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Edited by M. STERNE.

for the

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PRETORIA.

South African Veterinary Medical Association Annual Meeting held at Onderstepoort in September, 1937, under the Presidentship of Mr. S. T. Amos, F.R.C.V.S.

PRESIDENT'S ADDRESS.

It is my honour and privilege to give you a short review of the last year's working of your Association, and generally to allude briefly to some interesting points in the progress of the veterinary profession in South Africa.

There was undoubtedly, and there still may be, a feeling amongst some members of the profession that the progress was not altogether of as high a standard as members would naturally wish it to be, but I think that, when we think over the situation, we cannot but be pleased in many respects with what has been done by the profession in this country of ours.

One must remember, at the outset, that we are a comparatively young body, and we might even say to-day, that the veterinary profession is still in embryo in South Africa. South Africa demands special consideration from our professional point of view; the conditions are peculiar and very unlike those of many other countries. Diseases are many, distances are tremendous, and your veterinarians for such conditions have been, and still are, very few indeed.

I think one should remember that there existed in this country a very big prejudice against the so-called expert, and it will be a number of years before that prejudice is entirely broken down; but it will never be entirely broken down unless the veterinarian can prove his value to his community by his technical advice, his general understanding of the people concerned, and his tactful enforcement of the laws governing stock diseases, and this, may I remind you, is by no means an easy proposition. It is one that calls for constant care on the part of the administrating officer. The profession may be made or damned in any district by a thoughtless veterinarian, and to the younger members of the profession I would urge that the veterinarian, apart from his technical knowledge, should have a knowledge of human psychology, and that these two factors should always be used in harmony.

There is no doubt of the value of the work that the veterinarian has been able to do in this country. There is no doubt about the future utility of the work, and every year new milestones are being reached and passed. It only remains for us as members of the profession to see that each milestone is a monument to our usefulness to our community, and not just one that we have passed by never to look at again.

This year, in particular, one of the most important milestones has

been passed, namely the reciprocity which has been established between the Veterinary Faculty in the Union of South Africa and the Royal College of Veterinary Surgeons. This to some may mean nothing, but to others it is a very great achievement and I, personally, being a graduate of the Royal College of Veterinary Surgeons, am delighted to think that at last this portion of the British Empire acknowledges in its fulness the Diploma that was given to me as far back as 1897, wherein it was stated that I was free to practice the art of Veterinary Surgery in any portion of the British Dominions. I am sure it is equally pleasing to the graduate from this University of Pretoria to feel that he is now at liberty to take the M.R.C.V.S. as soon as he arrives in Great Britain. Reciprocities such as this can only lead to better and bigger values for everybody concerned, and in passing, I would like to pay tribute to the unremitting efforts made by Dr. du Toit and the members of the Veterinary Board to bring about this desirable end.

Another milestone that has been reached is the working of the Veterinary Act of South Africa. Everyone admits that the Act does not fully protect us, but it is a great step forward to have the Act actually on the Statute Book, and it is pleasing to be able to report again the vigilance of your Association and the Veterinary Board in trying to get passed the amendments which will give us a fuller protection in all our work. I will not enter into details of these amendments, because it is a subject that could be discussed for many hours. Sufficient be it, therefore, for me to refer briefly to the promises that we have already received from the Veterinary Board that definite amendments to the Act are to be hoped for within a short space of time.

The first prosecution under the Veterinary Act was undertaken by the Veterinary Board three months ago and a conviction obtained against a person who stated he was an M.R.C.V.S., when he had no such qualification.

Attention has been drawn to many instances where chemists have distributed certain drugs to the lay public although manufacturing chemists advertised that these drugs would only be supplied to the veterinary profession. To my mind this is a distinct breach of faith, and members who encounter such actions as these can help the position a very great deal if they will appraise the local president of the Pharmaceutical Society in their district of the facts. In Natal this has been done in several instances and, without any fuss at all, an improvement has been made.

Another frequent nuisance is the interference in veterinary work by chemists and dog traders generally, who have no hesitation in diagnosing and in selling a supposed remedy for the condition. An amendment to the Act is definitely necessary in this respect. I have personally encountered the interference of medical men in ordinary veterinary practice.

It is by no means uncommon for an owner of a horse with a damaged eye to want the opinion of the ophthalmic surgeon, and whilst I have no objection whatever to acting in consultation with a medical man, I have a definite objection to a medical man undertaking veterinary work without my leave, when I have already been in charge of the case. I find, also, that medical men are prone to operate on dogs, which, to my mind, is entirely wrong. I can understand a surgeon wishing to operate on his own animal, but to operate upon another person's animal is in my opinion a distinct contravention of professional etiquette, and when such conduct is encountered the best results can be obtained by firmly but courteously pointing out the mistake.

This brings me to the courtesy title of " Doctor". A referendum was taken during the past year and this showed a small majority against the use of the title. It was interesting to note how close the voting was, showing that in many districts veterinarians do definitely work under a disadvantage if they cannot be referred to as " Doctor", and this again is one instance that proves the unusual conditions prevailing in our country districts. I should not be at all surprised if, within the next few years, the question of the courtesy title were again raised, in view of the fact that there were almost as many votes cast for the proposal as against it.

Now, to come to the actual working of our Association. I would point out to members that the Association exists entirely for your benefit and the benefit of the profession in general. I must, first of all, pay tribute to the officials of the Association. I do not think the amount of work that is done by these men is fully appreciated by the members of the Association. Take, for instance, the excellent work done by Dr. A. D. Thomas during his period of editorship of our Journal. Few of us can realise the amount of time and thought that was given by this gentleman to the work of the journal, and he is deserving of our heartiest appreciation for bringing the journal through a very critical time, and I am sure I would be wanting in my duty if I did not make a special reference to him. Then take the work of your Secretary, Mr. van Rensburg. I can personally vouch for his unending work, and the courtesy that he has shewn to us all. Few of the members realise that this voluntary work is carried out entirely for their benefit and entirely at the expense of the energy of the volunteer.

The one blot on the Association is the non-payment of subscriptions by members. Is it fair to ask these volunteers to be still further overworked by having to make many appeals to members for their annual subscriptions? It is time that we all realised that the Association is working for the good of every member. If a man does not wish to become or to remain a member of the Association, then it is far better for him to remain a non-member, or to resign; but to put unnecessary

labour on the shoulders of the voluntary officials of a professional association is, to my mind, most unfair.

We must see that our benevolent fund is kept on the up-grade. None of us know when we may have to appeal to such a fund either for ourselves or our dependents, and if we are a profession worthy of anything at all, we should see that there is in existence a proper benevolent fund. I would therefore ask members to remember the fund whenever good luck comes their way.

Your Association has always had before it in proper perspective the emoluments payable to veterinarians throughout the country. The private practitioner is being helped by the establishment of a uniform chart of fees. The Government Veterinary Officer in common with other civil servants has been protected by the efforts of the representatives of your Association on the Pro-technical Section of the Public Servants' Association, and it is gratifying to note that, almost entirely due to the effort of the Director of Veterinary Services, the commencing salaries and the top salaries of the Government Veterinary Officers are appreciably higher than was proposed originally.

In conclusion I would urge all members of our profession in South Africa to try and work harmoniously together, never forgetting the objects of our profession and never forgetting that the greatest good can only be brought about if the greatest number of the members benefit thereby, and that although an individual success, or an individual superiority can be appreciated, it is a far greater thing if it is shared by everyone in the profession.

In this country we must be very careful indeed that our relation with the community brings respect to the veterinarian administrator and to the Veterinary Profession of the Union of South Africa.

XIIIth International Veterinary Congress, Zurich—Interlaken, August 21st to 27th, 1938.

The discussions will take place in Zűrich and in Interlaken. Special concessions on the railways have been procured for members attending the congress and a number of expeditions have been arranged to different parts of Switzerland. The reports and papers will be of a practical nature and will have a wide appeal to veterinarians, whether engaged in laboratory work, field work or in private practice. Certain sections of the discussions are of particular interest to the South African worker and it is gratifying to notice the prominence given to reporters from South Africa. Further particulars about fees, membership, fares and hotel tariffs can be obtained from the Secretary of the S.A.V.M.A. The Secretary has also a number of copies of the *Preliminary Announcement* of the congress and these can be obtained on request.

Jl. S.A.V.M.A.
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1938.

Speculations on the Incidence of Anthrax in Bovines.

By R. CLARK, B.V.Sc.

The object of this paper is not to prove any facts about the incidence of anthrax and I therefore offer no apologies for its vague and inconclusive nature. The article was written to crystallize my ideas on the subject and so to formulate some scheme for further investigation. It is, however, felt that by reading it I may stimulate discussion among my colleagues and so important information may be gained.

The local conditions under which an outbreak occurs are of very great importance and so local knowledge is essential before conclusions can be drawn from statistics. It is a very common experience in the field to receive notification that a smear sent in from a certain farm shows anthrax, and, when enquiries are made, to hear that the owner, suspecting anthrax, burned the carcass and dispatched the smear. Although this may have happened two or three weeks previously no further deaths have taken place. All the animals on the farm are then inoculated and no further cases occur. It is then very difficult to accurately assess the effect of the vaccination. Further, the owner is usually amazed that anthrax has appeared on his farm as he has been there for so many years and has never had it before.

A consideration of these facts led me to draw up Table I. This gives the details of the known outbreaks of anthrax in the districts of Vrede, Frankfort, Reitz, Bethlehem, Harrismith, Fouriesburg, Ficksburg and Ladybrand in the Orange Free State for the period 1933 to 1936.

It would be interesting to find out what would happen if immunization were not carried out and therefore a column is included giving the period in days, that susceptible animals were on the infected farm after the first death, *i.e.* after known infection. For this purpose it is assumed that the animals are immune fourteen days after inoculation.

It must further be remembered that this table only includes outbreaks where the diagnosis was confirmed by smear examination. There is no doubt that many cases of anthrax are never reported, especially in such areas as the one under consideration where compulsory forwarding of smears is not in force. Yet one is very seldom called out to investigate heavy mortality in cattle due to anthrax. It was also assumed that many owners of stock knew the disease and inoculated on their own without reporting it; yet now that a record is being kept of all orders for anthrax vaccine, this has not shown an extraordinary number of "suspicious

orders " nor large outbreaks of anthrax. Another noteworthy point is that practically all outbreaks showing a high mortality will be reported, while probably only a very small proportion of single deaths will be noted. The actual preponderance of single-death outbreaks is therefore probably very much higher even than is shown in the tables.

It will be noted from Table II that out of 100 outbreaks in which the total number of deaths is known, 47 showed one death, and 76 showed one to three deaths, while only 24 showed more than three deaths. This is in spite of the fact that the cattle were not immune for periods ranging from 15 to 42 days after the first appearance of the disease.

Table III shows that out of 39 outbreaks, where there were more than one death, in 20 cases (51%) all the deaths took place in one to five days, while in 28 cases (72%) all the deaths took place in 10 days or under. As the incubation period has been given as one to ten days, it might be argued that in these cases infection may have taken place at almost the same time.

In one outbreak with heavy mortality (22 deaths) it is known that the first carcass was cut up. On another farm where this happened three non-immunized animals introduced later died. In another case 53 pigs died after being fed the carcass of a bovine which had died of anthrax. On one farm floods preceeded an outbreak which caused the loss of 20 head of cattle. These facts show the high infectivity of carcass material.

The object of this preamble is to show that, provided the carcass of an anthrax animal is not cut up and distributed over the countryside, anthrax *usually* appears naturally almost as a sporadic disease.

Two queries therefore must be answered, namely :—

What is the usual mode of infection ?

What is the actual value of the vaccine under field conditions ?

Mode of Infection.

As biting flies do not appear to play an important rôle in the area under consideration, this means of infection will not be taken into account here.

A consideration of the foregoing facts leads one to the conclusion that the infection is not evenly distributed over the farm and that it is not equally accessible to all the cattle. The factor of individual resistance must of course be remembered here, but for the purpose of argument we will assume that the individuals of a non-immunized herd are more or less equally susceptible.

Infection through drinking water.

The aforegoing paragraph would appear to negative the suggestion that drinking water plays a big part in the spread of anthrax. If a dam

were infected one would expect either a large number of animals to contract the disease at the same time or isolated cases at short intervals. Neither of these is the rule. Also it will be seen from Table V that the outbreaks in March and April, when the dams in these parts are recently washed out and full, are as great as in August and September, when the dams are low and much mud is being stirred up. Moreover, sheep and horses usually water at the same place as the cattle do, yet the incidence of anthrax among these species is very low. The rarity of anthrax among sheep and horses in this area is of very great interest. It is often argued that as smears are not usually taken from sheep, the disease is not diagnosed. It can definitely be stated, however, that both in the eastern Transvaal and eastern Free State anthrax rarely assumes epidemic proportions among sheep. I make this statement as in both of these areas most of my time has been occupied investigating mortality in sheep and I have never yet found it due to anthrax, although I have always taken smears in cases where the disease could reasonably be expected. When a bovine dies of anthrax the sheep are very seldom inoculated, yet I have never received complaints that sheep are dying on the infected farms.

Infection through grazing.

If infection is through spores on the grass it might be argued that patches of grass would be infected where a death took place. Even then, under the intensive grazing conditions now in force, it could hardly be maintained that in 47% of cases only one beast happened to graze over this infected patch in a period of two to four weeks. The low mortality in sheep is also an argument against this mode of infection.

It has often been said that the young shooting grass brings up spores with it. The high incidence of outbreaks in September, October and November, shown in Table IV, would tend to substantiate this, but the second high incidence in February, March and April is not then explained.

Pica.

When all the above factors are taken into consideration it seems almost certain that in most cases infection in the bovine is due to the intake of a mass infection obtained from actual infected material, *i.e.* bones, skins, etc., and is connected with pica. This hypothesis would explain the sporadic nature of the disease, the fact that cattle are so generally affected and the fact that deaths so often occur in batches at short intervals. That Table V shows the fewest outbreaks in mid-winter might lead one to doubt this hypothesis, but it must be remembered that during this time cattle on the highveld are in the lands feeding on old mealie stalks grown on phosphorus fertilized land where old bones are generally not frequent. The high incidence shown in early summer when the grass is still short, the fall in December and January and the rise in February and March, when the grass is seeding, might all be taken to lend weight to the impression that pica and anthrax are connected.

In order to put this theory to the test Table IV was drawn up, showing the relative prevalence of anthrax and phosphorus deficiency in the different areas of the Union. The number of outbreaks in the different Senior Veterinary Officers' areas were kindly supplied by the Director of Veterinary Services. The figures cover the years from 1933 to 1936. The pasture analyses were taken from the 18th Report of the Director of Veterinary Services (1932) and sorted out into the same groups. The average of each group was then taken.

Very little significance is claimed for this table, but it is included as a possible indication for further investigation along this line. The areas are naturally too great and too varied to be taken as groups. Further, that most important point, the prevalence of the causal organism, is not taken into account. To my mind, this is the great problem, to demonstrate the natural occurrence of the organism. As later shown we often get cases of anthrax of unknown origin. In these cases, has the infection been recently introduced, or has it been present for a long time and not been picked up ? If the latter, how does it suddenly infect cattle and where do they pick it up ? It is naturally very difficult to elucidate these questions. The only point of interest in Table IV is the low incidence of anthrax in the Cape Eastern districts correlated with a high phosphorus content of the grazing as compared with other areas. On the other hand Cape Western area shows a low anthrax incidence with a low phosphorus average (1).

Regional Occurrence.

When the outbreaks were plotted on the map, it was found that about 50% of them fell into groups of from three to eight outbreaks. The remainder were dotted about the map. The dates of the appearance of the disease in these groups frequently run right through the period for which data are available, *i.e.* from 1933 to the present, but there are often several infections at short intervals. The following is a typical example of seven infected farms lying close to each other.

FARM NO.	DATE OF OUTBREAK.
1.	October, 1933.
2.	March, 1936.
3.	March, 1936.
4.	April, 1936.
5.	June, 1936.
6.	June, 1936.
7.	November, 1936.

(1) During the discussion, Dr. A. I. Malan pointed out that in the case of the Western Province, the phosphorus content of the pasture could not be taken as an index of pica among the cattle, as artificial feeding was the rule.

In my opinion where outbreaks appear on neighbouring farms and at short intervals, infection is usually spread by natives carrying meat about. Where there is a long interval between the outbreaks it may be due to an old infected bone being washed out or otherwise unearthed. Where anthrax appears suddenly in a clean area it is usually impossible to trace the source of infection.

Seasonal Occurrence of Anthrax.

Table V shows a drop in the number of outbreaks during the depth of winter, *i.e.* May, June and July. As stated previously, freedom from the disease in mid-winter may be attributed to the practice of putting the cattle into the lands during this time.

If we compare the numbers of outbreaks for each month with the rainfall we find that there is apparently no connection. The rainfall for the year does not appear to have any effect on the total number of outbreaks. Further, August shows the highest total of outbreaks (17) for any single month over the four years and only 0.1 inches of rain for the same period. The rainfalls given in Table V were taken at Bethlehem. The figures would doubtless vary considerably over the south-eastern Free State.

Value of Vaccination.

The value of vaccination is most clearly demonstrated in outbreaks where a large mortality takes place. In five such outbreaks, each of which showed five or more deaths, mortality ceased from 4 to 15 days after inoculation. These instances show that the vaccination protects against a mass infection probably in less than the recognised period of fourteen days. A further demonstration of the value of the vaccine is the fact that in ten cases non-immunized cattle brought on to infected farms died of the disease. It is of interest to note that only on one farm did these cases occur where there had been heavy mortality (8 deaths). This can be attributed to the fact that where there have been heavy losses the owner is careful to inoculate all new arrivals.

The routine is to carry out inoculation on infected farms every twelve months. Although the owners are warned to notify the veterinary office about all newly introduced animals and to inoculate them, it is certain that many cattle are frequently brought on to infected farms and kept there till the following annual inoculation. The fact that there are so few deaths among these cattle, shows that the freedom from the disease cannot be wholly attributed to the vaccine.

The cases where breakdowns in immunity occurred are also of great interest. Out of twelve such cases noted in Table I, eight were with vaccine Batch 91 (1936), one with Batch 93 (1936), one with Batch 97 (1936), and two with Batch 5 (1937). It would therefore appear that Batch 91

(1936) was deficient in antigenic properties. Single breakdowns with individual batches may always be attributed to one of the following factors, *viz.* the particular animal may not have been inoculated, individual idiosyncrasy or a gross infection. No batch can in any case give 100% protection.

Conclusions.

1. It is argued that most cases of anthrax in bovines are contracted through the chewing of anthrax-infected bones or other carcass material and not through infected grazing or water.

2. The cessation of deaths on many farms after a case or cases of anthrax which have been properly disposed of cannot be wholly attributed to the vaccine.

3. The value of vaccination in mass outbreaks is clearly demonstrated.

4. Anthrax is most prevalent in the summer, but rainfall appears unrelated to the incidence.

5. Many outbreaks of anthrax are probably carried from nearby infected farms, but the origin of most is obscure.

TABLE I.

Period in days during which animals were susceptible.	Period in days during which deaths occurred.	Number of deaths.	Contacts.	Period in days during which animals were susceptible.	Period in days during which deaths occurred.	Number of deaths.	Contacts.
21	4	8	80	20	?	4	170
16	?	5	28	19	1	1	186
18	17	4	16	22	1	1	66
19	1	1	230	19	1	1	140
15	7	22	130	17	9	4	140
17	1	1	150	?	60	11	150
16	3	2	50	28	?	3	?
21	5	4	153	19	?	4	537
15	2	2	120	19	?	6	95
15	2	2	83	?	1	1	78
22	1	1	185	15	1	1	120
20	10	7	78	?	1	1	340
22	1	1	18	22	1	1	420
20	1	1	150	29	?	2	33
19	1	1	32	19	3	2	507
26	2	2	200	18	1	1	700
23	24	6	219	22	1	1	120
20	5	7	185	30	1	1	452
18	1	1	357	29	?	28	427

TABLE I — (Continued).

Period in days during which animals were susceptible.	Period in days during which deaths occurred.	Number of deaths.	Contacts.	Period in days during which animals were susceptible.	Period in days during which deaths occurred.	Number of deaths.	Contacts.
15	1	1	220	27	?	2	160
16	1	1	155	26	1	1	84
28	14	?	715	37	19	2	68
15	3	3	250	31	4	3	142
18	1	1	178	16	4	2	300
16	4	3	54	19	2	2	203
15	1	1	181	26	1	1	576
17	1	1	19	24	1	1	300
31	1	1	68	31	1	1	261
16	3	2	61	22	8	2	65
25	1	1	425	19	25	5	50
15	1	1	100	37	1	1	160
20	3	3	146	24	?	7	52
28	1	1	500	19	6	2	86
35	19	4	203	31	1	1	173
16	6	3	116	22	1	1	133
15	2	2	127	20	1	1	179
18	3	5	200	21	?	2	170
15	5	20	480	25	1	1	175
17	?	3	27	38	?	2	78
18	?	3	85	27	13	3	133
19	?	3	262	17	1	1	160
14	1	1	32	19	?	3	84
20	1	1	170	16	1	?	445
24	9	3	29	27	1	1	132
23	1	1	60	15	1	1	123
36	?	2	50	24	?	3	46
24	1	1	7	35	?	7	48
23	2	5	67	42	1	1	31
17	11	6	211	16	18	2	1500
19	1	1	259	28	16	3	184
19	2	2	654	26	1	2	133
20	1	1	73	24	6	4	109

Each line represents a separate infected farm.

Column 1 represents the period in days between the first death and 14 days after inoculation.

Column 2 represents the period between the first and last death.

TABLE II.

Showing the number of deaths per outbreak.

Number of deaths.	1	2	3	4	5	6	7	8 to 28	
Number of farms showing above number of deaths.	47	18	11	6	4	4	3	7	Total 100.

TABLE III.

Showing the period between the first and last death in outbreaks with more than one death.

Period of deaths in days.	1 to 5	6 to 10	11 to 15	16 to 20	21 to 60	
Number of farms showing the above period.	20	8	3	5	3	Total 39.

TABLE IV.

Showing the incidence of anthrax and the average phosphorus content of the pasture in the different areas of the Union.

District.	Total Outbreaks.	Average P. content of Pastures.
Transvaal	655	.20 (low)
Natal	431	.15 (low)
O.F.S. and Bechuanaland	385	.19 (low)
Transkei	374	.17 (low)
Cape Western	31	.24 (low)
Cape Eastern	68	.35 (low)

TABLE V.

Showing Seasonal Occurrance of Anthrax.

Month.	1	2	3	4	5	6	7	8	9	10	11	12	Total
1933:													
Rainfall	2.8	3.6	3.0	1.3	—	—	—	—	—	1.6	4.6	4.5	21.4
Outbreaks	—	1	2	—	—	—	—	—	5	6	2	—	16
1934:													
Rainfall	6.8	3.4	5.3	1.2	3.2	—	1.5	3.8	—	3.9	9.6	5.3	44.0
Outbreaks	—	2	2	1	2	1	1	1	—	1	—	1	12
1935:													
Rainfall	2.3	1.6	5.5	1.6	0.1	—	—	—	0.1	1.6	2.2	1.7	16.7
Outbreaks	4	2	—	—	—	—	3	3	3	—	3	2	20
1936:													
Rainfall	2.7	3.5	4.0	1.6	3.0	—	—	—	—	2.8	7.8	3.1	28.5
Outbreaks	5	7	11	10	4	5	1	3	9	6	7	4	72
Totals:													
Rainfall	14.6	12.1	17.8	5.7	6.3	—	1.5	3.8	0.1	9.9	24.2	14.6	—
Outbreaks	9	12	15	11	6	6	5	7	17	13	12	7	120

THE ARMY VETERINARY SERVICE IN SOUTH AFRICA 1881—1914 (Concluded).

By H. H. CURSON.

CHAPTER III (Concluded).

(g) Special Features in Reports.

Matters of particular interest not referred to previously are as follows :—

Report for year ending 31.3.1904.—(i) In Great Britain concern is shown at the introduction of epizootic lymphangitis from South Africa, and on page 4 occurs the following passage : " up to 1902 it was unknown in the United Kingdom, and presumably South Africa, into which latter country it was introduced, in all probability, by mules from Italy or Cyprus." In P.V.O. Smith's report from South Africa, however, we learn (p. 11) " in Cape Colony and Natal *it was known* " before the War " and I have the assurance of Dr. Theiler that it was unknown in the Transvaal."

(ii) The O.C. A.V.S., Egypt, Major E. J. Lawson, is designated S.V.O. ! He states that the station veterinary hospital system is " working admirably ".

(iii) A suggestion that an A.V.D. officer should from time to time be allowed to study veterinary pathology and bacteriology in Dr. Theiler's laboratory, which " has a European reputation " was not only approved by the G.O.C. South Africa, but was " met in a most liberal spirit by the Agricultural Department of the Transvaal " ([78]). It was hoped to tackle specific ophthalmia and epizootic lymphangitis (p. 17).

Report for year ending 31.3.1905.—(i) The Army Council draws the attention of G.O.C.'s to the mortality, chiefly from pneumonia, among cavalry remounts " during the first two months after purchase "—usually from Ireland (p. 4).

(ii) Reference is made to the institution of an A.V.C. and to the establishment of station veterinary hospitals at Woolwich, Aldershot, and Bulford.

(iii) Attention is drawn to Theiler's method of immunising horses and mules against horsesickness.

([78]) Sir Frederick Smith on several occasions personally paid a tribute to Sir Arnold Theiler's assistance, particularly in the re-editing of his *Manual of Veterinary Hygiene*, first published in 1887.

Report for year ending 31.3.1907.—(i) In this return, which is Smith's first report as D.G., A.V.S., " the system of statistical analysis has been revised . . . and an endeavour made to emphasize those losses which may be regarded as preventable." A table dealing with sickness, mortality, and inefficiency in each unit in the United Kingdom " should be a true index of the horsemastership in each unit."

(ii) Smith draws attention to Lieut.-Col. Blenkinsop's able report from South Africa.

Fig. 26.
P.V.O. FREDERICK SMITH (1857 – 1929).
Taken from *Vet. Jl.*, Dec., 1907.

(iii) Blenkinsop recommends immunisation of mules against horse-sickness.

Report for year ended 31.3.1908.—(i) Smith emphasises the fact that " there is still great room for the prevention of disease and accident. In these matters we have not always made improvement, while in others, notably in sanitation, we have actually gone back ". He presents a chart showing that the principal causes of admission to hospital in the United

Kingdom (percentage of average strength) are in the following order: injuries (over 50%), lameness (over 20%), respiratory diseases, digestive diseases, skin diseases, and finally infectious diseases.

Report for year ended 31.3.1909.—(i) Smith reports that the death-rate and castings for disease in the United Kingdom "are materially reduced".

(ii) The return contains "the last Annual Report on the working of the Army Vaccine Institute", which was founded by Smith in 1888. "The supply is now being taken over by the Local Government Board".

(iii) In South Africa "not only are the deaths, destructions and castings for disease considerably reduced," but "epizootic lymphangitis, glanders, mange and osteoporosis no longer exist".

Report for year ended 31.3.1910.—Veterinary statistics are compared with those of foreign armies, mainly to the disadvantage of the British A.V.S.

(h) Special Orders and Reports.

When circumstances demanded, *e.g.* undue prevalence of a disease, special orders were issued by the authorities ([79]). These were :—

CIRCUMSTANCES.	AUTHORITY.
1. Marked prevalence of Mange.	*Rpt. A.V.D.*, 31.3.1904, p. 14.
2. Precautions for Horsesickness.	Do. p. 12
3. Precautions for Epizootic lymphangitis.	General Order, August, 1904. *Rpt. of A.V.D.*, 31.3.05, p. 14.
4. To keep mallein reactors for treatment. This was rejected in South Africa as hospitals were not finished ([80]).	War Office letter No. 116, Gen. No. 4990 (V.D.) dated Oct 28th, 1904. See *Rpt. of A.V.D.*, 31.3.05, p. 19.
5. Report by Capt. Lane on Osteoporosis.	Do. p. 28.
6. Precautions for Horsesickness.	Admin. Cir. Memo No. 21, published with General Orders. Nov. 15th, 1906.
7. Do.	Admin. Cir. Memo No. 27, published with General Orders. Dec. 5th, 1907.
8. "Forbidding docked horses' tails to be cut".	General Order, Oct. 1908. See *Rpt. of A.V.S.*, 31.3.1909.
9. Change of forage recommended, "*viz.* whole mealies for crushed and lucerne hay for oat".	*Rpt. of A.V.S.*, 31.3.1910, p. 77.

([79]) Not falling into this category was the order issued Apr. 5th, 1904, by the Board of Agriculture and Fisheries, United Kingdom, declaring epizootic lymphangitis a scheduled disease under the Contagious Diseases Act. The first case in the United Kingdom was reported on Sept. 17th, 1902, in a horse belonging to the A.S.C. stationed at Aldershot (*Rpt. of A.V.D. for year ending* 31.3.1904, *p.* 4.)

([80]) See *Vet. Rec.* of 13.12.02 for *Instructions for the Eradication of Glanders from Regiments and Units.*

(i) Veterinary Hospitals.

Number.—The experience gained in the war had thoroughly demonstrated the need for central veterinary hospitals in preference to regimental infirmaries. At the beginning of 1901, in addition to the large stationary hospital at Elandsfontein, there were 38 field hospitals "dotted all over the country and varying in strength from 300 patients to 3,000" (Smith, p. 197). At the end of May, 1902, these numbered 50. "By December, 1902, the fifty Field Veterinary Hospitals had been disbanded . . . The

Fig. 27.
P.V.O. JOSHUA A. NUNN (1853 - 1908).
Taken from *Vet. Jl.*, Mar., 1908.

stores in the various advance Depôts were distributed among the twelve Station Hospitals and four mobile hospitals so that each should possess an independent reserve in the event of difficulties arising. The base depôt at Capetown was retained ([81]), and into it all material surplus to the above requirements was returned". But by March, 1903, the figure "fell

([81]) The Base Veterinary Store at Capetown was closed during Col. Appleton's period as P.V.O. but when the Great War broke out the Base Depôt was reopened by the S.A.V.C. at Maitland, near Capetown.

to nine, the number then fixed as the future establishment for the country" (Smith, p. 224). These hospitals were of course located at the more important garrison centres.

"Early in 1904 an establishment of eight veterinary hospitals in South Africa was approved, but before any definite action could be taken it was decided that, as a reduction of the number of animals was probable, five hospitals would suffice for the requirements of that Command; these were formed by transfers from other units in a manner adopted at home,

Fig. 28.
P.V.O. LAYTON J. BLENKINSOP (born 1862).
Taken from *Vet. Jl.*, Feb., 1921.

the total establishment of N.C.Os. and men for that country being :—

Farrier Quartermaster-serjeants	5,	Serjeants	10,
Serjeant-farriers	5.	Corporals	18,
Shoeing-smiths	10,	Privates	30."

(*Blenkinsop and Rainey*, p. 2.)

"Towards the end of 1904 . . . the Treasury was approached with a view to the increase and reorganisation of its numbers and its division into *definite units*. This received sanction in February, 1905 . . . but only . . . by the sacrifice of fifteen officers . . ." (Blenkinsop and Rainey, p. 2). After this, officers "were allotted to stations and not to units" (Smith, 1937, p. 211). Later, 1906, "it was found that the term 'Veterinary

Field Sections I and II' was inconvenient and failed to convey any definite idea as to what constituted a unit of the corps. Consequently it was decided that the term 'Veterinary Section' should be used in future for what was previously a section of a field veterinary hospital and that such a veterinary section should constitute a unit of the corps, and be given a definite establishment of :—

Veterinary officers	2	Saddlers	1
Serjeants	1	Corporals	2
Farrier-serjeant	1	Privates	12
Shoeing-smiths	2	Horsekeepers	83

(specially enlisted or reserve)."

(*Blenkinsop and Rainey*, p. 3.)

During peace one veterinary officer would suffice, and the saddler and horse-keepers would be provided on mobilisation. In South Africa native personnel was also available.

As to the exact distribution of the veterinary hospitals definite information is not always available in the *Reports of the A.V.S.* According to *Das Militär Veterinärwesen* (Goldbeck, p. 72, 1908), there were in 1905 the following veterinary sections of the A.V.S. in South Africa :—

No. 9 at Pretoria with Capt. J. J. B. Tapley as O.C.
,, 10 at Standerton with Capt. G. P. Knott as O.C.
,, 11 at Potchefstroom with Maj. T. Marriott as O.C.
,, 12 at Middelburg (Cape) with Capt. P. J. Harris as O.C.
,, 13 at Bloemfontein with Capt. A. H. Lane as O.C.

