Jackal	Horse and donkey	Sheep	Genet cat	Wild cat	Mis-cella-neous	Total
1. Total positive	65	160	116	19	17	2	2	9	8	5	403
2. Histologically positive; biologically positive	53	88	90	6	13	0	2	7	7	3	269
2. (*a*) As a % of total	82	55	78	32	76						67
3. Histologically suspicious; biologically positive	0	14	1	5	0	0	0	0	1	1	22
3. (*a*) As a % of total	0	9	1	26	0						5
4. Histologically negative; biologically positive	12	54	25	8	4	2	0	2	0	1	108
4. (*a*) As a % of total	18	34	22	42	24	100					27
5. Histologically positive; biologically negative	0	4	0	0	0	0	0	0	0	0	4
6. Total of histologically suspicious and positive [i.e. (2) + (3) + (5)]	53	106	91	11	13	0	2	7	8	4	295
6. (*a*) As a % of total	82	66	78	58	76						73
7. Histologically suspicious; biologically negative	0	5	0	3	0	0	0	0	0	2	10
8. Total of histologically suspicious [i.e. (3) + (7)]	0	19	1	8	0	0	0	0	1	3	32
9. Histologically suspicious; biologically positive as a % of total number of histologically suspicious [i.e. (3) as a % of (8)]	0	74	100	63	0						69

*Table 7 is the sum of the results appearing in tables 4 (*b*) and 5 (*b*).

TABLE 8*

Total number of positive cases where the manner of death of the animal is unknown

	Cattle	Dog	Meer-cat	Do-mestic cat	Jackal	Horse and donkey	Sheep	Genet cat	Wild cat	Mis-cella-neous	Total
1. Total positive	198	131	35	19	8	1	4	8	7	12	423
2. Histologically positive; biologically positive	139	74	27	9	8	0	2	4	6	8	277
2. (*a*) As a % of total	70	56	77	47	100	0	50	50	86	67	65
3. Histologically suspicious; biologically positive	8	15	2	3	0	0	0	0	0	0	28
3. (*a*) As a % of total	4	11	6	16	0	0	0	0	0	0	7
4. Histologically negative; biologically positive	48	41	5	7	0	1	2	4	1	3	112
4. (*a*) As a % of total	24	31	14	37	0	100	50	50	14	25	26
5. Histologically positive; biologically negative	3	1	1	0	0	0	0	0	0	1	6
6. Total of histologically suspicious and positive [i.e. (2) + (3) + (5)]	150	90	30	12	8	0	2	4	6	9	311
6. (*a*) As a % of total	76	69	86	63	100	0	50	50	86	75	74
7. Histologically suspicious; biologically negative	0	4	0	5	0	0	0	0	0	4	13
8. Total of histologically suspicious [i.e. (3) + (7)]	8	19	2	8	0	0	0	0	0	4	41
9. Histologically suspicious; biologically positive as a % of total number of histologically suspicious [i.e. (3) as a % of (8)]	100	79	100	38	—	—	—	—	—	0	68

*Table 8 is the sum of the results appearing in tables 4 (*c*) and 5 (*c*).

TABLE 9*

A comparison of two staining techniques, Mann's Methyl Blue — Eosin (checked against a Methyl Blue — Phloxin Method) with Acid Fuchsin — Methylene Blue (Afmb.)

	Stain	Cattle	Dog	Meer-cat	Do-mestic cat	Jackal	Horse and donkey	Sheep	Genet cat	Wild cat	Mis-cella-neous	Total
1. Total positive.....	Mann	146	124	53	13	14	2	5	6	4	5	372
	Afmb	191	210	106	31	11	2	4	11	11	15	592
	Total	337	334	159	44	25	4	9	17	15	20	964
2. Histologically positive biologically positive	Mann	107	63	37	5	11	0	3	2	4	4	236
	Afmb	143	121	88	14	10	0	3	9	9	7	404
	Total	250	184	125	19	21	0	6	11	13	11	640
2. (*a*) As a % of total....	Mann	73	51	70	38	79	0	60	33	100	80	63
	Afmb	75	58	83	45	91	0	75	82	82	47	68
	Total	74	55	79	43	84	0	67	65	87	55	66
3. Histologically suspicious biologically positive	Mann	2	9	0	3	0	0	0	0	0	0	14
	Afmb	6	24	3	5	0	0	0	0	1	2	41
	Total	8	33	3	8	0	0	0	0	1	2	55
3. (*a*) As a % of total....	Mann	1	7	0	23	0	0	0	0	0	0	4
	Afmb	3	11	3	16	0	0	0	0	9	13	7
	Total	2	10	2	18	0	0	0	0	7	10	6
4. Histologically negative biologically positive	Mann	37	51	16	5	3	2	2	4	0	1	121
	Afmb	39	61	14	12	1	2	1	2	1	5	138
	Total	76	112	30	17	4	4	3	6	1	6	259
4. (*a*) As a % of total....	Mann	25	41	30	38	21	100	40	67	0	20	33
	Afmb	20	29	13	39	9	100	25	18	9	33	23
	Total	23	34	19	39	16	100	33	35	7	30	27
5. Histologically positive biologically negative	Mann	0	1	0	0	0	0	0	0	0	0	1
	Afmb	3	4	1	0	0	0	0	0	0	1	9
	Total	3	5	1	0	0	0	0	0	0	1	10
6. Total of histologically suspicious and positive [i.e. (2) + (3) + (5)]	Mann	109	73	37	8	11	0	3	2	4	4	251
	Afmb	152	149	92	19	10	0	3	9	10	10	454
	Total	261	222	129	27	21	0	6	11	14	14	705
6. (*a*) As a % of total....	Mann	75	59	70	62	79	0	60	33	100	80	67
	Afmb	80	71	87	61	91	0	75	82	91	67	77
	Total	77	66	81	61	84	0	67	65	93	70	73
7. Histologically suspicious biologically negative	Mann	0	1	0	0	0	0	0	0	0	0	1
	Afmb	0	10	0	11	0	0	0	0	0	6	27
	Total	0	11	0	11	0	0	0	0	0	6	28
8. Total of histologically suspicious [i.e. (3) + (7)]	Mann	2	10	0	3	0	0	0	0	0	0	15
	Afmb	6	34	3	16	0	0	0	0	1	8	68
	Total	8	44	3	19	0	0	0	0	1	8	83
9. Histologically suspicious biologically positive as a % of total number of histol. suspicious [i.e. (3) as a % of (8)]	Mann	100	90	0	100	—	—	—	—	—	—	93
	Afmb	100	71	100	31	—	—	—	—	—	25	60
	Total	100	75	100	42	—	—	—	—	—	25	66

* Where Mann's method is mentioned in the table, the results of sections stained both by Mann's and Methyl Blue — Phloxin methods are included (see text).
Table 9 represents positive cases where both histological and biological examinations were performed (table 3).

TABLE 10

Miscellaneous Animals*

Species	Number of Cases
Honeybadger	5
Duiker	3
Ground squirrel	4
Caracal	1
Rock rabbit	1
Kudu	1
Pig (domestic)	5
Goat	4
Man	2
Unknown	2
TOTAL	28

* The results of the histological examinations of these animals are included in the preceding tables under the heading "miscellaneous".

STATISTICAL ANALYSIS OF THE RESULTS

The statistical analysis is based on biologically positive cases.

(1) COMPARISON OF THE RESULTS OBTAINED WHERE THE HIPPOCAMPUS AND "BRAIN" WERE EXAMINED. (Tables 4 and 5 compared).

(i) *Histologically positive cases.*

	"Brain"	Hippocampus	Total
Histol. positive	84	556	640
others	98	226	324
TOTAL	182	782	964

$\chi^2 = 41{\cdot}18^{**}$

This value of χ^2 with df = 1 is highly significant at both 5% and 1% levels.

(ii) *Histologically suspicious cases.*

	"Brain"	Hippocampus	Total
Histol. suspicious	19	36	55
others	163	746	909
TOTAL	182	782	964

$\chi^2 = 9{\cdot}347^{**}$

This value of χ^2 with df = 1 is highly significant at both 5% and 1% levels.

(2) COMPARISON OF THE RESULTS OBTAINED BETWEEN ANIMALS THAT DIED AND THOSE THAT WERE KILLED.

(*a*) EXAMINATION OF THE HIPPOCAMPUS (TABLES 4 (*a*) AND 4 (*b*), COMPARED).

(i) *Histologically positive cases.*

	Died	Killed	Total
Histol. positive	72	245	317
others	32	93	125
TOTAL	104	338	442

$\chi^2 = 0{\cdot}416$

This value of χ^2 with df = 1 is not significant.

(ii) *Histologically suspicious cases.*

	Died	Killed	Total
Histol. suspicious	1	17	18
others	103	321	424
TOTAL	104	338	442

$\chi^2 = 3{\cdot}368$

This value of χ^2 with df = 1 is not significant.

(*b*) EXAMINATION OF THE "BRAIN". (TABLES 5 (*a*) AND 5 (*b*) COMPARED).

(i) *Histologically positive cases.*

	Died	Killed	Total
Histol. positive	22	24	46
Others	12	41	53
TOTAL	34	65	99

$\chi^2 = 6{\cdot}927^{**}$

This value of χ^2 with df = 1 is highly significant at both 5% and 1% levels.

(ii) *Histologically suspicious cases.*

	Died	Killed	Total
Histol. suspicious	4	5	9
Others	30	60	90
TOTAL	34	65	99

$\chi^2 = 0{\cdot}448$.

This value of χ^2 with df = 1 is not significant.

(*c*) TOTALS OF THE RESULTS OBTAINED FOR EXAMINATIONS OF THE HIPPOCAMPUS AND "BRAIN". (TABLES 6 AND 7 COMPARED).

(i) *Histologically positive cases.*

	Died	Killed	Total
Histol. positive	94	269	363
Others	44	134	178
TOTAL	138	403	541

$\chi^2 = 0{\cdot}086$.

This value for χ^2 with df = 1 is not significant.

(ii) *Histologically suspicious cases.*

	Died	Killed	Total
Histol. suspicious	5	22	27
Others	133	381	514
TOTAL	138	403	541

$\chi^2 = 0{\cdot}730$.

This value of χ^2 with df = 1 is not significant.

(3) COMPARISON OF THE TWO STAINING METHODS (TABLE 9).

(i) *Histologically positive cases.*

	Mann	Afmb	Total
Histol. positive	236	404	640
Others	136	188	324
TOTAL	372	592	964

$\chi^2 = 2{\cdot}361$.

This value of χ^2 with df = 1, is not significant.

(ii) *Histologically suspicious cases.*

	Mann	Afmb	Total
Histol. suspicious.....	14	41	55
Others.....	358	551	909
TOTAL.....	372	592	964

$\chi^2 = 4{\cdot}246$*.

This value of χ^2 with df = 1 is significant at the 5% level.

DISCUSSION

In the examinations at Onderstepoort the histological diagnosis is based entirely on the demonstration of *Negri* bodies in the brain; special emphasis being placed on their presence in the hippocampus. The present analysis shows the overall efficiency of the *Negri* body test for histological sections to be 66 per cent when the mouse test is positive. This figure compares unfavourably with those given by the Expert Committee on Rabies (W.H.O.) who reported that 10 to 15 per cent of cases that proved to be positive by mouse inoculation were negative by direct microscopic examination for *Negri* bodies[13].

The method of preservation of specimens precludes the making of impression smears upon arrival at the laboratory. In an attempt to improve the efficiency of the histological diagnosis, the making of impression smears in the field was instigated at one stage, but has fallen into disuse due to differences in techniques used in making the smears, and to the practical difficulties encountered in staining them[19]. Increased efficiency can, therefore, only be brought about by following the method for the examination of sections made from six separate sites of the brain as described by Lepine[12].

In this country the majority of animals suspected of being rabid are killed immediately, and the material submitted probably represents every stage in the symptomatology of the disease. Despite this, no statistical difference is shown between the histological diagnosis of those that died and those that were killed, with the exception of cases where the "brain" was examined. This is in direct contrast to the views generally held in this country and those expressed by Lepine[12] who considers that premature killing reduces the accuracy of the histological test.

The efficiency of the histological test for different species of animals varied considerably. Forty-three per cent of 44 cases of rabies in domestic cats was histologically positive; the highest percentage of histologically positive cases was for wild cats where a figure of 87 per cent was obtained. The figures for other species fall between these two extremes.

In biologically positive cases where the hippocampus alone was examined 71 per cent proved histologically positive and 5 per cent suspicious. The differences between these results and those where other parts of the "brain" were examined which gave percentages of 46 and 10 respectively, are highly significant.

The comparison of the staining techniques shows no difference in the histologically positive cases. A significant difference, however, was

revealed in the suspicious cases; the explanation for this is still unknown. The Acid Fuchsin — Methylene Blue method is considered superior to Mann's Methyl Blue — Eosin (checked against the Methyl Blue — Phloxin method) due to its simplicity and constancy.

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RABIES IN SOUTH AFRICA

FIELD CONTROL OF THE DISEASE

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SUMMARY

The rabies control methods employed in the Republic of South Africa are described. Mention is made of the relevant legislation and its practical application in dealing with both the localised and widely disseminated forms of the disease. Import requirements are also given.

INTRODUCTION

Two distinct epizootiological forms of rabies occur in the Republic of South Africa. On the central plateau the disease has established itself in a localised enzootic form which is kept going by small carnivora like the yellow mungoose, spotted genet, suricate and some of the wild cats. Domestic animals and human beings become involved sporadically without it ever assuming epizootic proportions in dogs. The first definite diagnosis in this area was made in 1928 in a yellow mungoose, although its existence had been suspected for some time. During 1950, however, a more easily disseminated form made its appearance in the Northern border districts of the Transvaal. The principal vectors proved to be dogs and jackal which spread the disease fairly rapidly through the more densely dog-populated areas of the Northern and Eastern Transvaal. It has now reached Natal.

The control measures are essentially the same for both forms and differ only in degree of application as will be described later. Basically reliance is placed on physical control of dogs and cats, supported by canine vaccination and control of susceptible wildlife populations where necessary. The successful application of these measures has been found to be dependant on the full co-operation of the general public and interested organisations such as other Government Departments, Local Authorities, Farmer Associations, and the indigenous Bantu population. Very wide publicity must be given to ensure early reporting of outbreaks and compliance with the control measures. The latter must naturally be backed by adequate legal provisions to ensure compliance with the law.

REGULATIONS APPLICABLE WITHIN THE REPUBLIC OF SOUTH AFRICA

The essential legal requirements of the Animal Diseases and Parasites Act, 1956 (Act No. 13 of 1956) are wellknown but it may not be out of place to give a brief resumé of the more important provisions applicable to rabies.

The Act provides that no person may move or permit to be moved any infected or suspected-to-be-infected animal except under authority of a permit. The same applies to movements of susceptible animals from, into or through any property where there is such an animal, and no person may impound an infected animal in a public pound. The owner must also see to it that the infected or suspected animal does not stray.

The Standing Regulations place the onus of reporting the disease on the owner or occupier of land, with an additional provision that the latter must notify his neighbours. At the same time the owner is expected to isolate the animals without delay, and prevent access thereto by unauthorised persons. Such animals may only be released or removed on the authority of a written permit issued by a State Veterinarian and it is the duty of the occupier or owner of the land to disinfect the place of confinement and other articles or things that have been in contact with the animal or animals, to the satisfaction of the State Veterinarian.

No person other than an officer or a veterinarian in the employ of a local authority may incise or open carcases of animals which have died or are suspected to have died from rabies. A written permit is required for the removal of the carcases or organs of such animals or those destroyed to prevent the spread of the disease. The disposal of carcases is the duty of the owner and it has to be done in a prescribed manner i.e. by burning or incineration or, if burning or incineration is not practicable, by burial (in quicklime if possible), at a depth of not less than six feet. Any residues of buried or incinerated carcases must be buried at a depth of not less than four feet. Finally no person other than an Officer may uncover, dig up or remove the carcases or any part of them from the place of burial.

Any person who has been in contact with an infected or suspected animal is required to disinfect his person and apparel as well as other articles which may have been in contact. Another human protection is that no person shall use or permit to be used, the milk of any animal infected or suspected to be infected with rabies.

The Chief of the Veterinary Field Services may, subject to the Minister's approval, order the destruction of an infected or suspected animal and it has to be carried out by or under the supervision of an Officer or any Police Officer. A State Veterinarian, may order the owner of an infected or suspected animal, or an animal which has been in contact or could possibly have been in contact with an infected or suspected animal, to confine isolate, secure, or muzzle, any animal in order to diagnose or to prevent the possible spread of the disease. He may do the same in any area where the movement of a particular species has been prohibited or restricted, and nobody may release such animals without his written permission, unless it was made applicable for a specified period.

Every owner having animals in an area where the movement of such animals is restricted or prohibited must maintain a register of such animals in a prescribed way to facilitate proper control on movements.

More specific regulations, in regard to infected or suspected animals, appear in Part XI of the Standing Regulations.

Nobody, other than a veterinarian or registered medical practitioner

may treat such animals, and it is the duty of the owner either to isolate and securely confine it to prevent it attacking human beings or other animals, or immediately to destroy it. When rabies has been definitely diagnosed he has no choice and must immediately destroy it. Carcases must, however, be isolated and protected from the weather until disposal instructions are obtained from an Officer.

Animals which have been in contact with an infected animal shall be destroyed by the owner unless the State Veterinarian is satisfied that effective isolation and confinement is practicable, and he gives the necessary authority. When an infected or suspected-to-be-infected dog has been at large and in the opinion of the State Veterinarian, could have been a source of infection to other animals, he may place a notice in a newspaper circulating in the area or by written notice, order the owners of all animals in a specified area to keep their animals securely confined and isolated for a fixed period. If any animal ordered to be confined, or any infected or suspected animal, is found at large by the occupier of land, it is his duty to seize it and place it in isolation and confinement, and report the matter without delay, to the nearest veterinarian or officer of the South African Police.

The manner in which an infected or suspected animal may be moved under permit, as required by the Act, is prescribed, i.e. only in a box or other container constructed and secured to the satisfaction of a State Veterinarian.

The Practical Application of Control Measures

The control measures recommended by the Expert Committee on Rabies of the World Health Organisation ,form the basis of the regulations applied in South Africa. They are:—

"(1) Registration, licencing and taxation of dogs.
(2) Elimination of stray animals.
(3) Restraint of dogs while the control campaign is under way.
(4) Mass vaccination of dogs free of charge.
(5) Provision of adequate facilities for diagnosis.
(6) Reduction in number of wildlife species where these are a reservoir of disease.
(7) A continual and energetic publicity campaign."

Localised outbreaks in the enzootic areas, where *Viverridae* are the principal carriers, are dealt with individually by the application of quarantine restrictions and the vaccination free of charge of all dogs within a radius of 15 miles. Egg-adapted live Flury-strain virus vaccine is used. When necessary the affected wildlife pupulation, which lives in innumerable burrows, is reduced by a standardised practice of gassing and trapping. These methods have proved successful without having to declare the specific localities as infected in the *Government Gazette.*

All regions infected by the form of the disease resembling epizootiologically the "Street Virus" type, have been gazetted as infected to allow of the control of movements, and ensure the regular vaccination of all dogs.

The areas involved consist of the Northern and Eastern districts of

Transvaal, the whole of the Natal province and the three Northern districts of the Eastern Cape Province.

All movements of dogs and cats are subject to permit. The movement of cats is discouraged. Dogs are allowed to be moved within the areas provided they have been vaccinated and are well-removed from any active infection. Introductions from non-infected parts of the country are permitted subject to the dogs having been vaccinated at least 30 days prior to entry, and cats on condition that they are permanently transferred to destination. Removals out of the declared areas are more difficult. From districts not yet infected dogs may be taken out under permit, 30 days after vaccination, but from areas considered more dangerous they are allowed out 180 days after vaccination or subject to detention in isolation and quarantine in the same way as imported dogs from infected countries. Puppies under 3 months of age are allowed out only from approved kennels. In the non-danger areas the puppies may be moved if the State Veterinarian is satisfied that they could not possibly have made contact with other carnivorous animals, but in the danger areas they have to be vaccinated when at least 3 months of age and are permitted to be moved a month later. Cats are not allowed out of any of the rabies-declared areas.

Compulsory free vaccination of all dogs over the age of 3 months is enforced in the gazetted areas. A tattoomark signifying the year of vaccination is placed in each animal's ear for future identification.

Outbreaks of the disease are dealt with in the same way as is done in the enzootic areas i.e. in accordance with the Standing Regulations. Apart from confining orders, a check-up is made of all dogs within a radius of ten to fifteen miles and revaccinations carried out where necessary. Where jackal are involved a controlled poison-bait method of destruction is employed to decrease their numbers as far as possible.

Discussion of Internal Control Measures

In South Africa the licencing of dogs is enforced by the Provincial Administrations. A certain number of unlicenced and stray animals are eliminated in the process, and generally it has a limiting effect on the number of dogs. Through circumstances beyond the control of the officials, there are, however, areas with a fair number of unlicenced dogs e.g. the more remote Bantu Reserves.

Prior to the application of mass vaccination in the Northern Transvaal in 1952, more reliance had to be placed on physical control and decreasing the density of the dog population, to stem the tide of infection. As Provincial Officials had powers to destroy unlicenced dogs, they rendered valuable assistance in this respect. After explaining the object of the measure to both the European and Bantu populations, hunts of unlicenced dogs were organised, and over 20,000 dogs destroyed which had a marked effect on controlling the infection.

Once vaccination had started this policy had to be reversed. Owners had to be encouraged to produce all dogs for vaccination, whether licenced or not, and any destruction of unlicenced but vaccinated dogs was liable to shake the confidence of dog-owners, particularly the Bantu.

Provincial Officials who had co-operated so well previously were no longer welcome at vaccination centres. A regrettable but unavoidable development, but the success of a vaccination campaign depends to a large extent on the co-operation of dog-owners.

Confinement of dogs in infected areas is fraught with many difficulties. In urban areas Local Authorities find it difficult to deal with stray dogs, because of lack of catching and impounding facilities and prosecution of negligent owners is often hampered by the necessary proof of ownership. In rural areas, particularly Bantu Reserves, it is even less successful unless frequent controlling inspections can be carried out. Owners are therefore, generally advised to destroy all unwanted dogs voluntarily and keep the others under close control. Official records are maintained of all dogs to keep a check on illegal movements and to ensure the early reporting or discovery of possible infection.

Vaccination of dogs is compulsory and free of charge in the proclaimed districts or when employed to combat outbreaks in other areas. Internationally recognised certificates are issued and the animals tattooed in the ears to indicate the year of vaccination. In the more developed parts of the country, private veterinary practitioners render valuable assistance within their practices. This is especially the case in Natal.

Experience in South Africa, and elsewhere, has shown that the success of canine vaccination is dependant on a number of factors: one of the most important being the co-operation of the owners, who unless properly informed, are sometimes completely ignorant of the implications of the disease. The assistance of all interested bodies has to be obtained and the widest publicity given by making use of radio talks, local newspapers, pamphlets, announcements in cinemas, addressing of meetings on the subject and catching notices displayed prominently in public places.

Whenever possible detail records of all dogs in the areas have to be kept for comparison with those inoculated. Vaccination centres have to be planned and organised in such a way that it is convenient for all owners and that nobody is unnecessarily delayed. The handling of the dogs must also inspire confidence. Last but not least is the proper care of the vaccine and its efficient application.

In the Eastern Transvaal 181,414 dogs have already been vaccinated while the total for Natal at the end of April, 1962, stood at 211,206, i.e. a grand total of 392,620 in the proclaimed rabies districts. Of these there are only 13 recorded cases of vaccinated dogs which have subsequently developed the disease: a negligable figure when the conditions under which the vaccine is sometimes used, is taken into consideration.

Early reporting and rapid diagnosis are, apart from the methods already described, essential pre-requisites to the eradication of rabies. The State Veterinary Services of the country is suitably organised for this purpose, and co-operates very closely with the Department of Health in this respect. All reported cases are immediately investigated, brain impression smears examined locally and specimens of brain and parotid salivary glands submitted to the Onderstepoort Research Institute for confirmation. Full details are submitted and whenever there are any human contacts the names and addresses of the persons involved are supplied to the Public Health Department.

As already mentioned wildlife plays an important part in the dissemination of the disease in South Africa, and control measures have been evolved for the purpose. Viverridae are dealt with by systematic gassing in their burrows to eliminate infected populations. The numbers of jackal and other carnivora in the bushveld areas are decreased by the controlled use of poison-bait. In spite of contrary experience elsewhere in the country it has proved efficient for the purpose e.g. during a trial period of 2 years some 21,020 poisoned baits were placed over a drag distance of 2,067 miles on 305 separate farms: 7,917 of these baits being picked up by jackal, 978 by other animals, and 515 dead jackal being found within a short distance of the baits. It was estimated that over 3,000 jackal were possibly destroyed.

It would appear that certain optimum population densities of wildlife are necessary for the existence of rabies, and when sufficiently decreased the disease disappears in spite of new populations migrating into the treated areas.

Import Control

The necessary provision has been made in the Animal Diseases and Parasites Act of 1956 (Act No. 13 of 1956) to safeguard the country against the importation of rabies. The Act requires that imported animals be accompanied by permits issued by the Chief of the Veterinary Field Services, and that they may not be introduced otherwise than in accordance with conditions stipulated in the permits.

The restrictions and permit requirements imposed are directly related to the rabies position in the country from which the animals originate. In all instances a certificate of health, issued by an authorised veterinarian of the exporting country is required, to which may be added a statement as to the country's freedom from rabies for 12 months.

Importations from most overseas countries and countries and territories on the African continent, where rabies occurs, are subject, on arrival in South Africa, to six months quarantine and vaccination at the owner's risk and expense. If owners desire to avoid the quarantine period, the animals have to be accompanied by certificates from veterinarians, approved by the Veterinary Departments of the countries of origin, to the effect that the animals were vaccinated with the Flury strain of avianised anti-rabies vaccine, potency tested and fully viable at time of injection, at least six months but not longer than three years, prior to introduction into South Africa. Certain other anti-rabies vaccines like Kelev, Vom, Pitman Moore and Pasteur are also recognised but where necessary the condition is added that the animals be revaccinated on arrival. If animals have been vaccinated with an approved vaccine within a period of six months, the remaining time will constitute the period of quarantine upon entry into the Republic. No dispensation is given in the case of dogs vaccinated before reaching the age of three months.

Carnivora and other animals capable of introducing the disease are treated in the same way as unvaccinated dogs. Generally speaking their importation is not allowed from infected countries.

The official quarantine stations are situated at CAPE TOWN,

DURBAN and JAN SMUTS AIRPORT. Owners have to bear the cost of feeding, care of the animals and any necessary veterinary attention, for which they must arrange through agents. Although feeding and care are not undertaken officially, owners are at liberty to make such arrangements with the superintendents of the stations. The charges usually vary from 25 cents to 40 cents per animal per day depending on the diet prescribed and the size of the animal.

Dogs and cats may be imported without quarantine from certain wellknown rabies-free countries provided the owner or exporter submits a sworn declaration that the animals had not been outside the country concerned during the immediately preceding six months or since birth. In addition the Commander of the carrying ship or aircraft must certify that the animals did not contact other animals and were not taken ashore at any port, en route to South Africa.

Movements from exposed, but not enzootically infected countries, as well as rabies-free parts of certain African Territories, are allowed quarantine-free introductions provided the animals have been vaccinated with recognised anti-rabies vaccines at least 30 days but not longer than three years prior to arrival in South Africa. If not vaccinated they are promptly vaccinated on entry and detained in quarantine at one of the stations mentioned for 30 days at the owner's risk and expense.

Vaccination of cats is recognised for import purposes from certain relatively rabies-free overseas countries and quarantine restrictions waived, but the maximum interval after vaccination is reduced to one year. In agreement with other African countries and territories it is, however, not encouraged or recognised for interstate movements and they must be crated and railed or conveyed by air to a quarantine station for 180 days detention, whether vaccinated or not, in the same way as unvaccinated dogs. This agreement is subject to review to conform with practice in other countries.

In-transit movements through the Republic are authorised by rail or air in official custody, subject to the usual health certificates and in compliance with requirements of the veterinary authorities of the countries to which the animals are proceeding. Movements by road are only permitted where dogs have been vaccinated in accordance with our requirements and provided the veterinary authorities of the country of destination authorise introduction in that way.

ACKNOWLEDGEMENTS

The writer wishes to thank the Chief of Veterinary Field Services for permission to publish this article.

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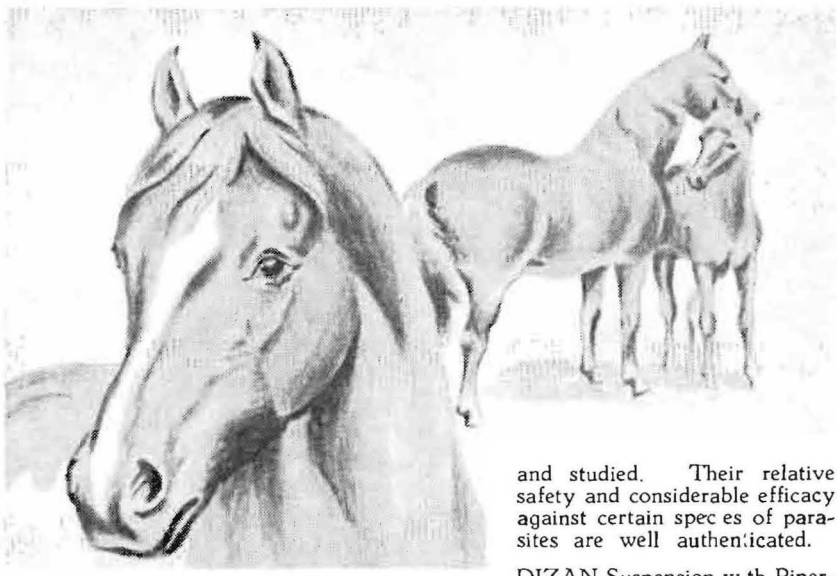

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VETERINARY CONGRESS 25th - 28th SEPTEMBER, 1962

RABIES IN SOUTH AFRICA

RABIES AND THE PRIVATE PRACTITIONER

A. F. TARR
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Received for publication, July 1962

SUMMARY

This contribution to the symposium describes some of the difficulties experienced by private practitioners during a rabies epizootic.

INTRODUCTION

This paper deals with a rabies outbreak in the Pietermaritzburg area during the latter part of 1961 and early 1962 as experienced by the private practitioner.

Emphasis will duly be laid on the symptomatology of the disease as was observed at the time.

CANINE ASPECT

Of all cases presented only one case of the "Furious form" was encountered, while many of the others were of the paralytic or socalled "Dumb rabies" type. However the majority of cases seen were clinically indistinguishable from the distemper complex and this presented a very real problem.

Temperatures were not significant but conjunctivitis, pharyngitis, tonsillitis and inappetance were generally encountered.

The most common courses of the diseases were:—

(*a*) Intermittent temperature for 4 to 5 days with a non-response to antibiotic or sulpha drug therapy, followed by progressive paralysis of the hindquarters.

(*b*) An intermittent temperature followed by meningo-encephalitic type symptoms.

(*c*) A combination of (*a*) and (*b*).

(*d*) Paralysis of the lower jaw and throat with profuse salivation and protrusion and prolapse of the tongue.

In all these cases there was at no stage any show of aggressiveness and the patients were tractable at all times. Only two dogs were destroyed at the owner's request due to unprovoked attacks on humans, but they were nevertheless easily handled at the surgery. Both cases were virtually normal at examination and were only diagnosed as positive rabies following routine specimens submitted to Onderstepoort.

Confusing associated factors were:

(i) Breed of dog e.g. the snappiness of Daschunds.
(ii) Bitches in season and the consequences.
(iii) Unreliable histories.
(iv) Disappearance of dogs e.g. one was recovered 14 miles from home and one or two cases disappeared entirely.

Feline Aspect

One case of rabies was encountered in a cat. The animal was attended three weeks after it had been bitten by a dog which was later confirmed as rabid. Abdominal herniation as a result of the bite was surgically corrected. Three weeks later the cat exhibited posterior paraplegia and in view of the history, was destroyed. The diagnosis of rabies was confirmed.

Bovine Aspect

One case was recorded in a cow. The animal was on heat, bellowed continuously, lost condition rapidly, developed an unsteady gait and walked in circles. This was followed by inability to swallow and profuse salivation and she was destroyed. Brain specimens proved positive and it was subsequently established that the cow had been bitten six weeks previously by a confirmed rabid dog.

Differential Diagnosis

Mention is only made of the conditions which gave rise to uncertainty during the outbreak.

Dogs:

(*a*) Distemper complex — N.B.
(*b*) Leptospirosis and nephritis complex.
(*c*) Acute tonsillitis/pharyngitis.
(*d*) Decayed teeth and tooth-ache.
(*e*) Foreign body in buccal cavity and pharynx.

Cats:

(*a*) Spinal cord lesions.
(*b*) Foreign body in buccal cavity.
(*c*) Ulceration of the tongue.

Bovine

(*a*) Nymphomania in the case encountered.
(*b*) Cerebral piroplasmosis.

Incubation Period

In this outbreak the unreliable incubation period of rabies was clearly illustrated e.g. The only case of the "furious form" encountered had been inoculated five months previously after having been in contact with a rabid dog.

A case of "dumb rabies" was encountered in a dog which had been inoculated 6 months previously. Further investigation indicated that a day before inoculation this animal had been in contact with a dog next

door which ultimately developed confirmed rabies. Subsequent contact was impossible due to barricaded premises.

In the cat case, the incubation period was probably six weeks.

Inoculation

A few remarks would not be out of place in this connection.

Site.—In our opinion the most convenient sites for inoculation are the triceps muscle in large dogs and between the semitendinosus and biceps femoris muscles in small dogs. This practice was carried out because of the danger of sciatic nerve damage and the proximity of the popliteal lymph-gland at the normally recommended site.

In all, over 1,000 dogs were done and there was not one case of abscessation or lameness brought to our notice.

Ear Tattooing

It is suggested that particular care be taken to avoid bloodvessels as profuse bleeding, othaematomas and obliteration of the tattoo could follow. Furthermore, the best tattoos resulted after the tattoo paste had been massaged into the perforations.

General Remarks

A very disturbing and uncomfortable aspect in dealing with possible rabies is the uncertainty which prevails due to the fact that most cases were clinically indistinguishable from the distemper complex. This was further aggravated by the absence of protective vaccination for the veterinarians concerned, until a very late date.

There is little doubt that several socalled distemper cases attended and subsequently destroyed, were in fact rabies. One might add that instinct became a facet of diagnosis in some cases.

The attitude of the public to the outbreak varied from sheer indifference and disbelief to one of utter panic. And the initial acute shortage of the vaccine certainly did not help to alleviate the position.

In the normal humdrum of every day life one does not comprehend the number of dog-bite cases in humans.

This brings us to a very vital and not always appreciated aspect of a rabies epizootic which places a particularly vital onus on veterinarians.

With urgent requests to report all such cases, it was found that an incredible number of persons were bitten every week. In the majority of cases the medical practitioners and hospital authorities concerned communicated with the veterinarian for an expression of opinion as to whether the victim should undergo prophylactic or rabies treatment. In many instances their opinions were accepted and acted upon.

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RABIES IN SOUTH AFRICA

RABIES FROM THE POINT OF VIEW OF THE MEDICAL OFFICER OF HEALTH OR THE MEDICAL PRACTITIONER

H. NELSON — Medical Officer of Health, Pretoria

Received for publication, July 1962

INCIDENCE

Rabies is an illness which occurs in practically every part of the world.

The following is a table extracted from the World Health Organisation Epidemiological and Vital Statistics Report[1] Volume 14, No. 11 of 1961, showing deaths from 1955 to 1960, in countries where the incidence was highest.

TABLE I

Country	1955	1956	1957	1958	1959	1960
Phillipines	195	162	189	215	212	265
Ceylon	137	136	156	127	28	N/A
Bombay, Madras, Punjab } India	294 76 65	35 147 69	N/A 124 62	N/A N/A 64	N/A N/A N/A	N/A N/A N/A
Colombia (America)	31	30	41	40	26	28
Congo (Africa)	42	40	32	39	N/A	N/A
Burma	35	44	55	N/A	N/A	N/A
Mexico	58	72	47	55	N/A	N/A
Turkey	35	17	54	61	50	59
South Africa	—	—	—	2	—	5

Note.—N/A indicates Not Available.

For the year 1961, there were twelve deaths in human beings in South Africa.

The following table gives details in connection with cases which occurred in South Africa:—

TABLE II

Year	No. of animals in which the illness occurred	No. of known human contacts	No. of deaths in human beings	The animal which caused the illness in the human being
1953.....	38	11	Nil	Nil
1954.....	101	39	Nil	Nil
1955.....	94	45	Nil	Nil
1956.....	93	46	Nil	Nil
1957.....	163	109	Nil	Nil
1958.....	109	55	2	1 Genet Cat, 1 Dog
1959.....	106	56	Nil	Nil
1960.....	79	43	5	3 dogs, 1 Goat and 1 Meercat
1961.....	315	181	12	10 Dogs, 1 Genet Cat and 1 Baboon
	1,098	585	19	

It will be seen from the above that fourteen of the patients who died as a result of rabies were bitten, or were presumed to have been bitten, by dogs.

From statistics throughout the world, bites by rabid dogs are the most common cause of rabies in human beings.

Although I have not been able to obtain any records of persons who were treated and recovered, it will be noted from the above table that from the beginning of 1953 to the beginning of 1961, there were 585 contacts, and it is presumed that most of them received some form of anti-rabies treatment, and that out of this total only nineteen died.

With the help of the State Health Department, I have been able to trace the *histories of the deaths amongst human beings* referred to in table II above.

The available details of these deaths are as follows:—

From the available records it is not certain that some of the cases which have been reported as rabies, are actually rabies.

The follow-up of the possible source of infection has been incomplete. It is also not certain whether the recorded infecting animal was really the cause of rabies in the patient, nor is it certain in all cases whether the patient died of rabies or not. On the other hand, there must have been quite a number of persons in remote parts of South Africa who died of rabies without the illness ever having been diagnosed and notified.

From the above table and also from the lack of information in regard to what happened to the 585 contacts referred to in Table II, it will be noted that our recorded information is very inadequate.

In the World Health Organisation Technical Report Series[2] there is a recommended form which should be completed in all cases of rabies. It would seem desirable that similar forms should be used in recording cases in the Republic.

Case No.	Date bitten	Date died	Biting Animal	Treatment	Part of body where bitten	Address	Remarks
1	12.458	5.5.58	Genet Cat	3 x 2 cc. Anti-rabies Vaccine	Unknown	Kuruman	All symptoms of rabies. No further details.
Observation.—It would appear that treatment was started too late, because he only had three injections when he died of rabies.							
2	6.10.58	22.6.59	Dog	Full course of anti-rabies serum	Unknown	Louis Trichardt	No further details; a doubtful case; age unknown
Observation.—The incubation is too long for this history to be correct. It is also an unconfirmed case. It would seem that what happened here is that the anti-rabies treatment probably saved the patient from contracting the illness, and that he subsequently died from some other cause.							
3	Unknown	19.7.60	Dog	Reported for treatment at a time after having been bitten. Had a septic finger on the right hand. Treated for wounds on finger. He disappeared and received no further treatment. A.T.S. 3,000 units. Penicillin 600,000 units	Finger, right hand	Dist. Tzaneen	He went home, and his brother informed the District Surgeon of his death subsequently. (Age approx. 48).
Observation.—Although this was recorded as a case of rabies, it is not at all certain from the history as to whether this patient died of rabies or not.							
4	8.11.60	2.12.60	Dog	Wounds were cauterised — 13 x 2 cc. Irradiated rabies vaccine absconded before treatment could be completed	Left hand	Dist. Messina	Died away from hospital. No further details. Age approx. 40
Observation.—This patient died in spite of having received almost a full course of treatment.							

Case No.	Date bitten	Date died	Biting Animal	Treatment	Part of body where bitten	Address	Remarks
5	8.11.60	10.11.60	Meercat	Died before treatment could be started	Arm and leg	Dist. Vryburg	The District Surgeon is of the opinion that death set in so soon as a result of the meercat biting the patient in one of the main arteries of the arm. Age 18 years.
Observation.—The District Surgeon notified this as rabies. Even though the bite was presumed to be right into an artery, it is doubtful whether the patient could die of rabies within such a short time.							
6	16.11.60	15.12.60	Dog	Wounds were septic as patient only arrived at hospital on 2nd December, 11 x 2 cc. of Irradiated Rabies Vaccine	Legs	Dist. Letaba	Dog attacked 5 Natives including the patient—killed by Natives. Age appox. 48.
Observation.—This also appears to be a genuine case — note the incubation period of one month — patient having been bitten on the legs. There was a long delay in treatment, although the patient received 11 injections.							
7	5.2.61	10.3.61	Genet Cat	Washed with Dettol; 13 x 2 cc. of Irradiated Rabies Vaccine	Finger, left hand, 1st attack finger right hand 2nd attack	Dist. Kuruman	Genet cat attacked 16 years old Native girl while sleeping. It was emaciated and apparently deformed. Age approx. 16
Observation.—This appears to be a genuine case where the patient had nearly a full course of treatment. Note the long incubation period where the patient was bitten on the hands.							
8	6.4.61	29.4.61	Dog	Only wounds treated	Face	Dist. Incwavuma	Age unknown, but it was a small child. Histologically proved positive
Observation.—This shows how short the incubation period can be if the patient is bitten on the face.							

Case No.	*Date bitten*	*Date died*	*Biting Animal*	*Treatment*	*Part of body where bitten*	*Address*	*Remarks*
9	6.8.61	8.9.61	Dog	Patient had prodromal symptoms on admission — far advanced—followed by rapid progression of rabies symptoms — died 59 hours after admission. 3 x 1½ cc. Irradiated Rabies Vaccine	Right leg, below knee	Dist. Ingwavuma	Wounds had healed on admission. Age 9.
Observation.—This seems to be a genuine case with a long incubation period, patient having been bitten on the leg below the knee.							
10	15.8.61	31.8.61	Dog	None—arrived with full symptoms of rabies and died 4 hours after admission—first symptoms, according to mother, appeared approx. 27.8.61	Forehead	Dist. Mahlabatini	Age 5 years
Observation.—Here again, a short incubation period, the patient having been bitten on the forehead.							
11	Unknown	17.8.61	Dog	Unknown	Right breast	Dist. Umbobo	Shown to be histologically as well as biologically a positive case of rabies — brain samples of patient taken. Age approx. 6
Observation.—Nothing is known about this patient, excepting that she died of rabies.							
12	Unknown	30.8.61	Dog	Unknown	Unknown	Dist. Umbobo	Same as above. Age approx. 25
Observation.—Nothing is known about this patient, excepting that she died of rabies.							

Case No.	*Date bitten*	*Date died*	*Biting Animal*	*Treatment*	*Part of body where bitten*	*Address*	*Remarks*
13	12.9.61	10.10.61	Dog	Died of other causes	N/A	Tzaneen	Cause of death: Pellagra
Observation.—This case is recorded as rabies, but from the above history it obviously is not so.							
14	28.9.61	6.10.61	Goat	No treatment given	Right hand	Dist. Postmasburg	Received a cut while removing grass from goat's hoof. No further details
Observation.—There are many doubts about the history of this case, like the shortness of the incubation period, the doubtful possibility of contracting the illness as a result of removing grass from a goat's hoof, and the fact that I subsequently heard that the goat did not have rabies. All this seems to indicate that the patient must have contracted rabies from some other source.							
15	14.9.61	27.10.61	Dog	Unknown	Hand, arm and shoulder, left side	Dist. Eshowe	Complained of pain in the throat and that he could not swallow. No neck rigidity. Reflexes normal. Respiratory system normal. Emotionally upset. Deteriorated. Began vomiting. Refused all food and fluids dribbled from the mouth. Became comatose. Died. Brain specimens. Biologically positive. Age 46.
Observation.—This looks very much like a genuine case. The incubation period is rather long for multiple lacerations as high up as they are.							

Case No.	*Date bitten*	*Date died*	*Biting Animal*	*Treatment*	*Part of body where bitten*	*Address*	*Remarks*
16	Unknown	8.10.61	Dog	Unknown. Apparently patient was so far gone that he died before treatment could be started	Unknown	Dist. Ubombo	No post mortem was done
Observation.—Tnere is no indication that this was a definite case of rabies.							
17	Unknown	17.11.61	Dog	Unknown	Unknown	Dist. Vryheid	Bitten by dog 1 year before death. Age approx. 35
Observation.—It can only be assumed that the diagnosis at death of rabies was authoritatively confirmed. The incubation period of one year is abnormally long, and if the patient did die of rabies, he may have been infected in the interim in some other way. This is probably what happened.							
18	1.12.61	25.12.61	Baboon — escaped	Treated by private doctor	Toe, arm, face	Dist. Pietersburg	No particulars of treatment available. Apparently rabies was not suspected until after positive rabies result from Onderstepoort after death. Age approx. 50
Observation.—This appears to be a definite case. It is not known what treatment the patient received. The patient had multiple bites on the toe, arm and face, resulting in this very short incubation period.							
19	1.12.61	28.12.61	Dog	Patient died three days after admission to hospital	Hand	Ntonjaneni	No further details
Observation.—This patient evidently had no treatment and the illness must have been advanced when admitted to hospital.							

Incubation Period

The incubation period is variable and depends on many factors. Deep bites, bites on the face and upper part of the body, and where lacerations are extensive, have a shorter incubation period. The amount of virus inoculated through a bite also influences the length of the incubation period, for instance, where successive persons have been bitten on more or less the same site of the body by the same rabid dog, the incubation period in those bitten last is longer because those bitten first received a larger dose of infected saliva.

For these reasons the incubation period of rabies in man varies between 10–70 days.

In dogs it is somewhat shorter, about 20–50 days and in puppies it can be as short as one week.

Risk of Infection after having been Bitten by a Rabid Dog

Because the treatment of rabies is always fairly drastic and with some vaccines not without danger, it is important to evaluate what the chances of developing the illness are in a person who has been bitten by a rabid or "suspect rabid" dog.

It is realised that this cannot be determined with accuracy and each case must be considered individually. In some outbreaks the infection rate, after being bitten by a dog which is known to be rabid, was found to be as low as 15 per cent. In others again it was as high as 70 percent. There are many factors, like the site and depth of the bite, the extent of the lacerations and the number of infecting organisms introduced, which account for this difference.

If a person is bitten on or near the face or head by a rabid dog, it must be assumed that he is going to develop rabies. If bitten on the trunk or extremities and lacerations are not deep, chances of infection are considerably less. If the bite has taken place through clothes, the chance of infection is still less and is estimated to be not more than from 3–5 per cent. If there are no bites, but there are scratches and abrasions which have been contaminated by saliva of a rabid dog, chances of infection are very small, about 1 per cent. If there are no bites, scratches or abrasions, chances of infection are practically nil.

Most dangerous of all infecting animals is the dog and the highest incidence of the disease is as a result of rabid dog bites.

On an average it is estimated that one out of every six persons who are bitten by a rabid dog become infected.

All these figures, however, do not help when one personally has to make a definite decision as to whether a person who has been bitten by a rabid or suspect rabid dog has to be regarded as having become infected or not.

In trying to arrive at a decision, the first consideration is whether the dog which bit the person is rabid or likely to be rabid. This is not always easy. The story we get is usually second-hand, without much knowledge about the condition of the biting animal. If it is alive and traceable this aspect could be followed up. If not, a decision may often

have to be made on pure guess work. If there is a history of an epidemic of rabies in an area it would influence the decision.

It is of the utmost importance not to destroy an animal which is suspected to be suffering from rabies. It should be caught and kept under supervision in order to see whether it develops rabies or not. Any dog which appears to be rabid or the heads of animals which have been killed because of suspected rabies, must be sent to a competent laboratory for diagnosis.

Points for Consideration in Arriving at a Decision as to whether there is a likelihood of a Human Being having been infected, and suggested lines of treatment

From a study of the special committee on rabies of the World Health Organisation, the following is a suggested guide:—

(1) Licks

Where unabraded skin has been licked by a rabid animal there need be no treatment other than careful washing the affected area with soap and water.

In licks where the skin has been abraded or scratched:

(*a*) If the suspect animal appears to be healthy, it should be kept under observation for a period of ten days or sent to a laboratory for diagnosis.

The patient must receive local treatment immediately and vaccine treatment is withheld until the illness is diagnosed in the biting animal, when it must be started immediately.

(*b*) If the animal shows signs suggestive of rabies, it must be kept under observation like in (*a*) above, but local treatment and vaccination must start immediately. If the biting animal has not developed rabies after the fifth day of exposure, treatment is stopped.

(*c*) If the animal is known to be rabid from the beginning or has escaped or has been killed, vaccination must start immediately.

(2) Bites

In mild exposures, like a bite through clothes or where the bite is not deep or where the bite is on the trunk or extremities:—

(*a*) If the animal appears to be healthy it must be kept under observation for a period of ten days and if during that time there are clinical or laboratory signs of rabies, vaccine treatment of the patient must start as soon as the diagnosis is made in the biting animal.

(*b*) If there are signs suggestive of rabies, the animal must be kept under observation, but vaccination of the patient must start immediately and must only be discontinued if the animal is still normal on the fifth day after exposure.

(*c*) Where the animal is known to be rabid or has escaped or has

been killed, but not subjected to laboratory examination, vaccination must start immediately.

(3) Severe Exposure

This refers to cases where there are multiple bites or face or head bites or where the bites are deep:—

(*a*) If the animal is healthy it must be kept under observation for a period of ten days and the patient must be given antiserum immediately. If during the period of observation there are clinical or laboratory signs of rabies in the biting animal, vaccination must be started immediately.

(*b*) If the animal shows signs which are suggestive of rabies, it must be kept under observation for ten days, serum must be given immediately followed by vaccination, but this must be discontinued if the biting animal is found to be normal after the fifth day after exposure.

(*c*) If the animal is known to be rabid or has escaped or has been killed, but has not been subjected to laboratory examination, the patient must be given serum immediately followed by a course of vaccination.

Apart from the above types of cases, it is also recommended that particular groups of individuals, such as veterinarians, dog handlers, laboratory workers or other people who are unusually exposed to risk of being bitten or otherwise infected with rabies, should also be vaccinated as a prophylactic measure. This has already been done to 180 laboratory workers, veterinary students and field personnel at the Onderstepoort Veterinary Laboratory, who were bled on April 27, 1962 prior to being given the first dose of Duck-egg vaccine. They each received 0.2 ml vaccine intradermally at 5–6 day intervals for four doses. They were bled again fourteen days after the last dose. A further dose is to be given in October, that is, five months after the last dose. They will then again be bled fourteen days later. The bloods are being tested for anti-bodies at the laboratories of the Poliomyelitis Research Foundation, and the results should be available after the last bleeding.

(4) Treatment

(*a*) *Local treatment*

In all cases where there is any suspicion at all that a person has been bitten by a rabid or suspect-rabid animal, local treatment of wounds should be given immediately.

This consists of washing the wound as clean as possible with ordinary soap and water, careful application of concentrated nitric acid to puncture wounds, and infiltration of the area with a local anaesthetic like procaine. If there is a strong suspicion that the animal is rabid, the wound area should also be infiltrated with hyper-immune serum.

(*b*) *Vaccine treatment*

A great deal of research has been done into dangers connected with vaccine treatment and the value of different types of vaccines.

The types of vaccine prepared, methods of preparation and toxicity of the different types have been dealt with elsewhere in this symposium.

Generally speaking, vaccine treatment is considered to be of real value in preventing death from rabies in human beings, because once the illness develops, death is inevitable.

The special committee of the World Health Organisation on rabies, states that—

"the most valuable recent report in this connection is that of Veeraraghavan (1959) on the incidence of rabies in persons exposed to bites of animals which definitely induced rabies in other susceptibles. The incidence of rabies in those who were completely treated (excluding cases developing less than 15 days after vaccination) as compared to those who refused treatment was 3·35 per cent, as against 49·2 per cent. Clearly the controls in this case are not ideal; however, ideal controls are virtually impossible to obtain, and the striking difference in the two groups lends further support to the long established belief that rabies vaccination in persons exposed to the disease is of real value.

Nevertheless, there remains a disturbingly high incidence of rabies in vaccinated persons. At least a partial solution to this problem would appear to have arisen with the development of anti-rabies serum. Aside from evidence of its effectiveness in animals, striking results in man have been reported by Baltazard and his colleagues (1955). In a group of 17 persons suffering severe head wounds from multiple bites by a rabid wolf, 5 received two injections of anti-rabies serum on the first and fifth days, 7 received one serum injection, and 5 received no serum. All 17 received the standard course of vaccine. There were no deaths in the first group, one in the second and three in the third. Incidentally, all eleven people (in another group) bitten elsewhere than around the head and neck, and receiving vaccine alone, survived. Since this observation it has become generally accepted international practice (World Health Organisation 1957) to administer anti-rabies serum as well as vaccine in the presence of severe bites or bites around the head and neck. Nevertheless, some deaths from rabies have occurred in individuals given combined serum and vaccine prophylaxis. Because of the risk that serum administration may block the effect of vaccine, the report of the latest W.H.O. Expert Committee on Rabies (World Health Organisation 1960) recommends the added precaution of giving supplementary doses of vaccine 10 and 20 days after the last usual dose, in persons receiving serum as well as vaccine. The efficacy of this schedule in man, however, remains to be determined.

Under expert observation records have been kept of patients who have been treated with hyper-immune serum only, with hyper-immune serum plus vaccine or who were altogether untreated. In some cases the records show that there is a 50 per cent increase in the number of cases who do not develop rabies because of hyper-immune serum together with vaccine treatment."

It must, therefore, be accepted that treatment is essential in patients who have been bitten by rabid or suspect rabid animals.

The question of what type of vaccine should be used is still controversial.

The Semple type of vaccine made on nervous tissue is regarded by many as producing more anti-bodies than any other known type of vaccine. This view, however, is not held by all. Some say that the Duck-egg vaccine is equally potent. Others again say that the Duck-egg vaccine is disappointing in its anti-body producing properties, although it is admitted that it does produce anti-bodies.

On the whole it would appear that Duck-egg vaccine may be somewhat less effective than the Semple type, but that the difference is not very great.

The dangers connected with the use of the Semple type vaccine are, however, very great. In South Africa the number of cases developing paralysis, after the administration of Semple vaccine, is said to be as high as one in 250. In the United States of America some say the incidence is about one in 500.

The more serious accidents connected with Semple vaccine are neuro-paralytic and include neuritis, myelitis and encephalo-myelitis, resulting in various types and degrees of paralysis, apparently caused by repeated injections of brain tissue substance. As far as can be gathered from the available information, four persons in South Africa died as a result of administration of the vaccine out of 1,000 who received it. It is not known how many became paralysed, the extent the paralysis assumed, how many recovered from the paralysis, and to what extent there was recovery.

On the other hand, the Duck-egg vaccine is as safe as any vaccine can be, and the incidence of post vaccination complications is rare, if not unknown. Considering the potency and dangers connected with each of these two types of vaccine, I have no hesitation in saying that the Semple vaccine should never, under any circumstances, be used if Duck-egg vaccine is available, because in this we have a vaccine which is safe and is a good anti-body producer.

How to use Duck-egg Vaccine

In using this vaccine as a prophylactic in veterinarians and other persons who might be exposed to rabies, the dosage and methods of administration advised by the manufacturers are intradermal injections of 0·2 ml given at five day intervals for four doses with a booster dose of 0·2 ml intradermally six months later.