According to the *Report for the year ending 31st March*, 1907, these five hospitals were still in existence, Pretoria and Bloemfontein being particularly important centres as Remount depôts were also present. In February, 1909, however, Standerton was evacuated as a military station, and the A.V.C. section was transferred to Potchefstroom and the native personnel discharged.

In the *Report for year ending* 31.3.1909 it is stated with regard to Pretoria that very little had been done "to improve the station veterinary hospital or the troop stables" (p. 69). At Potchefstroom "a small bacteriological laboratory" had been installed, and at Middelburg (Cape) the temporary buildings are said to be "totally inadequate and unfit for sick animals". At Bloemfontein the accommodation had been increased by 14 standings "and all the old temporary buildings . . . have been replaced by more permanent structures", *e.g.* new forge, transport stable, guard room, harness room, six-stall stable with forage barn, harness room for officers' horses, a cement platform for dressing purposes, a "foot bath, *post mortem* platform, boiler house and pharmacy store". Flies were troublesome during the summer, but "incinerators for the disposal of stable refuse are in use at all cantonments in the Command".

In the *Report for the year ending* 31.3.1910, the hospital at Potchefstroom was described as being " very complete and up to date ", that at Pretoria required isolation boxes, and at Bloemfontein a " small bacteriological laboratory " had been arranged in the hospital. At Harrismith, there was a temporary hospital with stable accommodation for 18 horses " while the average daily sick is 62 ". New stabling was being constructed for the Mounted Infantry, " on completion of which one of the old ones will be allotted to the Hospitals " (p. 81).

From the *Report for year ending* 31.3.1912, it is learned that No. 11 Section returned to the Home Command and that station veterinary

Fig. 29.
A.D.V.S. ARTHUR F. APPLETON (born 1861).

hospitals existed at Pretoria, Potchefstroom, Bloemfontein (Tempe), Harrismith, and a branch veterinary hospital at Artillery Barracks, Pretoria (p. 47). See figs. 22–25.

During Col. Nuthall's short period as Asst. D.V.S. (October 1913—August, 1914), there were four sections on a peace establishment, these being stationed at Potchefstroom, Pretoria (Artillery Barracks), Roberts Heights, and Tempe. Most of the officers and rank and file were at

Potchefstroom and Roberts Heights (letter dated August 10th, 1926).

Officers and men were located not only at the centres mentioned above, but at stations where sufficient work justified. During manoeuvres the various Sections (which also comprised a mobile section of five N.C.Os. and men, plus natives and transport) supplied the personnel of field veterinary hospitals and mobile sections ([82]). During the years ending 31.3.1909 and 1910 two of each were provided, and for the next year only one field veterinary hospital (No. 10 section from Potchefstroom) was required for the Camp of Exercise at Kroonstad.

Regarding *personnel,* Col. Nunn reported (*Rpt. for year ending* 31.3.1906) on "the paucity of officers, which has been brought to the notice of the G.O.C., South Africa, a copy of which was forwarded to you (*i.e.* D.G., A.V.S.) on December 11th, 1905. The situation remains the same . . ." He adds that civil aid would not be available in the event of hostilities or a bad epidemic, which factors, if they arose, would be bound to break down the A.V.S." in its present condition ([83]).

The following year Col. Blenkinsop mentioned there were available 21 executive veterinary officers, which number was sufficient for peace, "but it makes no provision for the strain of war, and presumably the veterinary service . . . is maintained for war . . ." ([84]). In discussing the Sections, the P.V.O. recommended "that two sections should always be stationed wherever there is a station veterinary hospital" (as in Great Britain) so as to release one complete section for manoeuvres, etc. In referring to the rank and file, attention was drawn to the A.V.C. being "made up of trained soldiers drawn from fighting units . . ." and that these men were armed "with cavalry swords, a very small proportion are mounted, and a cavalry sword is a useless weapon to a man on foot."

During the year ending 31.3.1912 the establishment of N.C.Os. and men was reduced from 74 to 60.

The creation of central veterinary hospitals was not always a simple matter, *e.g.* Smith (1927) records that at Roberts Heights, apparently in 1905, the site authorised was "on the edge of the sewage farm, well away from the Cavalry Lines. A mere accident brought this site to notice before building operations began. The explanation was that a veterinary hospital was something insanitary; the real fact was that, as it was not to be under Cavalry control, the regimental authorities pressed for it to be kept as far away from them as possible" (p. 228). Other objections were likewise raised.

([82]) Not approved officially until 1913.

([83]) Details regarding the A.V.S. officers in South Africa were sometimes published in the veterinary press, *e.g. Vet. Jl.,* Jan., 1904.

([84]) The Special Reserve of Officers A.V.S. was not created until 1909!

(j) Animal Management.

The Principal Veterinary Officer in his *Report for the year ending 31st March,* 1919, in discussing injuries inflicted *outside* the stable, states that they " are not so easy to deal with as the stable injuries " (p. 76). He adds that " grazing army horses in this country is most unsatisfactory, not only do we get a long list of casualties, but many cases of colic ([85]), vegetable poisoning and Biliary fever result from it. Is the little grass they get worth it ? Would it not be cheaper to increase their ration all round by 2 lbs. of green lucerne ?" (p. 76). In the *Report for year ending 31st March,* 1907 (p. 41) " every case of Horsesickness which occurred . . .

Fig. 30.
A.D.V.S. CHARLES E. NUTHALL (born 1862).

at the Artillery Barracks, Pretoria, was traced to the animals being out at night ". With regard to the value of stabling for preventing horse-sickness, the same Report (p. 41) adds, " If our stables in the horsesickness area had been built on the Indian system, of a central passage *to which* the horse faces . . . as at Middelburg, Cape Colony, we would have practical immunity . . . provided the animals were stabled at night

([85]) Especially " at Harrismith owing to the sandy nature of the soil " (*Rpt. A.V.S., 31st March,* 1919).

and the floors were not kept scrupulously clean. If horses are kept well under a roof in a dirty stable, no mosquito will bite them ". In order to prevent horsesickness, grazing was prohibited " except in fine weather ", *i.e.* when there was abundant sunshine (*Rpt. ending 31st March,* 1904, p. 12).

Even feeding on the picket lines had dangers greater than in the stable, for apart from injuries, there was the risk of sand and gravel being taken in with the food and causing colic. By the end of the year terminating 31st March, 1905, feeding off the ground had been practically abolished for horses, but not for mules.

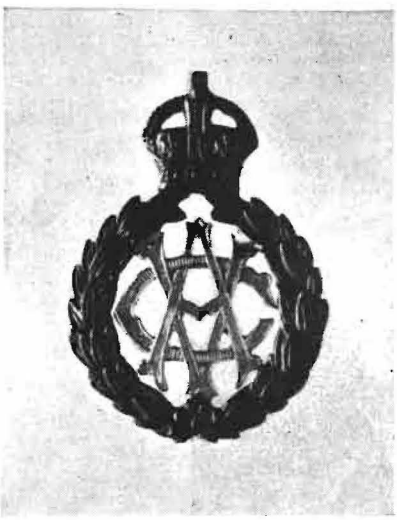


Fig. 31.
BADGE OF A.V.C. Adopted 1906.

Fig. 32.
BADGE OF R.A.V.C. Adopted 1919.

From the above it is clear that the Authorities did not attach much value to grazing.

During the construction of cantonments and stables there was an " extraordinary number of punctured feet from picked up nails. In one cantonment (Pretoria) the mules have had to be shod with protecting strips of iron across the foot to prevent these " (*Rpt. year ending 31st March,* 1905, p. 23). During the year ending 31st March, 1908, punctured wounds were also common, the explanation being " the recent very wet summer ", for not only is the horn softer, but " the mud is washed away, leaving the nails lying loose on the hard metalled surface ". Near a cantonment or town, nails from old packing cases and wood boxes were very common on the roads.

The position with regard to stables has been dealt with in a special

section. In the *Report up to 31st March,* 1907, the comment is made (p. 49) that " the present system of tying animals in stables with a long collar chain enables them to not only eat their bedding but frequently the greater part of their neighbours' food. This can be prevented by the Egyptian army method of tying animals to a ring running on a perpendicular bar under the manger."

In discussing the high admission rate for corns (higher in riding horses than draught), the P.V.O. (*Rpt. up to 31st March,* 1907) attributes this " to unsuitable pattern shoes having been in use for riding horses ". He adds that the Indian pattern has " recently been substituted for those of the heavy home pattern " which are broader webbed and therefore provide more cover. In the following year (*Rpt. A.V.S.,* 31.3.09, p. 64), through " a special pattern broad webbed shoe " and more careful shoeing, " the greatest improvement was seen in the Mounted Infantry Battalions". In the *Report up to 31st March,* 1910, the disadvantages of the Indian pattern, *e.g.* narrow web and softer iron, are emphasised.

The staple grain ration was maize and until 20th February, 1909, this was given crushed, but for several reasons ([86]) whole maize was later used. In addition, lucerne hay replaced oat hay and in the *Report up to 31st March,* 1910, the P.V.O. reported satisfactorily on the change. It was also found (*Report up to* 31 *March,* 1909, p. 61) that " where mealies are fed dry and with a small proportion of bran" and lucerne hay " we have a lower admission rate and mortality for digestive trouble . . . than where the mealies are soaked or even crushed, particularly if oat hay takes the place of the lucerne." In 1909 " a very large proportion of lucerne has been rejected by the Forage Boards on account of being badly saved ".

Colic was an ever present danger, especially among mules, which received less attention than horses; and, as the following figures show, the time of watering had a marked influence on the incidence and course of the complaint (*Report* 31.3.1909, p. 77).

	ATTACKED BEFORE WATERING.	ATTACKED AFTER WATERING.
Colic cases	38.36%	61.64%
Fatal cases	46.66%	53.34%

Digestive maladies were associated not only with improper feeding and watering but also with " the supply of very inferior veldt grass for bedding "! (*Report up to* 31.3.1908, p. 54.)

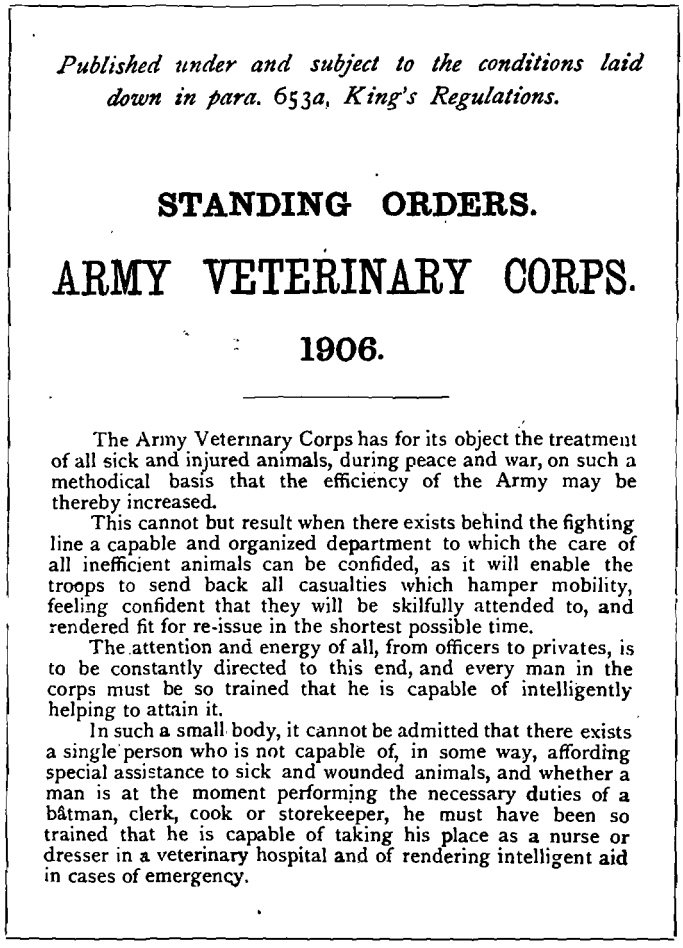
With regard to the disposal of stable refuse, in spite of incinerators being available at all cantonments, dung pits were sometimes employed, *e.g.* Potchefstroom (*Rpt.* 31.3.1909, p. 69). As a result flies were usually

([86]) Saving of expense for crushing, saving of wastage through crushing, on account of better keeping qualities, and as whole mealies were more easily obtainable in time of war.

troublesome during the summer, especially in the case where the tails of docked horses were cut. See General Order of October, 1908.

The estimated cost "of keep" for a horse was 2/- per day.

During the manoeuvres in 1907 "the horses of the 9th Lancers stampeded from their picket lines at night". The cause was not ascertained, but the animals were nearly all young and recently imported.



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STANDING ORDERS.

ARMY VETERINARY CORPS.

1906.

The Army Veterinary Corps has for its object the treatment of all sick and injured animals, during peace and war, on such a methodical basis that the efficiency of the Army may be thereby increased.

This cannot but result when there exists behind the fighting line a capable and organized department to which the care of all inefficient animals can be confided, as it will enable the troops to send back all casualties which hamper mobility, feeling confident that they will be skilfully attended to, and rendered fit for re-issue in the shortest possible time.

The attention and energy of all, from officers to privates, is to be constantly directed to this end, and every man in the corps must be so trained that he is capable of intelligently helping to attain it.

In such a small body, it cannot be admitted that there exists a single person who is not capable of, in some way, affording special assistance to sick and wounded animals, and whether a man is at the moment performing the necessary duties of a bâtman, clerk, cook or storekeeper, he must have been so trained that he is capable of taking his place as a nurse or dresser in a veterinary hospital and of rendering intelligent aid in cases of emergency.

Fig. 33.

PAGE 1, STANDING ORDERS, A.V.C. 1906.

This booklet ($4\frac{7}{8}$ x $7^{9}/_{16}$ ins.) of 35 pages gives details as to the duties of officers and N.C.O's. Appendices lay down the course of training for men and describe Army forms and books.

As a result six horses were killed and 148 admitted to hospital chiefly for barbed wire wounds (*Report up to 31st March,* 1908, *p.* 56).

One receives the impression that animal management, the basis of

animal husbandry, and preventive veterinary medicine, were thoroughly studied by the A.V.C. Within recent years the veterinary profession in South Africa has perhaps not paid sufficient attention to this aspect of our profession. See Blenkinsop's article " Veterinary Science in its relation to Agriculture ", *Transvaal Agr. Jl.* V, p. 599.

(k) Miscellaneous.

(1) The designation P.V.S., used by the military until 1891, was adopted by the Natal Civil Veterinary Division in 1896, the assistants being known as Assistant V.Ss. ([87]). In 1902, when a Division of Veterinary Science was created in the Transvaal, Stewart Stockman was designated P.V.S. and his assistants, as in Natal, D.V.Ss. At Union in 1910, the title P.V.S. was assumed by the head of the Veterinary Division (Administration) ([88]), *i.e.* Charles E. Gray. The designation Senior Veterinary Surgeon (Officer) still used in the Civil Veterinary Division of the Union Department of Agriculture was also formerly a military title.

(2) At least two A.V.D. officers, Messrs. T. Flintoff and R. C. Cochrane, served in the Civil Veterinary Division for a time, the former as Chief, Veterinary Division O.R.C. from 16th August, 1903, until shortly before his death at Felixtowe on 24th August, 1907. The latter was seconded in the Transvaal C.V.D. for a year during 1903–1904 (*Vet. Jl.*, June, 1903, p. 353).

(3) Vet. Lieut. R. C. Cochrane attended the inaugural meeting of the Transvaal V.M. Assn. at Long's Hotel, Johannesburg, on 16th February, 1903. At the second meeting on 26th June, 1903, at Long's Hotel, Capt. Eassie (Pretoria) and Lieut. Plunkett (Krugersdorp) were present. In August, 1903, the Association made representations to the War Office that compound rank be abolished, otherwise it would " endeavour to persuade the younger members not to enter the Army " (letter 13.8.03). Extraordinary to relate, compound rank existed in the Transvaal Volunteers until Union (31.5.10) ! Although at one time or another over a dozen officers A.V.D. were members of the Transvaal Vet. Med. Assn., not only were veterinary officers reluctant to join the Association, but in 1907 there were complaints that some practised privately. At the meeting of 28.12.07 (see History of Transvaal Vet. Med. Assn., *Jl. South Afr. Vet. Med. Assn.* II (1), 1931), the President read a letter from Col. Blenkinsop, P.V.O., indicating that such was contrary to regulations.

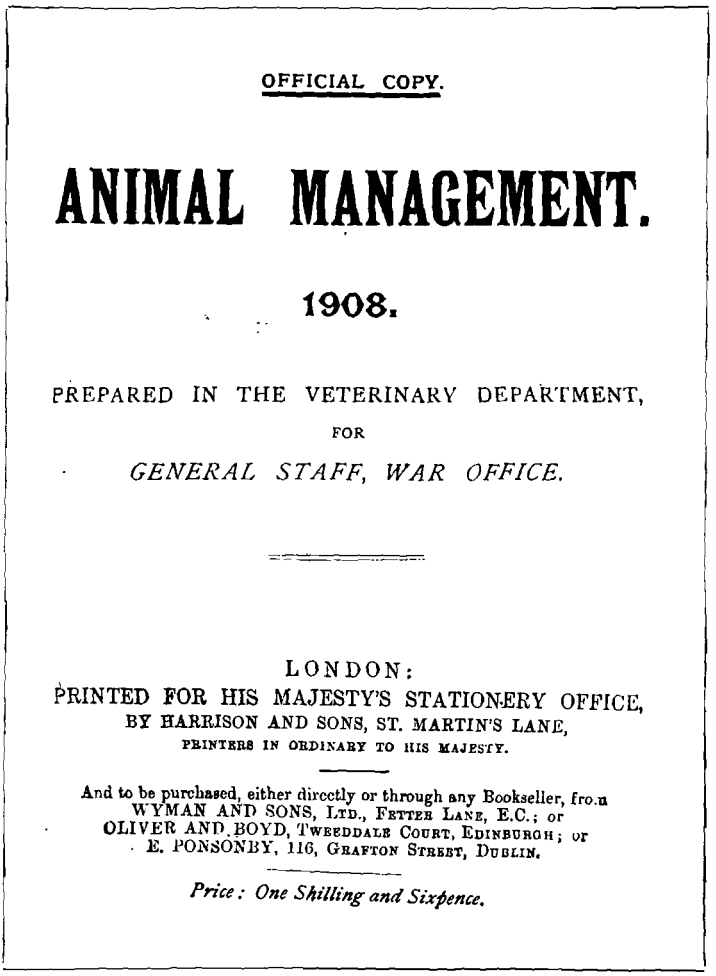
(4) Col. Blenkinsop was present at the 3rd Pan-African Veterinary Congress at Pretoria in January, 1909.

([87]) In 1898 the term A.V.S. was altered (in Natal) to District V.S.

([88]) Changed to Director of Veterinary Services (not to be confused with D.V.S. Union Defence Force) in 1927, when there was amalgamation of Administrative and Research Divisions.

(5) Col. Appleton P.V.O. was responsible for the establishment of the Pretoria Society for the Prevention of Cruelty to Animals. The booklet on *The Management of Horses, Mules, Dogs, Cats and Poultry* issued by the Society was compiled mainly by him. A copy is in the Library, South African Vet. Med. Assn., Onderstepoort.

(6) The last C.V.S. to be employed by the A.V.S. was apparently S. I. Johnston (1866–1929), who on 1.10.05 accepted a post with the



OFFICIAL COPY.

ANIMAL MANAGEMENT.

1908.

PREPARED IN THE VETERINARY DEPARTMENT,

FOR

GENERAL STAFF, WAR OFFICE.

LONDON:
PRINTED FOR HIS MAJESTY'S STATIONERY OFFICE,
BY HARRISON AND SONS, ST. MARTIN'S LANE,
PRINTERS IN ORDINARY TO HIS MAJESTY.

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E. PONSONBY, 116, GRAFTON STREET, DUBLIN.

Price: One Shilling and Sixpence.

Fig. 34.
TITLE PAGE, BOOK OF ANIMAL MANAGEMENT, 1908.
The book ($5^{1}/_{16}$ x 7 ins.) contains 343 pages of valuable information on animal management with plates of grasses and weeds.

Transvaal Civil Veterinary Division. See *Vet. Rec.* 11.11.05 and *J. S.A.V.M.A.*, Nov., 1929.

(7) The last officer A.V.S. to leave South Africa was Major A. F.

Deacon, who handed over as S.V.O. Cape (not to be confused with the civil post of like designation) to Capt. Crowhurst, South African Veterinary Corps in October, 1914.

(8) It is interesting to recall that about 30 years ago the Masonic Hotel, at the corner of Vermeulen and St. Andries Streets, was the leading hotel of Pretoria and there were held several veterinary dinners.

(9) As an illustration of the difficulties of historical research may be mentioned the comment of Sir Frederick Smith (who was himself a Director General A.V.S.) when the uncertainty regarding Whitfield and Southern Rhodesia (see Chapter I) was brought to his notice. His reply (letter of 18.4.27) was, " Whitfield is now dead and in the W.O. records, of which I have a complete copy, he is not given credit for any South African campaign. I may add, however, that these records have not always been faithfully kept " !

(10) In regard to uniform, much detailed study is necessary in order to be able to record the precise position. Smith (1927) has devoted an appendix to this matter, but many points are still obscure. The photographs reproduced in this history—several being taken from Smith (1927)—are therefore of particular interest. Smith makes no mention of a maroon flash which Sir John Moore (letter of 5.9.30) states he wore on his " Indian helmet " but " it was abolished ". He adds " the khaki helmet never had a maroon flash " ! Smith does not refer to the maroon gorget patch worn on khaki service dress even prior to 1899. According to the same authority khaki " had been worn on service since 1880, but it was not official " (p. 250). He is, however, in error when he states that " the last time the British Army went to war in red and blue tunics was . . . 1879 ". Red tunics and white helmets were worn in the First Anglo-Boer War (1880-1881). Justice can only be done to this intricate subject by a specialist.

(11) The autographs of a few of the officers referred to in this record are reproduced in a paper " Autographs of Veterinary Interest," which appeared in the *Jl. S.A.V.M.A.* IV (3), 1933.

(12) In conclusion, it is a pleasure to express indebtedness to those veterinary officers who responded to an appeal for information. Their names are : Sir Francis Duck, General Sir Frederick Smith, Col. R. Rowe, Col. A. F. Appleton, Col. A. England, General Sir John Moore, Brig.-General C. E. Nuthall, Col. R. W. Raymond, Col. S. L. Slocock, and Major G. E. Oxspring. The first three, unfortunately no longer with us, will always be remembered in South Africa.

(1) Conclusion.

This chapter brings to a close the record of the A.V.S. in South Africa. After a stay of nearly a century and a quarter the Imperial Army

was recalled, as were the legions of Imperial Rome, to defend the Mother Country in the Great War (1914–18). Replacing the A.V.S. in the local theatres of war was the South African Veterinary Corps (89), a unit made possible by the political union of the four Colonies in May, 1910.

When it is recalled that during 1881–1899 opportunities for development had been allowed to slip and that the consequences of this were

VETERINARY MANUAL (WAR), 1914.

CHAPTER I.

1. ORGANIZATION.

1. The Veterinary Service of an Army in the field is organized and controlled by a Director of Veterinary Services. Its function is to promote efficiency by preventing and reducing wastage amongst the animals of the Army.

2. The general principles governing this service are contained in Field Service Regulations, Part II, and more detailed instructions as to their execution are given in this manual.

3. Veterinary hospitals are located on the Lines of Communication for the treatment and care of sick and injured animals of the field army. Veterinary mobile sections are provided to relieve field units of all inefficient animals which may hamper their mobility, and convey them to railhead for transfer to veterinary hospitals.

4. Base and advanced veterinary store depots are formed for the upkeep of veterinary equipment.

5. The personnel allotted to the above units is laid down in War Establishments.

Fig. 35.

PART OF TITLE PAGE AND CHAPTER I OF THE VETERINARY MANUAL (WAR), 1914.

This publication, of same size as the Standing Orders, contains 9 pages. Details are given of the duties of officers and the organisation for war. The official number of the Manual is 40/W.O./2195.

shown in the Second Boer War (1899–1902), then the rejuvenation between 1902 and 1914 must be acknowledged to have been phenomenal.

(89) History still to be compiled.

Of greatest importance was the establishment of hospitals and the provision of personnel. So efficient did the A.V.S. become that during the Great War it surpassed the veterinary organisation of any other European Army!

The period of demobilisation in 1902 allowed the release of approximately 70 C.V.Ss. The part some of these men played in the subsequent veterinary history of South Africa will be described when a record of the various civil veterinary services is compiled.

A general idea has been given of the duties and difficulties of the officers of the A.V.S. Both the G.O.C. Imperial troops and P.V.O., A.V.S., were stationed at Pretoria.

CHAPTER IV.

Miscellaneous.

(a) Campaigns in South Africa, 1881–1914 (Table III). (b) Contributions to periodical literature. (c) General conclusion. (d) Bibliography.

(a) CAMPAIGNS IN SOUTH AFRICA, 1881–1914.

The following table gives a summary of the position :—

TABLE XII.

CAMPAIGNS IN SOUTH AFRICA, APRIL 1881–1918.

Date.	Campaign.	Imperial Cavalry Unit.	Award	Further Remarks.
1884–85	Bechuanaland	6th Dragoons	No award.	
1888	Zulu	6th Dragoons	" "	
1890	Mashonaland	—		
1893	Matabeleland	—		
1896	Matabeleland (Rebellion)	7th Hussars	See text.	See text.
1897	Mashonaland (Rebellion)	7th Hussars		
1897	Bechuanaland	Campaign undertaken by Cape Government.	Medal issued in 1900. A bar was also given.	See Williams (1909).
1899–1902	2nd Anglo-Boer	See Table I.	Queen's Medal and bars. King's Medal and bars.	See Smith 1912-14, and 1927 (p. 259).
1906	Natal Native	Campaign undertaken by Natal Government.	Medal issued in 1908 with bar, "1906".	See Stuart (1913).
1914–18	German South-West Africa, 1915.	Campaign undertaken by Union Government.	Imperial Govt. issued 1914-15 Star, British War & Victory Medals.	See Blenkinsop & Rainey (1925), (p. 406).

Additional information is given as follows :—

Rhodesian Campaigns 1890–97.

Thanks to the Staff Officer "A" Branch (Lieut. A. C. Walker), Southern Rhodesia Forces (his letter C.S. 518.20 of 28.8.1934) the following particulars are available :—

1. *Pioneer Column,* 1890.—Medal with Clasp " Mashonaland, 1890". Issued to all members of the Pioneer Column who were in or north of Fort Tuli between 1st June, 1890, and 12th September, 1890. If a holder of this medal also took part in the campaign of 1893 he was awarded a clasp " Matabeleland, 1893 ". If he took part in both or either of the 1896 and 1897 campaigns he was awarded a Clasp " Rhodesia 1896 ".

(For all campaigns Medal with three Clasps.)

2. *Matabele War,* 1893. Medal. A holder of this Medal who served in 1896 was awarded a Clasp " Rhodesia 1896 ", and a further Clasp " Mashonaland 1897 " if he served in that year.

3. *Rhodesia Rebellion,* 1896.—Medal. With a Clasp " Mashonaland 1897 " awarded to a holder of the Medal who served in 1897.

4. *Rhodesia Rebellion,* 1897.—Medal.

The cost of above medals was borne by the British South Africa Company, and the Defence Department is responsible for issue. See *The Rhodesia Herald Pioneer Number,* 12.9.1930, p. 27.

No details of the above are given in Gale and Polden's *Sheet of Official Naval and Military Medals and Ribbons* from 1793, which, however, shows the Cape of Good Hope General Service Medal (1900) and the Natal Rebellion Medal (1908).

Second Anglo-Boer War, 1899–1902.

(i) It is assumed that C.V.Ss. who served in South Africa (Table VI) were eligible for the two medals issued and that the C.V.Ss. who performed sea-transport duties were not.

(ii) By Government Notice 2307 of 21.12.1920 the issue of two medals to burghers who fought for the Republican Forces was approved. Particulars of the awards are not given in Gale and Polden's Sheet, but may be seen in *The Star* (Johannesburg) of 22.9.1928.

Great War, 1914–18.

(i) " The Rebellion which occurred in South Africa was not regarded as a campaign and members of the Government Forces, who took part, did not thereby qualify for the award of any medal " (Letter D.C. 962/Medals of 12.6.34 Department of Defence, Pretoria).

(ii) Apart from the German South-West Africa Campaign the

Union Forces played a major part in the German East Africa Campaign. Both the South-West and East Africa campaigns " qualify for the award of the 1914–15 Star in addition to the British War and Victory Medals. The Star was awarded by the Imperial Government ". (*Ibid.*)

(iii) " No medals were issued to Rhodesian troops excepting those who actually saw service in a theatre of war " (Letter C.S. 518.20 of 28.8.1934).

(iv) A considerable amount of confusion took place at the time of Union in regard to the disposal of official files. It is, however, gratifying to know that the following medal registers are available at Defence Headquarters, Pretoria : Natal Rebellion 1906, German South-West Africa, German East Africa, Egypt and France (Great War 1914–18). The last four registers contain the names of all South Africans who enlisted

Fig. 36.

GRAVESTONE OF CAPT. A. J. HASLAM, FORT SMITH, KENYA.

The inscription on one side reads, " In Memory of Captain A. J. Haslam, Army Veterinary Department, B.A., M.D., F.R.C.V.S., killed by the Wakikuyu, 23rd July, 1898. Aged 34 years." On the other side is : " Erected by his friends on the Staff of the Uganda Railway." Haslam was the first veterinary surgeon in East Africa. The photo was kindly sent by Mr. J. R. Hudson, M.R.C.V.S. (letter A. 9/15/1974 of 2.9.35 at Kabete, near Nairobi.

during the Great War with the Union Imperial Service Contingents and Defence Forces (Letter D.C. 962/Medals of 28.8.1934, Dept. of Defence, Pretoria).

(v) Much assistance has been received from R. C. Lange, Esq., of the Department of Defence, Pretoria, in connection with military history, and to him cordial thanks are due.

(b) Contributions to Literature.

Apart from the references given in the text, an attempt has been made to give a list of contributions of South African military veterinary interest during the period 1881–1914. Official reports are not included (90). Owing to missing numbers (Library of Veterinary Research Laboratories, Onderstepoort) the list is not complete.

1881 – 1899.

1884. RUTHERFORD, C. Osteoporosis and Splenic Abscess. *Vet. Jl.* **18** : 413.
1886. RUTHERFORD, C. Cases of supposed Horse-sickness in Natal. *Vet. Jl.* **23** : 324.
1887. RUTHERFORD, C. Paper before Norfolk and Eastern Counties V.M. Assn. on "South African Horse-sickness". *Vet. Jl.* **25** : 46; 113.
1888. NUNN, J. A. The Use of Antifebrin in Veterinary Practice. *Vet. Jl.* **27** : 97.
1889. NUNN, J. A. South African Horse-sickness. *Vet. Jl.* **29** : 74. (From India).
1894. NUNN, J. A. The Specific Fevers of Malarial Origin in Equines. *Vet. Jl.* **39** : 402. (From India).
1896. HAYES, M. H. South African Horse-sickness (Oedema Mycosis). *Vet. Jl.* **42** : 22.
1898. HASLAM, A. J. Diseases of British East Africa. *Vet. Rec.* 27.8.98.
1899. NUNN, J. A. Paper before Central V.M. Society on "Burns and Scalds". *Vet. Jl.* **48** : 122.

See also papers by Cammack, J. (p. 12) and Thomson, H. (p. 96) in Vol. 26, and Cammack, J. (p. 250) and Conti, G. (p. 249) in Vol. 28 of *Vet. Jl.*

1899 – 1902.

1899. — The Transport of Horses. *Veterinarian*, p. 916.
1899 – 1902. MOORE, J. A Veterinary Diary of the South African War. Summary by Dr. E. M. Robinson in *Jl. R.A.V.C.*, Nov., 1931.
1900. SPREULL, A., Jun. Narrative of a Veterinary Student whilst soldiering in South Africa. *Vet. Jl.* II, p. 200.
1900. COCHRANE, R. C. The Effects of Bullet and Shell Wounds on Horses. *Vet. Rec.* 9.6.1900.
1901. MARTIN, E. E. Ship pneumonia. *Jl. Comp. Path. Ther.* **14** : 373.
1901. COLEY, J. T. Clinical Notes of African Horse-sickness. *Jl. Comp. Path. Ther.* **14** : 373. See also *Vet. Rec.* 11.1.02.
1901. HUTCHEON, D. Military Horses and how to breed them. *Vet. Jl.* **3** : 187.
1901. WILLIAMS, W. O. Remount and Transport work in America. *Vet. Jl.* **4** : 144.
1901. WILLIAMS, W. O. Some of the Departments of the Remount Commission and their Work. *Vet. Jl.*, p. 209.
1901. CHERRY, C. C. Reminiscences of the South African Campaign. *Vet. Jl.* **4** : 358.
1901. LUND, J. M. Successful Treatment of Sand Colic in South Africa. *Vet. Rec.* 6.7.01.
1901. WILLIAMS, A. J. Rupture of the Carpal Interosseous Ligaments with Partial Dislocation of the Radiocarpal Articulation. *Vet. Rec.* 16.11.01.
1901. HARVEY, G. J. Horses at Sea. *Vet. Rec.* 3.8.01.
1901. HAYES, M. H. The Transport of Horses by Sea. *Vet. Rec.* 7.9.01.