As a prevention against the disease in persons who have been bitten by rabid animals or animals suspected to be rabid, it should be used in conjunction with hyper-immune serum.

Here again, there is some controversy as to how this combination should be spaced. All are agreed that hyper-immune serum must be given immediately, but some say that vaccination could be commenced on the following day, others that it should not be given until at least four days after the hyper-immune serum, and others again that it should not be given until about eight days afterwards.

All agree, however, that it should be given daily for fourteen days,

irrespective of which day, after giving hyper-immune serum, vaccination commences. The reason for these different periods in between the administration of hyper-immune serum and vaccination is because there are so many different opinions about how long hyper-immune serum will inhibit the development of anti-bodies by the vaccine. If it is true that hyper-immune serum will prevent the development of anti-bodies for as long as one week, and the vaccine is given the day after the administration of the hyper-immune serum, the only effective vaccinations will be those administered about one week later, and the first six or seven will be of no value, and as only fourteen are recommended with two booster doses twenty to thirty days later, the patient will only have about seven or eight effective immunising doses, which are not sufficient to produce adequate immunity.

There may be some truth in this, but it is difficult to prove that this actually happens in vivo. In any case, what is true for one person may not be true for another. There is certainly no doubt that starting with the vaccine one day after the hyper-immune serum can do no harm and can only do good.

If it is considered that the inhibiting effect immediately after hyper-immune serum is so great that half the vaccinations may be ineffective, this could be overcome by continuing with the vaccine for a longer period.

For these reasons, I suggest that the best line of treatment would be to give hyper-immune serum immediately and to follow it up the next day with Duck-egg vaccine daily for 21 days and not 14 days. By doing this we will cover the possible "ineffective" period immediately after administration of hyper-immune serum by a 21 day course, and yet give whatever possible protection right from the beginning. The larger number of vaccination can only do good and can do no harm. The dosage of hyper-immune serum is a minimum of 20 ml and that of the vaccine is 2 ml given subcutaneously.

Whatever method is used, it is essential that if hyper-immune serum is given with Duck-egg vaccine or with Semple vaccine, two booster doses of Duck-egg vaccine should be given one ten and one twenty days after the last vaccination in the series.

Anaphylactic Shock and Serum Sickness

In treating a patient with hyper-immune serum the danger of anaphylactic shock or the complications of serum sickness, which is connected with the use of any anti-serum or the introduction parenterally of any foreign protein into human beings, must always be borne in mind. It is important, therefore, that before giving any anti-serum, the possibility of allergic reactions should be carefully gone into. Patients with a history of severe reaction to previous injections, or a family or personal history of allergic conditions such as hay fever, asthma or who have allergic reactions to certain animals or plants, should be tested for sensitivity against the specific serum to be administered.

The test for sensitivity is by injecting 0.1 cc. of the anti-serum diluted in 1 cc. of sterile normal saline subcutaneously, and if the patient is sensitive an area of urticaria will appear at the site of infection. If the

reaction is positive, the same procedure has to be repeated hourly until there is no longer any reaction. In other words, the test for sensitivity is also the method of desensitisation. As soon as no reaction occurs the full dose of anti-serum can be given, but preferably in smaller and divided doses diluted with equal parts of normal saline.

The signs and symptoms of anaphylactic shock are very typical. It usually starts very suddenly within a few minutes, some times within a few seconds after injection, although there may some times be a delay for as long as thirty minutes.

The patient has a sudden feeling of anxiety and uneasiness. There is a sudden pounding headache with intense throbbing in the ears, often followed by rapid collapse of the cardiovascular system which may lead to sudden death in untreated cases. In severe cases the first signs are often sudden generalised convulsions with death within a few seconds.

Treatment consists of administration of Epinephrine one in a thousand, 0.3 to 1 cc. subcutaneously for less severe reaction, and 0.25 to 0.1 cc. intravenously every half to one minute for more severe cases until relief is obtained. Oxygen therapy is also given when there are respiratory difficulties.

Serum sickness is not such a dangerous condition, and it usually comes on eight to twelve days after inoculation, but it may come on as early as ten to twelve hours after the injection.

The same precautions in regard to anaphylactic shock should, however, be taken.

The signs and symptoms of the illness are rise in temperature, adenitis, particularly in the area draining the injection site, but it may become generalised, severe pains in many joints, a typical urticarial skin eruption which usually starts at the site of injection and spreads to other parts of the body.

As a rule the illness cures itself and treatment is symptomatic. Antihistamine or ephedrine given by mouth every four hours generally relieves symptoms.

It is most important to remember that when giving any anti-serum, it is imperative that there is adrenalin at hand for immediate administration, should it become necessary.

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A ROUTINE STAIN FOR RABIES

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SUMMARY

A diagnostic staining method is described for sections of brain material in cases of suspected rabies.

It is an Acid Fuchsin-Methylene Blue method based on formalin fixed material and using very dilute solutions of these stains. The method is straightforward and rapid and well within the capabilities of most technicians. The same technique and stains are used for both frozen and paraffin sections, giving comparable results.

The method has been in use in this laboratory for a number of years and results compare favourably with the methods previously used.

INTRODUCTION

In the majority of suspected rabies cases in South Africa brain material for histological examination is collected in the field. The time taken for many of these specimens to reach the laboratory renders the use of fixatives such as Zenker's impractical. Specimens are therefore collected in standard unbuffered 10 per cent Formalin.

Prior to 1957 two methods were used for staining *Negri* bodies in sections of hippocampus or brain. These were the Methyl Blue-Eosin method of Mann[1] and a Methyl Blue-Phloxin method developed in this laboratory.

As a routine diagnostic stain Mann's had several disadvantages. It was found that with formalin fixed, and particularly poorly fixed material the results were indifferent. In addition, differentiation in Mann's method is difficult and in the hands of the technician sections were often over-differentiated. The 18 to 24 hours staining required was too long, especially in urgent cases i.e. where human contacts were recorded, while the method could also not be adapted to frozen sections.

To meet the need for more rapid results in urgent cases, the Methyl Blue-Phloxin method was devised for frozen sections and was later adapted to material embedded in paraffin.

Although two staining methods were then applied to all rabies material it was still felt that the results were unreliable and many cases required restaining.

An attempt was therefore made to overcome these difficulties. The possibility of using other staining methods for *Negri* bodies was investigated, but in most of these special fixatives or, at the very least, adequate fixation of fresh material was of prime importance. As Acid Fuchsin

had proved to be a valuable stain for demonstrating *Negri* bodies it was decided to use this as a basis and to attempt to develop a reliable method which would better suit local requirements. Using this stain at various concentrations and in neutral, alkaline and acid media, it was found that more consistent results were obtained with longer staining in very weak solutions in acid medium. Various counterstains and differentiating media were also applied and from this preliminary experimentation the Acid Fuchsin-Methylene Blue method (Afmb) was evolved.

Materials

For the reasons stated above only material fixed in 10 per cent Formalin is available. A block suitable for embedding in paraffin wax is cut from the hippocampus, or if this is not present, two blocks are cut from other areas of the brain, the part being dictated by the nature of the specimen. In urgent cases similar blocks are cut for both frozen sections and paraffin embedding.

Staining solutions are made up as follows:—

Acid Fuchsin*

(*a*) *Stock solution*:

Acid Fuchsin	1 gm.
Distilled water	100 ml.

(*b*) *Working solution*:

Stock solution Acid Fuchsin	1 ml.
N/20 HCl (approximately)	99 ml.

Methylene Blue†

(*a*) *Stock solution* (Loeffler's alkaline):

Methylene Blue	0.3 gm.
96 per cent Ethyl alcohol	30 ml.

Dissolve the stain in the alcohol, thereafter adding:—

0.01 per cent aq KOH	100 ml.

(*b*) *Working solution*:

Stock solution Methylene Blue	10 ml.
Distilled water	40 ml.

Undiluted these stock solutions will keep for months. The working solutions are used in the staining sequence for one week and are then replaced by fresh solutions.

Method

A.—Paraffin Sections

The tissue blocks are processed in a tissue processor overnight and

* The Acid Fuchsin used is that of Coleman & Bell (certified).
† Various makes of Methylene Blue have been used.

embedded in paraffin the next morning. Sections are cut at three microns and are affixed to slides with albumin. Slides are left on the slide warmer for at least an hour before staining.

Staining technique:

(1) Deparaffinate in xylene (see (*a*) below).
(2) Hydrate through graded alcohols, 100%, 96% and 70%.
(3) Wash in distilled water.
(4) Stain in working solution Acid Fuchsin — 10 minutes.
(5) Rinse in distilled water 1–2 seconds (see (*b*) below).
(6) Counterstain in working solution Methylene Blue 8–10 seconds (see (*c*) below).
(7) Rinse in distilled water 1–2 seconds (see (*b*) below).
(8) Rinse in 96% alcohol (see (*d*) below).
(9) Dehydrate in 100% alcohol.
(10) Clear in xylene.
(11) Mount in Canada Balzam.

(*a*) Leaving the sections in xylene for periods up to an hour or longer improves subsequent staining but is not done in routine staining.

(*b*) In steps 5 and 7 distilled water only should be used since tap water immediately removes all traces of the Acid Fuchsin stain.

(*c*) The counterstain also acts as a differentiating medium for the Acid Fuchsin and the time factor in this step is of the utmost importance. After this step the section should appear pink and not bluish to the naked eye.

(*d*) The 96% alcohol removes any excess Methylene Blue from the section.

B.—Frozen Sections

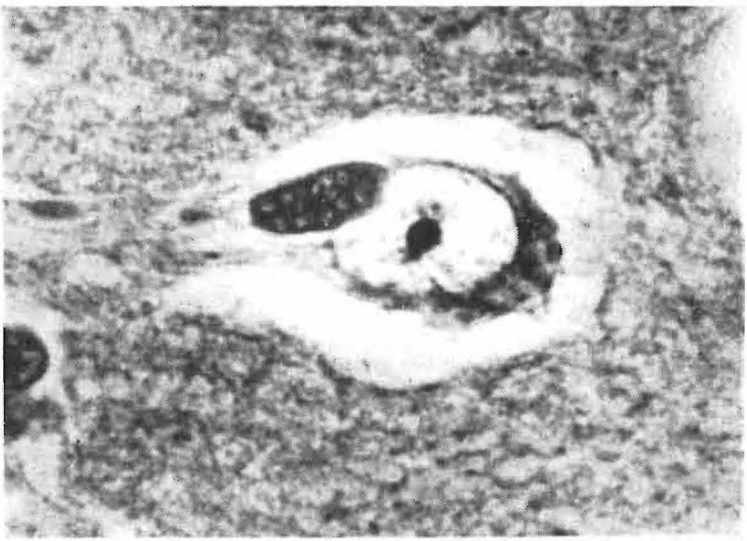
Sections are cut at ten micron, collected in distilled water and mounted from the water on to albuminised slides. These are drained of excess water, blotted if necessary and allowed to dry at room temperature for approximately 10 to 15 minutes.

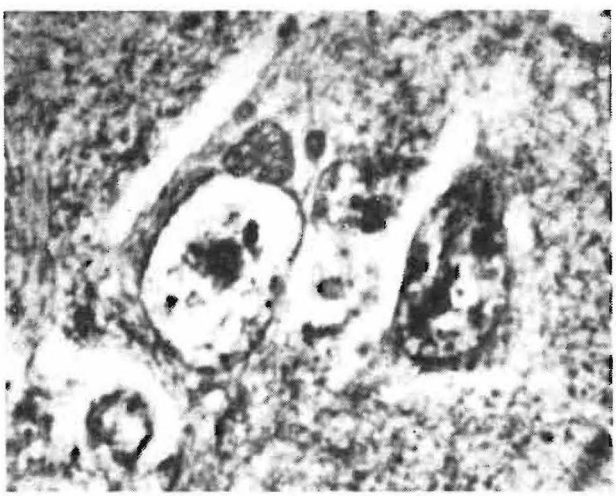
These slides are thereafter treated exactly as the paraffin sections above, commencing at step 4 i.e. they are dehydrated, cleared and mounted in Canada Balzam.

Both paraffin and frozen sections can be stained in bulk up to step 4. From the Acid Fuchsin each slide should, however, be counterstained separately.

Results:

Nuclei	blue
Nucleoli	purple
Erythrocytes	deep pink to red
Negri bodies	deep pink
Inner corpuscles of *Negri* bodies	usually unstained or blue
Cytoplasm	light pink or blue





Bovine hippocampus showing *Negri* bodies
Stained Afmb x 1200

Discussion

The method described is a diagnostic staining method based on formalin fixation for routine histological examination of sections of suspected rabies material. A comparison of the results obtained with Afmb on the one hand and Mann's and Methyl Blue-Phloxin on the other is discussed elsewhere in this journal[2].

The method is simple and rapid. The preparation of the stock and working solutions presents no problem and the staining technique is straightforward provided that the Methylene Blue counterstaining is carefully timed. Less than 90 minutes are required for cutting and staining a paraffin section, while in urgent cases frozen sections can be completed within 45 minutes. Apart from deparaffination and hydration similar techniques using the same staining solutions are followed for both frozen and paraffin sections, thus giving rise to comparable results. The definition in the paraffin section is however better due to the difference in section thickness.

On histological examination contrast in routinely stained sections is not high but *Negri* bodies are nevertheless easily recognised and can be found even in badly autolysed and poorly fixed material. The differentiation between nucleolus and *Negri* body is good and should present no difficulty. On the other hand there is little difference between the staining reaction of the inclusion body and erythrocytes. Erythrocytes assume a deeper and more even colour than the intracytoplasmic *Negri* body.

The basophilic inner corpuscles of the *Negri* body may be unstained in the routinely stained section but can be stained blue in well fixed material with careful counterstaining.

ACKNOWLEDGEMENTS

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STUDIES ON SPECIFIC OCULO-VASCULAR MYIASIS OF DOMESTIC ANIMALS (UITPEULOOG)

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SUMMARIES OF CONTRIBUTIONS TO THE ONDERSTEPOORT JOURNAL OF VETERINARY RESEARCH OBTAINED THROUGH THE PERMISSION OF THE CHIEF, VETERINARY RESEARCH INSTITUTE, ONDERSTEPOORT, (FROM WHOM REPRINTS OF THE ORIGINALS ARE AVAILABLE)

PART I — HISTORICAL REVIEW

Onderstepoort Journal of Veterinary Research, Vol. 1, *No.* 1, (*March*, 1962)

SUMMARY

Literally translated "Uitpeuloog" means bulging or protuding eye.

It will be shown that the disease is caused by the invasion of an aberrant host by larvae of one of the *Oestridae*, and therefore the name specific Oculo-vascular Myiasis is suggested for the disease commonly called Uitpeuloog.

As early as 1927 reports were received of a peculiar disease of domestic ruminants and horses from the Kalahari region of South West Africa. The reports were scanty but it appeared that the disease was characterised by exophthalmia which might be unilateral or bilateral. Frequently concurrent affection of the central nervous system was noticed. The occurrence of the disease was quite unpredictable though there appeared to be some correlation with the density of migrating game, such as the blue wildebeest (*Gorgon taurinus taurinus*), Springbuck (*Antidorcas marsupialis marsupialis*) and Gemsbuck (*Oryx gazella*).

When the disease did appear the morbidity and mortality might be so high as to make sheep breeding unprofitable and to result in the decimation of these flocks.

The first reports of Uitpeuloog were received in the Kuruman district of the Republic of South Africa and the eastern Sandveld area of South West Africa. Outbreaks occurred in 1927, 1935, 1937, 1941 and 1958, when the disease was also seen in adjacent areas of Bechuanaland. The disease however appeared fairly frequently between these larger outbreaks.

It was moreover found that when the disease occurred, it was usually encountered during the period August to September when insect life is most prolific and when certain trees are flowering. The disease seemed to appear suddenly, follow a rapid course, and disappear equally suddenly.

The enzootic area may be described as semi-arid, partly duned sand veld, although several severe outbreaks were reported from the limestone regions of the Keetmanshoop district of South West Africa. The average annual rainfall of the enzootic areas is 8 to 10 inches and the vegetation

varies from dominantly grassveld to a well balanced grassy bushveld. For the greater part of the year drinking water is pumped from boreholes and it is around the troughs and drinking places of these watering places that domestic animals congregate.

Sheep, cattle, goats and enquines are all susceptible, but a survey has shown that merino sheep, cattle, horses and goats are more resistant than karakuls, cross-bred karakuls, persians and afrikaner sheep. Sporadic cases have been seen in Springbuck and Steenbuck and farmers in the area insist that cases have occurred in dogs and man. The morbidity appears to vary from 15% to 75%; the mortality is variable but may reach 75% of affected animals.

Various theories concerning the aetiology of the disease were put forward. These varied from metabolic disturbances, poisonous plants, infectious agents to *Oestrus ovis* (Mabin 1936) and *Gedoelstia* (R. du Toit, 1950). Different forms of the disease were also suggested, e.g. the ophthalmic and nervous forms. Until the present series of experiments was undertaken no specific results had been obtained from the various investigations which had been conducted.

No treatment either prophylactic or theurapeutic was found to have any value and no evidence of the development of immunity had been observed.

PART II — EXPERIMENTAL TRANSMISSION

(Awaiting Publication in the Onderstepoort Journal of Veterinary Research)

Summary

It has been shown that thrombo-phlebitis and thrombo-arteritis with prominent eosinophilia are constant lesions pathognomonic for Uitpeuloog. The incidence of these lesions is not correlated with the severity of the eye lesions which previously were regarded as the pathognomonic symptom.

Uitpeuloog was transmitted by the subinoculation of large doses of fresh blood from natural cases in the acute phase of the disease. In one instance the infection was maintained by subinoculation in series for 3 passages when it was lost.

A successful transmission with blood from an apparently healthy wildebeeste is recorded.

In a single experiment brain material was found to be non-infective.

A frequent transmissible intercurrent infection was spirochaetosis due to *S. theileri* which is believed to play no part in the aetiology of Uitpeuloog as a clinical entity. Up to this stage of the investigations the definite aetiological agent had not been identified.

It is believed that nutritional and environmental factors are at most of minor aetiological importance.

Guinea-pigs and mice were refractory to infection.

PART III — SYMPTOMATOLOGY, PATHOLOGY, AETIOLOGY AND EPIZOOTIOLOGY

(Awaiting Publication in the Onderstepoort Journal of Veterinary Research)

SUMMARY

1. Of the domestic animals sheep, cattle, goats and horses are susceptible in that order of frequency and severity.

2. Three main forms of Uitpeuloog are recognised — the ophthalmic, encephalitic and cardiac forms. The symptoms are described.

3. The pathognomonic pathological lesions are thrombo-endophlebitis and thrombo-endarteritis with encephalomalacia.

4. It is shown that the immediate cause of the disease is the invasion of an aberrant host by first stage larvae of *Gedoelstia hassleri*, and *G. cristata* type I and II of the family *Oestridae*.

5. The portal of entry of the larvae is the eye. Two cases of the disease one being the fatal encephalitic form were produced by the supracorneal and conjunctival instillation of larvae dissected from the abdomen of a captured gravid female *G. cristata* type I.

6. Three recognizable types of larvae, types A, B and C, are described as emanating from *G. hassleri* and *G. cristata* type I and type II respectively.

7. The rôle of blue wildebeest and hartebeest in the aetiology is discussed and adult flies were reared from larvae collected from the nasal cavities of these species of game.

8. Attention is directed to the fact that in contradistinction to the susceptible species of domestic animals no macroscopic pathological lesions are produced in game.

9. The epizootiology of the disease is discussed.

10. A suggested pathogenesis in domestic animals and game is outlined.

11. The presence of unidentified species of *Oestridae* in the trachea of game is recorded.

FOOT AND MOUTH DISEASE IN GAME ANIMALS WITH SPECIAL REFERENCE TO THE IMPALA (*AEPYCEROS MELAMPUS*)

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Received for publication, July 1962

SUMMARY

A brief description of foot and mouth disease lesions in the impala in the Kruger National Park is given. The principal lesions are general malaise, vesicle formation on the dorsum of the tongue, the dental pad and the bulb of the heel. Lameness is associated with the foot lesions. A map shewing the routes of infection from the Kruger National Park to the cattle farming areas south and west of the Park is given.

INTRODUCTION

Various authors have, prior to this report, observed foot and mouth disease lesions in herbivorous game animals not only in Africa but also in other parts of the world. This report will give a brief description of some of the symptoms and lesions observed in game animals, particularly the impala (*Aepyceros melampus*), in the Kruger National Park outbreak during 1958–1959.

THE OUTBREAK

The presence of foot and mouth disease in the Kruger National Park was reported on 27 November, 1958. Clinical examination of infected impala on 3 December 1958 showed typical lesions of foot and mouth disease. Vesicular epithelium taken from two of the animals was sent to Pirbright, England. Here the diagnosis was confirmed, the virus type being S.A.T. 3. The disease spread rapidly and infected game and cattle on the southern and western borders of the Park. Typing of material from these animals also revealed the presence of virus type S.A.T. 3. Buffalo, gnu, kudu, sable and impala have been found to be infected. Because of the vast numbers of impala in the Park particular attention was paid to the disease in impala as they acted as indicators of the direction of the spread of disease. Such observations allowed the warning of possible cattle infection to be given long before the cattle actually became infected.

THE PATHS OF INFECTION

If, as has been suggested, the Kruger National Park is enzootically infected with foot and mouth disease, then it was important to learn

the paths of infection that caused foot and mouth disease in the cattle on the borders of the Kruger National Park. It was soon clear that the paths of infection were related to the grazing areas of the game animals. Experience in the Eastern Transvaal had taught the Division of Veterinary Field Services that there are five main routes of infection from game to cattle. These routes can be classified as:—

(1) The Komatipoort or Ten Bosch route.
(2) The Toulon route.
(3) The Gowrie-Jeukpeulhoek complex route.
(4) The Kempiana route.
(5) The Addger-Argyle route.

As a full and detailed discussion of these routes will be described in a later article suffice for the present to say that:—

(1) the Komatipoort route can be regarded as the route of Buffalo infection as this is the area where the greatest concentration of buffaloes is found as the area on both sides of the Komati river can be regarded as their natural grazing ground.
(2) the Toulon route be regarded as the impala route of infection because of the thousands of impala found within the confines of the confluence of the Sand and Sabie rivers.
(3) the Gowrie-Jeukpeulhoek complex because they form part of the habitat and natural grazing grounds of the blue wildebees (*Connochaetes taurinus*) found on these farms and the adjacent portions of the Kruger National Park;
(4) the Kempiana route because of the same reasons as found in the Gowrie-Jeukpeulhoek complex; and
(5) an exactly similar state of affairs in the Addger-Argyle route.

The last three named routes are three separate and distinct entities as these groups of blue wildebees confine themselves to their own grazing grounds. They intermingle only under conditions of extreme stress.

The Course of the Disease

Infected impala shew the following symptoms: general malaise, staring haircoat, tucked up body, rapid loss, of condition, anxious and sometimes haggard facial expression. Such infected impala tend to stand alone in the shade of trees; they show a disinclination to graze; are inclined to lie down and when grazing tend to lag behind the flock. Observations have shown that the disease in impala is associated with great pain. Animals with severe mouth lesions have been seen to feed carefully often sampling the herbage before plucking it. In a few cases "mouth smacking" sampling the herbage before plucking it. In a few cases "mout smacking" as seen in cattle has been observed. In these cases the animal holds its head on high rapidly opening and closing the mouth and at times drawing the upper lip back as if something was irritating the mouth. No salivation has been observed in impala. At the beginning of infection impala show a stiff, stilted gait. It can be described as careful walking on eggs. The movement is slow and painful, the body arched and the head held down. As the disease progresses definite lameness is observed in one or more limbs. This lameness can be so severe that a carrying leg lameness is

seen. This carrying leg lameness is often associated with secondary infection of the foot lesion. Animals with foot lesions often attempt to lick the foot. They show a tendency to shake the leg as if attempting to shake or brush off irritation. They have been observed to continually change the body weight from one foot to another, At times pain may be so severe that the foot is held in the air for short periods. In other instances they have been observed to stamp the foot on the ground.

In the impala foot and mouth disease lesions are found chiefly on the dental pad, the dorsum of the tongue and the bulb of the heel of the digit. Lesions have also been observed on the upper and lower lips, the nostrils, the hard palate and the lower jaw.

The dental pad lesion is perhaps the lesion most frequently seen in the impala. The vesicle very often involves the whole of the pad. In contrast to the tongue dental pad epithelium is hard and more easily handled. For typing of virus it is the epithelium of choice. Intact vesicles are white in colour. The mucous membrane is only slightly raised from the underlying corium. The corium has a raw, red appearance.

Tongue lesions are not seen in all infected animals. Unlike cattle the lesions are found on the dorsum of the tongue. In only a few cases have lesions been observed on the spatulate portion of the tongue. On the dorsum, the lesion may be single, multiple or diffuse. The mucous membrane of the vesicle is raised above the surface of the tongue. It is white in colour and soft and friable. The corium under the mucous membrane of such a vesicle is red in colour. In a diffuse lesion the impression is gained that each individual papilla has formed a vesicle and that these coalesced. Such a diffuse lesion can involve the whole of the anterior portion of the dorsum of the tongue. The lesions occasionally seen on the spatulate portion of the tongue show the mucous membrane white in colour and only slightly raised above the corium. The corium has a pink appearance.

In an infected animal not all the feet or all the digits are affected. In the impala no lesions have been observed in the interdigital space. The common site for vesicle formation is the bulb of the heel. The vesicle can be large or small. On rupture there is a clear cut separation between the skin of the coronary band and the horn of the hoof. Very often this extends right around the coronet. In a few instances this separation has been so severe that the horny hoof has become so detached from the underlying pedal bone tissue that the hoof has been shed. Separation between the horny hoof and the wall of the coronet at the site of vesicle formation leaves a pinkish-white surface. This separation leads to the formation of new horn and the shedding of the old horn. In South Africa this is known as "slipper formation" and in England as "thimbling". This descent of old horn is a valuable aid in ageing the infection. In the impala this descent is very rapid and the "slipper" can be at the point of the toe within six months. Vesicle formation at the bulb of the heel is very susceptible to secondary infection. Such infection can:—

(1) increase the severity of the infection and the descent of old horn thus giving the lesion an older age than it really is; and

(2) can become so severe that the whole of the foot as far as the fetlock joint becomes swollen, shows numerous supparating

abcesses, cause a carrying leg lameness and atrophy of the muscles of the affected limb.

Occasionally the only foot lesion seen is vesicle formation on the anterior sharp edge of the hoof at the junction of horny hoof and coronary band. This vesicle is never more than peasize. The underlying tissue is white in colour.

Observations have shown that in wet or cool weather symptoms appear to be more severe and the disease more virulent and infectious whereas in hot, dry weather the disease seems to be mild, of low infectivity, the symptoms slight and the lesions small.

As in domestic animals in South Africa foot and mouth disease is not fatal to adult impala but does cause mortality in impala lambs.

ACKNOWLEDGEMENT

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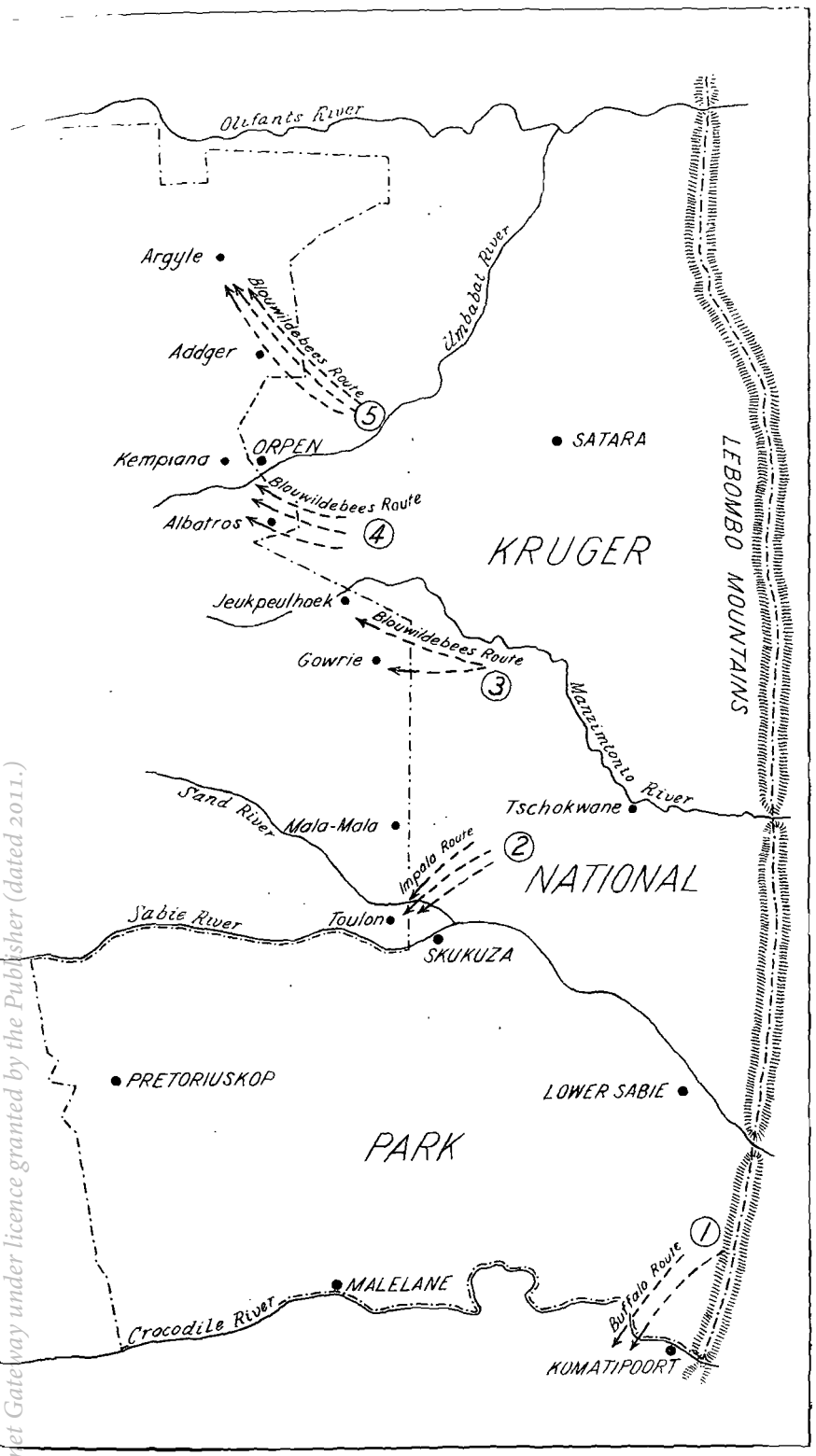

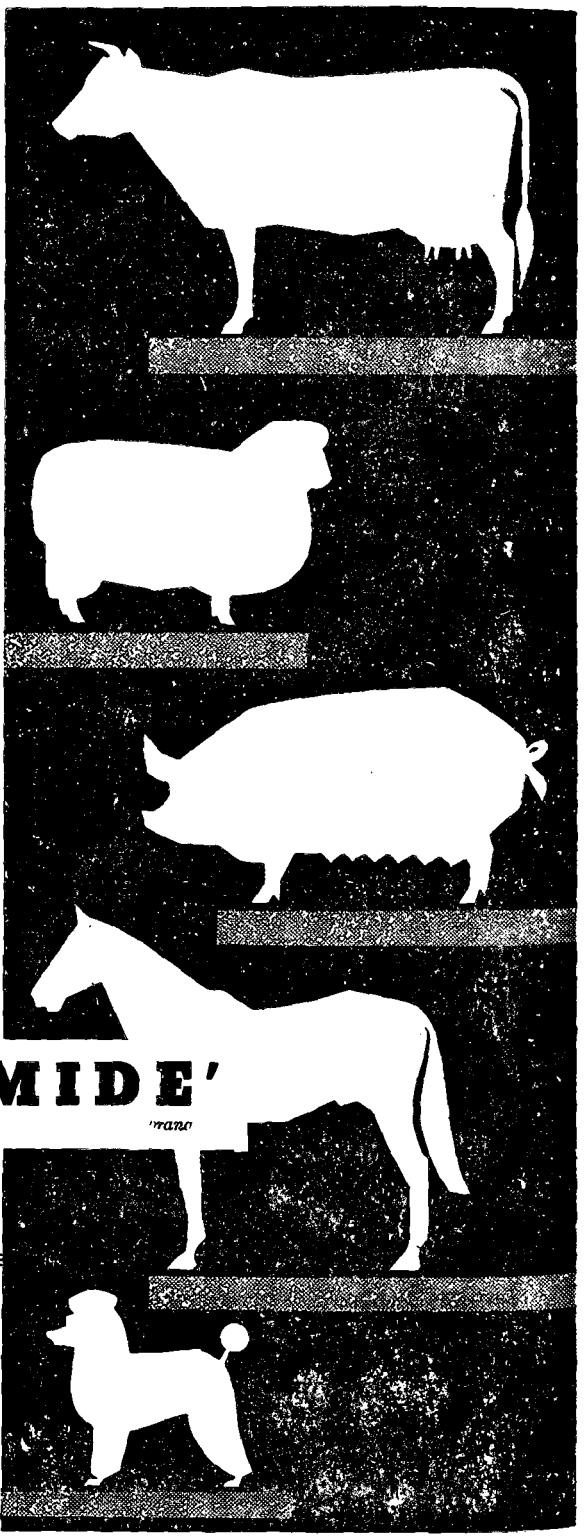

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FLOCK IMMUNISATION OF SHEEP AND GOATS AGAINST HEARTWATER

PART II: PRELIMINARY EXPERIMENTS ON FLOCK IMMUNISATION OF GOATS

J. D. H. POOLE — P.O. Box 7552, Johannesburg

INTRODUCTION

In part I of this paper two economical and safe methods of immunising sheep against heartwater were described[1].

The preliminary investigations reported here were undertaken in an attempt to establish:—

1. Whether similar methods as those described for sheep[1] could be applied to goats.
2. Whether treatment of infected goats before a reaction occurred ("blocking") would be a simpler and equally effective method of immunisation.
3. Which day after infection would be the most suitable for "block" treatment.
4. What dosage of chlortetracycline is necessary for "block" treatments.
5. What percentage of infected goats show a temperature reaction after artificial infection and of these what percentage will develop symptoms and die of heartwater.

METHOD

This experiment was undertaken in the Coerney area in the Zuurberg district of the Eastern Cape Province on a flock subjected to severe incidence of heartwater and in which very extensive losses from the disease had been experienced in the past.

A flock of 223 Cross-bred Angora/Afrikander kids aged 3 to 6 months were infected intravenously with 2.5 cc. of heartwater blood (Ball-3 strain) obtained from the Government Veterinary Laboratories at Grahamstown. The dosage of 2.5 cc. instead of the 5 cc. used for cattle has proved adequate after long usage in this area.

Rectal temperatures of all infected goats were taken daily from the 7th to the 42nd day after infection.

For all treatments chlortetracycline suspension in oil* was used by the intramuscular route.

Coloured and numbered plastic eartags were used for identification of the different groups and of individual goats within the groups. Temperatures were thus individually recorded.

EXPERIMENTAL GROUPS

Group I.—Forty goats were given a "block" dose of chlortetracycline at a dosage of 2 mgm. per pound bodyweight on the 5th day after infection.

Group II.—Forty goats were treated on the 5th day with 4 mgm chlortetracycline per pound bodyweight.

Group III.—Thirty-seven goats were treated on the 6th day with 2 mgm. chlortetracycline per pound bodyweight.

Group IV.—Thirty-five goats were treated on the 6th day with 4 mgm. chlortetracycline per pound bodyweight.

Group V.—Thirty-nine goats were treated on the 11th and 13th days after infection with 2 mgm. chlortetracycline per pound bodyweight regardless of temperature.

Group VI.—Thirty-two goats were left as untreated controls.

Results

Table I

Number of goats in each group reacting on various days after infection

The day on which the first rise in temperature above 104° F was noted was taken as the reaction day.

Day after Infection	Treatment					
	Group I	Group II	Group III	Group IV	Group V	Group VI
	2 mgm./lb. 5th day	4 mgm./lb. 5th day	2 mgm./lb. 6th day	4 mgm./lb. 6th day	2 mgm./lb. 11th and 13th days	Control
7	—	—	—	—	5	14
8	—	—	—	—	4	5
9	—	—	—	—	4	1
10	—	—	—	—	3	4
11	—	—	—	—	12	3
12	—	—	—	—	6	1
13	—	—	—	—	2	—
14	—	—	—	—	—	1
15	—	—	—	—	—	—
16	—	—	—	—	—	—
17	—	—	—	—	—	—
18	1	—	—	—	—	—
19	—	—	—	—	—	—
20	—	—	1	—	—	—
21	—	—	—	—	—	—
22	2	—	—	—	—	—
23	—	—	—	—	—	—
24	—	—	—	—	—	—
25	5	—	2	—	—	—
26	3	—	—	—	—	—
27	1	—	1	—	—	—
28	—	—	3	—	—	—
29	1	—	3	—	—	—
30	1	1	—	—	—	—
31	2	4	1	—	—	—
32	2	5	2	1	—	—
33	1	3	—	6	—	—
34	—	1	2	6	—	—
35	—	1	—	1	—	—
Total Number Reacting	19 (47%)	15 (37.5%)	15 (40.5%)	14 (40%)	36 (92%)	29 (90.6%)

Group I.—As can be seen in Table I, 19 (47%) of the goats in this group showed definite temperature reactions commencing between the 18th and 32nd days after infection.

Of these 19 reactors, 7 goats developed symptoms of heartwater. Five of these were retreated with chlortetracycline and two were not treated but allowed to die in order to confirm the diagnosis. Neitz[2] has pointed out that some temperature reactions after infection with heartwater blood may be due to other conditions. In both the animals which died typical rickettsial colonies were demonstrated in brain smears.

Group II.—Fifteen (37.5%) of the goats in this group reacted between the 30th and 35th days after infection. Of the 15 reactors, 12 required retreatment but here again 2 were left untreated and died, the diagnosis of heartwater being confirmed by means of brain smears.

Group III.—Fifteen (40.5%) of the goats in this group reacted between the 20th and 34th day after infection. Of the 15 reactors 8 required retreatment but 2 were left untreated and died.. The diagnosis of heartwater was again confirmed by means of brain smears.

Group IV.—Fourteen (40%) of these goats reacted between the 32nd and 35th days after infection. None of these reactors developed symptoms of heartwater and no retreatments were necessary.

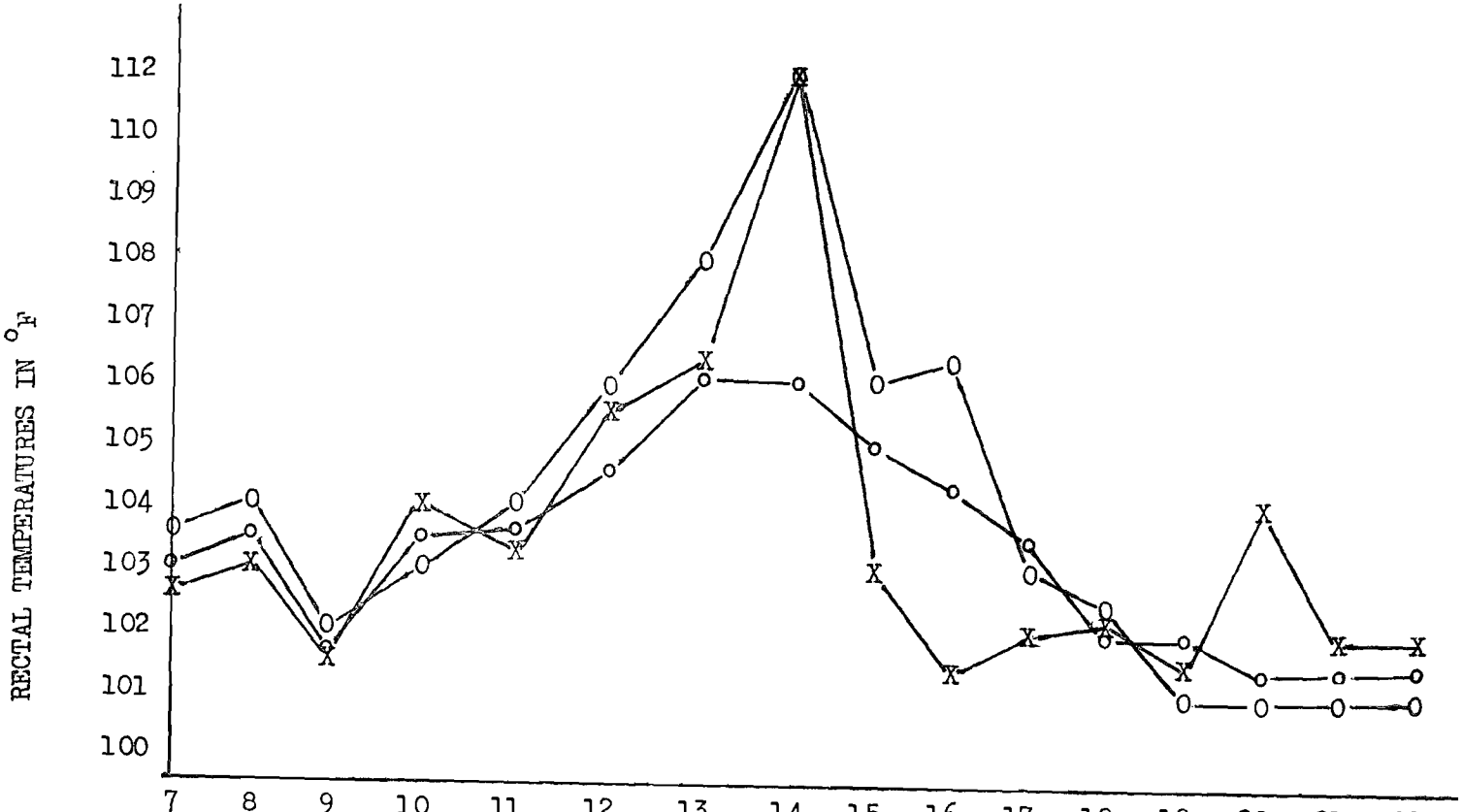
Group V.—The method used in this group was similar to that which has proved very successful in sheep[1] except for the fact that a slightly later treatment schedule was applied in goats (i.e. 11th and 13th days rather than 10th and 12th days) because previous experience has shown that goats react slightly later than sheep.

As can be seen from Table I, 92% of these goats reacted between the 7th and 13th days after infection. No goats in this group developed symptoms of heartwater and no retreatments were necessary.

It must be remembered that these were crossbred Angora/Afrikander goats, because subsequent experience on large numbers of pure-bred Angoras has shown that in these animals a third treatment may sometimes be necessary on the 14th or 15th day after infection. In the case of Afrikander goats subsequent experience has shown that the two treatments with chlortetracycline on the 11th and 13th days after infection are completely safe and effective.

Because of the success of this group, another flock of 3–6 month old crossbred goats were immunised in the same manner except that no temperatures were taken, the animals being handled only three times. No mortality due to heartwater was experienced during the immunisation process.

Group VI.—The 32 goats in this group were left as controls and given no treatment. Twenty-nine (90.6%) of them had temperature reactions between the 7th and 14th days after infection and 12 of the reactors (i.e. 37.5% of the total in the group) developed typical heartwater symptoms. The 12 goats which developed symptoms were used for a drug trial during which four of them died and the diagnosis was confirmed in each case by means of brain smears.



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An interesting fact is that 17 goats in this group showed definite temperature reactions but recovered without any treatment at all. Amongst those which recovered naturally, 4 goats developed extremely high temperatures. The temperature charts of 2 of these are reproduced below for the sake of interest. The peak temperatures of 112°F were estimations because in each case the ordinary clinical thermometers broke in the rectum due to the extremely high temperature. This phenomenon was repeated with two thermometers in each case. The average temperatures of other reactors in the group are given for purposes of comparison.

O—O Goat No. Red 36
X—X Goat No. Red 20
0—0 Average of Remainder

Discussion and Conclusions

Effects of "Block" Treatments

It is obvious from Table I that all "block" treatments applied had the effect of (*a*) prolonging the incubation period; and (*b*) reducing the number of reactors.

In Group IV which received 4 mgm chlortetracycline per pound bodyweight on the 6th day after infection, reactions only commenced 32 days after infection and only 40% of the goats reacted at all. More important, however, is the fact that none of the goats in this group developed symptoms. This method of "blocking" on the sixth day may prove a useful and easy way of immunisation. The advantage of such a method lies in the fact that animals need only be handled twice although the cost of the treatment material remains the same as in Group V which was treated at the time of reaction.

Groups I, II and III can be regarded as complete failures, due to the fact that retreatments were necessary in some goats in each of these groups. Since the idea was to try and simplify the immunisation process by completely "blocking" out all reactions severe enough to warrant retreatment Group IV was the only successful group.

Subsequent Immunity

This experiment was carried out in November just prior to the heartwater season. During the subsequent two months a very severe outbreak of heartwater was experienced on this farm. A total of 34 of the experimental goats died of what the experienced owner put down to heartwater. Unfortunately the writer could not be present on the farm to confirm all the post mortem diagnoses but four of them were confirmed by means of brain smears.

The deaths from this natural outbreak of heartwater were almost equally distributed amongst goats in Groups I, II and III. No deaths attributable to heartwater were experienced in Group V and only one unconfirmed death occurred in Group IV.

The indications are that for some unknown reason the "blocking" methods employed in Groups I, II and III did not confer a very good immunity and it is the writer's intention to do a much larger scale trial using the method used in Group IV to see whether a good immunity is in fact obtained by this method.

No mortality from heartwater has occurred in the flock subsequently immunised using the same process as in Group V, i.e. treatment at the time of reaction. This method of immunisation is therefore the only one which can at present be recommended as safe, easy and reliable.

ACKNOWLEDGEMENTS

The writer wishes to acknowledge with thanks, the unstinting co-operation of Mr. Bert Hayter, the owner of the goats used in this experiment.

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SKIN LESIONS IN SOUTH AFRICAN DOMESTIC ANIMALS WITH SPECIAL REFERENCE TO THE INCIDENCE AND PROGNOSIS OF VARIOUS SKIN TUMOURS

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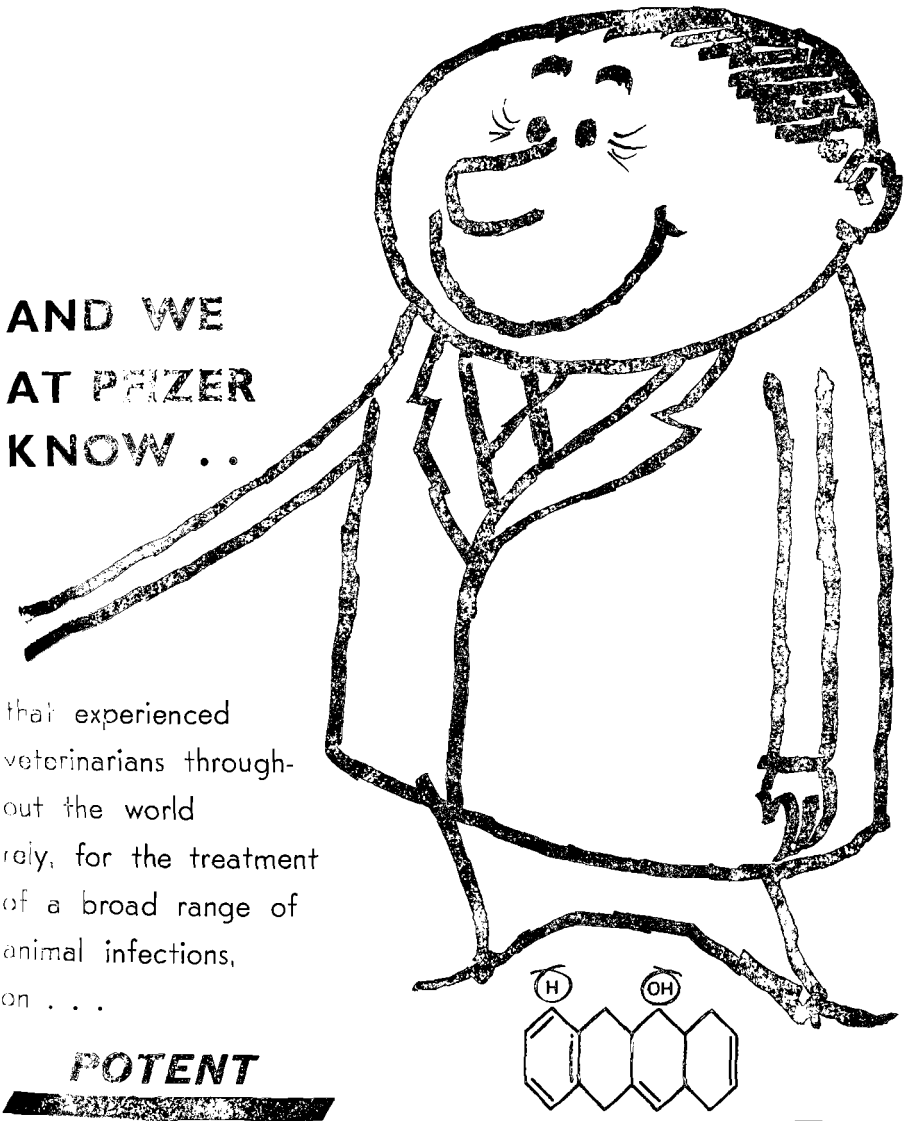
(Received for publication, July 1962)

SUMMARY

A brief review of the South African literature on recorded skin tumours is given. The differential diagnosis, incidence, most common sites and the clinical behaviour of skin tumours are discussed. A Table giving the incidence of skin tumours occurring in the various species of domestic animals during the last 22 months is included. Attention is drawn to the high incidence of tumours affecting the eyelid. The value of biopsies as an aid to the correct diagnosis and treatment of skin lesions is emphasized. Clinicians are encouraged to make use of biopsies in order to render a more satisfactory service to their clients.

Skin lesions are very common in this country. A wide range of aetiological factors may be responsible for the lesions. Contrary to the divergence of aetiological factors the gross appearance of the skin may be very similar in a number of unrelated entities and great difficulties may be experienced in reaching a correct diagnosis. On the other hand a correct diagnosis is essential for specific treatment and to furnish an informed prognosis. To improve diagnostic accuracy certain routine examinations are necessary. Smears, prepared from skin scrapings offer a very useful and simple method of arriving at a diagnosis and it is a rapid method which lends itself to use even under field conditions. It can be very useful especially for the diagnosis of some forms of ecto-parasitism, such as sarcoptic and demodectic mange, as also with certain dermatomycoses. Lumpy wool and Senkobo disease are good examples of this group. Most fungal infections are relatively superficial. All that is necessary is to remove the dry scab and to prepare a smear from the raw surface of the skin or from the bottom of the crust. When heat fixed and stained with Giemsa or Gram the typical double or multiple spore forming mycelia of the fungus may be recognised with ease in such preparations. However, with some of the deeper penetrating parasites like besnoitia, it may be simpler and more reliable to examine biopsies of the affected skin. This method is more time consuming but allows greater accuracy while also enabling one to exclude or diagnose other similar skin lesions resulting in a crustose dermatitis.

Nutritional distrubances or deficiencies exert a marked influence on the appearance of the skin. Good examples are Cu. deficiency leading to steely wool, nicotinic acid deficiency resulting in palagra and selenium poisoning. In all these cases biopsies may aid the diagnosis.



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Hormonal disturbances or endocrine dysbalance may produce characteristic alopecia, symetrical loss of hair and atrophy of epidermis being associated with this type of disturbance. The best known examples of this group are Sertoli cell tumours of the testicle, canine Cushings syndrome and hyperthyroidism, histological examinations of skin biopsies may be very useful in arriving at an accurate diagnosis and prescribing the correct treatment. This is especially applicable to the stubborn skin dermatoses occurring in old castrated or ovarectomised dogs. The absence of any recognisable aetiological cause and the typical atrophic appearance of the skin may suggest a hormonal deficiency and supplementation of sex hormones may lead to dramatic improvement of the hair coat.

Focal areas of skin thickening and the development of skin nodules are common findings. These lesions may be loosely classified into three groups: non neoplastic pathological hyperplasia, granulomas and neoplastic growths.

Acanthosis

Benign hyperplasias of the skin confused with neoplasms include acanthosis. This is a thickening of the epidermis as a results of hyperplasia of the Malpighian layer. It is regarded as a normal conpensatory attempt of the body to counter act a mild, usually chronic irritation. This may occur with or without accompanying hyperkeratosis or thickening of the keratin layer (stratum corneum) and usually the granular layer. When this thickening reaches fair preportions it may be regarded as a neoplastic growth, and is usually referred to as cornu-cutaneum. Histological examination is usually necessary for accurate diagnosis.

Epidermoid Cysts

Other non neoplastic skin lesions that may lead to confusion are epidermoid and dermoid cysts. They are usually firmly attached within the dermis and appear as small nodules which slowly increase in size. The overlying skin generally remains covered with hair until the nodule attains a large size. Incision of such nodules reveals one or more thin walled spherical cysts and gray, grumous, somewhat desiccated content. Surgical removal is indicated. On microscopical examination the cyst wall consists of flattend squamous epithelium surrounded by a collagenous capsule. The cyst contains concentrically and irregularly laminated masses of keratin, mixed with amorphous tissue debris. Sometimes the wall ruptures with a severe resultant foreign body inflammatory reaction. The origin appears to be an occlusion of the mouth of hair follicles. The dermoid cyst differs from the epidermoid cyst by the presence of skin adnexa in the wall. Fragments of hair may therefore be present in the cyst.

Acanthosis Nigricans

Acanthosis nigricans is a dermatosis of obscure aetiology and is recognised in man and the dog. Its principal clinical features in the dog are the presence of symmetrical patches of heavily pigmented, roughened,

thickened skin, particularly in the flanks, axillae, inguinal, circumanal and abdominal regions. The lesions may, in the advanced state, produce a disagreeable odour. Microscopically the lesion is dominated by acanthosis and hyperheratosis of the epidermis. Increased amount of melanin, suspected from the gross appearance, is not always a prominent feature.

Keloid

Keloid is a lesion sometimes seen in animals. It is really an excessive scar formation and is seen especially in cattle belonging to natives as a result of certain types of injury. Histologically, the keloid is made up of heavy bands of eosinophilic collagen.

Calcinosis Circumscripta

Calcinosis circumscripta is another unusual lesion seen in man and the dog. It consists of circumscribed masses of calcium salts in the dermis. They have a chalky-white granular focal appearance and a gritty consistency. Microscopically they consist of spherical masses of granules of calcium salts. These masses are surrounded by large numbers of giant cells. The aetiology of this condition is obscure but it may be some interference with the lymph flow in the dermis. Surgical removal is indicated.

Infectious Granulomas

Infecious granulomas are another group of lesions causing difficulty in the differential diagnosis of skin lesions. They may be the result of wound infections or chronic irritations. Well known examples of this group include: capped elbow, proud flesh and the granulomas caused by habronema larvae. Actinomycosis, actinobacillosis and nocardiosis are also often incorrectly diagnosed and confused with neoplasms. In all these cases an accurate diagnosis is only possible with histological examination of skin biopsy material.