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(90) See, however, review in *Veterinarian*, 1899, p. 925, on War Office publication : "Suggestions for the General Treatment of Horses, Mules, and Oxen on Service in South Africa". 56 pages.

1901. LUND, J. M. An Unnamed Disease in South Africa. *Vet. Rec.* 14.9.01.
1901. HOGGAN, T. R. R. An Unnamed Disease in South Africa. *Vet. Rec.* 12.10.01.
1901. COCHRANE, R. C. Phenomenal Fibrous Growths. *Vet. Rec.* 19.10.01.
1901. CROLE, D. Pseudo-Tetanus. *Vet. Rec.* 19.10.01.
1902. MOORE, R. The Management of Horses and the Duty of Veterinary Surgeons on Board Ship. *Vet. Jl.* **5** : 152.
1902. WILLIAMS, A. J. Poisonous Plants in South Africa. *Vet. Rec.* 11.1.02.
1902. SANDERSON, C. J. Disease in South Africa. *Vet. Rec.* 4.1.02.
1902. HAMILTON, J. R. R. Supernumerary Digits in a Pony. *Vet. Rec.* 5.4.02.
1902. TASKER, H. K. Specific Ophthalmia. *Vet. Rec.* 5.4.02.
1902. FLANAGAN, W. G. Enlargements on a Horse's Back. *Vet. Rec.* 17.5.02.

The most valuable contribution is the *History of the War in South Africa* by Sir Frederick Smith, published in the *Vet. Rec.* 1912–14.

1902 – 1914.

1902. COCHRANE, R. C. Glanders in South Africa. *Jl. Comp. Path. Ther.* **15** : 31.
1902. HUBAND, T. A. Losses among Horses in South Africa. *Vet. Rec.* 13.12.02.
1902. WILLIAMS, A. J. Rupture of the Carpal Interosseous Ligaments with partial Dislocation of the Radio-carpal Articulations. *Vet. Rec.* 26.7.02.
1902. MAJ. GEN. DOWNE. Report on Remount Operations in South Africa. *Vet. Rec.* 15.11.02.
1902. PARKER, T. Lymphangitis Epizootica. *Vet. Rec.* 29.11.02.
1902. MORGAN, J. W. Vomiting in a Horse — Recovery. *Vet.Rec.* 27.12.02.
1903. BLENKINSOP, L. J. Some Notes on Breeding Domesticated Animals, especially Horses. *Transvaal Agr. Jl.* **1** : 21.
1903. HEAD, A. S. The Wear and Tear of Horses during the South African War. *Jl. Comp. Path. Ther.* **16** : 299.
1903. WEBB, E. C. South African Horse-sickness. *Jl. Comp. Path. Ther.* **16** : 120.
1903. EASSIE, F. A System of Remount Management introduced during the past War. *Vet. Jl.* July.
1903. HODDER, A. Epizootic Lymphangitis. *Vet. Jl.* Sept.
1903. NUNN, J. A. Two cases of Nervous Affection of the Head in the Horse. *Vet. Jl.* Aug.
1904. GREENFIELD, H. Eye Diseases of the Horse in South Africa. *Vet. Jl.* May.
1904. COLEY, J. T. South African Horse-sickness. *Vet. Jl.* Aug.
1904. COLEY, J. T. South African Horse-sickness. *Vet. Rec.* 23.7.04 and 30.7.04.
1904. RUNCIMAN, B. Curious Loss of Hair. *Vet. Rec.* 8.10.04.
1904. RUNCIMAN, B. Systematic Disturbance in Epizootic lymphangitis. *Vet. Rec.* 22.10.04.
1905. JOHNSTON, S. I. Thrombosis in the Horse. *Jl. Comp. Path Ther.* **18** : 78.
1905. EASSIE, F. Some Observations on Tropical Biliary Fever. *Jl. Comp. Path. Ther.* **18** : 108.
1905. WEBB, E. C. The Question of the Co-relation of Biliary Fever in the Horse and the Subacute form of Horse-sickness. *Jl. Comp. Path. Ther.* **18** : 218.
1905. PRIME, T. F. An Extraordinarily high Temperature. *Vet. Jl.* July.
1905. PALLIN, S. F. G. Biliary Fever of Horses in India. *Vet. Jl.* Jan.
1905. NUNN, J. A. The Sick Horse in Peace and War. *Vet. Jl.* March.
1905. NUNN, J. A. Animals in Time of War and the Geneva Convention. *Vet. Jl.* June.
1905. MARTIN, E. E. The Possibilities of Hybridising. *Vet. Rec.* 25.3.05.
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1905. SCHOFIELD, W. E. A Case of ruptured Lung in the Horse. *Vet. Rec.* 8.7.05.

1905. SCHOFIELD, W. E. Complete Transverse Rupture of the small Intestine in the Horse. *Vet. Rec.* 16.9.05.
1906. LEANING, A. Death from Lightning. *Vet. Jl.* Jan.
1906. NICOLAS, J. Tumour attached to the Diaphragm of a Horse. *Vet. Jl.* Feb.
1906. WATKINS-PITCHFORD, H. Observations on the Mark vi ·303 Bullet. *Vet. Jl.* Mar.
1906. LANE, A. H. Open Temporo-Maxillary Joint. *Vet. Jl.* Mar.
1906. MARRIOTT, T. Two cases of Fractured Vertebræ. *Vet. Jl.* Mar.
1906. LEANING, A. Meningitis in a Horse, supervening on an Injury. *Vet. Jl.* Mar.
1906. LANE, A. H. Bone Disease amongst Horses in South Africa. *Vet. Jl.* May. See articles on Osteoporosis in *Agr. Jl. Cape of Good Hope,* Apr., 1905.
1906. HOLNESS, H. J. Three cases of Osteotomy in Cases of Splint Lameness. *Vet. Jl.* July.
1906. SCHOFIELD, W. E. Fracture of the Os Pedis in a Mule. *Vet. Jl.* July.
1906. LEANING, A. A Case of Pleuro-pneumonia supervening upon Bilious fever. *Vet. Jl.* August.
1906. HAZELTON, E. H. Animal Diseases following War. *Vet. Jl.* Sept.
1906. BURRIDGE, T. E. Case of Sand Colic in a Mule. *Vet. Jl.* Oct.
1906. ANDERSON, R. G. The Civil Veterinary Surgeon on Active Service. *Vet. Rec.* 10.2.06.
1906. HARRIS, P. J. A Monument to Horses in South Africa. *Vet. Rec.* 11.8.06.
1907. BLENKINSOP, L. J. Veterinary Science in Relation to Agriculture. *Transvaal Agr. Jl.* 5 : 599.
1907. TAPLEY, J. J. B. Some Accidents. *Vet. Jl.* Feb.
1907. SCHOFIELD, W. E. Fractures occurring in a Troop Horse and Mule. *Vet. Jl.* Feb.
1911. STEWART, H. A. Rupture of the Diaphragm and Strangulated Diaphragmatic Hernia. *Vet. Jl.* Aug.
1911. RAINEY, W. Double-sided Operation for Guttural Pouch Disease. *Vet. Rec.* 3.6.11. See also *Vet. Rec.* 22.7.11.
1911. RAINEY, W. Pitfalls. *Vet. Rec.* 12.8.11.
1911. RAINEY, W. Radiotherapy. *Vet. Rec.* 9.9.11.
1912. LANE, A. H. Horses for War Purposes, *United Services Mag.* Apr. (Quoted by Smith, p. 133).
1912. STEWART, H. A. Diffuse Lipomatosis in a Mule. *Vet. Jl.* Oct.
1912. RAINEY, W. Gastrophilus equi and stomach Staggers — A suggestion. *Vet. Rec.* 2.3.12. See *Vet. Rec.* 15.6.12.
1912. COLLYER, J. B. Irrigation of Joint Cavity. *Vet. Rec.* 11.5.12.
1913. RAINEY. W. The Passing of the Bran Poultice. *Vet. Rec.* 15.3.13.
1913. RAINEY, W. Acclimatisation of the Horse. *Vet. Rec.* 26.4.13.
1914. COCHRANE, R. C. Veterinary Hospitals during War. *Vet. Jl.* Jan.
——. SESSIONS, H. Two Years with Remount Commissions. Quoted by Smith (1912 - 14) on p. 224.

(c) General Conclusion.

This record should be considered as the continuation of the history of the Army Veterinary Service in South Africa described under the title "Matters of Veterinary Interest, 1795–1881" (*Jl. S.A.V.M.A.* 1935). By 1881 the veterinary profession had become well established in the Cape Colony and Natal and, from that date, histories could be compiled of both the military and civil branches. This history deals only with the

military side, *i.e.* with the Army Veterinary Service and in the narration of events not only have campaigns (Table XII) been described, but also the various cavalry movements (Table I).

It has been possible not only to preserve the names of all regular veterinary officers and most of the civilian veterinary surgeons attached to the A.V.D., but also to give at least the details of qualification. Of particular importance are the civilian veterinary surgeons who made South Africa their home after the Second Anglo-Boer War. Several of the photographs shown are or unusual historical interest.

While the contributions to veterinary literature are not of a high standard, it must be remembered that the duties of veterinary officers, like those of state field veterinarians, are purely routine and there is little or no opportunity for more spectacular work, *e.g.* research. Only those who have had experience can appreciate the monotony of regulatory duties. Indeed in the circumstances it is extremely creditable that through the efforts of the army veterinarian the mortality among horses and mules was considerably reduced when comparing the post-Boer War stage with the pre-Boer War stage.

The A.V.S., like all organisations, had its vicissitudes, but it is gratifying that after a generation had been wasted in not developing along right lines, that the final phase was characterised by initiative and sound organisation.

In conclusion, let us repeat the words which appeared in the *Official Souvenir, South African Industrial Exhibition,* held at Capetown in February, 1914 (*Vet. Rec.* 16.5.14) :—

" Probably no technical man has borne so much opprobrium, received so much opposition or been the subject of so much banter, yet pushed on more doggedly than the veterinary surgeon . . . But the veterinary surgeon, in the end, won his way, if not to the affection, at any rate to the intelligence of the great majority of the people ".

These words apply not only to the civilian veterinary surgeon, but also to the army veterinary officer!

Pristinae virtutis memores.

(d) Bibliography ([91]).

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([91]) Additional to the references detailed in the text.

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CORRIGENDA.

TABLE V.

Read F. B. Drage, died Brixworth, Northampton, 7.7.35, aged 68.
F. J. Short, died Torquay, 1.3.37, aged 79.
Jl. South African Veterinary Medical Association, Dec., 1937.

TABLE IX.

Read Nicholas, J.
Tapley, J. J. B., retired 1.12.37; Schofield, W. E., retired 15.4.34; Gibbs, H. E., retired 18.1.36; Burridge, T. E., retired 3.9.32; Leckie, V. C., retired 10.11.36; Hodgkins, A., retired 12.2.35.
Bone, H., qualified London and retired 20.5.34.
Tindle, R., Wright, W. N., and Taylor, E., all qualified London.
Anthony, W. S., retired 1.12.33.
Brookes, W. T., Andrews, J. O., and Audas, R. S., qualified 1905.
Marriott, T., died 25.5.37 Birmingham, *Vet. Rec.* 5.6.37.

Thanks are due to Dr. Fred. Bullock and Major Glyn Lloyd of the A.V.D. of the War Office for the above information.

REVIEW.

If anyone goes to the trouble of writing a text book (1) one feels the desire to give an encouraging review of it and one expects to be able to do so if the authors are members of the staff of a famous institute and state that the book is based on lectures given at that institute. On the other hand the reviewer has a certain responsibility and must guard against the dissemination and perpetuation of erroneous and misleading statements.

It is therefore with great regret that the book under review cannot be recommended. It contains so many erroneous statements that it is positively dangerous to students and will be misleading to the practitioner. Although issued under the title of "Veterinary Parasitology" the book covers only protozoology (26 pages) and helminthology (76 pages), with short chapters on technique, life history diagrams, host lists and an index. The characteristic of the book is extreme brevity and the selection of the material is not always very satisfactory. So more than a page is devoted to *Balantidium coli,* but *Anaplasma* is nowhere mentioned. Amongst the helminths the whole group of hookworms is conspicuous by its absence.

The following are some of the misstatements referred to above :—

p. 8. Sporozoa are "incapable of movement". It will remain a mystery to the student how they enter erythrocytes and other cells.

p. 12. *Theileria parva* is said to find its way via the bloodstream to the endothelial cells of internal organs in which it develops. Then Koch's bodies "occasionally find their way into the peripheral blood-stream and attack the red blood corpuscles".

p. 15. Diagnosis of east coast fever is made by examination of a blood film. This is just how some people miss east coast fever.

p. 25. "*Trypanosoma gambiense* (*syn. T. brucei, T. rhodesiense*)" is said to occur in various domestic and game animals. No reason is given for arriving at this synonymy and nothing is said about human sleeping sickness in relation to this trypanosome.

p. 31. It is stated that in piroplasmosis and theileriasis immunization is possible by injection of blood of recovered animals. Then follows "In the case of artificial immunization against *Theileria,* it is stated that the animals do not remain carriers as they do in *Babesia.* Whilst this procedure eliminates the disease from all the animals on one farm, it will be realised that, being carriers, they remain a potential source of danger to similar animals on adjoining farms", which is not exactly clear, especially for a student who usually believes everything he sees in print.

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(1) *A Guide to Veterinary Parasitology for Veterinary Students and Practitioners.* Southwell, T., and Kirshner, F. (1937). H. K. Lewis & Co., Ltd., London. pp. x + 143, 88 figures, 12 diagrams. Price 7/6.

p. 35. Of gravid segments of tapeworms it is said that "the segment ruptures, liberating the eggs into the lumen of the intestine". This will be rather misleading in relation to diagnosis.

p. 49. About the life cycle of *Dipylidium caninum* we learn that "owing to the fact that dogs roll about on the ground, the eggs of *D. caninum* are frequently found on the coat of the dog. These eggs are swallowed by the larval fleas and lice on the dog, and the cysticercoids are found in the adult fleas and lice". The authors apparently know nothing about the life history of fleas, nor will the student.

p. 63. Under liver fluke we read "Adenoma, rising from the epithelium of the ducts are not uncommon".

p. 71. *Larva* is defined as "A name applied to a baby nematode in which not even a trace of the genital organs has appeared". Yet one can see the genital primordium in "babies" which have just hatched and distinguish the sexes in third stage larvæ.

p. 72. Of nematodes it is said that "in many genera the male is degenerate". Probably the result of living in a dark intestine without air. The authors definitely do not like anthelminthic therapy and, not being veterinarians, prefer very impracticable preventive measures. They state twice that a dog with *Echinococcus* tapeworms should be destroyed immediately. In the case of poultry tapeworms it is a "comparatively simple matter" to collect and dispose of the fæces daily and this is much preferred to treatment. We are also rather discouraged from treating sheep for liver fluke, because "the effective treatment cannot be undertaken till it is too late". On the whole it must seem ridiculous to treat animals when "the treatment is in all probability quite ineffective and useless in 99 per cent. of species".

One can only conclude that it is a great pity that such an imperfect and misleading "Guide" should be allowed to appear, as it can do more harm than good.

H. O. M.

THE ASSOCIATION.

Council Meeting held at Polley's Hotel, Pretoria, on 26th October, 1937.

Present: S. T. Amos (Chair), C. J. van Heerden, P. J. du Toit, A. C. Kirkpatrick, C. Jackson, H. O. Mönnig, A. D. Thomas, H. H. Curson, A. M. Diesel, S. W. J. van Rensburg (Secretary).

The Chairman apologised for his absence from most Council meetings during the year and thanked Mr. van Heerden for carrying on in his absence.

(1) Minutes of meeting held on 2nd September, 1937, were taken as read and were confirmed.

(2) **Arising from these minutes:**

(a) *Dr. Schulz.*—Mr. van Heerden reported on the investigations carried out by him and the Secretary. The matter was discussed very fully and while great sympathy with Dr. Schulz was felt by all, it was realized that the Department had fulfilled its legal obligations towards him. It was decided that:

i. A letter be addressed to the Secretary for Agriculture expressing gratitude for what the Department had done in meeting the medical, hospital and travelling expenses incurred, but requesting that in view of the serious injuries incurred, the Department should also consider making an *ex gratia* payment to Dr. Schulz.

ii. The President, Mr. van Heerden and the Secretary see Dr. Schulz and explain the position to him.

iii. A vote of thanks to the Sub-Committee be recorded.

Dr. Curson asked for permission to leave the meeting at this stage.

(b) *Manager: Maitland Abattoir.*—The Secretary submitted correspondence with Major Keppel regarding the scale of salary pertaining to the above post. After discussion it was decided that it would not be opportune to take up this question with the City Council at present.

(c) *Resignations.*—The five submitted at the previous meeting were reconsidered. Decided:—

i. Maj.-Gen. E. Butler's resignation be accepted with regret.

ii. Mr. G. T. Cannon's resignation be accepted with regret and that he be supplied in future with the Journal in view of the fact that he was one of the oldest members of the Association.

iii. Mr. Diesel be asked to intreview Mr. F. Hutchinson.

iv. Mr. J. Forrest's resignation stand over pending a reply from him.

v. The Secretary reported that Mr. McNae had withdrawn his resignation.

(d) *Arrears.*—The Secretary reported that no replies to his registered letters had been received from Messrs. D. B. McCall and G. F. Cameron-Dow. Decided to refer these to the General Meeting with a recommendation that they be dealt with in accordance with the Constitution.

(e) *Book Fund.*—A letter was submitted from Dr. Thomas suggesting that the profits which had accrued be invested by the Finance Committee and that the interest thereon be devoted annually to a prize for the person who had contributed materially in any way whatever in advancing the cause of the veterinary profession. It was decided that:—

i. The scheme be submitted to the General Meeting with the suggestion that Council acts as a committee to administer.

ii. Council record its appreciation of the services rendered by Dr. Thomas and those colleagues who assisted him in carrying on the work of the Book Fund.

(f) *S.A.V.C. Organisation.*—Dr. du Toit reported on interviews he had had with officers of the Defence Force and suggested that in view of certain pending changes the matter be left in abeyance for the present. Agreed.

(3) **Reserve Fund.** The Secretary reported that he had been advised by the Post Office that the Association's holdings in Union Loan Certificates must be reduced to 1,000 units. It was accordingly necessary to withdraw £382 8s. plus accrued interest. Proposed by Dr. du Toit and agreed that £250 of this be invested at the discretion of the Finance Committee and the balance be transferred to the Benevolent Fund.

(4) **Cruelty to Animals.** The report of the Sub-Committee was discussed. Decided to forward this to the Director of Veterinary Services. The desirability of getting veterinarians appointed in research institutes where experimentation on live animals is carried on was emphasised.

(5) **Bacilliary White Diarrhoea.** The programme of B.W.D. testing was considered and it was pointed out that private practitioners were not debarred from bleeding. The President stated that this being the case he had no further objections.

(6) **General.**

(a) A letter was read from the Australian Veterinary Association, in which the latter congratulates the S.A.V.M.A. on obtaining reciprocity with the R.C.V.S. Decided to refer this to the General Meeting.

(b) *Admission of Animals to Onderstepoort.*—The Secretary submitted a report from a member regarding the admission of certain animals for treatment at Onderstepoort without a veterinary certificate. Decided to circularise the relative correspondence among members of Council and to consider the matter at the next meeting.

(c) *Veterinary Inspectors' Association.*—Mr. Diesel reported that the name of the above association has now been changed to " Stock and Assistant Stock Inspectors' Association ".

(Sgd.) *S. W. J. VAN RENSBURG,*
HON. SEC.-TREAS. S.A.V.M.A.

32nd General Meeting held at Onderstepoort, 27th-29th October, 1937.

Present: S. T. Amos (President), C. J. van Heerden, J. Nicol, S. Jackson, J. H. Mason, J. J. G. Keppel, R. Paine, R. Alexander, G. de Kock, J. R. Scheuber, O. T. de Villiers, J. R. Frean, A. D. Thomas, W. G. Barnard, H. G. Franz, J. Zwarenstein, A. E. Lund, N. C. Starke, A. M. Diesel, B. v. d. Vyver, J. H. R. Bisschop, R. B. Osrin, N. T. v. d. Linde, H. Theiler, W. O. Neitz, L. T. Edwards, W. S. B. Clapham, E. M. Robinson, D. Coles, E. T. Clemow, N. Barrie, L. Stonier, J. G. Boswell, A. Thiel, I. P. Marais, J. H. Cloete, P. J. Meara, G. C. v. Drimmelen, A. Tarr, W. J. Wheeler, G. D. Sutton, C. J. Erasmus, M. C. Lambrechts, P. J. J. Fourie, J. G. Bekker, Mrs. J. A. Robinson, M. C. Robinson, J. G. Williams, J. I. Quin, P. Snyman, R. Clark, C. Jackson, R. E. Hartig, H. Sigwart, P. J. du Toit, B. S. Parkin, S. W. de Villiers, D. G. Steyn, J. S. Watt, H. O. Mönnig, M. W. Henning, A. M. Howie, V. Cooper, M. Sterne, K. Schulz, H. P. de Boom, J. v. d. Walt, H. H. Curson, G. Martinaglia, J. Hobday, A. C. Kirkpatrick, C. T. Nilsen, J. H. Schoeman, G. Watt, G. v. d. Wath, S. W. J. v. Rensburg (Secretary).

Apologies for Absence. Messrs. F. A. Verney, W. A. Elder, A. S. Canham, S. H. Ewing.

1. **Minutes** of 31st General Meeting held on 29th and 30th October, 1936, were confirmed.

2. **Matters arising from these minutes.** The Secretary reported that effect had been given to the resolutions passed at the last meeting.

3. **New members.** The following were proposed, seconded and elected: J. A. Badenhorst, C. W. A. Belonje, J. W. A. Brooks, H. P. A. de Boom, M. de Lange, W. J. B. de Villiers, J. M. de Wet, D. A. Haig, H. F. T. Hellberg, J. L. Mainprize, M. J. N. Meeser, T. H. Sandrock, G. D. Sutton, S. J. v. d. Walt.

4. **Presidential Address.**

5. **Election of Council.** The following were elected for 1937-38:
   President: S. T. Amos.
   Vice-President: C. J. van Heerden.
   Hon. Sec.-Treas: S. W. J. v. Rensburg.
   Council Members: R. A. Alexander, H. H. Curson, A. M. Diesel, P. J. du Toit, A. C. Kirkpatrick, H. O. Mönnig, D. G. Steyn, A. D. Thomas.

6. **Resignations.** The following were considered: (a) Maj.-Gen. E. Butler: accepted with regret; (b) G. T. Cannon: accepted with regret. In view of the fact that Mr. Cannon was one of the oldest members of the Association it was decided to supply him with free copies of the

Journal in future. (c) F. Hutchinson and J. Forrest : decided to leave these two in abeyance until further representations had been made to them.

7. **Members in arrear.** The Secretary reported that two members, *viz.*, G. F. Cameron-Dow and D. B. J. McCall were over three years in arrear with their subscriptions and that no reply had been received to registered letters sent to each of them in terms of Section 7 (b) of the constitution. It was decided that their names be removed from the membership list in accordance with the above-mentioned section.

8 and 9. **The Secretary's report** as well as those of the Standing Committees for 1936 - 37 were approved by the meeting.

10. **Revision of Constitution.** The alterations and amendments proposed by Council and circulated to members on 8th October, 1937, were approved after some discussion and after the following changes were made :—

Rule 3 (*a*).—Dr. Fourie objected to the wording of this rule and moved as an amendment that the following be added : " and persons registered under Act 16 of 1923 ".

Dr. Alexander and the Secretary explained that the position of such persons was duly considered by the Committee and that it was felt that registered veterinarians who did not possess diplomas or degrees were not debarred, since such veterinarians had certain " veterinary qualifications," *e.g.* long experience, in virtue of which they were registered under the Act.

The amendment was lost by a large majority.

Rule 7 (*c*).—Substitute " 12 months " for " 6 months ".

Rule 9 (*a*).—Substitute " two years " for " one year ".

Rule 9 (*b*).—Add " and the Editor of the Journal shall be *ex officio* member of the Council ".

Rule 9 (*g*).—Substitute " to be elected by Council ", for " be elected by each committee ".

It was further agreed that Council should decide which four members are to retire at the end of the present year.

1. **General.** (a) ThePresident read a letter from the Australian Veterinary Association congratulating the S.A.V.M.A. on obtaining reciprocity with the R.C.V.S.

(b) Dr. de Kock suggested for next year's programme a discussion on milk from three aspects : i. safety; ii. cleanliness; iii. nutritive value.

(c) *Book Fund.*—The President read the proposals made by Dr. Thomas regarding the interest on the profits derived from the Book Fund for a prize to be given annually to the person who had contributed materially in any way whatever in advancing the cause of the veterinary profession. Agreed : i. to leave the control of this matter to Council; ii. to record the meeting's appreciation of the work done by Dr. Thomas

and those colleagues who assisted him in carrying on the work of the Book Fund.

The President suggested that the name of Dr. Thomas be associated with the prize to be awarded.

The following papers were then submitted :—

11.15 a.m. " Canine Hysteria " by Messrs. A. R. Thiel and J. G. Boswell. Opener Dr. B. S. Parkin.

12.30 p.m. " The Presence of Hydrocyanic Acid in Stock Feeds and other plants " by Dr. D. G. Steyn. Opener Mr. S. J. v. d. Walt.

2 pm. Demonstration on the international tuberculin test by Drs. de Kock and Robinson.

2.30 p.m. Scientific films by Messrs. Bayer Pharma, Ltd.

THURSDAY, 28th OCTOBER.

9 a.m. " Brucellosis " : (a) Contagious Abortion in Bovines " by Mr. W. G. Barnard. (b) " The Brucellosis Problem " by Dr. E. M. Robinson. Opener Mr. J. Nicol.

11.15 a.m. " Calf Paratyphoid " by Prof. M. W. Henning. Opener Mr. W. J. Wheeler.

12 noon. Short discussion on :

(a) Calf Diphtheria by Dr. E. M. Robinson.

(b) Trichomonas by Dr. E. M. Robinson.

(c) Swayback in Lambs by Dr. G. de Kock.

(d) The Influence of Locomotion on Grazing Animals, Dr. Quin.

2 p.m. Demonstration on international tuberculin test.

2.15 p.m. " Diseases of Chicks " by Dr. J. J. Bronkhorst and Mr. Coles. Opener Mr. R. Clark.

FRIDAY, 29th OCTOBER.

9 a.m. " Fluorine Poisoning in Man and Animals " by Dr. D. G. Steyn. Opener Dr. Thomas.

11 a.m. " Certain Aspects of Veterinary Problems as encountered Overseas " by Dr. G. de Kock.

12 noon. " Speculations on the Incidence of Anthrax in Bovines " by Mr. R. Clark. Opener Dr. M. Sterne.

2 p.m. Demonstration " Ventriculectomy laryngis," Dr. Quinlan.

3 p.m. Post Mortem : Tuberculin Reactors , Dr. E. M. Robinson.

3.30 p.m. *General* : (a) The President informed the meeting of Mr. F. J. Carless' serious illness. Agreed that a telegram be sent to Mr. Carless conveying the meeting's sympathy and wishing him a speedy recovery.

(b) Dr. du Toit suggested a discussion on the extension of the meeting to three days. The general feeling was that the extension was well justified. Several speakers suggested that the programme in future should include a free period to enable visitors to consult various officers

at Onderstepoort. Decided that this suggestion be conveyed to the responsible Committee.

(c) Dr. du Toit moved a motion of thanks to the President who, he said, had guided the deliberations with tact and good humour and had lent dignity to the proceedings. He expressed the hope that the President would see his way clear to carry the burden for many more years.

(d) The President appealed to the different branches of the profession to keep their relations on a high level. He felt that there was going to be a great advance in the profession in South Africa and that this must be shared by all members. The discussions had shown that there was a great need for more veterinarians in South Africa.

He moved a general vote of thanks to those who had read papers and given demonstrations, and to the Secretary and Assistant Secretary.

(Sgd.) *S. W. J. VAN RENSBURG,*
HON. SEC.-TREAS. S.A.V.M.A.

Special Council Meeting held at Onderstepoort, 29th October, 1937.

Present: S. T. Amos (Chair), C. J. v. Heerden, R. A. Alexander, D. G. Steyn, A. M. Diesel, A. C. Kirkpatrick, H. O. Mönnig, A. D. Thomas, P. J. du Toit, G. v. d. Wath, S. W. v. Rensburg (Secretary).

The Chairman welcomed the two new members of Council (Drs. Alexander and Steyn).

Editorship of Journal. The Secretary read a letter of resignation received from Dr. Jackson, and the Chairman reported on an interview he had had with Dr. Jackson. Decided that his resignation be accepted with regret and that Council's appreciation of the services rendered by Dr. Jackson while Editor of the Journal be conveyed to him.

Standing Committees. The following were elected :—

1. *Editorial* : P. J. du Toit, A. D. Thomas, C. Jackson, J. H. Mason and M. Sterne. Convenor : M. Sterne pending appointment of Editor.
2. *Finance* : B. S. Parkin, C. J. van Heerden, R. Alexander, G. v. d. Wath. Convenor : R. Alexander.
3. *Library* : P. J. du Toit and C. Jackson. Convenor : C. Jackson.
4. *General Purposes* : C. J. van Heerden, A. C. Kirkpatrick, H. O. Mönnig, H. H. Curson, P. J. J. Fourie. Convenor : H. O. Mönnig. Assistant Secretary : G. v. d. Wath.

Admission of Animals to Onderstepoort. The complaint lodged by a member having been circularised, a full discussion took place. It was decided that :—

i. In view of the fact that this question is now also receiving the attention of the Faculty of Veterinary Science, Dr. Steyn should see the complainant and explain the position to him.

ii. The Secretary write to the Faculty asking the latter to consider very carefully the question of competition between Onderstepoort and private practitioners and suggesting that the fees for treatment and hospital accommodation at Onderstepoort be increased to the level of that charged by private practitioners.

Further it was considered desirable to have a meeting of private practitioners in Johannesburg after the above matter had been considered by Faculty.

(Sgd.) *S. W. J. VAN RENSBURG,*
HON. SEC.-TREAS. S.A.V.M.A.

NEWS.

The Council of the South African Biological Society has awarded the 1937 Senior Captain Scott Medal to Dr. E. M. Robinson.

* * *

The Council of the South African Biological Society has also decided to present a silver medal to the outstanding student in the Faculty of Veterinary Science. This award has been established as a memorial to Sir Arnold Theiler.

* * *

The following candidates obtained the B.V.Sc. degree in December, 1937 — J. L. Doré, E. B. Kluge and L. J. F. von Maltitz.

* * *

Dr. N. Viljoen has been awarded the D.V.Sc. degree of the University of South Africa for a thesis on "*Cysticercosis in Swine and Bovines with Special Reference to South African Conditions.*"

* * *

Mr. R. du Toit, former secretary of the Association, has recently returned from America after a fifteen months' tenure of a Commonwealth Fund Service Fellowship.

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1938.

Canine Hysteria.

By A. THIEL, B.V.Sc., and J. G. BOSWELL, B.V.Sc., Johannesburg.

In presenting this paper on canine hysteria, we should like to emphasize the fact that we are dealing with a disease which, up to the present, has not been known to exist in South Africa to any great extent; but which nevertheless seems to be on the increase. Little research has been done on this problem as it occurs in South Africa; but overseas it has aroused great interest, and many very interesting observations have been made and a number of hypotheses put forward.

This paper will deal with the disease from the practitioner's point of view, and is based on clinical experience only, as no bacteriological or pathological research has been possible.

Definition.

The condition may be defined as a disease of dogs characterized by paroxysmal attacks of deranged consciousness or sensibility, manifested largely in barking, crying and running.

History.

Apparently the disease was first noted in the southern states of the U.S.A. in about 1916, and spread north to the northern states and Canada in about 1920. It first appeared in England in about 1924 and as many of the early cases occurred in south-west Lancashire and Cheshire it was suggested that it was brought as an infection from America through Liverpool. There is now no part of the British Isles from which cases are absent and it appears to be fairly well known on the Continent. As far as South Africa is concerned, we have very little information. We can only state that we have definitely encountered the disease, or a disease very closely resembling canine hysteria as described by overseas authors, in and around Johannesburg, although we understand that the condition also exists in other parts of the country. The importation of highly bred dogs from everseas probably takes place to a far greater extent into Johannesburg than into any other part of the Union, and possibly this explains why more cases are observed here in Johannesburg each year.