Neoplasms

Neoplasms affecting the skin are, contrary to general belief extremely common amongst our domestic animals, particularly those which are permitted to attain old age. This field has been greatly neglected in South Africa and only a few of the rather extensive series of neoplasms affecting the skin have been recorded in this country. Jackson records the following skin tumours in mammals: acanthoma, haemangioma, lymphangioma, haemangioendothelioma, sarcoid, basalcell epithelioma (carcinoma), cornu-cutaneum, fibroma, papilloma, melanoma, adenoma of sebaceous gland and perianal adenoma. In a recent publication de Kock describes the following skin tumours: perianal tumours, basal cell epithelioma (carcinoma), squamous cell epthelioma (carcinoma), reticulum cell sarcoma, neurogenic sarcoma, haemangioma and transmissible venereal tumours. Schulz has described a case of multiple acanthosis in the pig.

In the present series obtained during the last 22 months a remarkable variety of skin tumours have been diagnosed. It embraces almost the complete list of known skin tumours. This is an immediate result of closer

co-operation between the clinical departments and the department of pathology and a growing realisation of the value of biopsies.

In Table I the skin tumours are tabulated. Tumours affecting the eyelids are tabulated in Table II. In the majority of eyelid tumours the third eyelid or membrana nictitans was the structure involved.

Papillomatosis

Papillomatosis is a benign neoplastic growth occurring in almost all domestic animals. These warts consist of proliferations of epithelial cells supported by a stroma of connective tissue. They are all considered to be of a contagious nature and caused by a virus. In some species these warts are more precise lesions that refuse to grow anywhere but in a selected type of epthelium, for instance in the mouth of the dog. In others massively roughly keratinized warts indiscretely involve large areas of the skin.

In bovines cutaneous papillomatosis is more frequent than in any other animal. It is more common and severe in young animals, only partial immunity to reinfection develops, and neutralizing antibodies are not demonstrable in convalescent sera. The disease is generally self-limiting and recovery without treatment is common.

Papillomatosis is most frequent on the neck, chin, shoulder and dewlap. The typical wart appears as a rough, cauliflower-like mass of varying size and irregular shape, elevated above the skin surface and often attached by a stalk.

Fibropapillomatosis is another variety of this complex which occur on the genitalia of bovines. This condition is also considered to be of an infectious nature but successful artificial transmission has not yet been achieved. The fibromata also consist of epithelial and fibrous elements the latter being generally more prominent. They may recur after surgical excision but do not metastasize and are usually self-limiting.

In equines the counterpart of this lesion is known as sarcoids. The equine lesion is found most frequently on the skin of the head but can occur anywhere on the skin. They usually present a broad base and are not keratinized. They tend to become eroded. Complete removal is indicated; incomplete removal may lead to recurrence and deep lesions of a much more malignant appearance, which may offer problems in the histological diagnosis.

Canine oral papillomatosis is well known in young dogs. The condition is considered to be contagious with a duration of about 3 to 4 months. The lesions are generally multiple and may occur anywhere on the oral mucosa but do not extend below the epiglottis or into the oesophagus. Cutaneous papillomatosis occurs in dogs but apparently is not caused by the same virus that induces the oral lesions.

Papillomatosis is very well known in rabbits and are often referred to as Shope papillomas, because it was from these tumours that Shope originally isolated a virus as proof of the infective nature of papillomatosis. The virus probably attacks the epithelium at many neighbouring points in an area, inducing multiple papillomas that subsequently coalesce into a single mass. The growth does not enter into the dermis; therefore

proliferation extends outwards. The tumours are similar in wild and domestic rabbits, but larger in the latter. The papillomas are black or gray, and the upper surface is dry, well keratinized, and fissured. In some cases it developes into cutaneous horns. The papilloma of rabbits may undergo a carcinomatous change and subsequent invasion may occur. Oral papillomatosis also occurs in rabbits.

Papillomatosis of goats may occur at two sites — skin and udder. In the udder the lesion resembles cutaneous horns which may reach a length of 3 cm. These generally assume a rod-like shape but may develop into massive, discoid tumours with a tendency to develop into squamous cell carcinomas.

Melanoma

Melanomas are uncommon in domestic animals other than the dog, horse and Angora goats. They are also reported in the ox, pig, sheep and cat.

In dogs the tumour arises from melanoblasts in most areas of the body and possibly from the chromatophores of the iris. The benign form or benign melanoma (naevis, mole) must be distinguished from the malignant melanoma or melanosarcoma. Melanoma is not to be confused with melanosis, a non-neoplastic form of melanin pigmentation found in the skin, meninges, interna of the cardio vascular system, and elsewhere in the body. Melanosis appears as an unrelated black focal area, with the pigment in melanophores rather than in melanoblasts. Melanosis often disappears with age.

In the older literature melanoma in dogs were regarded as rare, but they are now considered fairly common, especially malignant melanomas. The incidence is highest in dogs over 7 years. Cocker spaniels, Scottish Terriers, Boston Terriers and Airedales are supposedly more susceptible. Contrary to what one would expect melanomas have not been reported in the Dalmatian.

They are more common in the mouth, particularly in the lips around the mucosal surface, gums, hard and soft palate and tongue. It is the most common malignant tumour in the mouth of the dog and the prognosis is always poor. The next most common site is the skin. The tumours are usually well defined, not encapsulated, firm or spongy and the colour may vary from almost black to almost without pigment. This is especially true of very malignant and undifferentiated melanomas. Metastasis usually occurs to the regional lymph node.

Melanomas in the horse occur in 80% of gray horses but are rare in horses of other colours. The incidence increase with age. The skin is usually involved and the most common sites are the perineum, lateral to the anus, and the inside of the root of the tail, also at the base of the ear, eyelids, scrotum, udder, sheath and limbs. The tumours are nodular, flattened or elevated. They are usually firm and there is a tendency for ulceration to occur. Most melanomas in horses are benign but malignancy may occur with metastasis to the regional lymph node.

In goats especially Angora and Swiss milch goats melanomas are an important tumour. They comprise 40% of all caprine neoplasms, white

goats being more likely to develop these tumours. In the goat melanomas show a marked tendency to metastasis and generally present a malignant histological appearance.

In other species of domestic animals melanomas are of no significance.

Mastocytoma

In general the dog is by far the most susceptible animal to skin tumours. The most common tumour of the dog's skin is mastocytoma, forming about 20% of all skin tumours found in this species. Mastocytoma is found mostly in the dermis of the skin. It is usually a solitary growth, but may be multiple. More than 30% of these tumours are found in the hindquarters of the dog (gluteal region, perineum, legs and feet). On the legs they are found principally in the lateral regions of the thigh and stifle joint while about 15% develop from the skin of the external genitalia. The tumours are usually round or oval, a few being pedunculated. They are poorly delineated from the surrounding tissues. Their consistency is ordinarily hard, though some are soft and resemble oedematous swellings. The cut surface of the tumour is lobulated, and characteristically light orange, pink or greyish white. The tumour consist of mastocytes, round or avoid cells with well-outlined cytoplasmic borders and centrally placed round nuclei. The cytoplasm contains darkly stained granules especially in slow growing tumours. Impression smears of the tumour will show these up with advantage. As a matter of fact mastocytoma is the only tumour which can be diagnosised with safety from such an impression smear. Eosionophiles are characteristic components of these tumours. The tumours are generally benign, but some 30% of cases, are malignant with a tendency to metastasize. Surgical removal is indicated but post-operative recurrence may be expected in about 20% of cases. This tumour is also occasionally noted in the cat.

Basal Cell Tumours

Another fairly frequent tumour found in the skin of the dog and cat is the basal cell tumour. These tumours arise from the basilar cells of the epidermis, cells of hair follicles and basilar cells of sebaceous glands. They resemble carcinomas but rarely metastasize and must be considered benign tumours. The most common site is the head and particularly the commisures of the mouth, skin around the eyes, concha of the ear, cheek and jaw. Another favourable site is between the claws. They are firm to rubbery in consistence, grayish-white in colour and often encapsulated and lobulated tumours. Surgical excision is indicated, although local recurrence occurs in a small percentage of cases. They generally respond very favourably to röntgen-ray treatment.

Haemangiopericytoma

Haemangiopericytoma, a newly recognised neoplasm of domestic animals almost peculiar to the dog, is another common skin tumour, occurring most frequent in the subcutis of the trunk and extremities and arise from the pericytes supporting the blood vessel walls. They are

generally encapsulated and not intimately adherent to the overlying skin or to the deeper tissues. They grow slowly, often requiring as long as six years to reach an appreciable size. They do not metastasize but in a large percentage of cases recur after surgical removal. Surgical removal should, therefore, be absolutely complete and a portion of normal surrounding skin requires to be sacrificed. This may not always be possible, especially on the limbs.

Perianal Gland Tumours

Perianal gland tumours are peculiar to the dog, and are among the most common tumours found in this species. Adenomas are much more frequent than carcinomas. They usually occur in dogs over eight years and the incidence is significantly higher in entire males. They occur as solitary or multiple tumours above, below or lateral to the anus. They are characteristically multi-nodular, well circumscribed and firm or rubbery. On cross section they appear as white or tan masses. The overlying skin is usually ulcerated. They usually respond very well to surgery, castration also being indicated.

Contageous Venereal Tumours

This tumour, first described in 1820, has long been prevalent in many countries. It is a common tumour in South African dogs, affecting usually the genitalia of both sexes. In a small number of cases these tumours may occur in an extragenital site of the skin. However, on histological grounds they cannot be distinguished from those occurring in the genital tract. It is one of the best known transmissible tumours.

This tumour is often cauliflower-like in shape but it may also be pedunculated, nodular or papillary. The neoplasms is firm and grayish red. The superficial portion is commonly ulcerated. Microscopically the tumour cells usually appear in solid sheets with only a delicate net work of fibrous tissue as a supporting stroma. The tumour consists of large round cells, which bear some resemblence to lympo-cytes. However, their nuclei, rounder or slightly indented stain more strongly than those of lymphocytes. Mitotic figures are numerous and give an impression of malignancy. Contrary to this impression however, the tumour rarely shows metastasis and recurrence of the surgical removal is unusual.

Malignant Lymphoma

Malignant lymphoma is one of the most common tumours affecting our domestic animals. The primary site is usually the lymphoid tissue of the spleen or lymph nodes, but in some few cases a primary lesion may occur in the skin. The tumour appears in the skin as a yellowish-gray raised nodule, not too clearly demarcated and intimately associated with the dermis. Microscopically it consists of dense aggregations of uniform immature lymphocytes. This tumour must always be regarded as potentially malignant and a guarded prognosis given.

Acanthoma

Under this heading is classified a group of epithelial tumours which are characterized by prickle cells and, often also by pearls or concentric keratinisations. The histological appearance closely resemble squamous cell carcinomas but the cells are usually less anaplastic. Clinically they also differ from their carcinoma counter part in that metastasis is but seldom seen. However, local infiltration may occur and surgical removal must be radical. This is by far the most common tumour affecting the eyelid in our domestic animals; the membrana nictitans being the common site. The ox and horse are most commonly affected.

Sebaceous Adenoma

Adenomas of the sebaceous gland are relatively common benign tumours found in the skin of dogs. They closely simulate normal sebaceous glands, often there is little but its size to distinguish it from a normal gland. They are generally small in size, 2 to 10 mm. in diameter, but some may grow even larger. They are usually slow growing but as a result of the secretory activities of the glandular cells they may rapidly increase in size when the excretion is unable to escape. The lesions are discrete, non-encapsulated, multi-lobulated, firm, grayish-white nodules. This multi-lobular appearance of the tumour is an important diagnostic feature. Microscopically they consist of packets of sebaceous cells separated by thin collagenous septa. Because of their lipid content they are vacuolated towards the centre of the lobule. They are usually completely benign but surgical removal is indicated from a cosmetic viewpoint.

Haemangioma

Haemangioma is a benign tumour of endothelial cells. The tumour consists of endothelium-lined blood spaces, filled with normal circulating blood. These tumours are benign and surgical removal is indicated with a good prognosis.

Fibroma

Fibroma is a benign tumour of connective tissue sometimes occurring in the skin. It is not of great significance as a skin tumour.

Lipoma

Lipomas often occur in the skin, usually in the subcutes, as soft yellowish-gray discrete masses. They are completely benign but may attain a fair size, and surgical removal is therefore indicated.

Hair Matrixoma

This skin tumour consists of a stroma in which lie numerous gland-like structures resembling hair follicles, but often with a cystic lumen or with an epithelial wall of excessive thickness. It often presents a lobulated appearance and has a tendency to calcification. Epithelial cells which

stain poorly but which maintain their outline (shadow or ghost cells), usually lying towards the centre of the lobule, are very characteristic and diagnostic feature of these tumours. The absence of any hairs serve to differentiate this structure from a dermoid cyst. It is a benign tumour but because of its size surgical removal is indicated.

Squamous Cell Carcinoma

Squamous cell carcinoma is a common malignant neoplasm affecting all domestic mammals but most commonly the dog. In the dog the skin of the trunk, legs, digits, scrotum and lips is particularly involved. The penis, base of the tail, vulva and perineal area are commonly involved in the ox and horse. Carcinomas of the top of the ear and the eyelid are sometimes observed in the sheep. The head and neck are predilection sites in cats.

Squamous cell carcinoma is often papillary with a cauliflower-like appearance. The tumour usually has a base broader than that of a papilloma. Carcinoma is subject to trauma and commonly displays ulceration and bleeding. They infiltrate widely and usually metastasize especially to the regional lymph nodes and lungs. The prognoses is poor.

Haemangiosarcoma

Haemangiosarcoma is a malignant tumour of endothelial cells. It occurs mostly in dogs, but is also found in other domestic animals. Particularly common sites are the skin and spleen, especially in the dog. It is poorly circumscribed, non-encapsulated, rubbery or spongy in consistency, and grayish-red in colour with dark red areas. Haemorrhages are a common complication. It is a highly malignant neoplasm, readily metastasing and often recurring after surgical removal. Metastatic nodules are regularly found in the lungs, regional lymph node, liver, peritoneum, heart and other internal organs.

Fibrosarcoma Neurofibrosarcoma and Liposarcoma

Fibrosarcoma neurofibrosarcoma and liposarcoma are merely the malignant counter parts of the benign tumours already described. They do not frequently occur in the skin, because of their malignant tendencies the prognosis should always be guarded.

Undifferentiated Carcinoma

This was a very malignant tumour with very anaplastic cells. It was therefore not possible to classify it.

Sebaceous Gland Adenocarcinoma

This is the malignant sebaceous gland tumour and it is not an uncommon skin tumour, particularly in the dog. It may attain a fair size, and there is a strong tendency for ulceration to occur. Very often a severe inflammatory reaction may be present due to the irritant effect of the sebaceous secretions. It is not always fully realized that lipids, even

endogenous lipids, have a very severe irritant effect on tissues and they may be responsible for a marked granulomatous reaction. These tumours do not usually metastasize but may recur following surgical removal. Surgery must therefore be of a radical nature.

Discussion

The number and variety of neoplasms diagnosed during a relatively short period of 22 months is ample proof of the frequent occurrence of these lesions in this country. These findings are even more significant when it is realised that no special efforts have been made to collect such specimens. This therefore serves to indicate the important role this aspect of Veterinary science may play in private practice. Particularly in urban areas, where large numbers of pets are kept and often allowed to reach old age, skin neoplasms may form an appreciable percentage of the practitioners daily work. A correct diagnosis of these lesions will lead to more accurate prognosis and will therefore play a part in improving client-practitioner relationships.

The gross appearance of these lesions is unfortunately often very similar. Nevertheless, with a certain amount of interest and correlation of observation the practitioner may become reasonably skilled at making a tentative diagnosis from the gross appearance. A knowledge of the most common sites, the incidence and the general behaviour of these growths will help him to reach an accurate diagnosis in a surprizingly large number of cases. However, this is only possible if the gross observations are correlated with the histopathological findings.

In order to obtain more accurate histopathological diagnosis, it is necessary to submit certain information with the biopsy material. It is essential to know the breed, sex and age of the animal as also the site, size and gross appearance of the lesion. To illustrate this point it may be mentioned that without this information it may be virtually impossible for the pathologist to differentiate between extragenital venereal tumours and malignant iymphomas of the skin. The age of the animal is of a vital importance here, because the former occur usually only in young animals while the latter are seen in older animals.

In performing a biopsy certain precautions should be taken. Usually a wedge-shaped piece of the tumour tissue is removed and where the angle of incision is too acute the biopsy will be too shallow and an essential portion of the tumour may not be included. Two parallel incisions, extending through the depth of the lesion will give much better results. It is also important to cut the biopsy tissue from the edge of the lesion, where active proliferation is occurring and not from the centre where only neorosis may be evident. This is especially important to the pathologist when he has to decide on the malignancy of the tumour. If possible, especially where material is collected at post-mortem examination, more than one section should be included, since one section may reveal changes providing a clue to the diagnosis which are absent from the others.

TABLE I

Tumours of the Skin

	Dog	Cat	Sheep	Ox	Horse	Goat	Pig	Monkey	Total
Hyperkeratosis	—	—	1	—	—	—	—	—	1
Epidermoid cyst	3	—	—	—	1	—	—	1	5
Acanthosis nigricans	1	—	—	—	—	—	—	—	1
Calcinosis circumsripta	3	—	—	—	—	—	—	—	3
Papilloma (saroid)	1	—	—	1	10	—	1	—	13
Melanoma	1	—	—	1	3	2	—	—	7
Mastocytoma	15	1	—	—	—	—	—	—	16
Basal cell carcinoma	7	1	—	—	1	—	—	—	9
Harmangiopericytoma	2	—	—	—	—	—	—	—	2
Perianal gland Adenoma	3	—	—	—	—	—	—	—	3
Contageous Venereal Tumours	1	—	—	—	—	—	—	—	1
lymphoma	1	—	—	—	—	—	—	—	1
Acanthoma	—	1	8	1	—	—	2	—	12
Sebaceous gland Adenoma	3	—	—	—	—	—	—	—	3
Haemangioma	2	—	—	1	—	—	—	—	3
Fibroma	1	—	—	—	—	—	—	—	1
Lipoma	2	—	—	—	—	—	—	—	2
Hair matrixoma	2	—	—	—	—	—	—	—	2
Squamous cell carcinoma	4	—	2	1	—	—	—	—	7
Haemangiosarcoma	2	—	—	—	1	—	—	—	3
Fibro sarcoma	—	—	1	—	—	—	—	—	1
Neurofibrosarcoma	1	—	—	—	—	—	—	—	1
Liposarcoma	—	—	—	—	1	—	—	—	1
Undif. carcinoma	—	1	—	—	—	—	—	—	1
Sebaceous adenocarcinoma	3	—	—	1	—	—	—	—	4
	58	4	12	6	17	2	3	1	103

TABLE II

Tumours of the Eyelid

	Dog	Sheep	Ox	Horse	Total
Acanthoma	—	1	6	4	11
Squamous cell carcinoma	—	1	1	—	2
Adenoma of Tarsal gland	—	—	1	—	1
Basal cell carcinoma	1	—	—	—	1
Haemangiosarcoma	1	—	—	—	1
TOTAL	2	2	8	4	16

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THE HEALTH OF THE WEANLING PIG

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INTRODUCTION

Two features of pork production in the Republic are of interest as exerting a decided influence on swine health and therefore, on production costs. Firstly, there are relatively few specialist pig farmers in the country, the bulk of production emanating from a large number of small producers with a few sows each as a farming sideline. Many of these people are merely temporary pigkeepers, and without proper housing facilities or any great knowledge of swine husbandry. It is this comparative ignorance which results in many animals being raised under unsuitable and uneconomic conditions. The second feature is the long-established conception of the pig as a consumer of waste and by-products such as mine, hotel and hospital swill, sweet potatoes, spoilt grains, skim milk and bakery, cannery and hatchery wastes. The incorrect use of such products results in the feeding of unbalanced inadequate rations and subsequent poor growth or frank deficiency disease, particularly among growing pigs.

These two features combine to produce among young pigs a significant amount of unthriftiness and ill-health directly attributable to unsound husbandry practices rather than to the prevalence of serious swine disease in the country. This situation is happily susceptible to rapid and far-reaching improvement by a veterinary profession able to evaluate the aetiological rôle of the husbandry shortcomings in disease outbreaks as part of its health promotion activities. The remainder of this paper is devoted to a brief consideration of some major environmental stress factors in relation to the health of the newly-weaned pig.

GENERAL CONSIDERATIONS

The excellent feed conversion efficiency of young pigs from weaning to porker weight is an important commercial asset, and growth setbacks during this period seriously endanger the profitability of pig raising. Careful management will, therefore, seek to protect the young pig from disease at weaning, particularly since the animal itself is immunologically rather vulnerable at this age.

Weaning weight offers a clinical assessment of future vigour and growth potential as well as an index of preweaning husbandry efficiency. While all weaners at 6 to 8 weeks of age suffer from some degree of anaemia[1] and immunological immaturity as a result of a relative hypogammaglobulinaemia[2], these inadequacies are especially prominent in the lighter pig weighing less than 25 pounds.

In large weaner groups the lightweight is prone to be bullied away from the feed trough and to suffer consequent underfeeding and reduced growth. Thus is a serious vicious cycle set in motion unless undersized animals are sorted into special groups at weaning. There appear to be three main causes for unsatisfactory weaning weights, viz. iron-deficiency anaemia, persistent gastro-intestinal upset as a result of poor housing[3] or lack of creep feeding.

Environmental Stress

Adverse environmental conditions or states of stress elicit accelerated adrenal cortico-steroid production in animals subjected to such conditions. An increased breakdown of tissue proteins results, together with reduced lymphocytosis and depressed gammaglobulin synthesis. The clinician will observe these changes as a failure to maintain a normal growth rate and an increased susceptibility to disease. There is also an increased requirement for minerals and B-group vitamins under stress conditions.

The first step towards avoiding the above undesirable and uneconomic sequelae lies in ensuring a trouble-free weaning period. Worming and castration procedures must not accompany weaning, dietary changes should be minimised at this time and a familiar environment retained where possible by the removal of the sow and not her litter to new quarters. The pathogenic implications of four stress factors commonly encountered in this country, viz. overcrowding, poor sanitation, parasitism and inadequate nutrition are discussed further below.

Overcrowding

The growing-fattening pig requires some 3 to 5 square feet of floor space, according to size, in addition to three square feet of dunging area. Where restricted feeding is employed, some 9 to 12 inches of trough length is required per pig. An adequate water supply is a valuable digestive aid where dry feeding is practised. The prophylactic value of bars on feed and water troughs as a means of reducing faecal contamination cannot be overemphasised, particularly under crowded conditions.

It is evident that the larger the weaner group the greater the stress states generated may become as the problems of hygiene and competition for trough and resting space become accentuated. The ideal would be to wean pigs in litter groups which are never mixed with other litters. This isolation eliminates savaging and limits the spread of diseases such as enzootic pneumonia and sarcoptic mange to an extent which justifies the increased housing costs involved. A significant epidemiological fact in regard to enzootic pneumonia is that the rate of lung healing and the incidence of secondary bacterial complications are proportional to the density of the pig population.[4] A similar relationship may be postulated for other infectious diseases where crowded conditions multiply the re--infection rate.

Overcrowding generates excessive savaging and bullying, with the lighter pig always the chief sufferer. For this reason correct sorting of weaner groups is important. Savaging is increased by cold or damp con-

ditions, particularly where bedding is in short supply or constantly being soiled due to lack of dunging space. Bite wounds offer portals of entry to numerous types of pyogenic organism, infection resulting in abscessation of snout or limbs. In South Africa the commonest organisms encountered in these lesions are *Spirochaeta suilla* and the fusiform organism[5], the scrotum also sometimes being involved in newly castrated hogs. Infected animals respond poorly to treatment and rapidly become stunted.

Tailbiting is another vice which may appear under crowded conditions, and has been stated to be increased by a protein-deficient ration. A single pig may be responsible for initiating an epidemic, and should, if possible, be promptly identified and segregated.

Sporadic cases of Glässers disease may be encountered under overcrowded conditions. The disease is due to *Haemophilus suis* infection and is characterised by great lameness and a reluctance to move out of the bed. The temperature is high, episcleral congestion is evident and the joints are extremely painful when handled. At post-mortem examination there is evidence of polyserositis, arthritis and tendovaginitis and frequently an accompanying meningitis. An unusual feature of the arthritis is the peculiar inspissated, cheesy masses found in affected joints.

Poor Sanitation

Excessive faecal accumulations in weaner sties may result from overcrowding, shortage of cleaning water or labour, badly drained or broken floors and deep litter housing where insufficient bedding material is available. Under these conditions constant faecal soiling of skin and feet increases the risk of spirochaetosis or other pyogenic dermatoses, while excessive faecal contamination of feed and water supplies usually results in enteric disease outbreaks.

Two serious enteric infections occurring under such conditions are paratyphoid and vibrionic swine dysentery. Both diseases are characterised by subclinical carriers of infection, but do not appear to be provoked to clinical appearance except under unhygienic conditions. Neither of these diseases can be satisfactorily controlled solely by chemotherapeutic means, but require strict isolation measures on daily-washed concrete floors to be brought under control. The isolation should be maintained until market weight since recovered pigs often relapse to become infective. The uneconomic longhaired runts which appear as a depressing sequel to most paratyphoid outbreaks should be promptly destroyed.

In the pig the chronic phase of many intestinal infections results in permanent bowel damage commonly designated as necrotic enteritis or "necro" in the literature, and recognisable clinically by the unthriftiness and persistent scouring or purging of affected animals. The irreversible, fibroplastic changes seen in the intestinal mucosa of these cases are indicative of the hopeless prognosis, and such animals should be destroyed as generally uneconomic, probably infective and always incurable.

Parasitism

Apart from the constant irritation they cause, lice are believed to transmit pox and *Eperythrozoon*[6] infections. Since a few larvae may survive if a single treatment with a suitable insecticide is applied, it is advisable to repeat treatment after 12 days. Sarcoptic mange is widespread and as a general rule weaners should be dipped before being mixed together. This disease tends to persist in a herd unless painstaking multiple treatments are applied. Demodectic mange appears to be of such limited occurrence that it does not constitute a serious health hazard.

Three species of intestinal nematodes are regularly identified in young pigs, viz. *Ascaris lumbricoides*, *Oesophagostomum dentatum* and *Trichuris trichura*, with *Ascaris* forming by far the greater part of the infestations seen. The latter two species are generally only found where pigs are continually maintained in camps under conditions approximating to those described for many American "hog lots". With the regular use of piperazine compounds, particularly for the treatment of pregnant sows, coupled with concrete-floored housing, it is possible to reduce intestinal parasitism to a low level. Deep litter housing, if unhygienic, may promote severe intestinal parasitism. All three species of nematode mentioned above may be present in such cases and may perhaps best be controlled by the feeding of the antibiotic hygromycin B in the feed for the whole of the fattening period, together with the provision of extra bedding to improve the hygiene.

It is of interest to note that heavy experimental Ascaris infections of young pigs[7] produced transitory coughing and no pneumonia in only a proportion of the experimental animals. In no case did the coughing *last more than* 4 *days*. It was inferred that persistent coughing was more likely to be caused by enzootic pneumonia or other causes than by Ascaris infestation. The control of Ascaris infestation in enzootic pneumonia infected herds is of particular importance since the American report[8] that migrating Ascaris larvae markedly enhance the size of enzootic pneumonia lung lesions.

In the Western Cape Province lungworm is of frequent occurrence in young stock raised outdoors. Another hazard of outdoor pigkeeping in all parts of the country is the serious loss occasioned by cysticercosis and hydatidosis in exposed animals.

Inadequate Nutrition

Abrupt feeding changes at weaning cause some hitherto unsuspected changes in the intestinal flora. Recent reports [9, 10] have elucidated the pathogenesis of two diseases of great importance in young pigs, gut or bowel oedema and haemorrhagic gastro-enteritis. Both conditions are associated with certain strains of haemolytic *Escherichia coli* normally found in small numbers in weaner pigs, and are predisposed to by feed and management changes at weaning as well as by adverse housing conditions.