Occurrence.

The disease occurs at all times of the year, at any age, and in both sexes; but it would seem to be more prevalent in highly bred animals and especially in young dogs. Overseas experience shows that convulsions or fits as well as hysteria have certainly increased in recent years in the

young of those breeds with fine narrow heads. In many breeds, the aim of the breeder appears to be to increase the length and decrease the width of the skull. Such alteration of the natural shape of the cranium seems likely to effect the growth and development of the brain itself, and may possibly lead to cerebral weakness and instability.

Aetiology.

Naturally it is on this aspect of the problem that the greatest interest is centred. Numerous hypotheses have been put forward and long discussions have taken place; but the whole question is still in the melting pot. Among the causes suggested are : (1) infection, (2) diet, (3) digestion, (4) deficiencies, (5) alimentary toxæmia, (6) internal parasites, (7) allergy, (8) circulatory disturbances, (9) faulty hormone balance, (10) environment, (11) sundry irritations and excitements, (12) discipline, (13) excessive heat loss.

We propose to deal in detail only with those on which we feel capable of expressing an opinion based on personal observation.

(1) *Infection.* — Let us consider firstly the question of infection. Although this hypothesis is favoured by many, we cannot support the view that the disease is of an infectious nature. Some workers claim that they have isolated and cultivated a specific organism which is capable of reproducing the disease by inoculation into non-affected dogs. Apart from the fact that no constant organism has been isolated and that some believe the cause to be a bacterium and others a virus, there are numerous cases which have never been in contact with other dogs, and an even greater number of cases where dogs have not contracted the disease after prolonged contact with affected dogs. However, we have observed cases of distemper which have commenced with hysterical attacks but which have later gone over to the true nervous form of distemper with fits and convulsions. This may be a misleading factor in arriving at a conclusion regarding the ætiology of the disease.

(2) *Diet and Digestion.*—With the march of civilization an increasing refinement of foods has taken place; the dog has undergone a gradual evolution from a carnivorous to an omnivorous animal and the natural foods he gets are refined and devitalized. These changes, accompanied by a much more restricted type of life and frequently accompanied in house dogs by delayed defæcation, must have had a profound effect on the digestive and nervous systems.

Many workers claim that the feeding of biscuit has a marked effect on the incidence of the disease and our experience tends to support this view. We have encountered numerous cases in dogs fed largely or exclusively on biscuit, which have made a complete recovery when the diet has been altered and biscuit eliminated. One such dog showed a recurrence of the disease when biscuit was again included in the diet

some time later. Against this theory it must be pointed out that there are probably thousands of dogs fed on a biscuit diet, which never show any signs of hysteria. Furthermore the elimination of biscuit does not always effect a cure; and finally, the disease may occur also in animals which have never received a biscuit diet.

Some workers maintain that since the digestion of animal protein takes place in the stomach in an acid medium and that since starches and sugars tend to lessen acid gastric secretion, the feeding of the two mixed may be the cause of indigestion leading to hysterical attacks. In practice, however, we have not encountered a single case of hysteria amongst racing greyhounds, which are normally fed on a mixture of minced meat (raw or cooked), vegetables and rusks; but it is interesting to note in this connection that attacks of acute enteritis are not infrequent. It is doubtful whether the feeding of the animal proteins and carbohydrates mixed can be held responsible for these outbreaks.

(3) *Intestinal Parasites.*—It is well established that a severe verminosis may have a marked effect on the central nervous system. We have attributed many cases of hysteria to verminosis as the removal of the worms often overcame the nervous symptoms.

(4) *Environment.*—There is very little doubt in our minds that environment plays a very great part in the causation of this disease. We have had cases which have become quite normal on being taken from their homes to kennels and which have remained normal for weeks on end without any special treatment, but have shown a recurrence of the attacks shortly after being returned to their homes. H. R. Hewitson, in the *Veterinary Record,* tells of a Labrador dog, four years old, which had a portion of the garden at the side of the house set apart for its kennel and exercising run, and which had been having attacks for about three weeks before he was consulted. He saw the animal in its pen and observed that even when quiet it had an expectant, watchful expression. As treatment for over a fortnight had no effect, the dog was removed to kennels. In two days it was normal and it had no further attacks during a seven weeks' stay. An hour after it returned home it was put in its pen and immediately had a severe attack. These occurred frequently for a week, after which the owner reluctantly agreed to move the kennel and run to another part of the garden. Five years later there had still been no recurrence of the disease.

(5) *Sundry Irritations and Excitements.*—Under this heading we would include such conditions as canker, abscessation of the anal glands, foreign bodies, severe constipation, teething, bad teeth, excitement and fright. In one instance a dog frequently had hysterical attacks when its owner patted or talked to it. On other occasions this same dog would suddenly have an attack when being taken out for exercise on the leash.

The excitement of being taken out was probably too great. At other times the dog was most docile and even lethargic.

Symptoms.

The symptoms are dramatic in their onset. The animal suddenly, and usually for no apparent reason, becomes restless, cries out, barks, whines and bolts headlong for freedom, the direction of its flight being governed only by the path of least resistance, though minor obstacles are taken or crashed through heedlessly. Out-of-doors the victims will scale very high walls and obstacles, and when free they often run until they fall from physical exhaustion. Indoors there is a tendency to seek seclusion in corners or under furniture. Both fæces and urine may be passed during this stage of panic. The pupils are dilated, the expression is vacant and the patient fails to recognise the owner or the owner's call. An hysterical patient seldom becomes vicious and seldom deliberately attacks anyone; but incautious persons have occasionally been bitten in attempts to corner the victim and catch hold of it.

The attack may last for only a minute or two, or may go on for half an hour or more, and is followed by some degree of exhaustion depending upon the severity of the attack. Further, new fits may follow or the patient may become normal and remain so. The attack may come on during sleep and quietude or in circumstances of noise, excitement or emotion.

Post-Mortem Changes.

In the few case where we have had the opportunity of making a post-mortem examination we have been unable to find any lesions.

Differential Diagnosis.

The symptoms are so characteristic that the disease can hardly be mistaken for any other condition. As already pointed out we may encounter these hysterical attacks in the nervous form of distemper, but these later go over to the typical fits and convulsions.

Hysteria and rabies may be confused by the layman, but in rabies the symptoms are more prolonged, and are followed by characteristic paralytic symptoms and the death of the patient in 7–12 days. Furthermore, rabies may be confirmed by the demonstration of Negri bodies and by subinoculation into susceptible animals.

Prognosis.

In the great majority of cases this is favourable. Approximately 95 per cent. of recoveries may be expected if the attacks are unassociated with a more serious condition such as distemper. The mortality in uncomplicated cases is probably less than one per cent. and when it does occur is usually due to asphyxia. It must be understood that by recovery we do not mean a state of complete freedom from any further attacks, but merely recovery from the existing attack.

Prophylaxis.

As already mentioned, the modern tendency of breeders to develop an unnatural type of dog with a long tapering skull has possibly an important influence on the incidence of the disease, and if breeders would allow animals to revert to their natural type and would cultivate the broad skull and robust physique, and if they would rear, feed, house and exercise their animals according to rational principles, there would no doubt be a great reduction in the incidence of the disease. As it is, little trouble is experienced with dogs that have received from puppyhood ample raw and natural food, and have been kept free from internal and external parasites or other irritations.

Treatment.

If possible the animals should be restrained, but often we find it far more satisfactory to leave the animal entirely alone until it has quietened down somewhat, when a hypodermic injection of a sedative such as morphine (¼ to 2 grains according to the size of the animal) or "Luminal" solution (Bayer) may be given. The average dose of the latter that we prescribe is 1 cc. of the 20% solution, but for toy breeds or young pups the dose is reduced.

If possible the patient should be isolated and placed in a quiet and preferably slightly darkened room. We cannot agree with the practice of placing animals in a completely darkened room, as this seems merely to increase the fear associated with the condition. The animals should be kept quiet for the next two or three weeks and all forms of excitement avoided.

When the acute symptoms have abated a laxative should be given, followed by a course of sedatives such as potassium bromide or luminal sodium. We prescribe ½ to 1 grain of the latter daily, according to the size of the patient.

It is of course most important to attempt to seek the cause of the attack, and to take the necessary steps to prevent a recurrence. It may be necessary to give an enema to overcome a severe constipation or to carry out treatment for worms should these be considered to be the cause. Teeth, ears, and anal glands should also be examined. The questions of environment, of diet, and of digestion should be considered and, if faults are discovered, these should be adjusted on rational lines. As a routine measure we always advocate the elimination of the biscuit diet.

In the majority of cases the treatment just described proves satisfactory, but naturally, treatment may have to be altered to suit the particular case.

Reference.

HEWETSON, H. R. (1936). Hysteria in dogs. *Vet. Rec.* **48** (41) : 1202-1218

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IX (2): 52-59.
1938.

The Genetic Nature of Susceptibility to Cancer.

By J. J. BRONKHORST, Ph.D., B.Sc. (Agric.), Onderstepoort.

During the present century the science of genetics has made tremendous strides, and the evidence which has accumulated clearly demonstrates how inheritance takes place, *i.e.* by means of genes situated at definite points on the chromosomes. For example, we know that hæmophilia is inherited as a single sex-linked recessive gene, but we do not know how the gene delays the clotting time of blood. Does it produce its effect by enzyme action, by direct chemical action or by catalysis ?

The science of genetics has made at least one contribution of enduring value to cancer research. This contribution has been made possible by the study of spontaneous and transmissible tumours both in inbred and in heterogeneous stocks of mice. As a result of these studies it seems to be well established that there is a predisposition to cancer, although the mode of inheritance of this predisposition has not been demonstrated with exactness or finality. This shortcoming does not, however, allow us to overrule or overthrow all the evidence accumulated within the last twenty years. Nevertheless, the demonstration of heritability does not answer the question of how normal tissue cells change to cancerous cells, and the solution of this problem will probably go hand in hand with the elucidation of the mode of action of the gene itself.

As early as 1907 Tyzzer speculated about the importance of inheritance in spontaneous and transmissible tumours. He emphasized the necessity for the selection and the establishment of uniform strains of mice for such studies, and debated whether general or specific factors were responsible for tumour formation; and even anticipated the multiple factor hypothesis which plays such an important rôle in the more recent discussions of the genetic nature of tumour susceptibility.

It is quite clear that all neoplasms do not behave similarly and for this reason we will deal with mammary gland tumours, lung tumours, and leukæmias under different headings.

Mammary Gland Tumours of Mice.

Strains of Inbred Mice.

Before any attempt at a genetic analysis of any character can be made, true breeding lines must be established. In plants, where self fertilization can be practised, this is a comparatively easy matter. The worker with animals has a less simple task : even brother and sister matings, the closest form of inbreeding, take about 17 generations to establish a fairly

good degree of genetic homozygosity. Several strains of mice bred in this way have been described. Bittner (1935a) gave details of his inbred albino strain of mice which is now generally referred to as the A stock. Sixty-four % of the breeding females which lived longer than four months developed *mammary gland carcinomata* at an average age of 12.3 months. The males of this strain which lived longer than 10 months succumbed to *primary lung tumours* at an average age of 15.2 months. Thirty-six % of the breeding females with mammary gland tumours also had primary or secondary lung nodules. The average age at death of females which did not show cancer was 9.1 months. This means that the majority died before reaching the most dangerous age, and it is interesting to speculate on what would have happened had they lived longer.

The same author (1935b) also described another strain, a black agouti stock, generally known as the C3H or Z strain, in which 75% of the breeding females over 4 months old developed mammary gland carcinomata at an average age of 10.7 months.

The breeding behaviour of another well-known high-cancer strain, the dilute brown (dba) stock of mice, was described by Murray (1934). Fifty-eight and one-half % of the 2,252 breeding females developed mammary cancers at an average age of 10.6 months, while the remaining 934 females which did not develop cancer died at the average age of 8.7 months. This is significantly earlier than the age at death of the cancerous mice. This strain of mice had reached such a degree of homozygosity that the number of heterozygous genes was probably smaller than the mutation rate. As a result, further inbreeding could not make the strain more homozygous since the production of new mutations balanced the effect of inbreeeding.

Strains of mice with a very low incidence of spontaneous neoplasms have also been described. Strong (1937a) described his JK strain, which was free of cancer for 22 generations. This strain was produced by brother-sister mating over a period of nine years. Only one cancer, a carcinoma of the lung, has ever been found in mice of this strain. The average age of death for the first ten generations was 20.4 months, and the succeeding generations died at the ripe old age of 22.3 months.

The establishment of pure breeding lines in a genetic study of this kind is essential preliminary work and the mere fact that this has been possible is evidence of the heritability of spontaneous tumours.

Cross-breeding Experiments.

Having described some of the established lines which show a low or a high incidence of cancer, we can now discuss the results of hybridization or cross-breeding of these strains.

Bittner (1936a) made reciprocal (*i.e.* C3H ♂ x I ♀ and C3H ♀ x I ♂)

crosses between the C3H strain which developed 78.8 % mammary tumours at an average age of 10.7 months, and the I strain where no mammary gland tumours had been observed. The results of these crosses are given in table I.

TABLE I.

RESULTS OF RECIPROCAL CROSSES BETWEEN HIGH AND LOW STRAINS OF MAMMARY TUMOURS.

Mating.	Number of Mice.	Percent. mammary tumours (of total).	Average age in months.
C3H♀ x I♂	36	91.7	10.4 ± .4
I ♀ x C3H♂	10	Nil	Nil

Thus when the mother came from the high tumour stock, 91.7% of the female offspring died of mammary gland tumours. The male from the high mammary gland tumour stock did not, however, transmit the tendency to develop breast tumours to his offspring.

Thus this appears to be a case of maternal inheritance; or, as the author put it, " An extra-chromosomal influence is chiefly involved in the production of breast tumours in mice". By extra-chromosomal influence is meant the transmission of a character by means other than the chromosomes, *i.e.* through the cytoplasm or during intra-uterine life.

Further evidence that an extra-chromosomal influence is involved in the inheritance of breast tumours in mice was provided by Murray and Little (1935, 1936). Reciprocal crosses were made between the dilute brown stock (dba), which has a mammary tumour incidence of 80–90%, and a black stock (C57), where the incidence of mammary tumours is nil. When the dilute brown was used as the female parent, 39.8% of the F1 and 35.5% of the F2 virgin females developed mammary tumours. In the reciprocal cross (dba ♂ x C57 ♀) only 6.06% of the F1, and 5.96% of the F2 virgin females developed breast tumours. Again reciprocal crosses did not agree, thus confirming the work of Bittner cited above.

As the theory of extra-chromosomal inheritance may be somewhat strange to the reader, it will be considered in some detail, in so far as it affects mammary tumour inheritance.

To substantiate their view of extra-chromosomal inheritance, Murray and Little secured data on various back-crosses. The following symbols were introduced to clarify the plan of the experiment.

C = Chromosomal material derived from the high mammary tumour strain.

c = Chromosomal material derived from the non-mammary tumour strain.

E = Extra-chromosomal influence derived from the high mammary tumour strain.

e = Extra-chromosomal influence derived from the non-mammary tumour strain.

The following four back-crosses were made. The contribution of the female is shown as the numerator of the fraction, the male contribution as the denominator.

I. $\frac{\text{Fl}\,♀\,ce}{C} \times \frac{♂\,C}{C}$ An F1 female derived from a non-cancer female by a high cancer male, back-crossed to a high cancer male.

II. $\frac{♀\,CE}{C} \times \frac{\text{Fl}\,♂\,c}{C}$ A high cancer female back-crossed to an F1 male derived from a low cancer female by a high cancer male.

III. $\frac{\text{Fl}\,♀\,ce}{C} \times \frac{♂\,c}{c}$ An F1 female derived from a non-cancer female by a high cancer male, back-crossed to a non-cancer male.

IV. $\frac{\text{Fl}\,♀\,CE}{c} \times \frac{♂\,c}{c}$ An F1 female derived from a high cancer female by a non-cancer male back-crossed to a non-cancer male.

The first cross contains *three* high mammary tumour chromosomal contributions and *one* non-tumour. The extra-chromosomal influence is non-mammary-tumour.

The chromosomal contributions of the second cross are exactly the same as in the first cross, the extra-chromosomal influence is from the high mammary tumour strain. The third and fourth crosses both contain three non-mammary-tumour chromosomal contributions, the only difference being that the fourth cross contains the extra-chromosomal influence from the high mammary tumour strain.

The total number of virgin female mice which died in each cross, together with the incidence of mammary tumours, is shown in table II.

TABLE II.

RESULTS OF FOUR TYPES OF BACK-CROSSES.

Cross.	Total mice dead.	Non-mammary tumour.	Mammary tumour.	Percent mammary tumour.
I	10	10	0	0.0
II	47	16	31	65.9
III	13	13	0	0.0
IV	23	7	16	69.5

The four crosses demonstrate the unimportance of the chromosomal contributions; in the first two crosses the chromosomal complexes for high

mammary tumours are exactly the same, but the incidence of mammary tumours follows the presence of the extra-chromosomal influence. The same thing happens in the last two crosses. The authors thus concluded that in the four types of back-crosses cited the incidence of mammary tumours in mice depends primarily on the direct transmission of the extra-chromosomal influence.

Further evidence of the effect of the extra-choromosomal influence in the transmission of breast tumours was given by Bittner and Little (1937). An abbreviated summary is presented in table III. These data are perfectly clear and substantiate what has been said before, that breast tumours are transmitted through the female only.

TABLE III.

EXTRA-CHROMOSOMAL INFLUENCE IN VARIOUS CROSSES.

Mammary gland tumour incidence in the original stocks.		Evidence of Extra-chromosomal Influence.			
Stock	Percentage tumours in breeding ♀♀	Mating.	F1 Number of mice.	F1 Percentage breast tumours.	F1 Remarks.
dba	58.7				
dba	76.0				
		dba♀ x C57♂	113	39.8	Virgin ♀♀
dba	76.3	dba♂ x C57♀	379	6.1	,, ,,
A	83.2	dba♀ x MBact♂	69	68.1	,, ,,
		dba♂ x MBact♀	27	7.4	,, ,,
C3H	78.0				
		A ♀ x CBA♂	44	90.9	Breeding ♀♀
C57	0.0	A ♂ x CBA♀	16	31.3	,, ,,
C57	1.7	C3H♀ x I ♂	36	91.7	,, ,,
		C3H♂ x I ♀	10	0.0	,, ,,
CBA	2.8				
		dba♀ x C57♂	7	100.0	Breeding
CBA	13.5	dba♂ x C57♀	67	3.0	and Virgin ♀♀
N	8.8	C3H♀ x N♂	46	97.9	Breeding ♀♀
		C3H♂ x N♀	18	27.8	,, ,,
MBact	2.0				
	Totals	High cancer ♀ low cancer ♂	315	68.9	
		Low cancer ♀ high cancer ♂	517	7.2	

Some evidence opposed to the extra-chromosomal influence theory of Little and his co-workers was advanced by Bagg and Jackson (1937), who devised the so-called " functional test," in which females were compelled to breed rapidly and not allowed to suckle their young. When

Little's low tumour stock C57 was subjected to this technique, the incidence of breast tumours increased from one to fifteen %. Thus by applying the "functional test" it was possible to detect females which were potential tumour mice. The mice which, in these circumstances, developed tumours belonged mainly to certain family groups, which was further evidence of the impurity of this low tumour strain. These workers also made reciprocal crosses between high and low tumour stocks, and in one of the completed experiments, F1 hybrid female mice whose mothers were derived from the C57 low tumour strain, showed 62% mammary cancer when subjected to the functional test, whereas under normal breeding conditions Little and his co-workers found a very low incidence of mammary tumours (see table 1). Bagg and Jackson concluded that if the extra-chromosomal influence were present at all, it was masked by the change in the physiological set-up of the experiment.

Bernstein (1936) pointed out that in the cross, low tumour female x high tumour male, deaths are high before the average cancer age is reached, and as all such females are classified as non-cancerous, the question arises as to what would have happened had these females lived until they reached the tumour age. After 540 days (when both non-tumour and tumour females were dying at approximately the same rate) there was no significant difference in the tumour mortality from reciprocal crosses.

The Extra-chromosomal Influence.

Influence of the Mother's Milk.

Little and his co-workers continued their work in an attempt to determine the nature of the extra-chromosomal influence. Progeny of the A stock, which normally developed a high percentage of tumours, were fostered on two strains, CBA and C57, in which the incidence was very low. Bittner (1936b and 1937) gave the results of these nursing experiments. Table IV gives a comparison of tumour incidence and average age between control and fostered groups of A stock breeding females.

TABLE IV.

THE CANCER INCIDENCE IN CONTROL AND FOSTERED A STOCK BREEDING FEMALES.

A Stock.	Controls.	Fostered by CBA ♀♀ (low tumour).	Fostered by C57 ♀♀ (low tumour).
Total number	788	91	41
Percentage breast tumours	83.2	23.1	4.9
,, lung ,, ...	3.7	33.0	46.3
,, non-tumorous	13.1	44.0	48.8
Average age at :			
Appearance of breast tumours	11.5 mo.	11.8 mo.	10.0 mo.
Appearance of lung tumours	15.6 ,,	16.9 ,,	18.0 ,,
Death of non-tumorous mice	11.8 ,,	14.9 ,,	15.7 ,,
Death of all mice	11.7 ,,	14.8 ,,	16.5 ,,

It is clear that the incidence of breast tumours in a high-tumour strain is reduced considerably when the mice are fostered by low-tumour mothers.

The author concludes : " The results obtained by foster-nursing of young from a high-breast-tumour line by females of a low tumour stock may offer an explanation for the extra-chromosomal influence in the transmission of mammary tumours. Lung tumour susceptibility, which may be transmitted through either parent, is not affected.'

The Influence of Hormones.

It was early determined by Lathorp and Loeb (1913-14) that pregnancy had a very pronounced influence on the incidence of cancer of the mammary gland. Lacassagne (1932) was able to induce cancer in the rudimentary mammary gland of the male by continual injections of female œstrogenic hormone. This has been confirmed by other workers, but only in strains where the spontaneous incidence in females is high.

In order to determine whether the difference between males and females is due to some inhibiting hormone or to some difference between male and female in the quantity or quality of secretion, Murray (1937) started an extensive experiment on parabiotic twins (mice of opposite sexes joined surgically so as to establish a common blood-circulation). Altogether 189 pairs were started, but a large number died during the first two months owing to operative shock, non-adaptation to being attached to each other, and to wounds incurred in struggling.

From about 8 months of age, deaths due to mammary tumours were high in breeding and virgin females, but none occurred in the parabions. This introduction of hormones from the male into the bloodstream of the female caused an upset in the sexual cycle of the female, and the ovaries were stimulated to a precocious and prolonged production of follicles which degenerated within the gland without forming luteal tissue. Since none of the parabiotic females developed mammary tumours, it seems that the proliferation and change to malignancy in the mammary gland may be due to the luteal fraction of the ovarian hormone. The difference in cancer incidence between breeding and virgin females may be due to the prolonged luteal phase of pregnancy.

Conclusion.

It seems reasonable to conclude that mammary gland tumours are inherited. The mode of transmission has not been definitely established, and other factors, such as the environment, both internal (gestation, lactation, and glandular secretions) and external (housing, feeding, irritation), play a definite rôle. This is not necessarily opposed to the genetic theory, as the modern trend of thought is that the individual inherits certain potentialities which are expressed or suppressed by the

action of the environment. Between conception, when the individual receives its genic complement, and the appearance of cancer, there is an extensive period of time ; and it is thus obvious that genes, to be effective at the onset of malignancy, must influence, either directly or indirectly, some phase of the normal physiology of the individual.

(*To be concluded.*)

NEWS.

Dr. J. G. Bekker has resigned from his post of Municipal Veterinary Officer, Pretoria, in order to join Dr. G. Kind, of Johannesburg, in private practice. Dr. I. P. Marais, of Onderstepoort, has been appointed Municipal Veterinary Officer, Pretoria.

* * * *

Lieut.-Col. Wakefield Rainey, C.B.E., M.R.C.V.S., formerly of the R.A.V.C., has been appointed secretary, handicapper and measurer of the Bloemfontein Turf Club. Lieut-Col. Wakefield Rainey was Assistant Director-General of Army Veterinary Services 1915–1919; Chief Veterinary Officer of the Fiji Islands 1919–21; and editor of the Official History of the Great War (Veterinary Services). This is the first time that a veterinarian has been appointed secretary of a turf club in South Africa.

* * * *

Dr. H. H. Curson has again been elected to represent the Association on the South African Veterinary Board, while Dr. H. O. Mönnig has been appointed to represent the Faculty of Veterinary Science.

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The Presence of Hydrocyanic Acid in Stock Feeds and Other Plants.

by DOUW G. STEYN, B.Sc., Dr.Med.Vet., D.V.Sc.

It is well known that some plants which are extensively used as stock-feeds may under certain soil and climatic conditions and at certain stages of growth contain dangerous amounts of hydrocyanic acid. As examples the following may be quoted: linseed cakes, Sudan grass, Transvaal "kameeldoring" pods (*Acacia giraffæ* Willd.), and kaffircorn. To these plants, lucerne (*Medicago sativa* L.) and *Atriplex semibaccata* R. Br., the so-called creeping salt bush, can be added.

Lucerne.

Lucerne is known to cause hoven when animals not accustomed to it eat excessive quantities, or when it is in the wilted or frosted state. Even lucerne hay has been known to cause hoven. Lucerne grown not only in different areas but even on different parts of the same farm may vary in its capacity for causing hoven. This is realised by many stock farmers in the Karroo. The remark is frequently made by some of them that they do not buy lucerne hay grown on certain farms, as it is liable to cause hoven in animals fed on it.

Death caused by lucerne has in the past been ascribed to hoven due to excessive fermentation causing the liberation of gases, such as hydrogen, nitrogen, carbon dioxide and methane. In the light of results obtained in experiments conducted by the author with specimens of wilted and fresh lucerne, it now appears probable that hydrocyanic acid may be a contributory factor in the causation of hoven and death in animals, especially when lucerne is eaten in the wilted or frosted state.

The experiments were conducted with specimens of lucerne collected from a small plot of lucerne growing in a private garden at Onderstepoort. Specimens of wilted and stunted lucerne, which were collected at 10 a.m. on 8/11/36 during a spell of very hot and dry weather, were submitted to the picrate paper test and found to contain an appreciable amount of hydrocyanic acid. On the other hand, not a trace of this poison was detected in specimens of fresh lucerne collected at a later date from the same plot about a week after heavy rains had fallen.

Rigg, Askew and Kidson (1933), who submitted a large number of pasture plants to the hydrocyanic acid test, found 0.0015 per cent. of this acid in lucerne. From their publication it appears that they conducted tests with fresh (unwilted) specimens of lucerne.

I should mention here that lucerne has, on quite a number of occa-

sions, not only in South Africa but also in other countries, been suspected of causing photosensitization. In these cases no clinical ivterus was noticed ; [Byrne (1937); Hausmann and Haxthausen 1929)].

For further information in regard to the toxicity of lucerne see Steyn (1934).

ATRIPLEX SEMIBACCATA *R. Br.* (*Creeping Salt Bush*).

This plant, which belongs to the family *Chenopodiaceæ,* is known to be a valuable feed in the more arid regions of South Africa. Some months ago specimens of the creeping salt bush growing in the Smithfield district were submitted to Onderstepoort for investigation, as the plant was suspected of having poisoned sheep. These specimens of salt bush, which were in a wilted state, were found to contain appreciable quantities of hydrocyanic acid. The plant was air-dried and found still to contain hydrocyanic acid, although the quantity had decreased during the process of desiccation. The hydrocyanic acid is probably present in the plant in the form of a cyanogenetic glucoside, as tests with the dry plant material without emulsin yielded negative results, whilst they were positive when emulsin was added.

Seed of the bush obtained from Dr. M. Henrici, Veld Reserve, Fauresmith, was subsequently sown in the poisonous plant garden at Onderstepoort. Tests conducted with fresh specimens of the plant, when it was about 2–3 inches high, did not show the presence of HCN. The investigations are being continued.

The fact that specimens of the wilted creeping salt bush were found to contain appreciable quantities of hydrocyanic acid should serve as a warning that the plant may be dangerous when eaten in large quantities in the wilted or frosted state.

TEPHROSIA MACROPODA *E. Mey.*

This plant, which is used as a fish poison by the Zulus, was sent in for investigation as it was suspected of having caused death in sheep. It arrived at Onderstepoort in a wilted state and was found to contain an appreciable amount of hydrocyanic acid. Consequently the possibility existed that the plant might cause poisoning when in the wilted or frosted state.

No hydrocyanic acid was detectable in specimens of fresh *Tephrosia Vogelii* Hook (fish bean) growing in the poisonous plant garden at Onderstepoort.

EUPHORBIACEÆ.

Acalypha indica L. (O.P. Herb No. 2730; 7.7.36).

Common names.—Indian Acalypha; Mukta-jhuri (Indian). Native name (N. Transvaal) : Machelikoane.

Origin.—Vetfontein, Northern Transvaal.

State and stage of development.—Slightly wilted and in the flowering stage.

Uses.—According to the British Pharmaceutical Codex (Editorial, 1934) the plant is a gastro-intestinal irritant. In small doses it acts reflexly as an expoctorant and in large doses as an emetic. It has been used as a substitute for ipecacuanha. The natives in the Northern Transvaal use the plant in the treatment of eye-diseases.

Constituents.—It contains an alkaloid, *acalyphine*, resin, tannin and volatile oil (Editorial, 1934, and Wehmer, 1929). Mr. G. Roets, B.Sc., of Onderstepoort, found that the plant contains hydrocyanic acid. An article on the cyanogenetic glucoside present in the plant is being prepared by him and Dr. C. Rimington for publication.

Experiments.

Rabbit A (2.0*Kg.*)—10 gm. of the slightly wilted plant per stomach tube at 11 a.m. on the 7.7.36. There was laboured respiration within two hours after drenching.

8.7.36 : Apathetic, not feeding, laboured respiration, and accelerated but forceful heart-action.

9.7.36 : Died at 7 a.m.

Post-Mortem Appearances.—Blood of a dirty chocolate-brown colour and not coagulated; pronounced œdema of the lungs; pronounced fatty degeneration of the liver; gastric mucosa covered with a thick layer of mucus. Stomach contents positive for hydrocyanic acid.

Rabbit B (2.5 *Kg.*)—20 gm. of the slightly wilted plant per stomach tube at 11 a.m. on 7.7.36.

There was pronounced dyspnoea and restlessness within one hour of drenching. The heart action was very much accelerated and became progressively weaker. Paresis and paralysis also set in commencing in the fore-quarters and progressing until the animal was prostrate and unable to rise. Dyspnoea was very pronounced. The animal struggled continuously and died with convulsions, probably due to asphyxia, one-and-a-half hours after drenching.

Post-Mortem Appearances.—Blood of an intense dark chocolate-brown colour and not coagulated; all internal organs of a light dirty-brown colour; pronounced hyperæmia and œdema of the lungs; very pronounced hyperæmia of the gastric mucosa; the mucosa of the stomach and small intestine covered with a large amount of mucus.

Rabbit C (2.1 *Kg.*).—5 gm. of the dry plant (dried in shade) per stomach tube daily from 12.8.36 to 27.8.36.

Excepting loss of appetite no symptoms of poisoning were seen in the animal until one hour after the 5 gm. dose administered on the 27.8.36. There was pronounced restlessness, dyspnoea, and general convulsions with the head drawn backwards. Death occurred in a state of paralysis one-and-a-half hours after the last dose on 27.8.36.

Post-Mortem Appearances.—Pronounced œdema and slight hyperæmia of the lungs; heart in systole; slight subacute catarrhal gastro-enteritis; urine of a reddish colour; blood dark brown in colour and not coagulated. Stomach contents positive for hydrocyanic acid.

Rabbit D (2.45 *Kg.*).—7.5 gm. of the dry plant per stomach tube at 9 a.m. on 12.8.36.

The animal developed symptoms of poisoning similar to those described in rabbits A and B and died fifteen hours after drenching. The post-mortem appearances were also similar. The urine was of an intense reddish-brown colour and fluorescent.

Rabbit E (2.75 *Kg.*)—12.5 gm. of the dry plant per stomach tube at 9 a.m. on 12.8.36.

The animal died one-and-three-quarters of an hour after drenching. The symptoms and post-mortem appearances were similar to those described above. There were a number of hæmorrhagic patches on the gastric mucosa.