Many normal pigs possess tissue — sensitising antibodies to *E. coli*[9]. Feeding and environmental changes at weaning were found to cause rapid increases in the numbers of haemolytic *E. coli* in the gut, thereby increasing

the absorption of *E. coli* polysaccharide from the bowel. The symptoms and lesions of oedema disease and haemorrhagic gastro-enteristis are believed to result from an anaphylactic reaction in sensitive pigs, rather than from a direct toxic effect following polysaccharide absorption.[10]

Canadian workers[11] have described a series of outbreaks of haemorrhagic gastro-enteritis associated with similar serotypes of haemolytic *E. coli* and have noted the useful control of the disease which was obtained by reducing the protein content of the ration. This evidence appears to offer some explanation of the frequent occurrence of oedema disease in pigs weaned to overgenerous allowances of skim milk. The newly discovered association of these two important diseases with anaphylaxis, high-level protein feeding and husbandry changes offers an opportunity for more rational control measures in the future.

The use of more than two per cent of calcium carbonate in weaner rations may produce two undesirable diseases. Firstly, the excess calcium may cause an iron deficiency to develop just when an increased iron intake is desired.[12] This effect is particularly marked in litters with low weaning weights, probably because their iron reserves are generally low. Affected animals show reduced haemoglobin levels and liveweight gains. The same worker[12] was able to demonstrate a five pound liveweight advantage eight weeks after the administration of 100 mg. of iron (as iron-dextran) at weaning to pigs not receiving any calcium carbonate in their diet.

Rations with a calcium content in excess of one per cent may produce an insidious unthriftiness often, but not always, accompanied by hyperkeratosis of the skin. This disease, known as parakeratosis, was first reported in the United States in 1953[13] and has been seen on numerous occasions by the writer during the past five years. Affected animals shew erythematous patches around the eyes and on the ears and belly in the early stages of the disease, and may also pass consistently soft faeces. Later the appetite and rate of liveweight gain become greatly reduced and a dirty brown dermatitis, marked by severe hyperkeratosis and some cracking of the skin, may spread over the back, belly, quarters and hindlegs. The disease is effectively prevented and also cured by the provision of 100 p.p.m. of zinc in the ration as an addition of eight ounces of zinc carbonate per ton of feed. Parakeratosis bears some clinical resemblance to sarcoptic mange but may be differentiated by the absence of pruritis. Recent German studies[14] of parakeratosis also report the anaemic changes caused by excess calcium mentioned above and demonstrate the likely co-existence of the two conditions in the same animal.

The feeding of mouldy maize to weaner pigs in the Northern Transvaal has recently been observed as a cause of the vulvovaginitis reported from Ireland[15] and the United States.[16] Affected gilts shew extensive vulval swelling which may be accompanied in some animals by partial vaginal prolapse. Prompt remission of symptoms occurred when the feeding of the offending maize was discontinued.

Swill may contain dangerous quantities of glass, porcelain and bone fragments and should preferably be handsorted for foreign bodies prior to being fed. As a feed it is usually far too bulky for weaner pigs and cannot be fed to animals less than 80 pounds liveweight without provoking digestive upset and consequent unthriftiness.

Swill, whey and buttermilk may at times contain increased amounts of sodium chloride sufficient to result in poisoning where the animals' water intake is restricted or where separate drinking water is not provided.[17] Affected animals may die after shewing signs of gastro-enteritis or may live long enough to exhibit nervous symptoms such as circling, pushing, blindness and epileptiform convulsions. The lesions of meningoencephalitis eosinophilica found in these cases are considered pathognomonic of salt poisoning.

Swill may be deficient in many essential nutrients and great interest attaches to a recent Australian report[18] describing paraplegia in swill fed weaner pigs as a sequel to demyelination associated with extremely low liver copper levels.

Crude fibre levels in excess of seven per cent in weaner rations will reduce the digestibility of such rations and may be expected to exert an irritant effect in the terminal ileum and ileo-caecal orifice. Some relative hyperplasia of the abundant lymphoid tissue found in this area may occur as a sign of this irritation. In experimentally vibrio coli infected pigs a recurrence of the symptoms of dysentery has been observed after increasing the fibre level in the ration[19].

Discussion

A swine repopulation programme utilising hysterectomy-derived, specific pathogen-free pigs[20] has been advocated as an ideal method of raising disease-free stock and thus increasing the profitability of pig keeping. The method demands special equipment and specialised training in the procurement and raising of such pigs and is, therefore, not cheap. The system requires careful experimental evaluation, since it is entirely possible that the benefits claimed may be due at least as much to improved husbandry standards as to the elimination of respiratory disease.[21] A close study of the swine disease problems of the Republic during the past five years has convinced the writer that an educational campaign to improve swine husbandry will prove far more rewarding at this stage than the introduction of a repopulation programme with specific pathogen-free pigs. The veterinarian should play a leading rôle in such a campaign.

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BACTERIAL HEPATITIS IN CATTLE

CAUSED BY *PASTEURELLA MULTOCIDA* var. ictero-hepatitidis

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F. DU CASSE — Senior State Veterinarian, Estcourt, Natal

Received for publication, July 1962

SUMMARY

This paper is a short report on the first bacteriologically confirmed outbreak of Bacterial hepatitis in cattle in the Estcourt district in Natal. A report on two experimental cases is also given.

The characteristics of the causative organism (P. multocida var. ictero-hepatitidis) are given and compared with the characteristics of related organisms.

INTRODUCTION

In January 1960, an outbreak of a disease among sheep in the Dannhauser district described as Bacterial Icterus was reported on. The etiological agent was isolated and a short description of the organism given[8]. Its pathogenicity was confirmed. Subsequent to this, further outbreaks occurred sporadically in most of the major sheep raising areas of South Africa and South West Africa. Sheep in the Eastern Transvaal Highveld, the "Highveld" of Natal and the South Eastern part of the Karoo were commonly affected. Occasional sporadic cases also occurred in sheep kept at Onderstepoort. The diagnosis was confirmed histologically and bacteriologically in all the outbreaks.

The organisms isolated from the different outbreaks have the same morphological and pathogenic characteristics. Some differences in fermentation reactions were displayed by the several isolates but in general they were so similar that they can all be regarded as a single species.

From reports received from field veterinarians it appeared that all the outbreaks were associated with some or other stress factor, e.g. sudden change in grazing, adverse weather conditions, and dosing with hepatotoxic anthelminthics.

From time to time bovine liver specimens were received at Onderstepoort for pathological examination. Many of these specimens showed lesions similar to those seen in bacterial hepatitis in sheep viz. focal necrosis, netrophile infiltration and varying degrees of pigmentation[7]. In most cases however, the information accompanying the specimens was rather meagre and no specimens for bacteriological examination were submitted. The result was that no information regarding the etiology could be obtained.

Epizootiology

A disease, suspected as being typical three day stiffsickness, was very prevalent in the Estcourt area in 1961. The diagnosis was however doubtful owing to the prevailing drought, and the fact that adult cattle appeared mostly to be affected.

Farmers reported outbreaks of typical three day stiffsickness, while affected animals remained untreated. Many animals were found dead without having shown any symptoms; some affected animals recovered uneventfully. Drought in the area was very severe and this may have played some rôle. Plant poisoning was however not suspected.

Only one sick animal, a Brahman bull in the Ladysmith district, received anti-mortem examination. It was found lying down, had a temperature of 104°F, exhibuted severe twitching of the lips, and a rapid pulse. On stimulation, the bull rose and staggered off, showing more incoordination than stiffness. It showed severe distress and went down again very soon. It did not appear to be affected by heartwater. Penicillin and streptomycin injections were advised, but the bull died within twelve hours, before the injections were given. Due to circumstances a post mortem examination could not be carried out.

From the post-mortem examinations the majority of cases appeared to have died of cardiac failure, with marked sub-epicardial and endocardial haemorrhages (mild mulberry hearts), and severe hyperaemia and oedema of the lungs. The liver and kidneys were generally congested. In many instances the liver was midly to severely icteric with small to extensive necrotic foci clearly visible on section. Lymphatic glands were often enlarged and moist and hyperaemic on section.

The animal from which the specimens were taken was an eighteen month old steer which died overnight without any previous signs of illness. At autopsy, slight hyperaemia and oedema of the lungs, slight abomasitis and petechial haemorrhages on the epicard and in the trachea, were evident. The kidneys were congested and blood exuded on section. The spleen was soft and pulpy. The brain and spinal cord showed an excess of reddish cerebro-spinal fluid. The liver was yellowish, somewhat enlarged and with necrotic foci throughout its substance. Blood and spleen smears were negative for protozoon parasites as were tests for arsenic and prussic acid.

Laboratory Examination of Specimens

Specimens of liver and lymph-node on ice, and heart muscle in 10% formalin, were received at Onderstepoort for bacteriological, virological and pathological examination. The specimens were taken from the abovementioned steer.

No growth could be obtained by direct culture of either the liver or the lymph-node.

Saline suspensions of the liver and lymph-node were separately injected into six mice and four guinea-pigs. The amount varied from .25 ml. to 1 ml. for mice and 1 ml. to 2 ml. for guinea-pigs. The injections were given subcutaneously and intraperitoneally. Two of the eight guinea-pigs died within forty eight hours. An organism identical to that

isolated from cases of bacterial hepatitis in sheep, was isolated from the lung, heart blood and liver of both guinea-pigs. All primary cultures were made on blood tryptose agar plates, which were incubated at 37°C in air. No organisms could be found in heart blood smears or spleen smears.

Colonies on blood tryptose agar were white, smooth, round moist and became confluent. On sub-culture the colonies became less moist and less inclined to become confluent. The organism was dependent on "V" but not "X" factor for growth. Satalism with *Staphylococcus aureus* could not be demonstrated.

No growth could be obtained on nutrient gelatine or nutrient agar, but small colonies developed on Loefler's serum and serum agar after 48 hours' incubation.

Stained smears of the colonies showed small gram-negative cocci and coccobacilli. In colonies older than 48 hours filamentous forms were seen.

Litmus milk was turned very slightly acid but no coagulation or reduction of the litmus occurred. The methyl red test was negative, nitrates were reduced to nitrites, indole and catalase were produced, but formation of hydrogen sulphide could not be demonstrated. Acid but no gas, was produced in maltose, and sucrose and traces of acid were also produced in glucose, mannite and trehalose. Lactose, dulcite, arabinose, rhamnose, inosite, sorbite, starch and glycerol were not fermented. Addition of 5% serum to the sugars had no effect on the fermentation reactions.

Histo-pathological examination of the heart showed slight endocarditis and focal myocarditis[1]. Unfortunately no other specimens for pathological examination were submitted.

No virus could be isolated from the liver and lymph-node specimens.

Experimental

Experimental reproduction of the disease in bovines was tried after the diagnosis on material from the field cases had been made. Unfortunately the organism could not be reactivated after freeze-drying, with the result that another strain had to be used. The strain used was No. 8467 which had been isolated from a severe outbreak of bacterial hepatitis in sheep in the Ermelo district.

Two six month old Afrikaner steers were used in the experiment. The organism was grown on blood tryptose agar in Mason tubes for 24 hours at 37°C. The growth was washed off with saline and the density adjusted to correspond to Brown's tube No. 5.

Steers No. DOB 356 and DOB 580 were injected sub-cutaneously with 10 ml and 15 ml of the suspension respectively.

1. *DOB* 356:

This animal showed no temperature reaction or other signs of illness and was still alive after two months. It nevertheless developed an abscess at the injection site and gradually lost condition.

2. *DOB* 580:

This animal also showed no temperature reaction and developed a phlegmatic swelling at the injection site which also developed into an abscess, from which the organism could easily be isolated.

It deteriorated in condition and eventually died twenty days after having been injected.

Post Mortem Lesions

Post mortem changes were limited.

The carcass as a whole, the subcutis and fat, showed marked yellow pigmentation. There was a slight ascites and severe cachexia.

The various organs showed the following pathological changes:

Skin: Large abscess at injection site.

Lung: Mild hyperaemia, oedema and emphysema.

Heart: Epi- and endo-cardial petechiae.

Liver: Tumor hepatis. Marked degeneration and yellow pigmentation. The bile in the gall bladder was extremely tenacious.

Spleen: Atrophy.

Kidney: Nephrosis.

Rumen: Atony.

Small intestine: General stasis and catarrh.

Colon: Hyperaemia and petechiae. Specimens of the liver, spleen, kidney and lymp-nodes were taken for microscopical and bacteriological examination.

Histo-Pathology[7]

Liver: Marked central necrosis: severe bile pigmentation; early stages of fibrosis.

Spleen: Severe haemosiderosis: Atrophy.

Kidney: No special changes.

Bacteriological Examination of Specimens

(*a*) Direct cultures of the various organs were made on blood tryptose agar for two consecutive days.

(*b*) Mice and guine pigs were injected subcutaneously and intraperitoneally with suspensions. Cultures of the heart, blood, lung, liver, kidney and spleen of all experimental animals that died were made and examined further for the presence of the causative organism.

The organism could be isolated by direct culture from the spleen, kidney and pus from the abscess, on the first day only. It could not be isolated directly from the other organs.

The results of the biological examination were as follows:

(i) *Liver:*

Mice: 2 out of 4 died: cultures negative.

Guinea-pigs: 2 out of 4 died: one positive.

(ii) *Spleen:* Second day negative:

Mice: 3 out of 6 died: Cultures negative.

Guinea-pigs: 2 out of 4 died: One positive.

(iii) *Kidney:* Second day negative:
Mice: 4 out of 6 died: Cultures negative.
Guinea-pigs: 3 out of 4 died: Cultures negative.

(iv) *Lymphatic glands:*
Mice: 4 out of 6 died: Cultures negative.
Guinea-pigs: 2 out of 4 died: One positive.

(v) *Pus from abscess:* Both days:
Mice: 6 out of 6 died: All positive.
Guinea-pigs: 4 out of 4 died: All positive.

(vi) *Ascitic fluid:* Only coliform bacilli could be isolated by direct culture and from experimental animals.

Discussion and Conclusions

From the above information it appears that bovines are subject to both natural and experimental bacterial hepatitis. The lesions are similar to those encountered in sheep and goats and apparently the disease in cattle is also associated with some stress condition, in this case, three day stiffsickness and drought.

The liver lesions seen in the experimental case were very similar to those seen in suspected natural cases. The natural cases were however more acute[7].

By adjusting the infective dose, one would undoubtedly be able to produce typical acute cases.

The organism isolated from the original material was identical to the organism described in a previous paper[8] and isolated from other outbreaks of the disease in sheep. The fermentation reactions were however some-what different but it must be pointed out that the fermentation reactions of the organisms isolated from sheep were also not constant. The only sugar which was consistently fermented by all strains was maltose. The organism is tentatively identified as *P. multocida* though its pathogenic characteristics more closely resemble those of *Haemophilus spp.*[3,4].

Further work on the identity of the organism and its antigenic relationship to similar organisms such as *Haemophilus ovis*[4], *Haemophilus agni*[3], and *Pasteurella multocida*[2,] and [5], is at present in progress.

Acknowledgements

We are indebted to the Chief, Veterinary Research Institute, for permission to publish this paper and to Dr. D. J. Smit for the examination of the pathological specimens.

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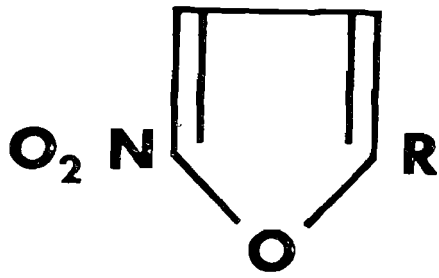

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CHINCHILLA FARMING IN SOUTH AFRICA

PART I

W. P. VAN AARDT — State Veterinarian, Grahamstown

Received for Publication, 21st May, 1962

HISTORICAL BACKGROUND

Chinchilla fur was worn by the early inhabitants of South America. The Spaniards first came upon the animal in 1527 in the South Coastal area of Peru, inhabited by the Chincha Indians, whom they were in the process of subjugating. Not knowing what the fleet little creature was, that seemed to be so prized by the Chincha's, the Spaniard's called these Chinchillas or little Chinchas. While the glint of gold and dream of jewels were uppermost in their minds, the beauty-appreciating Spaniards saw something in Chinchilla fur, to keep their other interests, the best of company.

The Incas, who ruled much of the Andean area, reserved most of this fur for their own use. Because of it's light-weight and pliancy it was especially suited for the extreme high altitude areas where these people lived.

Due to its durability as well as its other qualities, this fur was used to clothe warriors fighting at high altitudes, by giving them ample protection without handicapping them with heavy raiment. This particular quality is greatly valued now by the style-conscious who, instead of protection, seek sumptuousness and beauty without encumbrance.

This fur is to-day considered one of the most important furs in the future fur industry. This was demonstrated by the delight of the Queen of Spain when she received the first Chinchilla garment presented to any non-Indian. Most Queens still wear Chinchilla. Chinchilla fur has been worn by the style-conscious for over four centuries.

The interest in this fur built up over the centuries until even with the smaller markets of about 70 years ago, as many as 3,000,000 pelts per year are believed to have reached the world's fur trade. But this was not always in the interest of the Chinchilla.

Finally in 1880 a contest started between the New York fur interest and those of Europe, for this profitable fur. This was the beginning of the end of the wild supply. They were trapped out.

The Chilean conservationist, Frederico Albert, launched an unsuccessful campaign in 1899 to save and protect the Chinchilla in the wild. He published a magazine called "La Chinchilla" the object of which was to engender interest in saving the Chinchilla as a natural resource for Chile. What he knew, but what those who profited from the pelt trade chose to ignore, was the fact that once any creature is weakened in numbers below the ability to produce more than its natural enemies consume, that

creature is at that point, doomed. He failed. Later the animal was adjudged extinct. Several government ventures to do something before and after this period, were not successful.

In 1918 an American mining man, Mr. M. F. Chapman, arrived in Potuerillos, Chile, in the employ of the Anaconda Copper Company. Always having had an interest in animals, he readily bought a Chinchilla which a native offered him. Only later did he learn how rare it was. This intrigued him. He studied the animals and became one of the best informed men in the world on Chinchillas. He remained an authority until his death some years ago. He spent all his earnings on expeditions to those places where Chinchillas were still said to exist. The results were not encouraging.

On February the 22nd, 1923, he arrived at San Pedro, California, with only 3 females and 8 males. Nine years later all the known Chinchillas in the world were in 4 small sheds. The industry that once supplied 3,000,000 skins in a single year and had sunk to only three known females, had started to develop again. The industry is now selling between 75,000 and 150,000 skins annually. It will be some time before it can supply the millions needed.

When Chinchillas were rare, the industry moved towards the selling of "Breeder Pairs". It is only lately that there has been a move to sell the fur of these animals. The presence of an estimated 2,000,000 Chinchillas has encouraged the efforts of an industry, heavily filled with people, who are animal sellers.

Most sales of Chinchillas have been into the more populated areas of the world. Neither the climate nor conditions in New York, London or Birmingham, are suited to Chinchilla fur farming.

The first Chinchillas to Africa were shipped in 1956. These were 3 males; and they demonstrated that Chinchillas would live in Africa. Later a group, including females, were shipped out. These proved that Chinchillas would not only live but reproduce here. In November 1958 the first planned farm was established in Bulawayo, Southern Rhodesia. It was not long after this that Chinchillas were sent to the Republic of South Africa.

The production of Chinchilla fur on a commercial basis has now become fairly well established in the Republic of South Africa. At the moment there exists one firm e.g. The South African Fur Producers (Pty.) Ltd., (SAFPRO), who is out to establish the production of two and a half million pelts yearly in South Africa. The estimated value of these skins is R40,000,000.

ACKNOWLEDGEMENTS

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OPEN REDUCTION OF SCAPULOHUMERAL DISLOCATION IN A DOG

D. H. G. IRWIN — Department of Surgery, Faculty of Veterinary Science, Onderstepoort

SUMMARY

The history, clinical and radiological findings and open reduction of a dislocated shoulder joint in a dog are described. A plaster-cast was used to immobilise the joint for six days; satisfactory recovered ensued.

INTRODUCTION

The shoulder joint is less susceptible to traumatic dislocation than any other joint in the fore or hind limb of the dog. Although fracture through the scapular neck and of the upper third of the humerus is uncommon, it appears to be more frequent than scapulohumeral dislocation. Perhaps the mobility of the fore limb attachment to the chest permits sufficient "give" or play to the shoulder, to avoid fracture or dislocation.

ANAMNESIS

A six month old Schipperke-Pomeranian crossbred bitch was involved in a motor accident, in which the left shoulder was injured. The dog refused to use the left fore limb in spite of anti-inflammatory treatment. Five weeks later the patient was presented for examination and treatment.

EXAMINATION

The dog weighed five kilograms, and was in good condition. After clipping short the hair in the shoulder region it was easy to determine atrophy of the surrounding muscles, and severe restriction of mobility of the shoulder joint. A lateral radiograph showed that there was no joint space; in fact the articular surfaces were superimposed, establishing a diagnosis of dislocation. Closed reduction under deep barbiturate anaesthesia was unsuccessful, and open reduction was carried out.

OPERATION

All skin surfaces within eight centimetres of the shoulder joint were prepared for aseptic surgery.

The patient was anaesthetized with sodium pento-barbitone (Sagatal, May Baker) injected intravenously. A sterile stockinette sleeve of double thickness Tubegauz (Scholl Manufacturing Company) was applied to the whole limb and shoulder, starting from the distal end. This was secured

in position by spraying flexible collodion (Liquido-Plast, Hammer and Company) onto the skin at the porximal end of the limb and adjacent parts of the trunk. Further drapes were laid. A dorso-ventral incision about seven centimetres long was made antero-laterally with its middle over the joint. A branch of the cephalic vein was divided between ligatures, and the brachiocephalic muscle retracted antero-medially. The omotransversarius muscle was severed and the prescapular lymph node was retracted medially. The limb was pronated and by blunt dissection

the glenoid cavity was approached by cleaving the supraspinatus muscle in its long axis. The head of the humerus was lateral to the glenoid cavity. A little fibrous tissue was gently curetted from the glenoid cavity. By successively applying flexion, pronation and then extension, with one hand, and mediad pressure on the humeral head with the thumb of the other hand, the joint surfaces engaged. Two gut sutures were placed in an effort to close the joint capsule, which was barely recognisable. Maintaining limb traction distad throughout, the tissues were lightly approximated with gut, and the skin closed with silk. The wound was sealed by spraying flexible collodion over the area, and sticking sterile gauze to this.

Post-Operative Treatment

Whilst suspending the patient by strips of six-inch bandage, and maintaining traction on the affected limb, a plaster cast was applied, see fig. 1. Intramuscular injection of 300,000 i.u. penicillin was given, and repeated on the two days following operation.

Convalescence was uneventful. The cast and skin sutures were removed on the sixth day, and the patient was allowed home. Two weeks later the patient was presented for the discharge checkup. The shoulder joint showed good mobility, estimated at 85 per cent normal. The muscles of the region had picked up and the dog used the limb quite well. In an effort to overcome the "lameness of habit" which is so common in small dogs, a bandage was placed on the right forepaw. The owner was instructed to dose the dog two and one half grains (150 mgm) aspirin each evening, for two weeks, and the patient discharged. Enquiry four months later revealed that normal function had returned.

ACKNOWLEDGEMENT

The Chief, Veterinary Research Institute, is thanked for kindly allowing the publication of this note. Sister van Zyl is thanked for co-operation and assistance.

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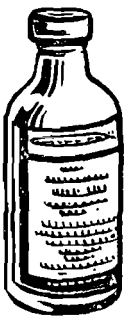

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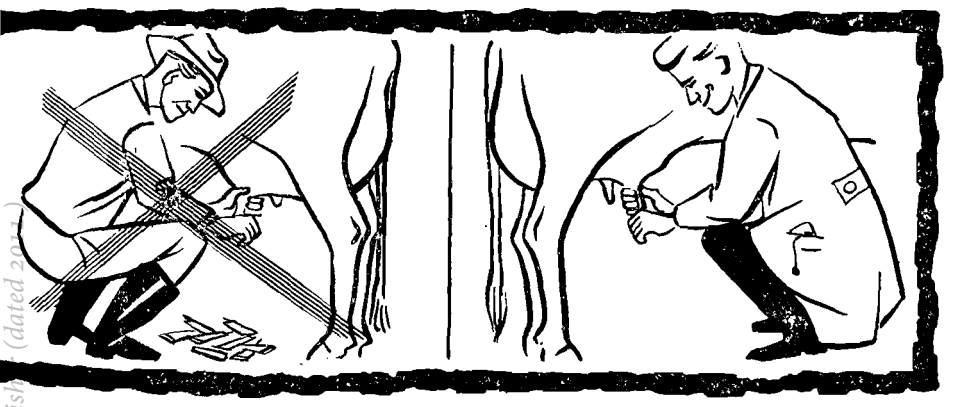

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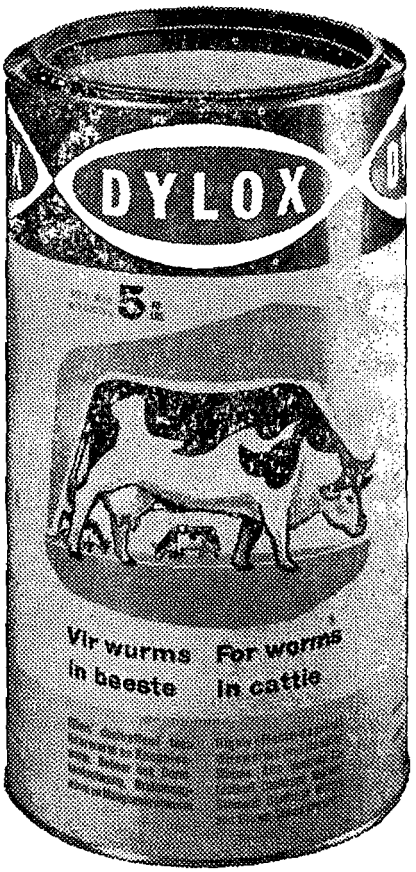

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J. S. Afr. vet. med. Ass. XXXIII (3) 1962.

POST-OPERATIVE THROMBO-EMBOLISM

A CASE REPORT

D. H. G. IRWIN — Department of Surgery, Faculty of Veterinary Science, Onderstepoort

INTRODUCTION

Thrombo-embolism is not an uncommon sequel to operations upon humans, particularly those performed in the pelvic region. Conversely, it is a rarity deserving of record in veterinary surgery. Hofmeyr[2] observed two cases of pulmonary thrombo-embolism, one in a cat and one in a bitch following Caesarian-section, in which the foetuses were putrid and post-operative convalescence was marked by prolonged recumbency. This report concerns a fatal case of pulmonary thrombo-embolism in a Jersey cow following total mastectomy.

SUBJECT

A Jersey cow, about seven years old, was presented for treatment of retained placenta and chronic hypertrophic mastitis. By the time the uterus was clear of infection and had undergone involution, it was apparent that the udder would never again produce milk. Moreover, its vast size so embarrassed progression that total mastectomy was indicated.

OPERATION

The technique employed was similar to that described by Hofmeyr[1] with the following exceptions: First, the initial skin incisions were made only one inch ventral to the reflection of the inner thigh skin onto the udder, as this was regarded adequate in this particular case.

Secondly, the lower half of the gland was removed without turning the cow onto the other side: this expedites the operation. This latter variation of the technique has been used in this Department by Hofmeyr when the existing pathology and other circumstances permit[2].

Finally, the subcutaneous abdominal veins were tied off at the latest possible stage of the dissection. Blood remaining in the udder was deliberately reduced through gentle massage of the gland before ligating the veins. The object of this procedure was to return as much udder blood to the system as possible.