Rabbit F (2.1 *Kg.*).—12.5 gm. of the dry plant per stomach tube on 12.8.36.

Death occurred one-and-a-half hours after drenching; the symptoms were similar to those described above.

Post-mortem appearances.—As described above, and in addition extensive hæmorrhage into the duodenal mucosa, which showed pronounced swelling; pronounced hyperæmia of the gastric mucosa.

The dark chocolate-brown heart blood was collected and examined spectroscopically by my colleague Dr. J. I. Quin. Hæmolysis was absent and no methæmoglobin bands were detected. It should, however, be pointed out that owing to the intense dark-brown discolouration of the blood it had to be diluted considerably to facilitate spectroscopical examination. Thus it is possible that the dark-brown discolouration was due to the formation of methæmoglobin, in spite of the fact that the spectroscopic analysis was negative.

As hydrocyanic acid is not known to cause such an intense dark chocolate-brown discolouration of the blood, it was thought that the plant might contain a second, or even a third active principle causing the discolouration of the blood and the gastro-intestinal irritation. There seems little doubt that the most pronounced dyspnoea seen in the experimental animals is due not only to the toxic effects of hydrocyanic acid but also to the discolouration of the blood, which reduces its oxygen-carrying capacity.

An experiment was therefore planned to determine whether the plant was still toxic to rabbits when freed from hydrocyanic acid.

The dry plant was ground, mixed with emulsin, and moistened. It was then incubated at about 40° C for a few days until only the slightest

trace of hydrocyanic acid was present, and again drenched to two rabbits as follows :—

Rabbit G (2.5 *Kg.*).—15 gm. of the dried incubated plant on each of four consecutive days.

At no time were any symptoms of poisoning discernible.

Rabbit H (1.9 *Kg.*).—10 gm. of the dried incubated plant on each of four consecutive days.

The animal developed no symptoms of poisoning.

From these results it appears that the active ingredient or ingredients responsible for the production of the dark chocolate-brown discolouration of the blood and the gastro-intestinal irritation were inactivated by the method applied to expel the hydrocyanic acid from the plant.

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The Immunization of Bovines against Lamsiekte.

By J. H. MASON, H. P. STEYN and J. H. R. BISSCHOP,
Onderstepoort.

Introduction.

The classical experiments of Theiler and his co-workers (1927) proved that, in South Africa, lamsiekte is caused by the ingestion of the toxin of *Clostridium botulinum,* type D, and occasionally by the ingestion of that of *Clostridium botulinum,* type C. The sequence of ætiological events is now veterinary medical history. South African soils and pastures are, in general, poor in available phosphorus and cattle grazing on such pastures suffer from a phosphorus deficiency and show pica. In an attempt to satisfy the craving, they eat any foreign matter which has a salty or brackish taste. Carrion such as rotten bones, parts of putrefying carcases, and even dead, decomposing sand tortoises, are devoured with avidity. If, perchance, such material contains botulinum toxin and if a beast ingests sufficient of it, lamsiekte may result. Theiler and his colleagues succeeded in breaking this sequence of events by overcoming the pica. By supplying the cattle with enough available phosphorus they succeeded, not only in reducing the incidence of lamsiekte to a minimum, but in improving growth, production and reproduction to such a marked degree that, to-day, progressive cattle farmers feed phosphatic supplements primarily because of these effects. To the less progressive farmer, phosphatic supplements are a necessary evil to prevent a greater evil. Because of this attitude, any attempt to combat lamsiekte by artificial immunization with a botulinum antigen alone would be ill-advised. Doubtless the incidence of the disease would be effectively reduced, but a large percentage of the cattle would retrogress productively to the low level prevailing in those days when a phosphatic supplement was not added to the ration.

We know of only one example where artificial immunization is used to combat a botulinum intoxication in farm animals, *viz.* botulism (sarcophagia) in sheep in Western Australia. However, in this instance, the pica leading to the intoxication cannot be overcome by adding calcium or phosphorus salts to the ration (Dr. H. W. Bennetts in a private communication).

The Present Experiment.

As a result of Theiler's investigations, a large-scale breeding experiment was started in 1925 at the Lamsiekte Research Station, Armoeds-

vlakte, near the town of Vryburg in the north-western part of the Cape Province. One aspect of the investigation was to ascertain accurately the effects of phosphatic supplements on development, production and reproduction in cattle. For this purpose about 200 cows (approximately two-thirds of the breeding stock) were placed on a suitable phosphorus-containing diet and constituted the "bonemeal herd". The remaining one-third of the original breeding stock, numbering 98 cows, received no supplement of any kind and constituted the "control herd". Because of the high incidence of lamsiekte in these control cows, the study outlined above became very difficult; therefore we decided to try to immunize this herd with botulinum formol-toxoid to save, if possible, this experimental material. We are of the opinion that the results obtained up to the present are sufficiently significant to warrant publication, although open (in some measure) to criticism.

Preparation of the Formol-toxoids and Laboratory Tests.

The strains of Clostridium botulinum used and the method of preparing the toxoids were described by Mason and Robinson (1935). For the purpose of this communication, the following points may be mentioned again. The C strain (178C) was isolated, at Onderstepoort, from a decomposed rat and the D strain, at Armoedsvlakte, from a bovine dead of lamsiekte. The germs were cultivated in horse-flesh broth (plus meat particles and 2 per cent. horse serum) for 7 days at 37° C, freed from meat and bacteria by pulping, "toxoided" by adding enough commercial formalin to make a 0.5 per cent. concentration and finally incubated until non-toxic (*i.e.* 0.25 cc., injected subcutaneously, non-lethal for the mouse; 10 cc., similarly injected, non-symptom-producing for the goat; and 30 cc., harmless for the ox).

The Immunization of Control Cattle.

Because no precedent existed, we decided to adopt the following scheme for immunization.

(a) *Initial injections.*

D toxoid	1st day	—	10 cc. subcutaneously.
	3rd week	—	,, ,,
	5th week	—	,, ,,
C toxoid	8th week	—	,, ,,
	11th week	—	,, ,,

(b) *Subsequent injections.*

To maintain the level of immunity each control animal received, once a year, 10 cc. of each toxoid. After a few years this procedure was changed to 5 cc. of each toxoid, every half-year.

Immunity Tests.

Tables 1 and 2 summarize the results of titrating the sera of two groups of animals for antitoxin; the first group was immunized, at Onderstepoort, with D toxoid only; and the second group was immunized, at Armoedsvlakte, with both C and D toxoids.

Table 1.

Antitoxic content of sera of Onderstepoort cattle immunized with D toxoid.

Bovine.	Immunization.	Date of bleeding.	No. MLD "D" toxin neut. by 0.1 cc. serum.
61	27.10.34 30 cc. 13.11.34 10 cc.	19.11.34	$1\frac{1}{2}$
1322	27.10.34 10 cc. 13.11.34 20 cc.	19.11.34	> 5
1454	27.10.34 10 cc. 13.11.34 10 cc.	19.11.34	< 1
6024	None	19.11.34	< 1
4527	None	19.11.34	< 1

(Neut. = neutralized.)

Table 1 shows that two injections of D toxoid led to the formation of demonstrable amounts of antitoxin in two out of three animals.

Table 2 shows that two or three injections of D toxoid stimulated the production of D but not of C antitoxin; after two injections of C toxoid C antitoxin was demonstrable. A little more than a year later (no further injections having been given) both the C and the D titres had dropped considerably, but the excellent response to another injection of each toxoid showed that the basal immunity was good.

Field Results.

Because of the results obtained (Table 2) with 10 animals taken at random, we had reason to believe that the antitoxin content of the sera of the remaining immunized controls would be of the same order and hoped this level would be high enough to reduce the incidence of lamsiekte greatly.

The occurrence of the disease depends mainly upon three factors :—

a. The accessibility of cadaver material;
b. The toxin content of such material;
c. The intensity of pica in the cattle.

The amount of carrion in the pastures is kept as low as possible. Once a year the whole of the veld is searched systematically and material such as tortoises, bones and pieces of skin are destroyed. Nothing is known of the toxin content of animal debris or of the variation from season to season or from year to year. Pica in the control cattle runs at 100 per cent., except during those few summer months when there is an

Table 2.

Antitoxic content of sera of Armoedsvlakte cattle immunized with C and D toxoids.

Bovine.	Injections 10 cc. sc. "D" toxoid.	Test "D" toxin 22.12.34.	Test "C" toxin 22.12 34.	Injections 10 cc. sc. "C" toxoid.	Test "C" toxin 21.3.35.	No further injections. Test "D" toxin. 27.4.36.	Test "C" toxin. 27.4.36.	Injection 10 cc. sc. "C" toxoid.	Test "C" toxin 4.6.36.	Injection 10 cc. sc. "D" toxoid.	Test "D" toxin 17.10.36.
1899	10.10.34 24.11.34	> 3	< 1	19.2.35 11.3.35	1½	< 1	< 1	27.4.36	20–40	22.7.36	2 – 4
1981	as 1899	> 6	< 1	as 1899	1½	< 1	< 1	14.5.36	10–20	30.6.36	2 – 4
1988	27.10.34 28.11.34 12.12.34	3–6	< 1	as 1899	> 10	2–4	< 1	14.5.36	10–20	3.7.36	16–32
2005	20.10.34 28.11.34 12.12.34	1–2	< 1	as 1899	1½	< 1	< 1	27.4.36	Dead		
2080	as 2005	> 6	< 1	as 1899	1	1–2	< 1	14.5.36	10–20	30.6.36	2 – 4
2286	as 2005	> 6	< 1	as 1899	1	< 1	< 1	as 2080	20–40	3.7.36	1 – 2
3139	as 2005	1–2	< 1	as 1899	? 1	< 1	< 1	as 2080	10–20	3.7.36	< 1
3160	as 2005	3–6	< 1	as 1899	1½	< 1	< 1	as 2080	60–120	3.7.36	< 1
3178	27.10.34 28.11.34 12.12.34	< 1	< 1	as 1899	? 1	1–2	< 1	as 2080	40–80	22.7.36	8 –16
3242	as 3178	1–2	< 1	as 1899	< 1	< 1	< 1	as 2080	? 1	3.7.36	1 – 2

(The figures under "Test 'C' or 'D' toxin" represent the number of M.L.D. of toxin neutralized by 0.1 cc. of serum.)

Table 3.

Death Rate from Lamsiekte in "Control," "Bonemeal" and "Toxoid Control" Cattle.

YEAR.	CONTROL CATTLE.			"BONEMEAL" CATTLE.			"TOXOID-CONTROL" CATTLE.		
	No.	Lamsiekte deaths.	% LS. deaths.	No.	Lamsiekte deaths.	% LS. deaths.	No.	Lamsiekte deaths.	% LS. deaths.
1927	43	7	16.28	131	1	0.76			
1928	68	6	8.82	242	3	1.24			
1929	75	18	24.00	356	3	0.84			
1930	60	9	15.00	387	1	0.26			
1931	76	3	3.95	486	nil	nil			
1932	106	12	11.32	632	1	0.16			
1933	104	3	2.88	778	nil	nil			
	552	54	*Commencement of Immunization of Controls.*	5012	10	0.34			
1934	100	nil	nil	875	nil	nil			
1935	86	nil	nil	866	nil	nil			
1936	107	nil	nil	750	2	0.27			
1937	108	nil	nil	708	1	0.14	8	2	25.00
1938 (up to 1.4.38)	93	nil	nil	814	1	0.12	6	2	33.00
	1171	0	0 % DEATHS.	4012	11	0.11 % DEATHS.			

Mean Group 1 = 11.75 (controls from 1927–1933).
Mean Group 2 = 0.00 („ „ 1934–1937).

Difference = 11.75 Significant at P = 0.2, *i.e.* in 2 out of 100 trials a difference as large as this may arise by chance.

Mean Group 3 = 0.47 ("bonemeals" from 1927–1933).
Mean Group 4 = 0.10 („ „ 1934–1937).

Difference = 0.36 Not significant. (P = ± 0.2).

abundance of green grass, high in available phosphorus. Even under these conditions, pica seldom falls below 60 per cent. Thus, unless the toxicity of the carrion material varies considerably from season to season, the conditions bearing on the incidence of lamsiekte at Armoedsvlakte remain fairly constant except from December to the end of March.

Table 3, divided into two parts, gives the results of the field immunization. The upper half, covering the pre-immunization period from 1927 to the end of 1933, records the total number of bonemeal and control cattle, year by year, and the annual losses from lamsiekte. The lower half gives the same information for the period 1934 to April, 1938, *i.e.* from the commencement of immunization to date. In this section, the bonemeal group remains unchanged; the control group has been immunized with both the C and the D toxoids. An additional group of " toxoid control " cattle was sent down to Armoedsvlakte in 1937. They were not immunized and did not receive any phosphatic supplement. Since lamsiekte is very rare in cattle less than one year old, the figures given for the total number of " controls " or " bonemeals " for any one year apply to bovines over one year of age.

Table 3 shows that the incidence of lamsiekte in bonemeal-fed cattle is very low, with no significant variation during the 4 years before as compared with the 4 years after the commencement of the immunization of the controls. In the years 1927 to 1933, the lamsiekte mortality in the controls varied from 2.88 per cent. to 24.00 per cent. Subsequent to immunization, no control died of lamsiekte. Of the 8 susceptible cows placed on the Station to serve as controls to the immunized group, 2 died of lamsiekte in 1937 and in the first three months of this year (1938) 2 more succumbed.

Conclusions.

Judged by the antitoxin content of their sera, cattle may be satisfactorily immunized against Cl. botulinum, types C and D by 2 or 3 subcutaneous injections of the formol-toxoids of the toxins of these germs. The level of immunity may be maintained by one annual or two semi-annual injections of these products.

The incidence of lamsiekte, in phosphorus-deficient cattle suffering from pica, was reduced to nil by employing such a method of immunization.

References.

MASON, J. H., and E. M. ROBINSON (1935). The antigenic components of the toxins of Cl. botulinum types C and D. *Onderstepoort Jl.* **5** : 65-75.

THEILER, A., P. R. VILJOEN, H. H. GREEN, P. J. DU TOIT, H. MEIER, and E. M. ROBINSON (1927). Lamsiekte (parabotulism) in cattle in South Africa. *11th and 12th Reports Dir. Vet. Educ. & Res.* Union of S.A. (II) : 821-1361.

Acknowledgment.

We have pleasure in recording our thanks to Messrs. T. F. Adelaar, B.V.Sc. and P. A. Barnard for the labour spent in abstracting, from the experimental registers, the data given in Table 3, and to Dr. G. B. Laurence for the statistical analysis of Tabl 3.

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The Immunization of Guinea-Pigs against the Toxins of *Cl. Botulinum*, Types C and D.

By MAX STERNE and J. H. MASON, Onderstepoort.

Mason and Robinson (1935) showed that there is a slight overlapping of antigens in the toxins of Cl. botulinum, types C and D. The sera of goats hyper-immunized with C toxin neutralized a few M.L.D. of D toxin, and D " hyperimmune " serum (also made in goats) neutralized one or two lethal doses of C toxin. These tests were made by mixing the toxin and antitoxin *in vitro* and titrating for unneutralized toxin by injecting the mixtures into mice. In the experiments recorded in this communication, guinea-pigs were immunized by one or more subcutaneous injections of formol-toxoid or of a toxoid-antitoxin-mixture and their immunity to C and/or D toxin tested by injecting the one or the other toxin into them. The strains of Cl. botulinum (178 C and D) and the formol-toxoids used were those noted by Mason *et al.* (This Jnl. p. 65) The test toxins were dried ammonium sulphate precipitates, and their M.L.D. for guinea-pigs were accurately worked out prior to the tests proper.

Experiments.

1. *Immunization against C toxin with C toxoid-antitoxin-mixture.*

As the C toxoid was toxic for guinea-pigs in a dose of 2.0 cc. we added sufficient homologous antitoxin to this amount to make it harmless and used this toxoid-antitoxin-mixture for immunization. Table 1 records an immunity test using this mixture as antigen and shows that one injection produced a definite immunity.

Table 1.

Immunization against C toxin with C toxoid-AT.-mixture.

Guinea-pigs.	Antigen	Test 1 month later with C toxin	Result
1		0.0004 cc.	L
2		,,	L
3	2.0 cc.	,,	L
4	C Toxoid-	,,	L
5	AT.-mixture	,,	L
6		,,	L
7		,,	L
8		,,	5
9 –12		0.0004 cc.	2
13	Controls	,,	3
14–16		,,	4
17		,,	L

(L = lived. 2, 5 = died after 2, 5 days; 0.0004 cc. toxin = about 1 M.L.D.).

2. *Immunization against D toxin with D or C antigens.*

The D toxoid was quite atoxic even in a dose of 5.0 cc. One injection of a 2.0 cc. amount failed to immunize the guinea-pigs against 1 M.L.D. of the toxin, but 2 injections (1 month's interval and the test 10 days after the second injection) enabled the animals to withstand a fatal dose. After 6 injections of C toxoid and one injection of 1 M.L.D. of C toxin 5 of 7 guinea-pigs were immune to 1 M.L.D. of D toxin (Table 2).

Table 2.

Immunization against D toxin with D toxoid or C toxoid-AT.-mixture.

Guinea-pigs.	Antigen	Test with D toxin	Result
1 – 2	One injection D. toxoid	0.001 cc.	o/n
3 – 5		,,	1
6		,,	3
7 – 9	Two injections D toxoid	0.001 cc.	L
10 – 14	Six injections C toxoid-AT.-mixture and one injection of 1 M.L.D. of C toxin	0.001 cc.	L
15		,,	2
16		,,	3
17 – 19	Controls	0.001 cc.	o/n
20 – 22		,,	1
23		,,	2
24		,,	3
25		,,	L

(Abbreviations as for Table 1. 0.001 cc. = about 1 M.L.D.; o/n = overnight.)

3. *Immunization against C toxin with D toxoid.*

Table 3 shows that 4 injections of D toxoid (20 cc. in all, given within 7 weeks) did not immunize guinea-pigs against 1½ M.L.D. of C toxin. There was, however, some increased resistance because all the controls were dead before any of the treated animals had died.

Table 3.

Immunization against C toxin with D toxoid.

Guinea-pigs.	Antigen	Test with C toxin	Result
1	4 injections D toxoid	0.0006 cc.	3
2 – 3		,,	4
4		,,	5
6 – 9	Controls	0.0006 cc	1
5		,,	2

Another group of guinea-pigs were immunized with D toxoid followed by D toxin. In all, 7 injections of toxoid (30 cc.) and 3 injec-

tions of toxin (1, 2 and finally 5 M.L.D.) were given. The results are recorded in table 4.

Table 4.

Immunization against C toxin with D toxoid.

Guinea-pigs.	Antigen	Test with C toxin	Result
1 – 5	7 injections D toxoid	0.0005 cc	L
6	and	,,	3
7	3 injections D toxin	,,	7 K
8 – 12		0.0005 cc.	2
13 – 17		,,	3
18 – 19	Controls	,,	4
20		,,	5
21 – 22		,,	L

(K = alive on 7th day, but very ill and killed.)

The results given in table 4 show that most of the "D" immune guinea-pigs withstood 1 M.L.D. of the heterologous toxin.

The D toxoid was probably much poorer, antigenically, than the C toxoid. This would explain the difficulty in eliciting C immunity with the D antigen.

Conclusions.

1. It is relatively easy to immunize guinea-pigs against the toxin of Cl. botulinum type C with a type C formol-toxoid and against type D toxin with a type D formol-toxoid.

2. Several injections of type C or of type D toxoid are required to immunize against the heterologous toxin.

References.

MASON, J. H., and E. M. ROBINSON (1935). The antigenic components of the toxins of Cl. botulinum, types C and D. *Onderstepoort Jl.*, **5**: 65-75.

MASON, J. H., H. P. STEYN and J. H. R. BISSCHOP (1938). The immunization of bovines against lamsiekte. This Jnl., p. 65.

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Types of Goats in Italian Territory in Africa.

By F. M. BETTINI, Florence, Italy.

ERITREA.

According to Marchi there are three principal types of Native goats in Eritrea.

(1) A small goat found widely distributed throughout Central Africa, which is probably comparable with the goat of the Neolithic Period (*Torf Ziege* Rütimeyer). The coat is generally brown or red in colour and is much lighter over the back.

(2) The desert goat : a leggy animal, usually polled, but sometimes with vestiges of horns. The ears are long and hang down. This animal is a good milker.

(3) A goat called *Sciucrie* by the Natives. This is probably comparable with the Copper goat (*Capra hircus kelleri*) to which group belong the Angora, Cashemir and Tibet goats. This animal is big, long and roomy. It has very long perpendicular horns and very long ears. The latter are curled up at their ends. The profile is strongly convex and the coat brown. This goat is also a good milker.

ITALIAN SOMALILAND.

There are two types of native goat in this territory.

(1) One type is called *Deg uen* by the natives. It is tall, thick-skinned and has long lop ears curled up at their ends. This goat is probably identical with the *Sciucrie* of Eritrea and like that animal is rather a good milker.

(2) This type is called *Deg yer* by the natives and is smaller than the *Deg uen*. It has a thin skin, horizontal horns and is good for eating.

Both these goats are most commonly white in colour. Rarely they may be red or spotted, but are never black. The males have horns, while the females are usually polled.

In Somaliland one also finds goats introduced from Arabia and Bengala.

LIBYA.

(a) *Tripolitania.*—The goats in this territory are commonly called " Arabian ". The coat is usually black or brown, but sometimes the head or neck may be white or spotted with white. The ears are long and pendulous and the horns thin and turned backwards. According to Pucci and Cugnoni this goat is comparable with the Copper goat (*Capra hircus kelleri*). One also finds goats introduced from Malta in Tripolitania.

(b) *Cirenaica.*—The goats in Cirenaica are more or less the same type as those encountered in Tripolitania.

Is the Syringa Berry (*Melia azederach* L.) Poisonous for Cattle?

By G. D. SUTTON, B.V.Sc.

As far as I know, the berry of the syringa has not yet been listed among the plants or parts of plants poisonous for cattle, and as the disease described in this paper embraces only one observation and as no experimental work was carried out, I realize that a definite conclusion cannot be drawn. It is, however, possible that this communication may stimulate others to investigate the problem in a systematic manner.

History and symptoms.—The animals affected were adult Friesland cows, in fair condition, in receipt of an adequate supplementary ration of green oats and concentrates. They constituted part of a dairy herd in Chase Valley, near Pietermaritzburg and became ill in October, 1937, during a rainy period following a drought. The owner stated that a number stopped feeding and appeared to be ill, but that within 24 hours all except two recovered. It was these two bovines that I examined. One was lying normally on the sternum, temperature 102° F, conjunctivæ dark and congested, a slight frothy discharge from the nostrils and respiration normal. When forced to stand it held the forelegs somewhat wider apart than normally and the head at a lower level. After standing in this position for about a minute, the cow showed a general trembling of the body and fell heavily to the ground. At this stage the respirations were markedly increased both in frequency and depth, but soon returned to normal. The second cow, like the first, was lying in a normal position, temperature 104° F, conjunctivæ dark and congested, a marked frothy discharge from the nose and respirations normal. It could rise to the full extent of the hind legs, but not of the fore limbs; these buckled over either at the fetlock or the knee. After a few seconds the muscles of the scapular region trembled markedly and the animal sank to the ground. The effort of rising caused a transitory increase in depth and frequency of the respirations. Subsequently this cow died and a post-mortem examination was conducted.

Post-mortem Findings.—Red areas up to three inches in diameter occurred on the omentum. The heart showed ecchymoses. The lungs were markedly hyperæmic and œdematous with frothy material in the bronchi, trachea and larynx. The spleen was enlarged and dark in colour and the liver was somewhat congested. The abomasum and the small intestine were slightly inflamed. The rumen contained large numbers of mature syringa berries and berry stalks. The remains of the berries were also present in large quantities in the abomasal and intestinal contents. Stained smears of the spleen pulp revealed nothing of diagnostic value.

Conclusions.—It is suggested that the illness of these animals was caused by the syringa berries. There is, however, insufficient evidence to incriminate them with certainty, and only the results of further research can decide the question.

REVIEW.

A review of the third edition (1935) of Dukes' Physiology* was given in *Jl. S.A.V.M.A.*, Vol. VI, p. 311.

The present edition has been revised and re-written and the useful bibliography given at the end of each section much extended.

As pointed out previously, this book on veterinary physiology meets a much felt want, since there are very few books in English on this important aspect of animal biology.

The sections on nutrition and reproduction should be of special interest to the veterinarian.

As a text-book for veterinary students the book can be strongly recommended.

The omission of a special section on the locomotion of animals is, however, to be regretted.

J. Q.

* *The Physiology of Domestic Animals*, by H. H. Dukes, D.V.M., M.S. Fourth revised edition 1937, pp. xiv + 695. Comstock Publishing Company inc., Ithaca, New York.

OBITUARY.

GERALD AUGUSTUS HAROLD BEDFORD.

It is with the greatest regret that we record the death of Mr. G. A. H. BEDFORD at Pretoria on January 28, 1938, after a short illness.

Mr. Bedford, who was entomologist at Onderstepoort for 26 years, was born in Harley, England, on the 9th May, 1891. He received his primary education in Brighton, Sussex, and subsequently studied entomology at the South Eastern Agricultural College at Wye, Kent. Here he became associated with the late Professor F. V. Theobold under whom he worked until 1912 and with whom he acquired the foundation of his very extensive knowledge of the Culicinæ.

At about this time, Sir Arnold Theiler, then engaged on the problem of South African horsesickness, requested Professor Theobold to recommend an entomologist who could assist in the study of this and certain other insect-borne diseases of livestock from the entomological point of view, and Mr. Bedford was nominated.

He assumed duty at Onderstepoort in February, 1912, on a three-year contract and, at the expiration of this period, was appointed to the

permanent staff as research officer. During the earlier years of his services with the South African Government, his activities were necessarily confined to the problem which was largely responsible for his appointment, namely, the discovery of the vector of horsesickness. A great deal of effort was expended on this work and a survey conducted of practically the entire range of mosquitoes occurring in South Africa. No definite results, however, attended this investigation and the problem is still unsolved today.

Mr. Bedford was a born systematist and soon acquired material sufficient for the commencement of the long series of publications which

have contributed so materially to our knowledge of the parasitic insect fauna of South Africa and which may be looked upon as fundamental reference works for all entomological investigations in the veterinary field in this country. At one time or another he studied practically all the parasitic orders of arthropods in South Africa and published a series of papers on the various forms occurring upon the domestic animals and on the whole of our large vertebrate fauna. This work led finally to his specialization in the order Anopleura and it will be generally agreed that he has come to be regarded as one of the foremost authorities on this particular order.

The loss of Mr. Bedford in the field of entomological investigation will be greatly felt by his many colleagues of the Royal Entomological Society of London, and the South African Association for the Advancement of Science. His clear, painstaking annotations of the parasitic arthropods which play such an important part in South Africa will always be before us and will serve as an invaluable guide to those who follow after.

As a colleague, Mr. Bedford was always willing to assist all who applied to him for help and in spite of chronic ill-health, he showed a cheerful exterior to the world.

R. DU T.

BASIL YOUNG.

Mr. Young, who was known to all and sundry as "BRIGHAM" (so that quite a number of people were under the impression that this was his Christian name), was a Londoner by birth and he always claimed to be a true Cockney. Most of his early days, however, were spent in Sussex with a farmer uncle, and no doubt this early association with animals induced him to take up veterinary studies. He served a pupilage at Battle, Sussex, with the late Mr. Harry Jarvis, M.R.C.V.S., one of the old-time country practitioners, who instructed his pupils in everything from washing greasy bottles to parturition cases; so that Mr. Young in his early days had a good grounding in general practice which stood him in good stead in after years. On off-days he enjoyed a bit of fox hunting or a ride in a point to point, for Young was no mean horseman, and was one of the first to jump at the Rand Show. He was a good boxer and always ready to oblige anyone who wanted a bout. Eventually he entered the R.V.C. Camden Town, and from there at the time of the Anglo-Boer War, he came to this country with the Yeomanry. He served for a time and then returned to England. Within a few days, however, he joined another Yeomanry unit, and returned to South Africa and served till peace was proclaimed. He was for a time in Zululand with the Veterinary Department. Later he was on a stud farm and held a trainer's licence under Jockey Club rules. Afterwards he practised as a veterinary surgeon, first in Germiston and then in Johannesburg. He died suddenly from heart failure on April 17th, 1938, in Johannesburg. A widow, three grown-up daughters and a grown-up son are left to mourn him.

B. R.

THE ASSOCIATION.

Special Meeting of Council held at Onderstepoort on 1st March, 1938.

Present. C. J. van Heerden (Chairman), P. J. du Toit, M. Sterne, D. G. Steyn, H. O. Mönnig, A. D. Thomas, S. W. J. v. Rensburg (Secretary).

This being a special meeting the minutes of the previous meeting were not read.

Assistant Veterinarian: Basutoland. The chairman explained that this meeting had been called to decide what action should be taken with regard to the above post which was being advertised at £450–25–£600.

After full discussion Dr. Thomas proposed and it was agreed that the conditions pertaining to the post be circularized, but that members be informed that Council did not consider the scale adequate and could not therefore advise members to apply for the post. The Chairman and the Secretary were instructed to draw up the circular.

Colonial Veterinary Service. A letter was read from the Hon. Secretary, Nigerian Veterinary Association, submitting copies of letters addressed to the *Veterinary Record* and to the General Secretary N.V.M.A. *re* the present unsatisfactory nature of scales of salary and conditions of service in the Colonial Veterinary Service.

It was decided :—

I. that members of the S.A.V.M.A. be warned not to apply for posts in the Colonial Service without making full investigations into the conditions of service;

II. that the unsatisfactory conditions applying to the Basutoland post be communicated to the Nigerian Veterinary Association with the necessary warning.

S. W. J. v. Rensburg,
Hon. Sec.-Treas. S.A.V.M.A.

Council Meeting held in Johannesburg, 13th April, 1938.

Present. S. T. Amos (President), C. J. van Heerden, H. O. Mönnig, M. Sterne, J. Quin, G. Martinaglia, V. Cooper, G. v. d. Wath, S. W. v. Rensburg (Hon. Secretary). Letters of apology for absence from Drs. H. H. Curson and A. D. Thomas were read.

The attendance of Drs. Quin, Martinaglia and Mr. Cooper as substitutes for Dr. Steyn and Messrs. Kirkpatrick and Diesel was approved.

(1) **Minutes** of meetings held on 26th and 29th October, 1937, were taken as read, and were confirmed. Those of the special meeting held on 1st March, 1938, were read and confirmed.

(2) **Arising from these minutes :—**

(a) *Resignations.*—The Secretary reported that Mr. Hutchinson had withdrawn his resignation, and that no reply had yet been received from Mr. Forrest.

(b) *Reserve Fund.*—The Secretary reported that £382 8s. 0d. capital plus £27 17s. 8d. accrued interest had been withdrawn from Union Loan Certificate holdings. Of this sum £250 has been placed on fixed deposit with the United Building Society and the balance of £160 5s. 8d. transferred to the Benevolent Fund.

At the request of the Chairman the meeting was informed that the financial position of the Association at present was as follows :—

(i) £800 invested in 1,000 Union Loan Certificates;
(ii) £250 on fixed deposit with the United Building Society;
(iii) £330 in the Benevolent Fund with the Standard Building Society; £300 being on fixed deposit;
(iv) a credit balance in the Bank of about £87. This was approved by the meeting.

(c) *Admission of Animals to Onderstepoort.*—The Secretary stated that the resolutions taken by the meeting on 29th October, 1937, had been carried out, and he understood that the Faculty was still considering the matter. Dr. Quin said that Faculty was giving very careful attention to this question and he was certain that it would be solved in a manner satisfactory to all concerned. This was confirmed by Dr. Mönnig. The Chairman quoted cases in which attempts were made to send animals to Onderstepoort apparently only because the cost of maintaining them at Onderstepoort was less than that at which a horse could be kept in a private stable, and he indicated the necessity for exercising great caution in accepting animals from areas in which veterinary services were available.

Mr. Cooper stated that certain drugs which were supposed to be supplied only to members of the veterinary profession were sold to the public.

The Chairman stated that since his remarks at the General Meeting there had been a distinct improvement in this respect in Durban. He said that the Secretary of the Pharmaceutical Society of South Africa had written to the effect that that Society would be pleased to assist our Association in suppressing such sales if individual cases were brought to its notice.

It was decided that members be circularized and asked to report to the local Secretary of the Pharmaceutical Society or to the Secretary of

the S.A.V.M.A. specific cases in which drugs were sold indiscriminately.

(2) **Book Fund Prize.** A letter was read from Dr. Thomas giving his views on this matter. The Chairman pointed out that the administration of a trust fund like this must be placed on a definite footing and that our legal advisers must be consulted.