The cow recovered satisfactorily from anaesthesia, walked back to her stable and ate and drank normally two hours later. Next morning the cow was noticed to be breathing with difficulty, although still standing and feeding. An hour later she was dead: this was about 20 hours after operation.

Autopsy

This revealed generalized cyanosis, and large areas with red to reddish-blue discolouration of the lung surfaces. Careful dissection of the pulmonary artery revealed firmly clotted blood in some branches. These clots were dark in colour and when dissected free from their vascular beds, retained the form of the vessel, as if they had been cast there-in. They were tough and rubberlike in consistency. In no other part of the body could similar clots be found, nor were there any other sites where the blood supply had been interfered with. In the normal appearing areas of the lungs, the blood in the vessels was clotted very little or not at all.

Professor Schulz of the Department of Pathology in this Faculty, confirmed the diagnosis of thrombo-embolism.

Conclusion

It is felt that the blood contained in the udder until it was massaged prior to ligation of the subcutaneous abdominal veins must have sludged and became the cause of the thrombo-embolism. In future the writer will not try to conserve for the body the blood contained in a member being amputated.

ACKNOWLEDGEMENT

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

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THE USE OF CRYSTALLINE METHIONINE AS A TREATMENT FOR LIVER DAMAGE IN RACEHORSES

(WITH SPECIAL REFERENCE TO SENECIO POISONING)

G. P. RETIEF — Private Practitioner, P.O. Box 123, Mooi River, Natal

Received for publication, 29 May 1962

SUMMARY

Two case-reports of liver damage in racehorses are recorded, where use was made of Crystalline Methionine intravenously with very good results.

INTRODUCTION

Senecio poisoning, or "Dunsiekte", in horses is extremely common in the Natal Midlands. The general attitude of owners in the district is that the horse will die in any case, so treatment is seldom attempted.

However, in desperation, treatment with Crystalline Methionine — ("Crystalline Meonine": Wyeth Laboratories) was tried in two cases with remarkable results. The two case reports are recorded here.

CASE NO. 1.—TWO YEAR OLD GELDING

History:

A two-year old gelding showed signs of constipation and the owner collected 2 oz. of Dihydroxyantraquinone — "Altan" (Maybaker), at the surgery and gave the horse ½ oz. as an electuary. The next day the author was called out as the horse's condition had deteriorated rapidly.

Examination: 7.12.1961.

The horse was very lethargic and stood with its head pushed up against a corner of the loose-box. It would not eat lush green-feed in the manager but preferred dry bedding. It yawned every now and then.

Mucous membranes	— injected	
Pulse	— 48 p. min.	Temp. 100.5° F.
Dung	— very loose	
Bloodsmear	— negative	

A *tentative diagnosis* of liver damage due to plant poisoning was made.

Treatment:

6,000,000 u. Penicillin i/m
1 gm. Streptomycin i/m
3,000 megm. Vit. B i/m
1 litre 10% Dextrose Saline i/v
½ gr. atropine s.c.

Examination: 8.12.1961.

The owner revealed that for the last two months the horse had been let out to graze in a paddock full of senecio. It was not used to grazing as it was usually kept and fed in its box.

Treatment:

10 c.c. Multivitamin prep i/m
100 mg. Hydrocortizone
1 litre 10% Dextrose Saline

A *definite diagnosis* of senecio poisoning was made.

Examination: 8.12.1961 (p.m.)

The condition of the horse had now deteriorated rapidly.
Temperature: subnormal.
Pulse: irregular, 90 p. min.
Mucous membranes: very injected.

Treatment:

Niketamide 2 vials i/m.
"Vetibenzamine" (powerful i/v anthistamine).

Examination: 9.12.1961.

The pulse had settled down again but the horse was very lethargic and could hardly stand. The owner was told that the prognosis was virtually hopeless, but permission was asked to try Crystalline Methionine i/v.

Treatment:

12.5 gm. Crystalline Methionine was dissolved in 1 litre 10% Dextrose Saline and given as a slow drip i/v.

That same afternoon the owner reported that the horse had gone out of the box and was tearing round a paddock with no apparent ill-effects.

Report on 15.12.1961 (*six days later*).

The owner reported that the horse was showing the same symptoms again.

Fifteen gm. Methionine in Dextrose Saline was administered. An immediate improvement was noted and since then the horse seems to be in perfect health. It is now in training and the owner owner hopes to race it soon.

Case No. 2: Yearling Filly

History:

The filly lagged behind the others when coming back from the

paddock. She suddenly collapsed on her forelegs and, after floundering a bit, regained her feet, only to collapse again a few steps further on. The author was called out immediately.

Examination: 14*th January*, 1962.

The filly showed the symptoms described above, and also kept her head down. She seemed to be unaware of her surroundings and moved in circles in her box.

A *tentative diagnosis* of liver damage due to plant poisoning was made.

Treatment:

10 gm. Methionine in 1 litre 10% Dextrose Saline as a slow i/v drip.
10 c.c. Multivitamin i/m.
2,000 megm. Vit. B_{12}.

14*th January* 1962 (*p.m.*):

Another 1 litre 10% Dextrose Saline was given i/v.
The filly looked much better but was still collapsing on her forelegs, although at longer intervals.

Report on 15th *January*, 1962 (*a.m.*).

The owner reported that the filly had only collapsed twice during the morning.

Treatment:

10 gm. Methionine dissolved in 10% Dextrose Saline i/v.

Report on 15*th January*, 1961 (*p.m.*):

The filly had only been slightly inco-ordinated once during the afternoon.

Treatment:

1 litre 10% Dextrose Saline.

Report on 16*th January*, 1961:

The filly was now eating well and looked perfectly normal and bright. No signs of inco-ordination could be seen.

Treatment:

10 gm. Methionine in Dextrose Saline i/v.
10 c.c. Multivitamin i/m.
5,000 megm. Vit. B_{12} i/m.

Result:

Complete recovery with no relapses.

Discussion

In the light of only two case reports, the use of Methionine as a cure for senecio poisoning cannot be conclusively proved. However, the results were so gratifying that it is felt that there is a definite place for Crystalline Methionine in horse-practice.

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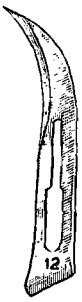

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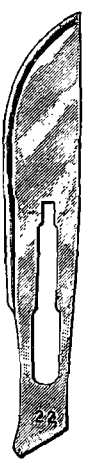

Nos. 20, 21, 22 and 23 fit No. 4 Handle.

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J. S. Afr. vet. med. Ass. XXXIII (3) 1962.

DIE KUDDE BENADERING IN VEEARTSENYKUNDE

K. M. VAN HEERDEN — Senior Staatsveearts, Middelburg (Kaap)

('N TOESPRAAK BY DIE 1962 JAARVERGADERING VAN DIE O.V.S., BASOETOELAND EN NOORD-KAAP-TAK VAN S.A.V.M.V. VOORGEDRA)

'n Veearts het in die verlede 'n belangrike leemte in die boerdery gemeenskap gevul as hy in die vermoë was om siek diere gesond te maak. Dit was, en tot 'n sekere mate, is nog 'n baie belangrike rol wat spruit, deels uit sy veeartsenykundige opleiding, en deels uit die gewoonte om fooie te vra vir elke besoek afgelê. Volgens laasgenoemde het die praktisyn dus gekonsentreer op gevalle wat met een, of uiters 'n paar besoeke, gefinaliseer kan word soos kalwings, kastrasies en die behandeling van individuele siek diere. Die belangrike kudde probleme wat te doen het met onvoldoende produksie en wat baie tyd en aandag verg, word dus nie aan geraak nie, en die boer word ook nie opgelei om aan te dring op sulke dienste nie.

Verreweg die grootste deel van ons boerdery inkomste is afkomstig van bees en skaap kuddes, waar die diere hul bestaan te danke het aan die feit dat hul lede van 'n groep is, wat in sigself 'n ekonomiese eenheid is, met die doel om 'n maksimum inkomste te lewer teen 'n minimum uitgawe. Dit rus dus op net sulke gesonde besigheidsbeginsels as enige van ons moderne industrieë en meer so vandag waar winsgrense aanmerklik vernou is.

Die progressiewe boer is meer geïnteresseerd in 'n goeie aanwas en kudde gesondheid, dan in die behandeling van individuele siek diere, en dit is hier waar die veearts, wat in 'n plattelandse praktyk belangstel, sy belangrikste rol kan speel. Behalwe 'n kennis van spesifieke siektes moet hy ook vertroud wees met die nuutste vorderings in nutrisie, genetika, diere versorging en bestuur, waaronder ook die finansiële implikasies van die spesifieke boerdery val. Dit is nie nodig dat hy 'n outoriteit op al hierdie gebiede moet wees nie, maar hy sal dan weet wanneer om hulp op 'n kooperatiewe basis in te roep. Wanneer op hierdie basis gewerk word is dit duidelik dat daar nie 'n helder skeidslyn bestaan tussen die veeteelt beampte en die veearts nie; eintlik bestaan daar 'n groot mate van oorvleuling. Dit is miskien logies dat die funksies van 'n veearts gedefinieer word as die funksie om die produktiwiteitspeil van die kudde te verhoog tot „normaal", terwyl die veeteeltbeampte metodes moet ontwerp om die „normale" produktiwiteitspeil te verhoog.

Sommige van die belangrikste probleme vandag in die veeindustrie gebied, is die gevolg van laegraadse, kroniese, of swak omskrewe siekte sindrome, waarvan die etiologiese komponente 'n mengsel van aansteeklik, voeding en bestuursfaktore is. Hierdie probleme vra vir studie, eerstens om die belangrikheid te omskrywe in terme van ekonomiese belangrikheid, tweedens om die komponente te evalueer en definieer om sodoende tot 'n diagnose te kom, en derdens om op 'n behandeling te besluit volgens die

belangrikste komponent. Behandeling mag veranderinge in bestuur, voeding of ras behels, instede van toediening van medisynes.

Wanneer diagnose op 'n kudde basis gedoen moet word, word twee groepe van simptome dikwels opgelet wat verskil van die gewone klassifikasie van siekte as sulks, maar wat vir alle praktiese doeleindes belangrike kudde siektes is. Ons kan dit noem:

(*a*) bestuursfoute; en
(*b*) latente siektes .

Bestuursfoute is bv. waar ramme in 'n groot kamp tussen ooie loop en waar sommige ooie nie in kontak met die ramme kom wanneer hul op hitte is nie. 'n Tweede voorbeeld is waar ramme nie ekstra kos kry voor hulle na ooie moet gaan nie en dus nie 'n reserwe van saad opbou om 'n redelike persentasie ooie te kan dek nie, of waar diere swak doen omrede kamp nie verander word nie, of oorstok is.

Latente siektes is bv. waar 'n brucella besmette kudde 'n natuurlike immuniteit opbou en al die diere heeltemal gesond lyk. Sodra daar egter introduksies bykom van nie-immuun diere, word die introduksies besmet, met gevolglike finansiële verlies, wat aborsies en vassittende nageboortes betref. Skape mag op 'n weiveld loop laag in molybdenum of hoog in koper inhoud, en hierdeur groot hoeveelhede koper in die lewer opberg. Hierdie diere vertoon heeltemal gesond totdat skielike spannings faktore, lewer beskadiging, of toediening van koper met wurm doserings, skielike vrektes veroorsaak.

Hierdie is 'n paar probleme van 'n kuddepraktyk en, soos gesien kan word, vra dit 'n totaal verskillende benadering as die benadering in 'n individuele diere praktyk.

Gedurende die afgelope 20 jaar het die epizootiese siektes onder beheer gekom en het die klem in lewende hawe produksie geleidelik verskuif na die velde van nutrisie, genetika en wetenskaplike diere produksie. Die belangrikste vorderings in hierdie velde is deur wetenskaplikes gedoen wat nie veeartse is nie. Soos die belangrikste infeksiesiektes egter onder kontrole gebring word, is dit juis hierdie velde wat moet ontwikkel en die sleutel tot die toekoms hou, soos bakteriologie en parasitologie dit 50 jaar gehou het.

Dit is nodig dat ons as veeartse meer moet weet van hierdie vakke om sodoende die basiese ondersoek op 'n plaas te kan doen en dan die eienaar van goeie raad te voorsien.

In ons kursus mag dit miskien raadsaam wees om nie soseer te konsentreer op groot en klein diere nie, maar eerder op die verskillende spesies soos bv. (waar hy dan volkome vertroud moet raak met die hele boerdery patroon).

1. *Stedelike diere* — honde, katte, perde en die diere wat as enkelinge behandel word. Hierdie is eie aan die klassieke veeartsenykundige praktyk en basiese medisyne en operasies kan hier onderrig word.

2. *Melkbeeste en varke* — wat gewoonlik geassosieer word as 'n boerdery eenheid.

3. *Skape en vleisbeeste.*

4. *Pluimvee.*

Hierdie is uitstekend om studente te onderrig in die grondbeginsels van kudde praktyk.

As ons substasies in die hart van die skaap, vleisbeeste en melkbeeste areas kan hê wat kan funksioneer as praktyk, navorsing en opleidings eenhede, kan studente 'n gedeelte van hul senior jaar by sulke stasies deurbring en sodoende waardevolle ondervinding opdoen.

Ten laaste, as die praktisyn sy klieënt kan oorhaal om vir sy dienste op 'n fooi per jaar basis te betaal, het hy die beginprobleme oorkom en is besig om 'n volwaardige kudde praktyk op te bou.

ERKENNING

Die Hoof, Veeartsenykundige Velddienste word bedank vir gesag om hierdie toespraak te publiseer.

A LAWYERS ADVICE TO VETERINARIANS CALLED TO GIVE EXPERT EVIDENCE IN THE COURTS

(Made available by D. J. Louw, Bloemfontein)

It has been the experience that veterinarians have been called upon to give expert evidence in the Courts and not been paid the witness fees. A court action of this nature could involve lengthy consultations with legal people and also much time in the Court itself at the hearing. The purpose of this memorandum is to advise what steps must be taken to ensure some measure of security for the payment of such fees. The usual method of placing evidence before a Court is for the witness to appear in person and give his evidence viva voce. Such witness is compelled to attend personally at the trial by the service upon him of a subpoena. The procedure for compelling the attendance of a witness varies according to whether the witness is in an area of jurisdiction of the Trial Court or outside, but for the purpose of this memo we will give you a brief outline of the position. A subpoena is normally issued in the case of the Supreme Court by the Registrar and in the case of the Magistrate by the Clerk of the Court. This document calls upon and orders the person to appear before the Court on the day of trial to testify concerning the matters in dispute. Should the witness fail to comply with a subpoena served upon him he will liable to a fine and imprisonment for contempt of Court. This liability however is dependent on the following:—

1. That he was summoned a reasonable time before the Trial;
2. that his reasonable expenses have been paid or tendered to him; and
3. that he has no lawful excuse to absent him in Court.

What a reasonable time is before trial is dependent on the circumstances of each particular case and will be influenced by the distance of the place of residence of the witness from the Court and the means of transport. Sufficient time must be allowed to the witness to make arrangements for his own business. Thus to serve a veterinarian with a subpoena an hour or so before the time at which he has to appear in Court is not reasonable and if he fails to attend on such a subpoena he will not be regarded as being in default. Similarly the question of what reasonable expenses are, will depend to some extent upon the status of the witness and on the travelling expenses and subsistance necessary to maintain the witness on his journey to the place of trial. The tariff in respect of reasonable expenses is altered from time to time and for the purpose of this memo it is not necessary to elaborate any further. *A lawful excuse* is presumably any excuse which would commend itself to a Court as going to show that the witness was not wilfully absenting himself but had been unavoidably prevented from being present for example by dangerous illness, dislocation of travelling facilities and such like events.

It must be brought to your attention that *such subpoena in no way compels a witness to consult with the legal counsel on whose behalf he has been subpoenaed* and it is this very aspect which places you in a position to negotiate with the litigating party who requires your services in regard to your fees. In the normal course of events where you may be required to give evidence you will be requested to attend a pre-trial consultation with the legal representatives of the party for whom you will give evidence and it is at this stage that a definite arrangement must be made with the party and his legal representative *in regard to your fees. It might prove to be useful to your Society as a whole if some measure of uniformity is decided upon in regard to a tariff for such matters.* Before agreeing to attend such a pre-trial conference, your fee both for the consultation and for your actual evidence in Court must be agreed upon between yourself and the litigating party. The lever which you have is that no Counsel will be prepared to examine a witness in a Court of Law on expert matters such as the type of evidence which you will be called upon to give, without first having the opportunity of consulting with such witness. No party can compel a witness to consult with him before the Trial, and should no arrangement or agreement be made in regard your fees, you simply withdraw from the matter, and if subpoenaed, appear in Court. Counsel would be very foolish, and indeed in most circumstances it will prove disasterous to him, not to have the opportunity of consulting before hand with an expert witness. Such a witness could in fact destroy his case and this is the most important feature in so far as you are concerned. It might be deemed advisable for you to frame some form of tariff whereby your fee is laid down on a hourly basis, and which would then entitle you to your fees both for the consultation and the actual time spent at the Trial. It must be born in mind that at the Trial long hours might be spent waiting before you are called as a witness and in respect of this time you should also be protected. The crux of the matter therefore is that should no agreement be reached in regard to your fees before consulting and before going to trial your attitude must simply be a negative one and you must refuse to disclose any information which you have.

SHOULD WE FORMULATE SUCH FEES? (ED.)

CIRCULATION OF INFORMATION TO MEMBERS

The S.A.V.M.A. undertakes on request, to circularise its members on all urgent and important matters, at R5.00 *per intimation for members and* R10.00 *per intimation for non-members.*

The circular letters are usually dispatched to members every 14–21 days.

BRANCH MEETINGS

NATAL BRANCH

ANNUAL GENERAL MEETING HELD AT ALLERTON ON 13TH JUNE, 1962

PRESENT:

Drs. L. C. Blomefield, G. Martinaglia, B. T. Paine, P. S. Snyman, A. R. Thiel, L. R. Morford, R. Paine, A. M. Diesel, J. L. Dore, I. S. Canham, H. E. Holtz, A. F. Tarr, R. A. Solomon, J. M. O'Grady, G. P. Retief, P. N. Collier, W. C. Viljoen, P. P. Wacher, H. F. Strydom, G. M. H. Shires, W. B. Hobbs, R. J. Bezuidenhout, D. D. McMillan, G. K. Shaw, D. G. Clow, D. E. Osborn, D. J. le Roux, A. J. Louw, R. Bangay.

VISITORS:

Prof. K. van der Walt, Dr. K. L. Malkin.

APOLOGIES:

Drs. B. C. Jansen, W. G. Barnard, B. du Casse, A. S. Canham, S. G. Turner.

The Chairman, Dr. Blomefield, welcomed all members and visitors and expressed particular pleasure in having Dr. R. Paine present. He also made mention of the absence of Dr. J. Zwarenstein after his number of years as an active member of the Branch.

The Chairman then offered a special welcome to Dr. A. M. Diesel and Prof. K. van der Walt and asked Dr. Diesel, the secretary of the S.A.V.M.A. to open the meeting.

OPENING ADDRESS:

In his opening address Dr. Diesel referred to the value of the S.A.V.M.A. Journal to the profession and also showed how extensive its distribution had become. He then suggested active support to the Journal particularly in so far as advertisement is concerned. Dr. Diesel also made mention of the Veterinary Act, the registration of Veterinarians and some of the problems which have been encountered recently in this respect.

In thanking Dr. Diesel for his opening address the Chairman expressed his gratitude that Dr. Diesel could spare the time to come down to Natal for this occasion. He was sure this gesture was greatly appreciated by the branch members.

CHAIRMAN'S REPORT:

The Chairman then delivered his report in which he made mention of the success of the Durban Congress of 1961 and the activity which had led to this success. He made a few remarks on the Rabies outbreak late in 1961 and the consequent control campaign. Dr. Blomefield went on to warn of the dangers of possible disease introduction with the immigration of Europeans from the emergent African States. The Chairman also

referred to our general "Professional Status" and went on to express his appreciation at the noticeable improvement in the organisation of the S.A.V.M.A. and felt that much credit was due to our newly appointed full time Secretary. Finally he thanked his Committee and Secretary for their loyal support during the year.

TRAVELLING ALLOWANCE FOR COUNCIL MEMBERS:

Dr. P. S. Snyman suggested that some contribution be made to Natal Branch members who served on Council to cover some of their expenses in attending meetings. Dr. Canham proposed that the matter be referred to the parent body but Dr. Diesel pointed out that Council could not carry all expenses, and it would impede the serviceability of the individual. Dr. Snyman then proposed that the annual subscription be raised to R2.00 to meet some of these costs. This motion was seconded by Dr. Solomon and carried. A lengthy discussion followed during which Dr. Tarr said that he felt an elected Council member could make some personal sacrifice and give something back to the profession. Dr. Dore then proposed that we increase our assistance if Council will help. This proposal was carried.

ELECTION OF OFFICE BEARERS:

After both Drs. Snyman and Blomefield had declined to stand for the chairmanship. Dr. Tarr was elected having been proposed by Dr. O'Grady and seconded by Dr. Jenkins.

Dr. Dore was elected as Vice Chairman, the proposer being Dr. Canham and the seconder Dr. Blomefield.

Dr. Jenkins was then proposed as Secretary by Dr. Blomefield seconded by Dr. Solomon. Dr. Viljoen declined to stand for a further term.

The Committee was chosen by ballot and the following members were elected: Drs. Thiel, Hobbs, Solomon, Blomefield and Viljoen.

Dr. Tarr then took the Chair and thanked his fellow members for his election. He then extended a personal welcome to Dr. R. Paine.

At this point Dr. Diesel suggested that a letter of good wishes be sent from the Natal Branch to Dr. Chase who had recently undergone a major operation.

CLINICAL PAPERS AND DISCUSSIONS

NEPHRITIS IN SMALL ANIMALS:

Prof. K. van der Walt delivered an outstanding paper on the nephritis-complex in small animals. Question-time was postponed to allow for fuller discussion.

B.H.C. POISONING IN BOVINES AND TRILENE as an Anaesthetic Agent in the Pig:

Two short papers on these two subjects were read by Dr. Jenkins.

FORUM

SMALL ANIMAL:

The panel for this forum consisted of DR. J. DORE, DR. I. CANHAM and DR. L. MORFORD. The subjects dealt with were briefly as follows:

(1) Cortisone therapy for dog skin conditions and the duration of treatment.
(2) Sporadic fits in dogs.
(3) Blood-transfusion in biliary fever cases.
(4) Complications encountered with oophorectomy.
(5) Treatment of so-called "Leptospirosis".
(6) Blindness as a symptom and complication of Biliary fever.

All these subjects enjoyed considerable discussion by the meeting.

LARGE ANIMAL:

The panel being DR. A. F. TARR, DR. R. A. SOLOMON and DR. P. WACHER. The subjects submitted for discussion were:

(1) Impacted ingesta in the mouths of cattle.
(2) Rumen impaction.
(3) The spongy eczematous conditions encountered on cows heels.
(4) Acute Lantana poisoning.

Once again a lively discussion resulted with many members taking part.

Numerous questions and discussions evolved from Prof. van der Walt's paper e.g. Leptospirosis was an interesting talking point and Drs. Dore, Martinaglia and O'Grady amongst others joined in.

GENERAL

Dr. I. Canham requested that record be made of the Branch's appreciation and thanks for the Committee's and sub-committee's work in organising the Durban Congress.

The Chairman thanked the S.D.V.S., Dr. Blomefield for the use of the room and facilities at Allerton.

The members all expressed the wish to have the Forum discussions continued in the future.

Dr. Tarr mentioned that evening meetings would soon be arranged at which a guest speaker would deliver a talk on a subject of interest to veterinarians.

The Chairman then declared the meeting closed.

JAARVERGADERING, NOORD-KAAPLANDSE EN BASOETO[6] LANDSE TAK VAN DIE S.A.V.M.N.

23 JUNIE 1962

Die O.V.S., Noord-Kaap en Basoetolandse tak van die S.A.V.M.V. het eenparig by die Algemene Jaarvergadering die volgende resolusies aangeneem ter oorweging deur die Raad van die S.A.V.M.N.:

1. Dat die wet op Misstowwe, Veevoedsels, Saad en Middels, (Wet No. 36 van 1947), ondersoek en gewysig word, sodat *Veemiddels* nie hieronder sorteer nie, maar die middels onder dieselfde beheer geplaas word waaraan genoemde middels onderhewig is by die bepalings van die Aptekerswet.

Hierdie wet laat onder die huidige omskrywing toe dat middels wat onder die Aptekerswet slegs op preskripsie verkrygbaar is tog wel as veemiddels in die gewone handel deur leke verkoop en gekoop mag word, met gevolglike onoordeelkundige gebruik van byvoorbeeld antibiotika.

2. Dat die belange van die privaat praktisyn gehandhaaf sal word, wanneer Streeksdiagnostiese-sentra opgerig word.

VERSLAG AANGAANDE DIE VERRIGTINGE BY DIE JAARVERGADERING VAN DIE O.V.S., BASOETOLAND EN NOORD-KAAPSE TAK GEHOU TE BLOEMFONTEIN OP 23 JUNIE 1962, OM 9.30 VM.

Ses-en-twintig lede het die vergadering bygewoon, waarvan ses nuwe lede was.

Dr. A. M. Diesel, Sekretaris: S.A.V.M.V. het die vergacering bygewoon as verteenwoordiger van die moederliggaam. Sy teenwoordigheid is baie waardeer daar hy hom die moeite getroos het om net vir die dag af te kom.

Gaste wat die wetenskaplike deel van die vergadering bygewoon het, was: Prof. de Villiers (Dekaan van die Landboufakulteit, U.O.V.S.); Mnr. T. Mostert (Assistent-Direkteur: O.V.S.-streek — Grondbewaring en Voorligting); Mnr. I. Barr (Redakteur van die „Farmer's Weekly").

Die besigheidsvergadering is die oggend afgehandel. Vervolgens 'n kortlikse samevatting van die belangrikste besprekings:

Melding is gemaak dat die Departement van Landbou-Tegniese Dienste 'n reorganisasie ondergaan, veral wat betref die navorsingspersoneel en bedenkinge of veranderings ten goede is vir die Veeartsenydienste is gelug.

Die skaal van fooie is bespreek veral wat betref mylfooie. 'n Beroep is op lede gedoen om 'n eenvormige fooi te vorder.

Betreffende siviele regsgedinge noem die voorsitter dat wetsadvies ingewin is. Indien lede as getuie moet optree, kan mylfooie sowel as professionele fooie per dag gehef word van die party wat die lid as getuie laat dagvaar.

In sy voorsittersrede het hy veral klem gelê op veeartse in die handel, met betrekking tot die etiese kode. Die voorsitter het genoem dat daar nie 'n verandering van die etiese kode moet plaasvind nie, maar dat Veeartse in die betrekkings die etiese kode moet nakom.

Na bespreking is 'n resolusie aan die moederliggaam gerig met betrekking tot die Wet op Misstowwe, Veevoedsel, Saad en Middels van 1947 indien voege dat Veemiddels, soos antibiotika, uit die wet verwyder moet word en ingesluit word in die Aptekerswet, sodat 'n beter beheer uitgeoefen kan word. Onoordeelkundige gebruik van die middels vind tans plaas deurdat leke dit verkoop en koop.

Die Komitee wat vir die volgende jaar verkies is, is soos volg:

Drs. D. J. Louw (Voorsitter); W. J. Ryksen (Onder-Voorsitter); H. G. J. Coetzee (Ere-Sekretaris/Penningmeester); C. H. Flight, N. Barrie en G. H. J. Stevens (Addisionele lede).

Die volgende wetenskaplike voordragte is die namiddag bespreek:

(i) *Die Kuddebenadering in Veeartsenykunde.*—Sommige aspekte van belang in die toer deur Engeland en Amerika.
— Dr. K. M. van Heerden.