It was decided that certain points be referred to the General Purposes Committee for consideration and that this committee should have power to act as far as the constitutional side was concerned. Further it was decided that the scheme evolved be subsequently circularized to members of Council, and that the latter be asked to make nominations.

(4) **Alteration of Constitution.** It was pointed out that in the revision of the Constitution at the last general meeting the duration of the terms of office of the President, Vice-President and Hon. Secretary-Treasurer was not defined. It was decided that the necessary proposal be introduced at the forthcoming general meeting, after the legal advisers had been consulted.

Dr. Martinaglia asked permission to leave the meeting.

It was decided by drawing lots that the following four members of Council would retire at the end of the present year : D. G. Steyn, A. M. Diesel, A. D. Thomas, A. C. Kirkpatrick.

(5) **New Members.** The following were proposed and it was unanimously decided to recommend their acceptance to the next general meeting : H. A. Crawshaw, J. L. Doré, E. B. Kluge, J. Wakefield Rainey, L. J. F. von Maltitz.

(6) **Annual General Meeting.** It was decided that the meeting be held in about the fourth week of October and that the General Purposes Committee arrange the date, programme, etc., in consultation with the officers concerned.

(7) **General.**

(a) *Expert Evidence.*—The Secretary said a member had reported that a Public Prosecutor had stated that the evidence of an inspector is sufficient in a cruelty case, and that it was not necessary for a veterinarian to be called. Mr. van Heerden pointed out that the law on this point was not the same in South Africa as in England. In the latter country a veterinarian " must " be called by the Crown, while in South Africa he " may " be called.

(b) *International Veterinary Conference.*—It was decided that Drs. Du Toit and Quin represent the S.A.V.M.A. at Zürich in August and that they should convey our greetings to the International Veterinary Conference.

S. W. J. v. Rensburg,
Hon. Sec.-Treas. S.A.V.M.A.

Extract from Minutes of the Annual General Meeting of the Natal Branch of the South African Veterinary Medical Association, held at Allerton Veterinary Research Laboratory, Pietermaritzburg, on 9th June, 1937.

Present. Messrs. A. M. Diesel, W. A. Dykins, S. van Rensburg, C. J. van Heerden, J. Nicol, R. Paine, C. M. Sharpe, Col. Harber, Dr. P. R. B. Smith, W. E. Footner, S. H. Ewing, W. G. Barnard, N. C. Starke, N. Boardman, J. Zwarenstein, F. B. Wright, L. C. Blomefield, R. W. Rossiter, H. G. Franz, C. J. Schoeman, A. F. Tarr, W. J. B. de Villiers, M. de Lange, J. L. Mainprize, G. D. Sutton, D. A. Haig, S. T. Jackson.

The president, Mr. Diesel, welcomed the members and asked them to signify in the usual way their sympathy with Mr. and Miss Amos in their recent sad loss.

The secretary then read his report, after which it was unanimously agreed to take the minutes of the last Annual General Meeting as read.

After reading the financial statement, the secretary said that the position, unfortunately, was not as satisfactory as it appeared because members who had been in arrears with their subscriptions had preferred to resign rather than to bring these up to date. Mr. Dykins proposed and Mr. van Heerden seconded the acceptance of the financial statement.

The following new members were proposed, seconded and welcomed : D. A. Haig, G. D. Sutton, A. F. Tarr, M. de Lange, J. L. Mainprize, W. J. B. de Villiers.

The following office bearers were elected :—

President : A. M. Diesel.

Vice-President : R. Paine.

Secretary : L. C. Blomefield.

Committee : N. C. Starke, C. M. Sharpe, W. A. Dykins, and J. Zwarenstein.

General.

The meeting congratulated Mr. L. R. Morford on his engagement to another veterinarian, Miss Amos.

Mr. Jackson proposed that a general collection of all outstanding subsciptions should take place at the meeting, as there were always a number of expenses to be met. Mr. Blomefield seconded and the motion was unanimously carried.

Mr. van Heerden proposed that if a member be in arrears for more than two years, the secretary should address a registered letter to him drawing his attention to this fact and that if no response were then received, the secretary should report this at the next meeting. Mr. Dykins seconded the motion which was carried.

The president then delivered his address after which the following papers were presented and fully discussed.

(1) " Streptococcic mastitis with special reference to diagnosis and control " by S. W. J. van Rensburg.
(2) " Curative treatment of mastitis " by G. D. Sutton.
(3) " Cows and cowsheds from a public health point of view " by Col. A. F. Harber.
(4) " The practical application of the double intradermal tuberculin test in cattle " by S. T. Jackson.

COLONIAL VETERINARY SERVICES.

The attention of members is drawn to the minutes of Council Meeting held on 1st March, 1938, published in this issue. Members are advised not to apply for posts in the Colonial Service without first making the fullest investigation and without first communicating with the Secretary, S.A.V.M.A.

Note added in proof.

Since going to press we have been informed by the Secretary of the S.A.V.M.A. that the post of Assistant Veterinarian, Basutoland, will be at the scale £500—£700 plus allowances and free pension. In the circumstances, the objections previously raised fall away.

CORRIGENDUM.

The Immunization of Bovines against Lamsiekte.

By J. H. MASON, H. P. STEYN and J. H. R. BISSCHOP, Onderstepoort.

Vol. IX — No. 2. Page 69, Table 3 :

" Significant at P=0.2," should read " 0.02."

Jl. S.A.V.M.A.
IX (3) : 85–124,
1938.

Tick-Borne Diseases.

By W. O. NEITZ and P. J. DU TOIT, Onderstepoort.

The relationship between ticks and redwater in cattle was suspected by stock-owners in America as early as 1869, but it was only in 1893 that Smith and Kilbourne demonstrated that *Boophilus annulatus* was the vector of this disease. Since then many more dramatic discoveries have been made in connection with the transmission of diseases by blood-sucking arthropods, especially mosquitoes, lice, fleas and flies.

Ticks play a far greater rôle in the transmission of animal than of human diseases. In recent years it has been realized more and more that the reservoirs of human tick-borne diseases, *viz.* spirochætosis, rickettsiosis, and tularæmia, are rodents and carnivores. The infection of man by ticks must, in the majority of cases, be regarded as accidental. However, several species of the genus *Ornithodorus* not only feed on rodents and other mammals, but in certain localities live in human habitations and thus have become completely dependent on man for a host.

Although tick-borne diseases are more prevalent in the tropics, the temperate zones also have their share of them. Arthropod vectors have adapted themselves to different climatic conditions. Some species, namely *Rhipicephalus sanguineus, Boophilus australis,* and *Argas persicus,* occur practically all over the world, whereas *Amblyomma hebræum* is restricted to the warm, moist, bushy country of South Africa. A knowledge of the distribution of ticks often makes it possible to predict what diseases may be expected to occur in certain localities.

The Ixodidæ were the first known vectors of disease, but it is only in recent years that complete studies have been made of the development of the parasites in these intermediary hosts. Far greater attention has been paid to the life cycle of the Plasmodidæ in mosquitoes, and of Trypanosomata in tsetse-flies, fleas, and keds. The reasons for this apparent neglect are (1) difficulties in the technique of sectioning, (2) the occurrence of symbionts which complicate the interpretation, and (3) the fact that in some cases it is necessary to examine a number of successive stages, *viz.* the eggs, larvæ, nymphæ, and even the adults, before the complete development of a piroplasm can be established. In the case of mosquitoes and tsetse-flies the cycle is completed in the same stage as that in which the vector becomes infected.

The object of this paper is to enumerate briefly the various ticks

concerned in the transmission of diseases. For this purpose several tables based on the method indicated by du Toit (1918) are appended. It is hoped that these lists will be of value to the veterinarian. If the tables are compared with the records of Knuth and du Toit (1921), it will be noticed that a large number of new vectors, as well as new diseases have been added. For the sake of completeness, vectors other than ticks concerned in transmission are mentioned in the text. In addition a table giving the incubation period following natural and artificial infection is appended.

The vectors to be discussed fall under two families, the Argasidæ and the Ixodidæ. Not only do these families differ morphologically, but their biological characters are distinct. The Ixodidæ moult twice before reaching maturity and the females die after laying a large number of eggs. The Argasidæ on the other hand moult several times (even in the adult stage), engorge frequently, and lay several batches of eggs.

The Argasidæ transmit spirochætosis and ægyptionellosis. Recently it has been claimed (Rastigaieff, 1936) that this family is also capable of transmitting anaplasmosis and theileriosis in sheep. The Ixodidæ are responsible for the transmission of piroplasmosis, babesiosis, theileriasis, rickettsiosis, spirochætosis, tularæmia, some virus diseases, and *Hepatozoon* infection in dogs.

Spirochætosis.

1. *Relapsing fever.*

In central and eastern Europe relapsing fever caused by *Spirochæta obermeyeri s. recurrentis* is transmitted by the human louse *Pediculus humanus*. The infection does not result from the bite, but from scratching into the wound the spirochætes contained in the fæces.

In Spain, Africa, America, Asia and Australia, relapsing fever is transmitted by the genus *Ornithodorus*. Four different species of spirochætes transmitted by ticks are recognized today, *viz. Sp. duttoni, Sp. hispanica, Sp. venezuelensis* (*neotropicalis*), and *Sp. persica*. Other species have been described, but many authors believe that they are merely varieties of one or other of the abovementioned species.

Ross, Milne, Dutton and Todd were the first to show that the genus *Ornithodorus* transmits spirochætosis to man. It is realized today that the reservoirs of relapsing fever are wild rodents. Manson-Bahr (1935) mentions that *Sp. crociduræ* of the shrew mouse is identical with *Sp. duttoni*. Baltazard (1937) isolated a strain of spirochæte, from wild *Ornithodorus erraticus* caught in Morocco, which he believes to be *Sp. duttoni* var. *crociduræ*. In Panama, Clark and Durm have found a wild squirrel monkey *Leontocebus geoffroyi* naturally infected with *Sp. venezuelensis*.

Koch found that spirochætes can pass through the egg to the next and subsequent generations. Infected ticks are able to transmit relapsing fever for a period of 18 months. The microorganisms multiply chiefly in the Malpighian tubules of the tick. They pass out of the tick in its fæces and the scratching of tick bites makes it easy for the spirochætes to enter the skin of the host.

Sergent (1933) observed *Rhipicephalus sanguineus* naturally infected with *Sp. hispanica,* and Sergent and Levy (1935) record a case where a human being became infected with *Sp. hispanica* as a result of a bite by a male *Rh. sanguineus.* Brumpt (1936) was able to transmit *Sp. hispanica* by means of *Rh. sanguineus* to guinea pigs. It is not yet clear whether this tick plays an important rôle in transmitting relapsing fever.

2. *Spirochætosis in birds.*

Spirochæta anserina is found practically all over the world. Under natural conditions the genus *Argas* is the vector. The infection is capable of passing through the egg to the next and subsequent generations. Infection results from the bite, the ingestion of infected eggs or tampans, or by rubbing infected fæces into a wound.

In Austria Gerlach (1925) showed that the fowl mite *Dermanyssus avium* is the vector. Rastegaieff (1936) working in Russia failed to transmit *Sp. anserina* with *Dermanyssus gallinæ.*

3. *Spirochætosis* (Spirochæta theileri).

Successful transmissions of *Sp. theileri* were obtained by Theiler (1905) in South Africa with *Boophilus decoloratus,* in North Africa by Crawley (1915) with *B. annulatus* and by Brumpt (1919) with *B. australis.* In all these ticks the infection passes through the egg and is given off in the next generation. The genus *Boophilus* is a one-host tick and it is not quite clear whether the larvæ, nymphæ or adults are the transmitters.

Theiler (1909) proved that *Rhipicephalus evertsi* is a vector of *Sp. theileri.* The infection is acquired by the larval-nymphal stage and given off by the adults. The spirochætes then pass through the egg and the parasites are given off by the larvæ.

PIROPLASMOSIS.

1. *Redwater in cattle* (Piroplasma bigeminum infection).

Piroplasma bigeminum was the first protozoon parasite shown to be transmitted by an arthropod. Today five species of the genus *Boophilus,* three species of *Rhipicephalus,* and one species of *Hæmaphysalis* are known to be vectors of this parasite. In the case of the one-host tick *Boophilus,* the infection passes through the egg and is given off by the larvæ in the next generation. Theiler was able to show that *B. decoloratus*

retained its infection to the second generation. Successful transmission with *B. microplus,* which occurs in the Argentine, was obtained by Regendanz (1936).

According to Theiler the larvæ bred from infected adults of *R. appendiculatus* are capable of transmitting the disease. Infected nymphæ transmit the disease in the adult stage. In the case of *R. evertsi* stage-to-stage transmission as well as passage through the egg to the larva takes place.

Sergent (1931) found that *R. bursa* is also a vector of *P. bigeminum.* Both stage-to-stage transmission and transmission through the egg to the larvæ, nymphæ, and adults take place. Knuth (1915) found that infected nymphæ of *H. cinnabarina* transmit the infection in the following stage. Zeller and Helm (1923) demonstrated that the infection in adults which had fed on a reacting animal passed through the egg stage and was given off by the adults.

The first records of the life cycle of *P. bigeminum* in the tick are those of Koch (1903) and Christophers (1907). Dennis (1932) studied the development in *B. annulatus,* but according to Reichenow (1935) several stages described are not those of *P. bigeminum,* but those of symbionts. Regendanz (1936) carried out his observations in *B. microplus.* The process of development very closely resembles that of *P. canis* in *D. reticulatus.* He failed, however, to find any parasites in the salivary glands. According to him, the description by Dennis of the piroplasms in the salivary glands is not convincing.

2. *Babesiosis of cattle* (Babesia bovis infection).

Kossel, Weber and Miessner (1903) transmitted *B. bovis* with *Ixodes ricinus.* They demonstrated both the stage-to-stage transmission and the passage of the parasite through the egg to the larval stage. In England, *B. bovis* (*divergens*) is probably transmitted in the same way.

Gouseff, Rastegaieff and Soussko (1936) found that *Ixodes persulcatus* which has become infected in the adult stage is capable of transmitting the infection as larva, nymph and adult in the next generation. It is of interest to note that in their experiments they found that a single adult male was able to infect an ox. These authors differentiate between *B. bovis* and *Francælla occidentalis,* and show that the latter parasite is transmitted by *I. persulcatus* in the same way as *B. bovis.*

3. *Piroplasmosis of sheep* (Piroplasma ovis infection).

Motas (1904) states that the adults of *Rhipicephalus bursa* that became infected as adults in the previous generation are capable of transmitting *P. ovis.* Rastegaieff (1933) transmitted this disease with the adults of *Rh. bursa* which had become infected in the larval-nymphal stage.

4. *Babesiosis of sheep* (Babesia ovis infection).

Rastegaieff (1933) transmitted *B. ovis* with the adults of *Rhipicephalus bursa* which had become infected in the larval-nymphal stage.

5. *Piroplasmosis* (P. caballi infection of the horse).

Marzinowsky and Bielitzer (1909) showed that the adults of *Dermacentor reticulatus* which had fed on a reacting horse transmitted the disease as adults in the next generation. Du Toit (1919) demonstrated that nymphæ infected as larvæ transmit the infection, and that the adults resulting from these nymphæ were also infective. Markow (1935), working in Russia, infected the nymphæ of *Dermacentor silvarum* and transmitted *P. caballi* with the adults. Du Toit (1919) was unable to get transmission with *Ixodes ricinus*.

6. *Nuttalliosis* (Nuttallia equi infection in horses).

Theiler (1906) transmitted *N. equi* in South Africa with the adults of *Rh. evertsi* which had become infected in the larval-nymphal stage. Knuth, Behn, and Schulze (1918) found that in Rumania *Rh. bursa* is the vector. The infection is picked up by the larvæ and given off in the next stage. Carpano (1913) obtained transmission with *Rh. bursa* in Italy, and Pricolo in Macedonia with *Rh. sanguineus*.

According to Springholtz-Schmidt (1937) there are eight species of the genera *Rhipicephalus, Dermacentor* and *Hyalomma* which are vectors of *N. equi*. No mention, however, is made of which stage of the tick transmits the disease. Nikolsky (1933) showed experimentally that *Hyalomma mauretanicum* is a vector in North Caucasia. The experiments of Markoff and Abramoff (1934) prove that *Dermacentor silvarum* is a transmitter in Russia. According to Koneff and Degtereff (1935) the ticks responsible in Khakasia are *D. silvarum, D. niveus* and *D. nuttalli*. In central Asia Agrinsky (1935) found *Hyalomma marginatum* and *Rh. sanguineus* to be transmitters of *N. equi*. Potschetschneff (1937) comes to the conclusion that in Kiew (U.S.S.R.) *D. silvarum* is the vector. Du Toit (1919) failed to transmit *N. equi* with *Ixodes ricinus*.

7. *Biliary fever in dogs* (Piroplasma canis infection in dogs).

Lounsbury (1901), working in South Africa, found that the adults of *Hæmaphysalis leachi* which had engorged on a reacting dog transmitted *P. canis* as adults in the next generation. Christophers (1907) transmitted biliary fever with the nymphæ and adults of *Rhipicephalus sanguineus* bred from infected adults. His observations were confirmed by Nuttall in England, and by Brumpt (1920). Reichenow (1935) obtained hereditary transmission (transmission through the egg) with larvæ, in a dog infested with several thousand seed ticks. Shortt (1936) in India showed that in addition to the hereditary transmission, stage-to-stage

transmission (*i.e.* from larvæ to nymphæ, and from nymphæ to adults) is possible.

Nocard and Motas (1902) suspected *Dermacentor reticulatus* of being a vector, but the adult to adult transmission was first demonstrated experimentally by Brumpt (1931). Regendanz and Reichenow (1932 and 1933) observed that the nymphæ bred from infected adults can be infectious. Brumpt and Larrouse (1922) obtained transmission with the adults, but not with the larvæ and nymphæ, of *Dermacentor venustus* bred from infected adults.

The first attempts to study the life cycle of *P. canis* in the tick *Rh. sanguineus* were made by Christophers (1907). Regendanz and Reichenow (1932) described the developmental stages of this parasite in *D. reticulatus.* The parasites were observed in the gut, the epithelial cells, the ovaries, and finally in the salivary glands of partially engorged adult females and males. Regendanz and Muniz (1936) found numerous piroplasms in the salivary glands of the nymphæ of *Rh. sanguineus.* Shortt (1936) examined the life cycle in infected nymphæ of *Rh. sanguineus.* The parasites enter the phagocytic cells of the epidermal layer of the gut. From here they migrate into the muscles and muscle-sheaths, where division takes place. No further changes occur until the adult tick commences to feed, when the parasites migrate to the salivary glands where intense multiplication takes place, giving rise to the infective sporozoites.

Brumpt (1937) failed to demonstrate any stages of *P. canis* in infected *Hæmaphysalis leachi* and *Rh. sanguineus,* but in *D. reticulatus* the parasites could be traced in the cells of the digestive tract, muscles, ovaries, ovules, and finally in the salivary glands. Brumpt also records an interesting observation in connection with the behaviour of *P. canis* in the tick *Rh. sanguineus.* Infected ticks which had been reared for five generations on hedgehogs were still capable of transmitting biliary fever to a dog. He suggests that *P. canis* is a parasite of ticks, transmitted hereditarily in them, which only accidentally gives rise to developmental forms which are attracted to the salivary glands and are capable of infecting dogs.

8. *Babesiosis in dogs.*

Babesia gibsoni infection of foxhounds and jackals has been recorded from India and China. The attempts by Patton (1910) and Ras (1926) to transmit this parasite with *Hæmaphysalis bispinosa* were unsuccessful. Swaminath and Shortt (1937) successfully transmitted *B. gibsoni* with *H. bispinosa.* Adults which were allowed to feed on infected dogs were capable of transmitting the infection as larvæ or nymphæ. Infection also resulted with adults that became infected as nymphæ. Ras (1926) failed to transmit *B. gibsoni* with *Rh. sanguineus,* but Sen (1933) reports successful transmission with the nymphæ of this tick which had fed on

infected dogs as larvæ. The adults derived from the same source did not produce the infection.

9. *Ægyptionellosis.*

Ægyptionella pullorum has been recorded from Egypt, Palestine, South Africa, Belgian Congo, and Yugoslavia, chiefly from fowls, but also from ducks and geese. Before *Æg. pullorum* was recognized as a distinct parasite, Galli-Valerio (1909) succeeded in transmitting "Balfour's intra-corpuscular bodies" by the bite of *Argas persicus*. Balfour (1911) infected fowls by injecting emulsions of *A. persicus*. Bedford and Coles (1933) successfully transmitted *Æg. pullorum* to healthy chickens with adult *A. persicus* which became infected as adults in South Africa. These ticks remained infective for a period of up to 162 days. Their attempts to transmit this disease with *Ornithodorus moubata* and *O. parengueyi* were negative.

Theileriosis.

1. Theileria annulata *infection in cattle.*

Galuzo (1934) showed experimentally and by field observations that *Th. annulata* is transmitted by *Hyalomma dromedarii asiaticum* in central Asia. Further observations by Galuzo (1935) and Galuzo and Bespalov (1935) in Kazakstan and Tadjiskistan in Russia showed that *H. detritum rubrum* is a vector. In both species the infection is acquired in the larval-nymphal stage and given off by the adults.

2. Theileria dispar *infection in cattle.*

Sergent, Donatien, Parrot and Lestoquard (1928) found that the adults of *Hyalomma detritum mauretanicum* are the transmitters of the North African theileriosis. Hereditary transmission does not occur. The same workers (1936) noted that larvæ could become infected and that, if moulting were delayed, the parasites might develop so that the nymphæ became infective.

Frotheringham and Lewis (1936) successfully transmitted *Th. dispar* with the adults of *H. impressum near planum* in East Africa. Negative results, however, were obtained with *Rh. appendiculatus*. Sergent, Donatien, Parrot and Lestoquard (1936) studied the life cycle of *Th. dispar* in the tick *H. mauretanicum*. At the end of an acute attack gametocytes appear which, when ingested by the tick, undergo binary division, but no sexual conjugation was noticed. The resulting zygotes are found in the epithelial cells of the intestine of the nymph. Here they encyst and remain so in the intestine during the hibernating period of 6–8 months. After the moult the zygotes lose their cysts and migrate to the acinous salivary glands. After attachment to a host, sporonts appear in the salivary glands. These sporonts give rise to sporoblasts which in their turn

produce sporozoites which find their way into the salivary ducts. In the experiments it was shown that ticks do not become infective until 60 hours after attachment and that, generally, transmission takes place from the third day.

3. Theileria parva *infection in cattle.*

East Coast fever occurs in east, central and South Africa. Towards the end of 1902 Lounsbury (1903) first transmitted *Th. parva* to susceptible cattle by means of *Rh. appendiculatus.* Subsequently Lounsbury (1906), and Theiler (1905–7) proved, in many experiments, that besides this vector, *Rh. simus, Rh. capensis,* and *Rh. evertsi* were capable of transmitting this disease. Their experiments with *Hyalomma ægyptium,* however, gave negative results. Frotheringham and Lewis (1936), working in Kenya, repeatedly showed that *Hyalomma impressum near planum,* a species of tick closely allied to *Hyalomma ægyptium,* is capable of transmitting east coast fever as nymphæ infected in the larval stage, and as adults infected in the nymphal stage.

Th. parva is never transmitted through the egg. The larvæ therefore are never infectious. In the three-host ticks the infection is picked up by the larvæ or nymphæ and given off by the nymphæ and adults respectively. In the two-host ticks only the adults are infective under natural conditions. An important point is that the infected ticks loose their infection whether they feed on a susceptible or an insusceptible animal. The infection is lost 72 hours after the attachment of the tick.

Mettam (1934) and Mettam and Carmichael (1936) described a disease in cattle which they term " turning sickness ". They believe this disease is a form of east coast fever. Ticks fed on cattle suffering from " turning sickness " were able to transmit east coast fever to healthy calves.

Theiler and Du Toit (1928) showed that *Th. parva* can be transmitted by intrajugular injection of emulsified infected adults and nymphæ of *Rh. appendiculatus.* The transmission succeeded only with adults which had recently moulted and which had been feeding for 72–100 hours. The transmission with nymphæ did not succeed with every batch of ticks, nor with all ticks belonging to one and the same batch.

The life cycle of *Th. parva* in the tick *Rh. appendiculatus* has been studied by Cowdry and Ham (1932) and by Reichenow (1937). The latter author does not agree with the observations of Cowdry, who based his work on the life cycle of the *Plasmodia* in mosquitoes. Reichenow failed to find the male and female elements of the parasite in the gut, and has further pointed out that certain structures in the salivary glands described by Cowdry as developmental stages of *Th. parva* are in reality symbionts.

Failure to transmit east coast fever was reported by Lounsbury (1906) and Theiler (1905–7) with *A. hebræum* and *Boophilus decoloratus*, by Montgomery (1915) with *Rh. pulchellus*, and by Frotheringham and Lewis (1937) with *Rh. sanguineus*, *Rh. pulchellus*, and *A. variegatum*.

4. Theileria mutans *infection in cattle*.

According to Theiler (1907) *Th. mutans* is transmitted by *Rh. appendiculatus* and *Rh. evertsi*. In both vectors successful transmission was obtained with adults which had fed on a reacting animal in the previous stage. Reichenow (1935) states that Miessner obtained *Boophilus annulatus* from the United States of America and succeeded in transmitting *Th. mutans* with them to a susceptible animal in Germany. In a report by the New South Wales Department of Agriculture it is stated that theileriosis in a benign form has a distribution which coincides with that of the common scrub tick, *Hæmaphysalis bispinosa*, which in all probability is the vector responsible.

5. *Theileriosis and Anaplasmosis of sheep and goats*.

Rastegaieff (1933) found that *Theileria ovis* (*recondita*) and *Anaplasma ovis* are transmitted by the adults of *Rhipicephalus bursa*. Rastegaieff (1935, 1936) mentions that she successfully transmitted *Th. ovis* and *Anaplasma ovis* to goats and sheep in Russia with the nymphæ and adults of *Ornithodorus lahorensis*.

If these observations are correct, then this is the first record that a species of *Theileria* and *Anaplasma* are transmitted by ticks not belonging to the family Ixodidæ. Rastegaieff (1937) infested a goat with the adults of *Dermacentor silvarum* bred from females collected in the Ukraine from sheep infected with *A. ovis* and *Th. ovis*. After four to five days both species of parasites appeared in the peripheral blood. It is very doubtful in this case whether transmission resulted from the ticks since the incubation period mentioned (4–5 days) is extremely short.

ANAPLASMOSIS.

1. *Anaplasmosis of cattle* (Anaplasma marginale *infection*).

Probably the first successful transmission of this disease was obtained by Smith and Kilbourne (1893) with *Boophilus annulatus*. Anaplasmosis as a disease distinct from piroplasmosis was first recognized by Theiler (1910), who demonstrated the hereditary transmission by *B. decoloratus*. The following year he found *Rhipicephalus simus* to be a vector. According to Brumpt (1922) *B. australis* transmits the disease in Brazil. In Argentina, Rosenbusch and Gonzales (1927) transmitted anaplasmosis with *B. microplus*. In Germany, Helm (1924) showed experimentally that *Ixodes ricinus* and *Hæmaphysalis cinnabarina punctata* are capable of transmitting the disease.

Sergent, Donatien, Parrot and Lestoquard (1928) found that in Algiers *Hyalomma lusitanicum* and *Rh. bursa* are vectors. Transmission by the latter tick was confirmed by Brumpt (1931). In the United States of America Rees (1930, 1932 and 1934) was able to infect cattle by allowing *Rh. sanguineus, Dermacentor andersoni, D. variabilis,* and *Ixodes scapularis* to engorge on susceptible cattle. Boynton Herms, Howell and Woods (1936) have shown that *D. occidentalis* and *D. albipictus* are vectors.

It will be seen from table I that there are no less than 16 species of ticks capable of transmitting anaplasmosis. The transmission through the egg has been demonstrated in nine species. The authors of this paper twice failed to transmit *A. centrale* with *B. decoloratus* which had been allowed to feed on actively infected cattle.

Studies of the life cycle of *A. marginale* in *Boophilus microplus* were reported on by Regendanz (1933). In the salivary glands of infected ticks he was able to see granules varying from 0.1 to 0.4 microns in size. The granules were distinct from the symbionts usually found in ticks, and absent in ticks which had not fed on a reacting animal. Cowdry and Rees (1935) examined the salivary glands of infected *D. variabilis* and *D. andersoni,* but could not find any granules such as those described by Regendanz.

2. *Anaplasmosis in sheep.*

See Theileriosis in sheep.

3. *Eperythrozoonosis in cattle.*

Donatien and Lestoquard (1937) obtained adult ticks of an undetermined species of *Hyalomma* from Iran. These ticks were allowed to feed on an ox which subsequently showed *Eperythrozoon wenyoni, Rickettsia bovis* and *Theileria dispar* in the peripheral blood. Neitz and Quinlan (1934) on the other hand found that two calves infected with lice, *Linognathus vituli,* but which had been reared under tick-free conditions developed a relapse to *Ep. wenyoni* after splenectomy. From the observations of Donatien and Lestoquard, therefore, it is not clear whether they were able to transmit Eperythrozoon or whether a relapse resulted from the associated parasites.

Rickettsiosis.

1. *Heartwater in ruminants.*

Heartwater (*Rickettsia ruminantium*) occurs in east, central and South Africa, and Madagascar. Lounsbury (1899) was the first to demonstrate the transmission by *Amblyomma hebræum.* The infection is picked up either by the larvæ or nymphæ and transmitted by the subsequent stages. The infection, however, does not pass through the egg as

is the case with rocky mountain spotted fever. Infected nymphæ do not loose their infection if they feed on a susceptible, immune, or insusceptible animal. This observation is very important because infected nymphæ can be spread over large areas by various species of animals. *A. hebræum* is still capable of picking up heartwater fourteen days after the recovery of the animal, but not after 35 days [Alexander (1931)].

Theiler and Du Toit (1928) showed that *R. ruminantium* can be transmitted by intrajugular injection of emulsified infected nymphæ of *A. hebræum*. The experiments with fed nymphæ were positive in eight out of nine cases whereas similar experiments with unfed nymphæ succeeded once only.

Cowdry (1926) found that *R. ruminantium* in the tick is strictly limited to the epithelial cells of the intestine and to the lumen of the gut. Infection probably takes place by regurgitation of the parasites from the gut into the wound caused by the tick.

Daubney (1930) successfully transmitted heartwater with adult *A. variegatum* collected in the veld in East Africa. Unsuccessful attempts at transmission were made by Lounsbury (1901) with *Rh. evertsi, B. decoloratus* and *Ornithodorus savignyi*. Du Toit (1928), Alexander (1929), and Neitz (1937) failed to transmit heartwater with *Amblyomma marmoreum* commonly found on tortoises in South Africa. Negative results were also obtained by Alexander (1929) with *Amblyomma nuttalli*.

2. Rickettsia bovis *infection in cattle.*

Donatien and Lestoquard (1936, 1937) described *Rickettsia bovis* in the blood of cattle bitten by adults of an undetermined species of tick of the genus *Hyalomma,* received from Iran.

3. Rickettsia canis *infection in dogs.*

Donatien and Lestoquard (1935) exposed dogs to a heavy tick infestation (*Rh. sanguineus*). In these animals they were able to demonstrate *Rickettsia canis* during life and post mortem. They were also able to set up this infection in monkeys (*Macacus*) by injecting emulsified engorged adults which had fed on reacting dogs, and also with unengorged emulsified larvæ derived from such ticks.

4. Rickettsia ovina *infection in sheep.*

Lestoquard and Donatien (1936) described a parasite, *Rickettsia ovina,* of the monocytes in the blood of sheep from Turkey and Algeria. From a sheep reacting to this infection they collected engorged adults of *Rhipicephalus bursa*. Emulsions prepared from these ticks when injected into apparently healthy sheep produced a febrile reaction, and *R. ovina* appeared in the peripheral blood. From this experiment it is suggested that *Rh. bursa* is the vector.

5. *Tick-borne fever in sheep and goats.*

Tick-borne fever of sheep, associated with louping-ill, was first described by Gordon, Brownlee, Wilson and Macleod (1932) in Scotland. They found the vector to be *Ixodes ricinus*. Macleod and Gordon (1933) showed that both the nymphæ and adults are infective, and that the infection does not pass through the egg stage. During their investigations they found the goat to be susceptible. Macleod (1936) confirmed the previous observations and showed that nymphæ do not loose their infection when they are allowed to feed on susceptible or insusceptible animals. The disease can also be set up by injecting partially engorged ticks emulsified in serum-saline. On the other hand the injection of emulsified unfed ticks is not capable of setting up the disease.