(ii) *Gevalsverslae — Hanekam (Nitriete) Vergiftiging. Lusernkuilvoer Vergifting in Perde.*
— Dr. C. H. van Niekerk.

(iii) *Interessante waarnemings met die verskuiwing van wit renosters vanaf Natal na die O.V.S.*
— Drs. T. A. T. Louw en P. de la Harpe.

(iv) *'n Paar gevalle van verdowing met Succiniel cholien chloried in wilde diere met behulp van 'n pylspuit-geweer.*

'n Suksesvolle dag is afgesluit met 'n gesellige skemerkelkparty in 'n plaaslike hotel.

DANKBETUIGING

Die Raad van die Suid-Afrikaanse Veterenêr-Mediese Vereniging wil graag sy waardering betuig aan die ondergenoemde twee liggame vir die gulhartige en waardevolle dienste wat hulle tydens sy kongres gelewer het:

1. AAN DIE RAAD VAN BEHEER OOR DIE VEE- EN VLEISNYWERHEID

Vir 'n besondere aangename geskenk en prysenswaardige gebaar.

2. AAN B.P. SUIDELIKE AFRIKA (EDMS.) BEPERK

Vir die uitstekende dienste gelewer ten opsigte van openbare luidsprekers, band-opname fasiliteite en verbindingsstelsel wat aansienlik tot die gladde verloop van die Kongres sal bydra.

3. DIE HOOF, NAVORSINGSINSTITUUT VIR VEEARTSENYKUNDE EN DIE DEKAAN VAN DIE FAKULTEIT VEEARTSENYKUNDE

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MEMOIRS

DR. P. L. LE ROUX, D.SC., M.R.C.V.S.

Phillipus Lodewicus le Roux died suddenly on the 11th May, 1962, at his home in Bury Saint, Edmunds, Suffolk, England.

Dr. le Roux was born on the 7th November, 1897, on his father's farm Pokkraal, six miles from Worcester. Educated at Rawsouville and the South African College School, he studied Veterinary Science at the Royal (Dick) Veterinary College and Edinburgh University, becoming an M.R.C.V.S. and B.Sc. in July, 1923. A doctorate was conferred on him in 1936.

After a year's post graduate study in the Edinburgh University Department of Parasitology, Dr. le Roux returned to South Africa to join the Division of Veterinary Services. He was posted to Allerton Laboratory, Pietermaritzburg, later to the Ermelo Experimental farm, and finally to the Helminthology Section at Onderstepoort. He left South Africa in 1931 to take up a Veterinary Research Appointment in Northern Rhodesia, and from this ill-health forced him to retire in 1946. He then joined the Staff of the London School of Hygiene and Tropical Medicine, where he was appointed Senior Lecturer in Helminthology, and in 1957 Reader in Medical Parasitology.

Although le Roux did valuable work in many fields, his greatest interest was in Helminthology, so much so that he was reluctant to publish

articles on other subjects. One remembers him in his early years at Onderstepoort haunting the post-mortem hall in quest of Schistosomes. Little wonder that he came to be recognized as a world authority on the Schistosome group of parasitic worms, and also on agnatic snails, which act as intermediaries of flukes of veterinary and medical importance. From 1924 onwards he published numerous articles on helminth parasites of domestic and wild animals.

In Northern Rhodesia, where he became something of a legend, he covered a wide field, working particularly on rinderpest, contagious bovine pleuropneumonia, and the acyclical transmission of trypanosomiasis. There, too he described "pseudo-urticaria in cattle" — the first reference to a disease later encountered in South Africa in a more virulent form and now known as Lumpy Skin Disease.

For several years he was associated with the Food and Agriculture Organisation of the United Nations, his efforts being greatly appreciated by his colleagues and those whom he trained from many parts of the world. His last assignment for the F.A.O., described by Dr. W. Ross Cockrill as an outstanding success, was towards the end of 1961 "as one of a small band of instructors who ran the Training Centre in Livestock Parasitology, held at the Veterinary College, Insein, Burma". In a letter received only three days before le Roux's death, the Director of Veterinary Services, Burma, spoke in glowing terms of his work there, and added "although Dr. le Roux is an old man, his energy and enthusiasm are truly amazing".

Professor Buckley, of the London School of Hygiene and Tropical Medicine, wrote that at the time of his death "he had been as always, very active in the Veterinary World, especially in the field of Helminthology, and was working hard, perhaps too hard, on various research projects. He never spared himself".

Gin le Roux (after his Onderstepoort days he was generally know as "Piet") was a striking and unusual personality. Himself a farmer's son, he appreciated the problems of the Stock owner, and the importance of practical measures in the prevention and control of disease. With his long memory, enquiring mind, very keen powers of observation, and enormous capacity for work, he was well equipped for Veterinary research. All who knew him at any time and anywhere were impressed by his enthusiasm and energy; he drove himself to the limit, and although he expected much from others he preferred to do things himself. His person and his clothes often bore evidence of his delvings into carcasses and intestinal contents, which perhaps accounted for his utter contempt for pretence and window-dressing. Restless, impatient, quick-tempered, and outspoken, in his early days he was often in trouble, but that worried him not at all, because he was completely unimpressed by authority and had a cheerful disregard for administrative control. His keen sense of humour was apt to burst forth on serious occasions. Many will remember the somewhat rasping laugh, which some called a "cackle", and the penetrating comment or biting criticism which often accompanied it: no wonder that some found that laugh irritating. But very many will remember his kindness and unobtrusive helpfulness in material as well as in scientific matters. Once in the old O.P. days, when the examination of

smears was worse than a hardship, several thousand smears, unaccounted for, were eventually found in a cupboard, where a cleaner had tidily packed them away. The explanation of the Research Officer to whom they had been issued was that his daily allotment of smears had accumulated, and when he went to examine them one Sunday, they were no longer on his table, and he therefore concluded that le Roux had examined them for him. That was the sort of thing Gin would do.

In 1924 le Roux married Dr. Margaret Bowie, by whom he had three daughters; this marriage was dissolved in 1932. In Northern Rhodesia he married Ella Clarke, who bore him two daughters. To his widow and daughters we extend our very sincere sympathy.

The news of le Roux's death came as a shock to many in many parts of the world. We have lost a good friend and the Veterinary profession has lost one of its most gifted workers.

ALBERT GEORGE GRIST

The passing of Albert George Grist at his home in Devon, England, on 9th May, 1962, at the age of 91 years, recalls the conclusion of a very important chapter in the history of veterinary activity in South Africa.

Albert Grist was born in Torquay, England on 26th March, 1871. He qualified as a Veterinary Surgeon at the London Veterinary College in 1892 and came to South Africa as a member of the Royal Army Veterinary corps at the time of the Anglo-Boer war.

He was extremely fond of riding, jumping and hunting and was in his earlier years, an accomplished horseman. He rode in a gymkhana at the age of 73. He judged at many jumping competitions.

He was stationed at Bloemfontein for very many years and was later transferred to Queenstown and the Eastern Cape.

He retired in 1927 and went farming for a few years, but in 1937 settled at Cowies Hill, near Durban, where he built up a lucrative practice and remained for 10 years.

While in England in 1936 he took a post-graduate course at the London Veterinary College.

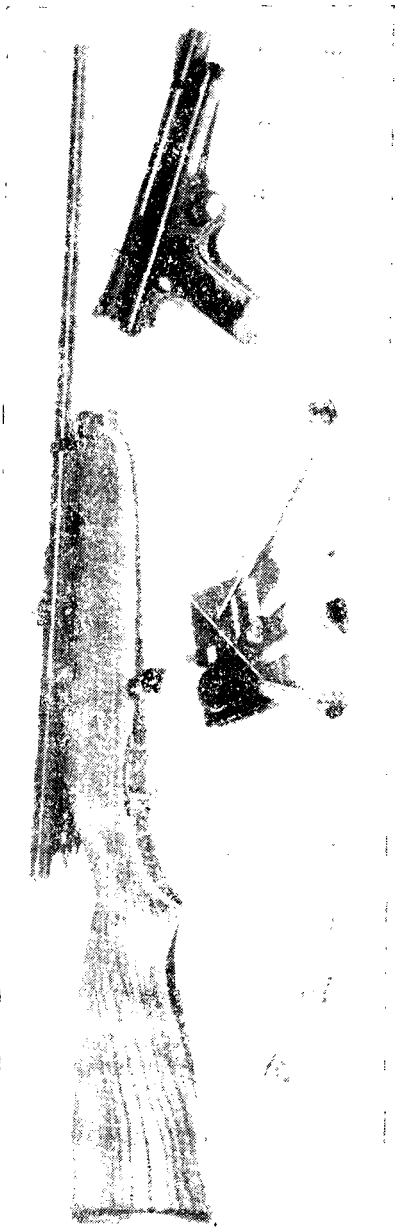
He returned to England temporarily in 1955 and permanently in 1959.

He was the last of the group of veterinarians who served the interests of veterinary science immediately after the Anglo-Boer war. This group was mainly those who settled in the country after being discharged from the Royal Army Veterinary Corps.

Their main interests were in horses, as can well be appreciated. They were followed by a group who were chosen for their wider knowledge of veterinary interests.

Some of the members of this group are happily still with us. Like the first group, they too served the interests of the country exceeding well.

Albert Grist leaves a wife to mourn his loss. To her we extend our deepest sympathy.



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BOOK REVIEWS

GENETIC ASPECTS OF DAIRY CATTLE BREEDING

by IVOR JOHANSSON

1st Edition. 260 pages. Price R4.00

Oliver and Boyd. Edinburgh & London

Dr. Johansson was professor of animal breeding at the Royal Agricultural College of Upsala, Sweden, from 1933 until his retirement in 1958. During those 25 years he became one of the acknowledged leaders in the bio-mathematical approach to animal breeding; an approach which has placed this subject on a scientific foundation, hardly imagined a generation ago.

In 1958-59, Prof. Johansson was a guest lecturer on animal genetics in the department of dairy science at the University of Illinois. The lectures he gave there form the basis of this book.

The first chapters of the book deal with population genetics, immunogenetics and monozygous twin investigation; research which has yielded valuable results on dairy cattle breeding. The next chapters deal with factors which influence milk production, namely fertility, body size and type, udder development and milkrate, while breeding methods and the general principles for estimating the breeding values of animals, including progeny testing, are discussed in the last chapters.

Throughout the book, the reader is impressed by the authors wide grasp and knowledge of his subject, by his outstanding knowledge of the literature and by his ability to co-ordinate and marshal known facts and theories. This book is indeed a notable achievement with which to end a notable research and teaching career.

J.H.R.B.

THE MOTOR APPARATUS OF THE MAMMARY GLAND

BY M. G. ZAKS

Oliver & Boyd, Edinburgh & London. 1962; 190 pages; 37 illus.; 16 tables. Published price, 40/-

The original of this book by Professor Zaks of the Sechnenov Institute of Evolutionary Physiology Leningrad, is in Russian. The translation into English is by D. G. Fry B.A. and the editing by A. T. Cowie, D.Sc., M.R.C.V.S., Ph.D. of the Shinfield Dairy Research Institute, Reading.

The book deals with some of the most important and least understood aspects of lactational physiology. Its perusal clearly shows that this field has been more intensively studied in Russia than elsewhere. This does not imply that the author has ignored research done in other countries. On the contrary, the work of prominent investigators outside Russia is extensively quoted and their results were duly considered in formulating conclusions.

The title does not reveal the full scope of the publication, which embraces the storage function of the mammary gland, the physiology of milk ejection, the production of fat and other constituents of milk, the relationship between milk removal and its synthesis, and the relationship between mammary gland function and methods of milking.

Further there is a brief description of the elastometric method for studying the physiology of the mammary gland in women.

It is quite certain that no previous publication on this subject has contained such a mass of new information and revealed more intensive basic study of the mammary gland.

Important new concepts substantiated by experimental proof are presented on such vexed questions as the storage capacity of the udder, intra-mammary pressure, ejection of milk from the alveolar system into the cistern, the reflex humoral and peripheral mechanisms responsible for milk ejection, and the many factors influencing quantity and quality of milk.

On the other hand many of the fallacies on important aspects such as the nature of milk ejection and factors that inhibit or control it are exploded, as are some of the popular beliefs regarding variations in milk yield and composition in the same cow. Thus the fear of many husbandrymen that feeding while milking distracts the cow and lowers the efficiency of the specific stimuli of the udder is shown to be without foundation, and the same applies to the "creaming" theory for the increase in fat percentage during milking.

This book is an indispensable aid to the research worker into all aspects of lactational physiology, to the veterinarian who wants to understand the functional aberrations encountered in the udder, to the genetecist concerned with the heritability of milk quality and quantity: and to the dairyman who aims at obtaining the maximum quantity and highest quality of milk from his cows.

S.W.J. v. R.

PHYSIOLOGY OF REPRODUCTION AND ARTIFICIAL INSEMINATION OF CATTLE

G. W. SALISBURY and N. L. VAN DEMARK

W. H. Freeman & Company, San Francisco. 1961; 630 pp.; 170 illus.; Published price $12.50

The purpose of this book by two world famous authorities in the field of bovine reproduction is to present the pertinent literature of the past few decades for the benefit of all interested in the breeding of cattle: research workers, veterinarians, animal husbandrymen, artificial inseminations, technicians, stock owners, students and others.

When it is noted the bibliography given at the end of each chapter covers a total of 71 pages and contains nearly 2,000 references, it is appreciated that they have well succeeded in this task. This by no means implies that the book is a mere review of the relevant literature. It also

incorporates the author's findings and conclusions based on their long and wide experience as research workers and teachers, and it is in this that its real value lies.

The book is divided into three main portions: the first describes the anatomy and general physiology of the reproduction systems of the cow and the bull; the second deals comprehensively with semen and its components, and the third describes the many factors which influence the conception rate.

Most of the information contained in the first portion is readily available in other books on reproduction and is not of much assistance to the research worker or qualified person. Yet its inclusion in a work of this type is essential for the benefit of the untrained student, technician and others who have not yet received this elementary instruction.

The real value of this work, however is in the last two sections. Nine of the ten chapters covering 265 pages which constitute the second portion deal with semen: its components, morphology, metabolism, collection, evaluation, quality, dilution, preservation, and its physiology in the female genital tract; the remaining chapter gives a full account of the technique of insemination and related factors. This is admittedly an outstanding section which surpasses anything previously published on semen. It is obvious that most of the information imparted here emanates from the extensive research carried out by both authors on this particular branch of reproductive physiology, a field in which they are recognised authorities.

The third portion comprising five chapters over 175 pages gives a very full description of the different factors which may lower reproductive efficiency in both cows and bulls. It is gratifying to note the emphasis placed in good management and that attention is also drawn to the rôle played by psychological disturbances.

One adverse criticism that can be levelled against an otherwise comprehensive description of the factors involved in the depression of bovine fertility is the scant consideration given to the various infections to which the genitalia are subject. It may be that these have been largely brought under control by artificial breeding in America, but in most other countries they still constitute the principal cause of herd infertility. Only four, namely brucellosis, vibriosis, trichomoniasis and leptosipirosis in addition to non-specific infections, are briefly considered. The student in Africa would thus search in vain for information on the principal venereal disease of bovines in this continent, namely, contagious epididymitis and vaginitis, or on the various types of viral infection of the genitalia which have been described in recent years.

The research worker may consider the coverage of reproductive endocrinology to be inadequate, but the person who wants more can always have recourse to the numerous publications mentioned in the bibliography. On the other hand one notes with satisfaction that the authors have avoided the pitfall which claims so many victims among current less experienced writers in breeding problems, in that they are not over enthusiastic over the use of hormones in an attempt to improve reproduction efficiency. They rather advocate tackling the root causes of the physiological disturbances of which hormonal unbalance is but a

sequel, and warn against injudicions hormonal treatment, declaring with full justification that "many workers have found that no treatment at all is as effective as giving inappropriate hormone treatments" (p. 524).

This book is one which should occupy a prominent place in the library of everyone interested in bovine reproduction.

S.W.J. v. R.

REPRODUCTION IN THE DOG

by

A. E. HARROP

Published by Bailliere, Tindall and Cox, London. 1960

This book has been written not only for the research worker and the practitioner but also for the dog breeder.

If the book may be criticised, it may be said that a weakness lies herein. As however it is the first comprehensive book on Canine Reproduction in any language, it is a must for any veterinarian engaged in Canine practice.

Perusal of the table of contents, indicates the field covered, viz:-

Anatomy (macro and micro) of the organs of reproduction.
Physiology of reproduction.
Mating and pregnancy.
Parturition — normal and complicated.
Canine paediatruis.
The effect of Nutrition on Reproduction and Infertility in the dog and bitch.

It is hoped that future editions, will expand on the last chapter, which is the one requiring perhaps greater emphasis for the veterinarian.

This book can be strongly recommended for the bookshelf of any veterinarian in practice.

S. v. H.

LEECHES (HIRUDINEA). THEIR STRUCTURE, PHYSIOLOGY, ECOLOGY AND EMBRYOLOGY

By

K. H. MANN. 1962

International series of monographs on Pure and Applied Biology-Zoology Division, Volume II. 201 pages, 113 figures. Pergamon Press 45/-

To the general zoologist this book reads as easily as any thriller. The most striking feature being the lucid presentation of the subject matter.

The introductory chapter in itself gives a good indication of the fare presented. The chapters dealing with the morphology, classification and embryology are excellent in that much of the information so studiously learnt from textbooks, is pleasantly and painlessly strung together. The

adaptations of these animals to their environment is stressed in the relevent chapters on physiology, ecology and locomotion, although the latter suffers somewhat in comparison. Throughout the book comparisons are made with the annelida and the differences and similarities are stressed. There are two appendices dealing with systematics, one with freshwater and terrestial leeches, and the other, written by Prof. Knight-Jones, with marine leeches.

The author has certainly proved that the Hirudinea warrant more attention from both the general zoologist and the parasitologist. After reading this book the reader will agree with its author — "Leeches are fasinating animals, full of strange zoological paradoxes."

A.V.

ANIMAL PSYCHOLOGY

By

R. H. SMYTHE, M.R.C.V.S.

Published by Charles C. Thomas, Springfield, Illonois, U.S.A.

This book gives an interesting account of animal behaviour and a comparison with human ways and actions.

The author first gives his views on how to study animal behaviour, pointing out that behaviour should be studied when the animal is free in its natural environment and not when confined to cages, or kept in strange or unnatural surroundings. The effect caused on the behaviour of animals by differences in sensory perceptions such as sight, hearing, smell and sensitivity to ground vibrations as well as the range of vision, which is governed by the height of the eyes above the ground, is considered. The importance of hormone control of behaviour and instinctive behaviour for self preservation is discussed. Attention is drawn to differences in behaviour prior to and after puberty especially when the mating urge is strong. The usual behaviour of the horse, dog and cat is described extensively and that of some other animals briefly. The importance of maternal training is mentioned. The next part is about the mind, the advantage the human has in speech for training his young, the brain, thinking, instinct, needs, reaction to stimuli, the escape reaction and how animals learn. After this, animal perception or how animals become aware of objects, assess their significance and communicate with each other is described in fishes, snakes, lizards, insects, shrimps, birds, bats and the horse. A classification of animals into groups according to the time of day or night when they are most active is included. Pain in animals is discussed. Then comes a section on the homing instincts of dogs, the homing and migration of birds with the factors which could govern these movements. Social instincts such as congregation into groups, mass action against enemies, order of precedence, leaders, imitation, group behaviour to protect females, play group example, parental-training, mating, pairing, nest building, care of young, territory occupied as well as instinctive actions for self preservation including the importance of the sense of smell for recognizing friends, enemies, parents or offspring

and for pro-creation are described. The importance of contact between animals and between man and animals is mentioned. Finally instances of psychic, intelligent and temperamental behaviour of animals are given.

The book will be found interesting and useful by those who are interested in or have to look after animals and of practical value for those who have to handle animals and whose safety may depend on knowing what an animal is likely to do next or how it will react to stimuli. Most of the domesticated animals are mentioned but there is more about the horse and dog than the others. More information about habits of cattle, sheep, and goats and pigs would be useful. The author has covered a wide field and possibly attempted to deal with too much in one book by including birds, fishes and insects as well as wild animals likely to be in contact with or tamed by man. G.D.S.

HANDBOOK ON TROPICAL DISEASES

Issued by

THE BRITISH VETERINARY ASSOCIATION, 7 MANSFIELD STREET, LONDON W.1.

Price 30/-

This book has seemingly been prepared to serve as a nucleus of information to workers who find themselves in tropical countries, surrounded by a host of veterinary problems, all seriously menacing the health of the animal population.

The diseases are indexed and arranged in alphabetical order and listed as viral, bacterial fungal, reckettsial, protozoal, climatic endo- and ectoparasitic.

This arrangement is a great help in a handbook and gives the reader a quick and easy reference to tropical disease classification, thus enabling him to acquaint himself readily with his problems and encourage him to study them in any particular environment.

Over fifty diseases and nearly twenty climatic and parasitic threats to animal health in the tropics are covered in the 231 pages which the handbook contains. The information is therefore condensed to the essential facts.

The information is accurately given, and well presented, though in some instances, brief.

The chapters on internal and external parasites cover an enormous field in the thirty pages devoted to the subject.

A very useful description of the operation of cattle dipping is given under the heading of Control of Ticks. For those who have spent some time in tropical countries, cattle dipping has become a very commonplace practice, but to those who are new to the control of tick-borne diseases in the tropics the handbook is a valuable introduction to the problem.

There is a lot one can criticise in the book, largely due to the fact that scientific advancement frequently runs ahead of the opportunity to arrange its publication.

A short chapter on the legislative control of the proclaimed diseases in tropical countries and a reference to the export of animal products

would add to the usefulness of the book as an aid to the Veterinary Administrator engaged in the State Service of tropical countries.

The British Veterinary Association is to be congratulated on its effort to supply a handbook on tropical diseases and the contributors responsible for the information contained in it are to be particularly complimented.

It will be necessary to review the information contained in the handbook from time to time and as the publication is a soft cover the expenses in doing this would be greatly reduced. A.M.D.

VETERINARY SERVICES IN WORLD WAR II

MEDICAL DEPARTMENT, U.S. ARMY

This book is issued by the Office of the Surgeon General of the Medical Department of the United States Army. It was prepared and published under the direction of Lieutenant General Leonard D. Heaton. The Surgeon General, United States Army. The Editor in Chief was Colonel John Boyd Coates, Jr., M.C. and the Editor Colonel George L. Caldwell V.C., U.S.A. (Ret.). Its author being Lieutenant Colonel Everett B. Miller, V.C., U.S.A.

The volume is beautifully bound and well printed on excellent paper and comprises 800 pages with 102 illustrations, which are reproduced photographs, 10 maps, 8 charts and 58 tables. It is divided into twenty chapters and is well indexed.

As the title suggests, it deals mainly with the activities of the Veterinary Corps in World War II, although a few chapters are set aside to describe the Evolution of Military Veterinary Medicine 1775-1916 and the Development of the Army Veterinary Services 1916-1940.

It is interesting to note that in World War I an estimated 20 per cent of the Veterinary Corps personnel was utilised to inspect the Army's subsistence supply, whereas in World War II between 90 and 95 per cent were used for this purpose. Between 1940 and 1945 more than 142 billion pounds of meat and dairy products were inspected by the Veterinary Corps. The book covers in detail all the activities of the Veterinary Corps in World War II. For instance there are chapters on Laboratory Service and Research. (There were 11 such Laboratories in the United States and 23 units overseas).

Animal Procurement, Animal Care and Management, Transportation of Animals (by ship, railroad, truck, aeroplane and road march); evacuation and hospitalization, army dogs, army signal pigeons, animal farms, captured animals and privately owned animals are all carefully attended to.

There is no doubt that the author devoted much time and effort to the preparation of a permanent record of the activities of the United States Army Veterinary Service in World War II: a record which is outstanding and brings much credit to that Corps.

In the words of Lieutenant General Leonard D. Heaton: "It sets a pattern for emulation by those responsible for planning Veterinary programs for the future." J.Z.

PUBLIC RELATION SERVICE

NEWS OF MEMBERS

1. Dr. L. W. van den Heever has returned from his trip overseas and reports as follows on the Bicentenary celebrations of the World's First Veterinary School — *Ecole Nationale Veterinaire de Lyon*, France.

"During my recent visit to several European veterinary faculties I also visited Lyons and had the privilege of attending their 200th celebrations.

As requested by Council, I presented the Director of the School with a congratulatory address from the S.A.V.M.A. (copy appeared in March, 1962 issue of the Journal) at an official ceremony at which most of the countries of the civilised world were represented.

The Director thanked the Association for the address and requested me to convey his appreciation to the Association. He also presented me with the enclosed commemorative medallion with the request that it be handed to the South African Veterinary Medical Association.

The inscription on the medallion reads:

'1962

The Bicentenary of the World's first veterinary School, founded at Lyon by Claude Bourgelat in 1762' ".

(NOTE.—The Medal will be on exhibition at the next Congress—Ed.)

2. Dr. R. B. Cumming, now living in Australia (University of New England, Armidale, N.S.W.) writes as follows (11th July, 1962).

"My apologies for the long delay in writing to you about the Australian Vet. Association's A.G.M. in Melbourne.

I attended all the functions which were very well organised and the refreshments were quite outstanding, particularly the wine-tasting evening: As the S.A.V.M.A. representative, I received complimentary tickets to the various functions which was greatly appreciated by me.

The meeting was very successful, particularly the session on pathology. By and large pathology in general and virology in particular have been neglected in Australia by Veterinarians but there is a rapidly widening interest in there field again.

All the papers and proceedings of the meeting are recorded in the Australian Vet. Journal, so I need'nt comment on them. The session on C.R.D. was well attended (about 40 vets) and we had a useful discussion.

My battle to have our degree recognised in Victoria is progressing slowly and I am rapidly gaining support, outside Victoria, for my point of view. I am fairly confident that the degree will be recognised within the next two years.

The 12th World's Poultry Congress is being held in Sydney in August and I am looking forward to meeting the South African contingent. One of the five symposia is on C.R.D. and I have been invited to speak along with Dr. H. A. Adler of California and Dr. H. P. Chu of Cambridge. I feel out of my depth in such exalted company."

3. Dr. "Mike" Brown has returned from a course on Nuclear physics and isotopes arranged at Cornell University. We trust he will tell us all about it at some convenient time.

INTIMATIONS

1. NOTIFICATION OF PROCLAIMED DISEASES

The notification of proclaimed Animal Disease will henceforth be intimated by circular letter and not by publication in the Journal.

2. REQUESTS TO AUTHORS

Reprints of *Request to Authors*, published in the June 1962 issue of the Journal (page 274-276) are available from the Secretary.

3. The College of Veterinary Medicine Budapest, Hungary, celebrates its 175th Anniversary during September 1962.

The Council of the S.A.V.M.A. has sent a message of congratulations to the Dean of the College.

BACK NUMBERS OF THE JOURNAL REQUIRED BY THE EDITOR

The Editor will be glad to purchase the following back numbers of the Journal at 75c each.

1959 Vol. XXX (4) — December Issue.
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Bluetongue	3 months
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Fowl Pox	3 months
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Lumpy Skin Disease	3 months
Rabies	3 months
Newcastle Disease	3 months
Distemper	1 month*
Enterotoxaemia	6 months
Lamb Dysentry	6 months
Lamsiekte	6 months
Black Quarter	6 months
Anthrax	6 months
Calf Paratyphiod	6 months
Brucella (Str. 19)	1 month
Brucella (Rev. I)	1 month

*The expiry period of 1 month for Distemper vaccine is applicable when the vaccine is stored at 4°C in an ordinary refrigerator. If the vaccine is stored in a deep-freeze at 18; the vaccine can be kept for 3 months.

Although the expiry periods for the other vaccines have been calculated for storage on the shelf, it is nevertheless advisable to store all vaccines, which are not used immediately, at temperatures ranging from 4 to 8°C.

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