6. *The tick-transmitted Rickettsiæ of man, dogs and rodents.*

Since the description of Rocky Mountain spotted fever, several other tick-borne ricketsial diseases have been described, namely São Paulo typhus in South America, " fièvre boutonneuse " in the Mediterranean region, and tick-bite fever in East and South Africa. The causal organisms of these diseases have been described as distinct species, but there is today a great deal of evidence showing that these diseases are closely related. In order to avoid confusion the mode of transmission of these diseases will be dealt with under separate headings.

(*a*) *Fièvre boutonneuse* (Rickettsia conori *infection of man and dog*).

Conor and Brugh (1910) described an eruptive fever in man as "fièvre boutonneuse". In 1930 Durand and Conseil succeeded in infecting volunteers by inoculating them with an emulsion of ticks (*Rh. sanguineus*) collected from a dog belonging to a patient suffering from the disease. Similar results were obtained by Brumpt (1930) with ticks collected from patients. The same author also showed that the infection passes from the adult *Rh. sanguineus* to the egg stage, and also from the nymph to the adult. Combriesco and Zotta (1932) bred larvæ from infected adults and demonstrated the presence of the virus in the larvæ by injecting an emulsion into a volunteer. Up to the present *Rh. sanguineus* is the only tick known to be a transmitter of " fièvre boutonneuse ". The virus in the tick behaves in the same way as the virus of Rocky Mountain spotted fever and both stage to stage, and generation to generation transmission have been demonstrated. The common reservoir of this infection is probably the dog. Combiesco and Zotta (1932), and Andréoli and Charlet (1934) describe accidental infection with boutonneuse fever through the conjunctiva, of laboratory workers and of persons who have removed ticks from dogs and crushed them between the fingers.

(*b*) *Sao Paulo typhus* (Dermacentroxenus brasiliensis *infection of man*).

Fialho (1932) gives an account of the occurrence of a form of typhus in São Paulo which was first observed in 1924. He showed, experimentally, that *A. cajennense* is a vector of this disease. Monteiro and da Fonseca (1932) confirmed this observation and in addition proved that the virus is transmissible hereditarily. Monteiro (1934) concludes from the results of cross-immunity tests that São Paulo typhus and Rocky Mountain spotted fever are very closely related. He was also able to demonstrate the causal organisms in the epithelial cells of the intestinal diverticula of ticks. Regendanz and Muniz (1936) transmitted this disease with the nymphs and adults of *Rh. sanguineus* which developed from larvæ fed on infected animals.

Brumpt (1936) showed that the virus can remain viable in *Ornithodorus turicata* for a period of 28 months. This tick is not capable of transmitting the disease while feeding. The presence of the virus in the tick was demonstrated by injecting the emulsified ticks into guinea-pigs.

(*c*) *Tick-bite fever.*

The name tick-bite fever was originally given by Nuttall to a disease first described by Sant'Anna in 1908. Sant'Anna records that the fever resulted from the bite of *Amblyomma* sp. and *Rhipicephalus* sp. Pyper and Dau (1933 and 1934) state that this disease is conveyed to man by the larval ticks of *A. hebræum, Rh. appendiculatus* and *Boophilus decoloratus*. Gear and Douthwaite (1938) found that the dog tick *Hæmaphysalis leachi* is also a vector of this disease. The injection of emulsions of this species of tick collected from dogs produced symptoms of tick-bite fever in guinea-pigs. Alexander, Mason and Neitz (1938) fed nymphæ of *Hyalomma ægyptium var. impressum*, collected from a hare, on a guinea-pig and produced symptoms similar to those of tick-bite fever. The resulting adults also transmitted this infection, but the larvæ and nymphæ bred from the engorged females did not convey this disease to guinea-pigs.

(*d*) *Rocky Mountain spotted fever* (Dermacentroxenus rickettsi *infection of man and rodents*).

The earliest studies relating to the transmission of Rocky Mountain spotted fever were made by Ricketts (1907). He found *D. andersoni* and *D. albipictus* to be vectors of this disease. Subsequently Maver (1911) showed that *D. rickettsi* is also transmitted by *D. variabilis, Amblyomma americanum* and *D. parumapertus marginatus*. Parker (1923) showed that *Hæmaphysalis leporis palustris* is a natural carrier of spotted fever virus. Dyer, Rumreich and Badger (1931), and Parker and Green (1931 and 1932) demonstrated, on several occasions, that *D. variabilis* is a carrier of this disease in nature. Parker, Philip and Jellison (1933)

transmitted spotted fever virus with *D. occidentalis, D. parumapertus marginatus, Rh. sanguineus, A. americanum* and *A. cajennense.*

The species of ticks discussed above as transmitters of this disease include five of *Dermacentor,* one of *Hæmaphysalis,* two of *Amblyomma,* and one of *Rhipicephalus.* Stage to stage transmission has been shown in all these ticks. Generation to generation transmission has been proved in all but *D. parumapertus marginatus* and *A. cajennense.* An interesting observation has been made by Philip and Parker (1933) who showed the sex to sex transfer of spotted fever virus in *D. andersoni,* particularly from infected males to normal females.

It has frequently been shown that spotted fever virus can be transmitted to guinea-pigs by injecting emulsions of infected eggs or ticks intraperitoneally. Brumpt (1936) allowed *Ornithodonis turicata* to feed on guinea-pigs reacting to spotted fever. These ticks failed to transmit this disease while feeding on susceptible guinea-pigs, but in emulsions prepared from such ticks the virus could be demonstrated for periods up to 620 days.

Moore (1911), working with *D. venustus* and *D. modestus,* found that the minimum duration of feeding necessary for ticks to infect a guinea-pig with spotted fever may be as short as 1¾ hours. Infection always resulted when ticks were allowed to feed for 20 hours. Clean ticks feeding on an infected guinea-pig became infected after they had fed for approximately 24 hours.

Virus Diseases.

1. *Louping-ill.*

The sheep-tick *Ixodes ricinus* has long been suspected of being the vector of louping-ill. Successful transmission, however, was only obtained after the cause had been established by Pool, Brownlee and Wilson (1930), and by Greig, Brownlee, Wilson and Gordon (1931). Whether or not Stockman (1916, 1918 and 1919) transmitted louping-ill cannot be stated definitely, because it is not certain whether he was dealing with louping-ill or tick-borne fever.

MacLeod and Gordon (1932) showed that louping-ill can be transmitted by the bite of nymphæ or of adults of *Ixodes ricinus* which have engorged in their previous stage on reacting sheep. Alexander and Neitz (1933 and 1935) transmitted louping-ill with the nymphæ and adults of *Rhipicephalus appendiculatus.* They showed that the infection does not pass through the egg to the next generation. The infective nymphæ tend to loose their infection after feeding on immune animals, though some of the resulting adults may still be infective. The same occurrence was noted after feeding infective nymphæ on susceptible animals, but, particularly

if sheep are the hosts, there is a danger of the nymphæ re-acquiring infection before detachment.

Rivers and Schwentker (1934) describe four cases of accidental laboratory infection in man. Natural transmission by ticks to man has not been observed, but this possibility must not be lost sight of.

2. *Nairobi sheep disease.*

In 1917 Montgomery described a tick-borne gastro-enteritis of sheep and goats transmitted by *Rh. appendiculatus*. Successful transmissions were obtained with adults infected as nymphæ, and with larvæ bred from adults which had fed on reacting sheep. Daubney (1931) confirmed these observations and in addition found that nymphæ infected as larvæ are capable of transmitting the disease. Another interesting observation by these workers was that infection in the vector is retained only to the following stage, unless a second meal of infective blood is obtained before completion of the transmitting feed, which will ensure a carry-over of infection for a further stage. It appears that after the transmitting feed the infection dies out during the moult.

Daubney and Hudson (1934) succeeded in infecting sheep by means of nymphæ and adults of *Amblyomma variegatum* which had fed in the previous stage on reacting sheep. They could not demonstrate the passage of the virus from infected females through the egg to larvæ. With *Rh. bursa* transmission was effected by one adult which had been infected as a nymph. Attempts to confirm this failed. Negative results were obtained with *Rh. evertsi*, *Rh. simus* and *Rh. pulchellus*.

3. *Rabbit papillomatosis.*

Larson, Shillinger and Green (1936) placed nymphæ of *Hæmaphysalis leporis palustris* on a rabbit suffering from papillomatosis. After 24 hours 50 of these ticks were removed and allowed to feed on a healthy rabbit for five days. This rabbit developed typical papillomatosis 16 days later.

PASTEURELLOSIS.

Tularæmia (Pasteurella (Bacterium) tularense *infection*).

Tularæmia occurs in America, Russia, Norway, Sweden, and Japan. It is chiefly a disease of the rural population and of fur-hunters. Infection results from handling infected carcasses or from the bite of blood-sucking ectoparasites.

The reservoirs of this disease are wild rodents : the cotton-tail rabbit (*Sylvilagus*), the jack rabbit (*Lepus*), the new-shoe rabbit (*Lepus*), the ground squirrel (*Citellus*), the muskrat, wild rat, mice, and game-birds. Philip, Jellison, and Wilkens (1935) record an outbreak of tularæmia in 1,320 sheep heavily infested with *Dermacentor venustus*. Of these sheep

40 per cent. were infected and 200 died.

The bacteria can be transmitted mechanically by the horse-fly *Chrysops discalis, Chr. noctifer,* the stable-fly *Stomoxys calcitrans, Tabanus septentrionalis,* the bed-bug *Cimex lectularius,* the squirrel flea *Ceratophyllus acutus,* the squirrel louse *Linognathoides læviusculus,* the rabbit louse *Hæmodipsus ventricosus,* and the mouse louse *Polyplax serratus.*

Ticks can also act as vectors. In the United States of America eight species of ticks are known to be transmitters. Parker (1933) showed experimentally that in all of these ticks the infection, when picked up by the larvæ, is retained in the adult stage and in three species of ticks the infection has been found to pass through the egg to the next generation. In Russia Golov (1934) suspects that *Dermacentor silvarum* can transmit tularæmia. Dissection of *D. andersoni* showed that *B. tularense* is distributed throughout the body, namely, in the gut, the fæces, the epithelial cells, as well as in the body fluids.

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TABLE I.

TICK-BORNE DISEASES.

Disease	Animals affected	Caused by	Country	Transmitter	No. of Hosts	Larva	Nympha	Imago	Egg	Larva	Nympha	Imago
REDWATER	CATTLE	*Piroplasma bigeminum*	N. AMERICA S. AMERICA MANY COUNTRIES S. AMERICA ASIA	*Boophilus annulatus* *Boophilus microplus* *Boophilus australis* *Boophilus argentinus* *Boophilus calcaratus*	1	×	×	×		>		
				Boophilus decoloratus	1	×	×	×		>		
										×	×	×
			SOUTH AFRICA	*Rhipicephalus appendiculatus*	3					>		
				Rhipicephalus evertsi	2	×	×	>				
										×		
			NORTH AFRICA	*Rhipicephalus bursa*	2					>		
			EUROPE	*Hæmaphysalis cinnabarina punctata*	3							
HÆMOGLOBINURIA	CATTLE	*Babesia bovis*	EUROPE	*Ixodes ricinus*	3							
			U.S.S.R.	*Ixodes persulcatus*	3							

NOTE.—× indicates the stage in which the infection is acquired; > indicates that in which it is transmitted.

TABLE I.—*Continued.*

TICK-BORNE DISEASES.

Disease	Animals affected	Caused by	Country	Transmitter	No. of Hosts	Larva	Nympha	Imago	Egg	Larva	Nympha	Imago
EAST COAST FEVER	CATTLE	*Theileria parva*	AFRICA	*Rhipicephalus appendiculatus*	3	×——	>					
				Rhipicephalus capensis								
				Rhipicephalus simus			×——	>				
				Rhipicephalus evertsi	2	×——	×——	>				
				Hyalomma impressum	2 or	×——	>					
				near planum	3		×——	>				
	CATTLE	*Theileria dispar*	NORTH AFRICA	*Hyalomma mauretanicum*	2	×——	×——	>				
			EAST AFRICA *	*Hyalomma impressum*	2 or							
				near planum	3		×——	>				
	CATTLE	*Theileria annulata*	TRANSCAUCASIA	*Hyalomma detritum rubrum*	2	×——	×——	>				
				Hyalomma dromedarii asiaticum	2	×——	×——	>				
	CATTLE	*Theileria mutans*	AFRICA	*Rhipicephalus appendiculatus*	3		×——	>				
				Rhipicephalus evertsi	2	×——	×——	>				
BILIARY FEVER	HORSE	*Piroplasma caballi*	U.S.S.R. & ITALY	*Dermacentor reticulatus*	3			×——	——	——	>	—— >
			U.S.S.R.	*Dermacentor silvarum*		×——	>	—— >				

* Experimentally.

TABLE I.—*Continued.*

TICK-BORNE DISEASES.

Disease	Animals affected	Caused by	Country	Transmitter	No. of Hosts	Larva	Nympha	Imago	Egg	Larva	Nympha	Imago
BILIARY FEVER	HORSE	*Nuttallia equi*	SOUTH AFRICA	*Rhipicephalus evertsi*	2	×	×	>				
	SHEEP	*Piroplasma ovis*	RUMANIA U.S.S.R.	*Rhipicephalus bursa*	2	× ×	× ×	> >				×
	SHEEP	*Babesia ovis*	U.S.S.R.	*Rhipicephalus bursa*	2	×	×	>				
	SHEEP	*Theileria ovis*	U.S.S.R.	*Rhipicephalus bursa*	2	×	×	>				
	DOG	*Babesia gibsoni*	INDIA	*Rhipicephalus sanguineus*	3	×		>				
				Hæmaphysalis bispinosa	3		×	>		×	>	>
BILIARY FEVER	DOG	*Piroplasma canis*	SOUTH AFRICA	*Hæmaphysalis leachi*	3			×				>
				Dermacentor riticulatus	3			×			>	>
			EUROPE	*Dermacentor venustus*	3			×				>
			MANY COUNTRIES	*Rhipicephalus sanguineus*	3			×		>	>	>
						×	>					
							×	>				

TABLE I.—*Continued.*

TICK-BORNE DISEASES.

Disease	Animals affected	Caused by	Country	Transmitter	No. of Hosts	Larva	Nympha	Imago	Egg	Larva	Nympha	Imago
	FOWL	*Ægyptionella pullorum*	MANY COUNTRIES	*Argas persicus*	Many			×>>				
GALLSICKNESS	CATTLE	*Anaplasma marginale*	SOUTH AFRICA N. AMERICA S. AMERICA S. AMERICA	*Boophilus decoloratus* *Boophilus annulatus* *Boophilus microplus* *Boophilus australis*	1	×——	×——	×——>				
			SOUTH AFRICA	*Rhipicephalus simus*	3			×——>				
			NORTH AFRICA	*Rhipicephalus bursa*	2		×——>					
			N. AMERICA	*Rhipicephalus sanguineus*	3	×——>						
			NORTH AFRICA	*Hyalomma lusitanicum*				×——>				
			EUROPE	*Ixodes ricinus*	3			×——>				
			N. AMERICA	*Ixodes scapularis*	3	×——>						
				Dermacentor andersoni	3	×——>						
				Dermacentor variabilis	3	×——>						
							×——>					
				Dermacentor occidentalis	3	×——>						
				Dermacentor albipictus	3			×——>				
			EUROPE	*Hæmaphysalis cinnabarina punctata*	3		×——>					
								×——>				
	SHEEP	*Anaplasma ovis*	U.S.S.R.	*Ornithodorus lahorensis*	Many	?×——>?						
							?——?——>?					

TABLE I.—*Continued.*

TICK-BORNE DISEASES.

Disease	Animals affected	Caused by	Country	Transmitter	No. of Hosts	Larva	Nympha	Imago	Egg	Larva	Nympha	Imago
NAIROBI-SHEEP DISEASE	SHEEP AND GOATS	*Ultravisible virus*	EAST AFRICA	*Rhipicephalus appendiculatus*	3	×	×	×				
				Amblyomma variegatum	3	×	×					
LOUPING-ILL	SHEEP HORSE CATTLE	*Ultravisible virus*	GREAT BRITAIN	*Ixodes ricinus*	3	×	×	×				
			SOUTH AFRICA *	*Rhipicephalus appendiculatus*	3	×	×	×				
RABBIT PAPILLOMATOSIS	RABBIT	*Ultravisible virus*	AMERICA	*Hæmaphysalis leporis-palustris*	3		×					
HEARTWATER	RUMINANTS	*Rickettsia ruminantium*	AFRICA	*Amblyomma hebræum*	3	×	×	×				
				Amblyomma variegatum	3		×					
ROCKY MOUNTAIN SPOTTED FEVER	MAN WILD RODENTS	*Dermacentroxenus rickettsi*	N. AMERICA	*Dermacentor venustus*		×	×	×				

* Experimentally.

TABLE I.—*Continued.*

TICK-BORNE DISEASES.

Disease	Animals affected	Caused by	Country	Transmitter	No. of Hosts	Larva	Nympha	Imago	Egg	Larva	Nympha	Imago
ROCKY MOUNTAIN SPOTTED FEVER (Continued)	MAN WILD RODENTS (Continued)	*Dermacentroxenus rickettsi* (Continued)	NORTH AMERICA (Continued)	*Dermacentor variabilis*	3	×—>	×—>	×—>—>—>				
				Dermacentor occidentalis	3	×—>		×—> ×—>—> ×—>				
				Dermacentor paruma-pertus marginatus	3	×—>—>	×—>					
				Dermacentor andersoni	3	×—>—>	×—>					♂×♀*
				Dermacentor albipictus	3		×—>					
				Dermacentor modestus	3	×—>—>		×—>				
				Rhipicephalus sanguineus	3	×—>—>		×—>—>—> ×—>—>—> ×—>				
				Amblyomma americanum	3	×—>—>		×—>				
				Amblyomma cajennense	3	×—>—>						
				Hæmaphysalis leporis-palustris	3	×—>—>						

* Sex to sex transfer by coitus.

TABLE I.—*Continued.*

TICK-BORNE DISEASES.

Disease	Animals affected	Caused by	Country	Transmitter	No. of Hosts	Larva	Nympha	Imago	Egg	Larva	Nympha	Imago
SAO PAULO or BRAZILIAN TYPHUS	MAN AND RAT	*Dermacentroxenus brasiliensis*	BRAZIL	*Amblyomma cajennense*	3		×	>				
								×		>		
				Rhipicephalus sanguineus	3	×	>	>				
FIÈVRE BOUTONNEUSE	MAN AND DOG	*Rickettsia conori*	MEDITERRANEAN COUNTRIES	*Rhipicephalus sanguineus*	3	×	>	>		>		
							×	>		>		
								×		>		
TICK-BITE FEVER	MAN DOG WILD RODENTS	*Rickettsia species*	SOUTH & EAST AFRICA	*Hæmaphysalis leachi*	3							
				Amblyomma hebræum	3	× ?	× ?	× ?		>		
				Boophilus decoloratus	1	× ?	× ?	× ?		>		
				Hyalomma ægyptium var impressum	2 or 3	× ?	>	>				
TICK-BORNE FEVER	SHEEP GOAT		SCOTLAND	*Ixodes ricinus*	3	×	>	>				
							×	>				
	CATTLE	*Rickettsia bovis*	IRAN	*Hyalomma species*	2	×	×	>				

TABLE I.—*Continued.*

TICK-BORNE DISEASES.

Disease	Animals affected	Caused by	Country	Transmitter	No. of Hosts	Larva	Nympha	Imago	Egg	Larva	Nympha	Imago
	SHEEP	*Rickettsia ovina*	NORTH AFRICA	*Rhipicephalus bursa*	2	×	×	?				
	DOG	*Rickettsia canis*	NORTH AFRICA	*Rhipicephalus sanguineus*	3	? ×	? >	? >		?		
	CATTLE	*Eperythrozoon wenyoni*	IRAN	*Hyalomma species*	2	? ×	? ×	?				
SPIROCHÆTOSIS	CATTLE SHEEP GOAT HORSE	*Spirochæta theileri*	SOUTH AFRICA	*Boophilus decoloratus*	1	×	×	×		>		
				Rhipicephalus evertsi	2	×	×	>				
				Boophilus annulatus	1	×	×	×		>		
				Boophilus australis	1	×	×	×		>		
SPIROCHÆTOSIS	POULTRY	*Spirochæta anserina* (*S. gallinarum*)	MANY COUNTRIES	*Argas persicus*	Many	×	?					
				Argas miniatus			×	>				
				Argas reflexus				? >				
				Argas victoriensis					×	>	>	>
							>	>				

TABLE I.—*Continued.*

TICK-BORNE DISEASES.

Disease	Animals affected	Caused by	Country	Transmitter	No. of Hosts	Larva	Nympha	Imago	Egg	Larva	Nympha	Imago
RELAPSING FEVER	MAN MONKEY AND SMALL WILD RODENTS	*Spirochæta duttoni*	AFRICA	*Ornithodorus moubata*	Many							
				Ornithodorus mòrocanus	Many							
RELAPSING FEVER	MAN MONKEY GERBILLES	*Spirochæta persica*	IRAN NORTH WEST INDIA	*Ornithodorus tholozani* *Ornithodorus lahorensis*	Many Many							
RELAPSING FEVER	MAN MOROCCAN HEDGEHOG DESERT FOXES JACKALS	*Spirochæta hispanica*	SPAIN MOROCCO	*Ornithodorus morocanus* *Rhipicephalus sanguineus*	Many 3							
RELAPSING FEVER	MAN S. AMERICAN MONKEY MARMOSETS RATS	*Spirochæta venezuelensis*	CENTRAL & SOUTH AMERICA	*Ornithodorus venezuelensis* *Ornithodorus talajé*	Many Many							

TABLE I.—*Continued.*

TICK-BORNE DISEASES.

Disease	Animals affected	Caused by	Country	Transmitter	No. of Hosts	Larva	Nympha	Imago	Egg	Larva	Nympha	Imago
TULARÆMIA	MAN RHESUS MONKEY WILD RODENTS GUINEA PIGS GAME BIRDS SHEEP	*Bacterium (Pasteurella) tularense*	N. AMERICA	*Dermacentor andersoni*	3	×	→	→		→		
				Dermacentor occidentalis	3		×	→				
				Dermacentor variabilis	3	×	→	→		→		
				Dermacentor parumapertus marginatus	3		×	→				
				Hæmaphysalis leporis-palustris	3	×	→	→		→		
				Hæmaphysalis cinnabarina	3							
				Rhipicephalus sanguineus	3	×	→	→				
				Amblyomma americanum	3	×	→	→				
	DOG	*Hepatozoon canis*	AFRICA INDIA	*Rhipicephalus sanguineus*	3	×	→					
						×		→				
							×	→				

TABLE II.

TICK-BORNE DISEASES.

Genus	Species	Parasite	Host
ARGAS	*A. miniatus* *A. reflexus* *A. victoriensis*	*Spirochæta anserina*	Poultry
	A. persicus	*Spirochæta anserina*	Poultry
		Ægyptionella pullorum	Poultry
ORNITHODORUS	*O. moubata*	*Spirochæta duttoni*	Man, etc.
	O. morocanus	*Spirochæta duttoni*	Man, etc.
		Spirochæta hispanica	Man, etc.
	O. lahorensis	*Spirochæta persica*	Man, etc.
		Theileria ovis?	Sheep & Goat
		Anaplasma ovis?	Sheep & Goat
	O. talaje	*Spirochæta venezuelensis*	Man, etc.
	O. venezuelensis	*Spirochæta venezuelensis*	Man, etc.
AMBLYOMMA	*A. americanum*	*Dermacentroxenus rickettsi*	Man, etc.
		Bacterium (Pasteurella) tularense	Man, etc.
	A. cajennense	*Dermacentroxenus rickettsi*	Man, etc.
		Dermacentroxenus brasiliensis	Man, etc.
	A. hebræum	*Rickettsia ruminantium*	Ruminants
		R. species (Tick-bite fever)	Man, etc.
	A. variegatum	*Rickettsia ruminantium*	Ruminants
		Nairobi sheep disease	Sheep & Goats
BOOPHILUS	*B. annulatus*	*Piroplasma bigeminum*	Cattle
		Anaplasma marginale	Cattle
		Spirochæta theileri	Cattle, Horses, etc.
	B. australis	*Piroplasma bigeminum*	Cattle
		Anaplasma marginale	Cattle
		Spirochæta theileri	Cattle, Horses, etc.
	B. argentina	*Piroplasma bigeminum*	Cattle
	B. calcaratus	*Piroplasma bigeminum*	Cattle

TABLE II—*Continued.*

TICK-BORNE DISEASES.

Genus	Species	Parasite	Host
BOOPHILUS (*continued*)	*B. decoloratus*	*Piroplasma bigeminum*	Cattle
		Anaplasma marginale	Cattle
		Spirochæta theileri	Cattle, Horse, etc.
		Rickettsia sp. (Tick-bite fever)	Man, etc.
	B. microplus	*Piroplasma bigeminum*	Cattle
		Anaplasma marginale	Cattle
DERMACENTOR	*D. albipictus*	*Dermacentroxenus rickettsi*	Man, etc.
		Anaplasma marginale	Cattle
	D. andersoni *	*Dermacentroxenus rickettsi*	Man, etc.
		Anaplasma marginale	Cattle
		Bacterium tularense	Man, etc.
	D. modestus	*Dermacentroxenus rickettsi*	Man, etc.
	D. occidentalis	*Dermacentroxenus rickettsi*	Man, etc.
		Anaplasma marginale	Cattle
		Bacterium tularense	Man, etc.
	D. parumapertus marginatus	*Dermacentroxenus rickettsi*	Man, etc.
		Bacterium tularense	Man, etc.
	D. reticulatus	*Piroplasma caballi*	Horse
		Piroplasma canis	Dog
	D. silvarum	*Piroplasma caballi*	Horse
	D. variabilis	*Dermacentroxenus rickettsi*	Man, etc.
		Anaplasma marginale	Cattle
		Bacterium tularense	Man, etc.
	D. venustus *	*Dermacentroxenus rickettsi*	Man, etc.
		Piroplasma canis	Dog
HÆMAPHYSALIS	*H. bispinosa*	*Babesia gibsoni*	Dog
	H. cinnabarina	*Bacterium tularense*	Man, etc.
	H. cinnabarina punctata	*Piroplasma bigeminum*	Cattle
		Anaplasma marginale	Cattle

* *D. andersoni* and *D. venustus* refer to the same species.

TABLE II—*Continued.*

TICK-BORNE DISEASES.

Genus	Species	Parasite	Host
HÆMA-PHYSALIS (*continued*)	*H. leachi*	*Piroplasma canis*	Dog
		Rickettsia species (*Tick-bite fever*)	Man, etc.
	H. leporis-palustris	*Dermacentroxenus rickettsi*	Man, etc.
		Bacterium tularense	Man, etc.
		Rabbit papillomatosis	Rabbit
HYALOMMA	*H. detritum rubrum*	*Theileria annulata*	Cattle
	H. dromedarii asiaticum	*Theileria annulata*	Cattle
	H. impressum near *planum*	*Theileria dispar*	Cattle
		Theileria parva	Cattle
	H. ægyptium var. *impressum*	*Rickettsia sp.* (*Tick-bite fever*)	Man, etc.
	H. lusitanicum	*A. marginale*	Cattle
	H. mauretanicum	*Theileria dispar*	Cattle
	H. species	*Rickettsia bovis*	Cattle
	H. species	*Eperythrozoon wenyoni*	Cattle
IXODES	*I. persulcatus*	*Babesia bovis*	Cattle
	I. ricinus	*Babesia bovis*	Cattle
		Anaplasma marginale	Cattle
		Tick-borne Fever	Sheep & Goat
		Louping-Ill	Sheep, etc.
	I. scapularis	*Anaplasma marginale*	Cattle
RHIPICE-PHALUS	*Rh. appendi-culatus*	*Piroplasma bigeminum*	Cattle
		Theileria parva	Cattle
		Theileria mutans	Cattle
		Nairobi sheep disease	Sheep & Goats
		Louping-Ill	Sheep, etc.

TABLE II—*Continued.*

TICK-BORNE DISEASES.

Genus	Species	Parasite	Host
RHIPICEPHALUS (*continued*)	*Rh. bursa*	*Piroplasma bigeminum*	Cattle
		Babesia motasi	Sheep
		Babesia ovis	Sheep
		Theileria ovis (*recondita*)	Sheep & Goat
		Anaplasma marginale	Cattle
		Rickettsia ovina	Sheep
	Rh. capensis	*Theileria parva*	Cattle
	Rh. evertsi	*Piroplasma bigeminum*	Cattle
		Theileria parva	Cattle
		Theileria mutans	Cattle
		Nuttallia equi	Horse
		Spirochæta theileri	Cattle, Horse, etc
	Rh. sanguineus	*Babesia gibsoni*	Dog
		Piroplasma canis	Dog
		Anaplasma marginale	Cattle
		Dermacentroxenus rickettsi	Man, etc.
		Dermacentroxenus brasiliensis	Man, etc.
		Rickettsia conori	Man, etc.
		Rickettsia canis	Dog
		Hepatozoon canis	Dog
		Spirochæta hispanica	Man, etc.
		Bacterium tularense	
	Rh. simus	*Theileria parva*	Cattle

TABLE III.

Disease	Parasite	Incubation period in days		Remarks
		Artificial transmission	Natural transmission	
Anaplasmosis	*Anaplasma centrale*	25–42	?	
	Anaplasma marginale	14–30	27–100	
	Anaplasma ovis	21–35	?	Period of 2–5 days quoted in the literature cannot be correct.

TABLE III—(*continued*).

Disease	Parasite	Incubation period in days		Remarks
		Artificial transmission	Natural transmission	
Eperythrozoonosis	*Eperythrozoon wenyoni*	5–10	?	
	Eperythrozoon ovis	2–21	?	
Pasteurellosis	*Bacterium tularense*	1–2	1–9	
Ægyptionellosis	*Ægyptionella pullorum*	10–13	10–15	
Piroplasmosis	*Piroplasma bigeminum*	6–15	8–21	
	Piroplasma caballi	5–10	10–13	
	Piroplasma canis	3–15	8–12	
	Piroplasma ovis (*Babesia motasi*)	6–10	7–8	
	Piroplasma trautmanni	5–8	?	
Babesiosis	*Babesia bovis*	4–12	9–20	
	Babesia gibsoni	5–21	14–16	
	Babesia ovis	6–8	7–8	
Nuttalliosis	*Nuttallia equi*	5–10	8–14	
Rickettsiosis Heartwater	*Rickettsia ruminantium*	6–15	9–21	
Rocky Mountain Spotted Fever	*Dermacentroxenus rickettsi*	4–5	7–10	
Fièvre boutonneuse	*Rickettsia conori*	4–6	7–9	

TABLE III—(*continued*).

Disease	Parasite	Incubation period in days		Remarks
		Artificial transmission	Natural transmission	
Sao Paulo Spotted Fever	*Dermacentroxenus brasiliensis*	4–5	7–10	
Tick-bite Fever	*Rickettsia species*	4–6	5–7	
Spotted Fever	*Rickettsia prowazeki*	4–8	5–14	
Tick-borne Fever		3–13	3–6	
Rickettsiosis	*Rickettsia bovis*	7–12	?	
	Rickettsia ovina	7–10	?	
	Rickettsia canis	7–10	?	
Spirochætosis (Relapsing fever)	*Spirochæta obermeyeri*	2–6	2–10	
	Piroplasma duttoni, etc.	2–6	2–12	
Spirochætosis of birds	*Spirochæta anserina*	2–3	4–6	
Spirochætosis of domestic animals	*Spirochæta theileri*	2–6	16–35	
Theileriosis	*Theileria annulata*	9–10	9–17	
	Theileria dispar	10–14	14–18	
	Theileria parva	10–12	9–25	
	Theileria mutans	21–42	15–30	
	Theileria ovis	21–28	?	Period 2–5 days mentioned in the literature cannot be correct.
Virus Diseases	Louping-Ill	2–5	2–6	
	Nairobi sheep disease	3–4	3–6	
	Rabbit papillomatosis	6–12	16	

Superfoetation in an Elephant.

By G. PFAFF, B.Sc., M.R.C.V.S., Insein, Burma.

The huge bulk of the elephant is probably responsible for many of the curious ideas current about this animal. It is, for instance, a common practice to dress wounds with ten per cent. tincture of iodine, apparently on the assumption that the microorganisms on the elephant are hardier than those on smaller animals. Many find the period of gestation a matter of intriguing curiosity and I am often asked : " It is true that elephants take years and years to have a calf ? " But it is the position assumed during copulation which arouses most curiosity. A European who has witnessed the act is still something of a phenomenon. Even many who are conversant with elephants think that the position of the vulva, which is well forward on the ventral abdominal wall, and the great weight of the elephant must make copulation an acrobatic achievement.

Steel (1885) relates that " on medals and other native works of art the animals are represented *sub coitu,* the female literally standing on her head." The same author quotes a writer in the *Oriental Sporting Magazine* who witnessed the act, the female standing in a trench, which was straddled by the male. But even Aristotle (cited by Steel) noted that during sexual excitement erection of the clitoris brings the vulva of the elephant into a position favourable to congress of the sexes. Moreover, the weight of the male elephant is no handicap because this is no greater burden to the female than the weight of the guinea-pig is to her mate. Actually during copulation the male elephant, unlike the stallion and the bull, takes most of his weight on his hind legs.

Œstrus in the elephant is accompanied by no apparent change in the external genitalia and by little or no change in behaviour. This probably explains the wide variation in the duration of pregnancy given for this animal. Steel, quoting various observers, gives the gestation period as 20 months 18 days (Corse), 18 months for a female calf and 22 months for a male calf (Sanderson), 583 to 680 days (writers in " The Asian "), while Evans (1910) records periods from seventeen to twenty-four months, and adds that Burmans and Karens consider that pregnancy may extend to thirty months. He also records one case of a male calf being born twenty-two months after the previous parturition.

European firms engaged on timber extraction in Burma own some five thousand elephants, each of which has at least one attendant and is under close observation. About 250 calves are born each year.

If a rider notices that when released a female makes up to a male and the two wander off together and keep company, a watch may be kept

and copulation witnessed, or the following day the hoof marks of the male may be seen on the back of the female, and these facts are noted in the history book of the elephant. An examination of a large number of such records shows a wide variation in the duration of pregnancy. One cow calved nine months after copulation, and some cows went twenty-seven months. This wide variation may be explained on the one hand by a service being recorded when it did not take place, and on the other hand by service occurring during a later heat than the one noted. Also pregnant cows are sometimes served, some because they are willing to take the bull and others because they are raped by tuskers on musth. According to these records the duration of pregnancy is round about twenty-one months, though it is generally accepted by elephant men that the duration of pregnancy is slightly longer for male than for the female calves.

The birth of elephant twins, although uncommon, is not unknown. Two such cases have been reported to me. One set of twin females died soon after birth. The other pair, a male and a female, both survived. It is interesting to note that the female is now, at the age of sixteen, a mother.

I have to thank Mr. R. H. Baillie, of the Bombay Burmah Trading Corporation, Limited, for bringing to my notice a case af particular interest. An elephant, Mokamai by name, owned by a Karen, Saw Tha Kwa, was captured ten years ago, and is now about twenty-one years of age. In the hot weather of 1934 she was served by Pakalo, but produced no calf. On the 17th December, 1936, Mokamai gave birth to a male calf, which died sixteen days later of an infection of the umbilicus, and four months later, on the 14th April, 1937, she produced a female calf which was still alive in December, 1937.

Weakly or premature elephant calves do not survive long, and as the first calf lived for sixteen days and died from a known cause it could not have been premature. If both calves resulted from the service in the hot weather of 1935, which may be regarded as having taken place in April, 1935, the gestation period for the male calf was twenty months and for the female calf twenty-four months. The second calf was not of abnormal size, so it seems to me unlikely that both calves resulted from the fertilisation of ova formed during the same œstral period. The case is probably one of superfœtation. The following letter, which vouches for the facts cited above, was sent by the Karen, Saw Tha Kwa.

Dated Toungoo, the 25th Nov., 1937.

The Manager,
B. B. T. C.,
Pyinmana.

Sir,

With reference to your letter dated the 22nd of November, 1937, I beg to reply to your question as follows :—

1. That in 1934 at the elephant grazing ground at Myitngachaung east of Toungoo, Mokamai had sexual intercourse with Pakalo.
2. That in 1935 at the elephant grazing ground of Nanchoon east of Pyinmana the said Mokamai enjoyed sexual intercourse with a male elephant named Par Si.
3. On the 17th December, 1936, a male elephant was born to Mokamai with injury at the bowel and died on 1st January, 1937.
4. On the 14th April, 1937, Mokamai gave birth to a female elephant, and is still alive.
5. Three years before having sexual intercourse with Mokamai, Pakalo had sexual pleasure with another female elephant but had no issue.

All the statements made by me above are true to my own personal knowledge and by my own eye sight. I have never seen Mokamai giving birth any elephant prior to that except the two small elephants in question.

(Signed) SAW THA KWA,
Contractor.

Institute Village, Toungoo.

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The Genetic Nature of Susceptibility to Cancer (*concluded*).

By J. J. BRONKHORST, Ph.D., B.Sc. (Agric.), Onderstepoort.

Lung Tumours in Mice.

The work on lung tumours in mice is not as extensive as that on the mammary gland neoplasm. The observations on the inheritance of lung tumours made by the workers at the Roscoe B. Jackson Memorial Institute are usually complicated by the appearance of mammary-gland tumours. The latter develop earlier than the lung tumours and the affected individuals are, on account of their early death, useless as material for the study of the incidence of lung tumours. Lynch (1926) made some preliminary observations on lung tumours and found that the greatest incidence was at the age of two years and over, and that sex, if at all effective, had comparatively little influence. In crosses between a low and a high-tumour strain, cancer appeared in 50 per cent. of the F1, and 25 per cent. of the F2 mice. This suggested that the factor determining the incidence of pulmonary tumours might be a dominant one. The author thought the factor might be variable, and argued that, where offspring from unaffected parents developed cancer, these parents actually were "tumour mice," but that the conditions of the environment did not favour the expression of cancer.

Lynch (1931) crossed a male mouse, from a pure strain with a high incidence of spontaneous lung tumours, with several females from a low-tumour strain. By the time the mice were 18 months old, lung tumours had appeared in 52 per cent. This shows clearly that the male mouse transmits pulmonary tumours to his offspring, although this is not the case with breast tumours. Several of the F1 males were back-crossed to females of the original strains, namely strain D which gave a high incidence of pulmonary tumours (34 per cent. of 222 mice over six months of age), and strain 1194 in which the incidence of lung tumours amounted to 6.7 per cent. of mice even after twelve months of age. These back-cross data are presented in Table 5. As might be expected, the incidence of lung tumours was highest in the back-crosses to the high-tumour females, and the difference is satisfactorily significant at all ages. From the back-cross data, no conclusions as to the number of genetic factors involved could be drawn.

One of the greatest difficulties in the genetic study of cancer susceptibility is the impossibility of classifying by inspection. Many of the mice die before they reach the age at which tumours become apparent. Others

TABLE V.

INCIDENCE OF LUNG TUMOURS IN BACK-CROSSES TO HIGH AND LOW LUNG-TUMOUR STRAINS.

		Backcross to Strain D (high tumours)		Backcross to Strain 1194 (low tumours).			
Age in months.	Total mice.	Number of mice with tumours.	Percentage mice with tumours.	Total mice.	Number of mice with tumours.	Percentage mice with tumours.	Difference between percentages.
7–12	125	11	8.8 ± 1.7	87	2	2.3 ± 1.1	6.5 ± 2.0
13–18	149	39	26.2 ± 2.4	96	4	4.2 ± 1.4	22.0 ± 2.8
19–24	79	43	54.4 ± 3.8	24	7	29.2 ± 6.3	30.3 ± 7.3
25–31	51	37	72.5 ± 4.3	11	3	27.3 ± 9.1	45.3 ± 10.0
Total	404	130	32.2 ± 1.6	218	16	7.3 ± 1.2	24.8 ± 2.0

again live past this age without developing neoplasms, but such individuals cannot definitely be classified as non-tumourous, since the requisite secondary environmental influences may have been non-operative.

The discovery that pulmonary tumours could be induced by tarring provided a means of checking the results regarding spontaneous lung tumours. This method was applied to the strains in question. At 13 months of age the mice were killed and the percentage of lung tumours found was 22.4 and 85.4 in the low and high lines respectively. Not only did the tumours become evident at an earlier age, but the incidence was increased considerably. Certain mice were mated prior to tarring, and the F1 tumour rate after tarring was 78.6 per cent. In two back-crosses the tarred progeny showed lung tumour incidence of 81.1 per cent. and 39.5 per cent., depending on whether the high or low tumour parent was used. Thus the results on spontaneous tumours are confirmed, and the familial incidence is again very evident.

Conclusions.

Lung tumours are transmitted through male and female alike, thus differing from mammary gland tumours. The exact number of genetic factors remains undetermined, though it is evident that the multiple factor hypothesis is the most probable explanation.

Unknown environmental factors play an important rôle in the onset and malignancy of pulmonary tumours.

Leukæmia.

The term " leukemia " is used by MacDowel and Richter (1935) for the sake of convenience, and includes various conditions involving malignant leucocytic infiltrations. The great majority of their cases were leukæmias of the lymphatic type. Out of a total of 543 positive diagnoses, 450 were lymphatic (nineteen of these lymphosarcomatous) and six myeloid, while in eighty-seven cases the diagnosis was doubtful. *Post-mortem* changes were largely responsible for the doubtful diagnoses.

This strain of mice, known as strain C 58, was inbred for 27 generations and a very high degree of genetic uniformity thus obtained. Among the definite diagnoses, 90.1 per cent. were leukæmia and 10 per cent. negative. This variability was almost certainly non-genetic, because the non-leukæmia mice were scattered at random through the different families and branches of the pedigree and, further, matings between the non-leukæmic mice produced just as large number of leukæmic offspring as matings between affected mice. Thus genetic constitution could not be held responsible for the failure of the ten per cent. to develop leukemia. This small proportion must have met conditions which prevented the manifestation of the hereditary tendency. One could say that in strain C 58 heredity was nine times as potent as non-genetic variables in controlling the incidence of leukæmia.

The age distribution of the two groups is interesting. Non- leukæmic mice lived longer, and thus it cannot be argued that they failed to develop leukæmia because they died prematurely.

Another strain, the StoLi (Storrs-Little), in which leukæmia occurs in only one per cent. of mice, has also been studied extensively.

Thus in these two strains, which were used in the hybridization experiments, one showed an incidence of 90 per cent. leukæmia and the other one per cent.

Table VI gives the F1 from reciprocal matings between 11 males and 17 females from strain C 58, and 6 males and 21 females from strain StoLi.

TABLE VI.

F1 FROM RECIPROCAL CROSSES BETWEEN HIGH AND LOW LEUKEMIC STRAINS.

Sex	Number of offspring from						Percentage definite diagnosis of leukemia in mice from		
	C58 mothers (high tumour)			StoLi mothers (low tumour)			C58 mothers	StoLi mothers	Diff.
	+	—	?	+	—	?			
Male	41	32	13	26	38	13	56.2	40.6	15.6
Female	45	21	14	19	23	21	68.2	45.2	23.0
Total	86	53	27	45	61	34	61.9	42.5	19.4 ± 4.3

+ = Positive, — = negative, ? = doubtful.

We note that the incidence of leukæmia is reduced by approximately one half of the first filial generation. Since any F1 generation is genetically as uniform as the two parental strains, it is hard to understand why only fifty per cent. should succumb to leukæmia, especially if one remembers that the genetic constitution of every F1 mouse is exactly the same. If leukæmia were induced by a simple dominant, every mouse should die; on the other hand, if it were a simple recessive, then the disease should skip one generation.

It is of course possible that hybrid vigour due to the crossing of two highly inbred strains, and modifying factors, may be a reason why only half the progeny show cancer in the F1 generation. It is more likely, however, that the increased variability in the F1 is due to non-chromosomal variables. One important influence is transmitted solely through the mother: for when C 58 (high tumour) mothers are used, 19.4 per cent.

more of the offspring show leukæmia than when low-tumour mothers are used. This difference is statistically significant, but it is not as large as in the case of mammary-gland carcinomata. We have seen that Little and his co-workers explained their findings on breast cancer as the result of extra-chromosomal influence transmitted through the mother's milk. This, however, is not the case with leukæmia, since MacDowel and Richter eliminated this possibility by foster experiments. Apart from the purely genetic factors which determine leukæmia in mice, there thus remain unidentified non-chromosomal influences which may be transferred through the cytoplasm or during intra-uterine life.

The genetic uniformity of an F1 generation derived from the crossing of two pure strains has already been stressed. This uniformity was demonstrated directly by back-crosses of leukemic, negative, and doubtful cases, to the pure bred StoLi strain. In all crosses the occurrence of leukæmia was between 43 and 50 per cent., thus proving that the genetic constitution of F1 mice was the same, whether they died of leukæmia or not.

In back-crosses of the F1 progeny to the pure-bred StoLi strain, the non-chromosomal influence of the female was again demonstrated. When the F1 hybrid was the father, 19.8 per cent. of the offspring developed leukæmia; when the F1 hybrid was the mother, 46.5 per cent. succumbed to leukæmia. The difference 26.7 ± 3.8 must be considered significant.

If the maternal influence is excluded (difference between reciprocal crosses), the incidence of leukæmia is reduced about one-half when the total heredity from strain C 58 is reduced one-half. This is again reduced by one-half in the back-cross. The rôle of heredity is thus unquestionable and can be expressed in quantitative terms more or less on the lines of Galton's Laws, although this takes no account of the transmission in terms of genes. The only way of getting further evidence would be by testing the breeding behaviour of every back-cross mouse by making a further back-cross. Thus the mouse's genetic constitution for leukæmia would be judged by the breeding behaviour of its offspring, rather than by its own tendency to develop leukæmia.

Strains of mice similar to C 58 (but with a lower incidence of leukæmia) have been reported by Doprovolskaia-Zavadskaia (1932), McCoy Hill (1930), and Slye (1931). The last is the only worker who has ventured to put the genetics of leukæmia on a genic basis. She believes that all neoplastic conditions, including leukæmia, depend on a single recessive Mendelian gene. In a later report (Slye, 1937) she states that a number of recessive genes might be involved. The consensus of opinion, however, is that there is insufficient evidence for such a conclusion.

Transplantable Tumours.

The discovery of transplantable tumours opened a wide field of research, and the use of such tumours to investigate susceptibility to cancer

was one of the earliest methods employed. From about 1890 many conflicting reports have been published. This is easily understood, because no uniform strains of mice were established, and the tumours investigated were of different types. Furthermore, the science of genetics was still in its infancy, and co-operation between pathologists and geneticists was lacking. Among the early workers on the problem we find such well-known names as Jensen, Loeb, Borrel, Ehrlich, Murray, Hertwig, Tyzzer, and Little. Since 1918, most of the work on the genetic aspect has been done by Dr. L. C. Strong of the Bussey Institute (1922, 1926a, 1926b, 1934, 1936). Strong started his work with two mammary-gland adenocarcinomata which were histologically indistinguishable form each other, although the reaction of these tumours to the same host was different. Several stocks of mice were tested, and the dilute brown (dba) strain was found to be uniformly susceptible to both transplants, whereas another inbred strain, known as the Bagg albinos, proved to be uniformly resistant. The F1 progeny of the two strains grew both tumours progressively and at a greater rate than the original inbred susceptible strain. In the F2 generation, the two tumours gave different ratios of susceptible to non-susceptible mice; the first tumour gave a ratio indicating three factors and the second a ratio indicating two factors. This means that at least two mendelian factors (which have been named Ast and Bst) must be present, simultaneously, for mice to be susceptible to the second tumour. The other adenocarcinoma will grow when three factors are present, *viz.* Ast, Bst, and Cst. From this Strong developed his genetic theory for the transplantation of neoplastic tissue, which he stated as follows : "The fate of the implanted tumour tissue when placed in a given individual (host) is brought about by a reaction between the host, determined to a large extent by its genetic constitution, and the transplantable tumour cell, controlled to some extent by certain intrinsic factors."

A criticism of the theory was that it did not apply to certain tumours which could be transplanted to all mice irrespective of their heredity (the so-called non-specific tumours). In other words, all the genes normally present in a mouse are required for susceptibility to these non-specific tumours. Strong, however, after injecting hundreds of mice with such a tumour eventually found three mice which were resistant. Six years of careful selection from these enabled him to establish a line completely resistant to this transplant. Thus he showed that the so-called non-specific tumours were no exception to the general rule.

The transplantability of a tumour is distinct from its proliferative ability. The problem of growth-rate is more complex than susceptibility, and thus more difficult to analyse. We have seen above that where a susceptible and a non-susceptible strain were crossed, the F1 proved to be susceptible, and the growth-rate of the tumour was faster than in the original inbred susceptible strain. Strong attributed this to the phenomenon

of hybrid vigour. There is some indication that multiple genetic factors may determine growth-rate of neoplasms, since it has been possible to select various substrains which will grow the same transplant at significantly different rates.

The reaction of tumour tissue to a given host is relatively constant, but changes in the constitution of the tumour cell may occur. In one of his experiments, Strong (1926a) isolated two apparently identical tumours from the same mouse and injected them into a heterogeneous stock of mice. By careful selection, he was able to isolate substrains which (1) grew both tumours progressively, (2) grew the one, but not the other, (3) were resistant to both, and (4) showed a temporary tolerance to one, but grew the other. If we assume then that the one tumour was similar to the original tissue from which it arose, then the other one must have been different because it reacted differently. It is therefore justifiable to conclude that the genetic constitution of the tumour cell may deviate from the genetic constitution of the host tissue from which it arose. In one of the substrains, spontaneous regression of one of the transplants took place in all mice. The host tissue thus provided a favourable environment for the establishment, but not for the continued growth of this particular tumour. Susceptibility and progressive growth thus seem to be distinct genetical phenomena.

In another experiment, Strong (1926) transplanted a certain carcinoma into a large number of F2 mice and determined that the successful growth of the transplant depended on the simultaneous presence of about six mendelian factors. After a year of constant results, there was a significant change in results, and the above six-factor hypothesis no longer fitted. Several transplants were then selected and continued simultaneously. One gave a two-factor ratio, a second gave a one-factor ratio, a third was non-specific, and a fourth gave the same results as the original. The author concluded that a genetic change in the nature of a mutation ("a change or shift in the genetic or biological constitution of the tumour cell which is perpetuated through the process of cell division") within a single tumour cell was responsible for the changed reaction between transplant and host. This confirmed a previous conclusion, *viz.* that the tumour cell may deviate from the genetic constitution of the normal tissue from which it arose. This change was not confined to transplantability alone, but also affected the proliferative energy (growth-rate).

Conclusions.

From the data presented, certain conclusions regarding the nature of transplantable tumours can be drawn. The behaviour of the tumour tissue during the process of transplantation is apparently controlled by the biological constitution of the tumour cell. The constitution of the tumour cell is relatively constant, resulting in constant results when transplanted,

and also constant growth-rates. Notwithstanding this stability, sporadic changes in transplantability and in growth rates do sometimes occur during the process of transplantation.

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The Horn Fly (*Lyperosia Minuta*) in South Africa.

By R. DU TOIT, Onderstepoort.

Before discussing the problem in this country, it is advisable to investigate the extent to which *Lyperosia* species have been responsible for economic loss in other countries, and what measures have been adopted for their control and eradication. The countries which chiefly concern us, and into which the flies have been introduced and have succeeded in establishing themselves, are the United States of America, the Hawaiian Island group, the Philippines, and Australia.

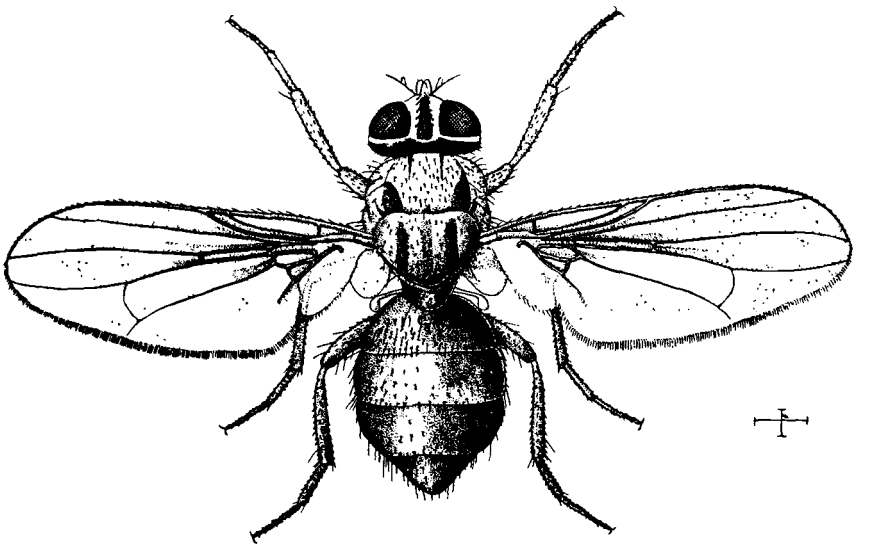
In North America, the Hawaiian Islands, and the Philippines the species concerned is *Lyperosia irritans,* commonly known as the horn fly, due to its habit of settling principally around the base of the horns of cattle. This species was originally introduced from Europe, where it has been known to exist for a very great number of years, but where its numbers have never been such as to cause any great alarm. In North America, however, the fly increased enormously and is today regarded as a serious pest of cattle, especially along the eastern sections of the country. Its distribution is patchy, however, and it shows a marked seasonal variation with more or less sporadic outbreaks, presumably due to the extreme seasonal fluctuation in temperature and humidity. Its effects upon cattle are regarded in the same light as those of *Stomoxys* (the blood-sucking stable fly) and attempts at controlling it have been limited to the use of repellant and insecticidal sprays. In the Hawaiian Islands and the Philippines the position appears to be somewhat similar, and little information likely to assist the work in this country has originated from those countries.

In Australia the position more closely resembles that in South Africa. The species involved is *Lyperosia exigua,* commonly known there as the buffalo fly, due to its marked affinity for buffaloes in the Netherlands Indies, whence it was introduced. It is worth while examining the position in Australia in some detail, because the problems of distribution and spread of the fly in that country are remarkably similar to our own.

The Australian records date back to 1825, when buffaloes were introduced from Java into Melville Island off the north shore opposite Darwin. In 1838, buffaloes were brought from this island to Darwin. on the mainland, and with them came the buffalo fly. The fly remained localized around Darwin for almost a hundred years, apparently showing little tendency to spread. It was reported on in 1912, but not until about

1925 was it recognized as a problem. By this time it had spread principally towards the west and from 1928 until about 1932 it advanced by 50 to almost 100 miles per year. Shortly after 1930 the spread in an easterly direction commenced, and by 1932 it had reached the north-western boundary of Queensland, where some of Australia's finest cattle country is to be found.

The observations of the Australian workers indicate that the buffalo fly depends for its existence on a relatively high annual rainfall and atmospheric humidity, coupled with fairly high temperatures; 20 inches of rain per annum represent about the minimum at which the fly can exist. The spread of the fly to the west and south is likely to be impeded by



The Horn Fly *Lyperosia minuta Bezzi.*
(Scale indicates natural size.)

natural barriers where climatic conditions are unfavourable to it, but there seems to be little to stop its steady advance to the east. It is generally believed in Australia that the fly is capable of spreading itself over considerable distances by its powers of flight alone, and the case is cited of the appearance of the fly on Mornington Island in the Gulf of Carpentaria which, at its nearest, is 20 miles from the mainland. Abundant evidence exists showing that the fly did not come to the island on cattle.

The work on the control of the buffalo fly has been directed chiefly towards preventing its initial spread and, subsequently, towards limiting of the numbers attacking cattle; as it is realised that little hope of total eradication exists. The danger of introducing the fly into the rich dairying

districts of the south-west by shipment of cattle by sea from the north to Perth and Freemantle has been fully appreciated, and elaborate spraying devices have been erected at the ports of embarkation, to free such animals from flies before loading. This campaign has been eminently successful, but its application appears to be limited to conditions such as these, as no repellants have yet been discovered which have a sufficiently lasting effect in preventing animals from becoming re-infested almost immediately. On the east side similar spraying devices have been installed at the railheads from which cattle are trucked to the south, but here considerably more difficulty has been experienced because of the constant danger of the cattle becoming re-infested almost at once, or on the initial stages of the journey.

Most of the work of eradication, however, has centred on the biological control of the fly. It was noted in Java and the Netherlands Indies, where the fly originated, that it never became a serious pest, presumably because it was kept in check by its natural enemies. Nieschulz, in 1928, pointed out the possible benefit to be derived from the use of small parasitic wasps (notably species of *Spalangia*) which parasitize the pupæ. He was consequently appointed by the veterinary department of Buitenzorg, at the request of the Australian Government, to investigate, and he recorded four species, one of which was particularly active. In 1929 Mackerras and Windred were sent to Java, and they made a careful survey of *Lyperosia* and its parasites, in the chain of islands, bringing the known number of species up to twelve. At this time Professor Handschin of Basle, Switzerland, was also appointed to go into the question of the biological control of *Lyperosia.* His work is interesting : he found that the Javanese species of *Spalangia* lives approximately 27 days, lays 168 to 170 eggs, each in a separate pupa of *Lyperosia,* and that the life cycle occupies 18 to 21 days. He found that, at Buitenzorg, the parasitization of pupæ in nature was about 8 per cent., but that in some areas, notably mountainous regions, it was as high as 46 per cent. Several parasites of *Lyperosia* (also *Spalangia*) were found in North Australia where the parasitization of the *Lyperosia* pupæ averaged one per cent. to 6.8 per cent. These species lived only about 15 days. Handschin then crossed the two species to increase the parasitization of the pupæ. A Javanese male *Spalangia,* crossed with a north Australian female *Spalangia,* gave a cross which lived up to 32 days and produced approximately 240 eggs. The reverse cross, however, that is, an Australian male with a Javanese female, gave a progeny which lived only 10 days and produced about 100 eggs.

A breeding station for the production of large numbers of the parasites was established in north Australia, and enormous numbers of the parasites have been liberated, over a number of seasons, in various heavily infested localities. The results have been most disappointing, and no

appreciable reduction in the numbers of the fly has been observed.

Various other natural enemies of *Lyperosia* have also been studied, with equally disappointing results. Species of *Hydrotæa,* which are believed to play a part in Europe in keeping down the numbers of *Lyperosia* were used. These are non-blood-sucking flies, the larvæ of which are predatory on those of *Lyperosia*. Various *Coleoptera* which infest dung were studied to test their ability to destroy the immature stages of *Lyperosia;* one, a staphylinid, competes vigorously with the larvæ of *Lyperosia* in the dung under experimental conditions.

Some of the observations made in Australia and in Java are worth examining in more detail, for the light they throw on the life history of *Lyperosia* and on other aspects of the problem. Very little information exists in this country on the life history of the species found here.

The Buffalo Fly, *Lyperosia exigua* de Meijere, which, incidentally, closely resembles our own species, *Lyperosia minuta* Bezzi, is a small greyish brown blood-sucking muscid about half the size of *Stomoxys calcitrans*. It remains very closely associated with its host during the course of its life, copulation taking place on the animal, and the fly only leaves to deposit eggs in the freshly dropped dung, after which it returns to its host. Under favourable conditions the eggs hatch in from 18 to 24 hours, and the larvæ, which closely resemble those of the house fly, but are considerably smaller, live in the dung for three to five days, burrowing down into the moister layers as the surface dries. The pupæ are of the usual barrel-shaped muscid type, light reddish brown in colour, and are found in the drier portions of the dung or in the ground under or around the pad. The pupal period lasts three to five days, after which the adults emerge. Under favourable conditions the whole of the life cycle may be completed in from seven to ten days, but it is considerably increased by a drop in temperature or other unfavourable climatic conditions.

It has been shown by Krygsman and Windred in Java that buffaloes exert by far the strongest attraction for the fly; next in order come zebu, then the European breeds of cattle, then horses, and to a lesser extent dogs, while in severe infestations even man may be attacked. As far as the attraction of the dung is concerned the same order holds : buffalo, zebu, cattle. The fæces of carnivora exert no attraction for the fly. Moisture in itself exerts a very strong attraction as does also a stream of warm air. The flies are positively phototropic; but the main source of attraction, so far as dung is concerned, is the smell. These two workers conducted a long series of experiments and showed that the power of attracting the flies possessed by fresh fæces of herbivores was due to amines, and that under normal conditions the dung lost all power of attracting the flies after a period of about three days. One other point which is significant, and which bears out our own observations in this

country, is that cattle are attacked in the following order, according to both the Javanese and Australian observers : old bulls, oxen, cows, and, to a considerably lesser extent, young cattle. No explanation has yet been advanced for this observation.

In South Africa the records of the identification of *Lyperosia* date back many years. As mentioned previously the species involved is *Lyperosia minuta* Bezzi, which has been recorded from Albany (Cape Province), Transvaal (?), Rhodesia, and portions of the Sudan and India. It seems as if a more or less local focus of the fly has existed in the Albany district for a great many years, not causing any alarm amongst stock owners and even going unnoticed by many. It is well known that cattle exposed to the attacks of the fly over a long period develop a tolerance, and Australian workers have also shown that a concentration of a thousand flies per beast constitutes the threshold below which cattle show practically no ill effects, but above which definite signs of irritation are manifest. Then the animals rub themselves and produce the characteristic lesions. The tolerance developed by cattle in the Albany district is probably a reason why farmers have not been very interested in the horn fly. The infestations, however, are severe; in fact, so severe that our attention was first directed to the fly by the effects produced by it on sheep. Sheep are attacked only when running with cattle, and when the concentration of flies on cattle is such that no more can be accommodated on them, and an overflow on to sheep occurs. (Sheep dung is an entirely unsuitable breeding medium for the fly.)

A gradual, almost unnoticed, spread of the horn fly has occurred during the last few years, along the coastal belt in a north-easterly direction, until in about 1935 or 1936 the fly had reached Natal. During the summer of 1937-1938 the pest had assumed rather alarming proportions in the south-western corner of Natal, and a survey of the distribution of the fly in Natal conducted during March, 1938, revealed that it had firmly established itself in the Port Shepstone and Elliot districts. The distribution, however, was somewhat patchy and confined to the valleys of the coastal belt, where humidity and temperature conditions were favourable. Advances of the fly into the adjacent areas of the Mount Currie, Umzimkulu, and Umzinto districts were observed, and localities were encountered where the fly had made its appearance, for the first time, during the present season. This clearly demonstrates that a steady spread of the fly is taking place.

Large numbers of horn flies were encountered in some of the infested herds visited, and in some cases fully 60 per cent. of the cattle showed the characteristic lesions at the bases of the horns or on the inner aspects of the ears, sides of the neck, and withers, and at either side of the root of the tail. The lesions themselves are very superficial, and consist of bright-red raw granulating surfaces which are moist to start with, due to

the exudation of serum. The average lesion is about 2.5 to 3.75 cm. in width by 6.3 to 7.6 cm. in length; but on a small proportion of the cattle lesions of up to 45 cm. in diameter are encountered. Both range and dairy cattle are attacked, the fly displaying its close association with its host in both cases, and even following the latter class of stock into the byre.

In the eastern province of the Cape Province and in the Transkei the fly has been reported all along the coastal belt as far as the southern limit of the Albany district. Reports have been received of considerable extensions inland and specimens have been received from comparatively dry areas such as the Eley Location in the Victoria East District, 70 miles from the coast.

Although there is little likelihood of the horn fly spreading inland to the arid Karroo areas, there seems to be little to stop its present advance into the remainder of the coastal belt of Natal and even Zululand; and when one considers that the fly already occurs in Rhodesia, and might be capable of adapting itself gradually to drier conditions, the position must be regarded as serious.

Although the " fly worry " produced by the horn fly, and the skin lesions resulting from the irritation and from the animals' rubbing themselves against various objects may not be regarded as being very harmful themselves, such skin lesions represent a potential site for screw worm infestation. Such infestation of wounds by maggots can very easily more than double the economic loss caused by the horn fly and can very greatly complicate ordinary farming practice, by introducing a factor which necessitates the constant attention of the cattle owner. It was noted during the survey referred to previously that the screw worm fly, *Chrysomyia bezziana,* has made its appearance in sections of the Natal south coast and although no lesions produced by the horn fly were actually found to be affected by screw worms, the possibility that such may occur must not be lost sight of.

It will be appreciated that the control of the horn fly is an extremely difficult matter. In any plan of campaign designed to control a pest of this nature certain lines of action immediately suggest themselves.

1. Destruction of the adult fly itself.
2. Destruction of the larval stages.
3. Rendering conditions unsuitable for the adult and larval stages to maintain themselves.
4. Prevention of spread.

1. *The destruction of the adult fly.*—(a) This may be attempted directly by means of some substance capable of killing the adult when brought into contact with it. The Australian workers have used lethal sprays with a very great measure of success, but such sprays are expensive and their use more or less limited to special occasions such as the shipping of cattle, *e.g.* where cattle are not immediately subjected to re-infestation.

This method would not be practicable under our ordinary farming conditions, but might have limited application in the case of dairies, where cattle could be sprayed upon entering the byre.

(b) Biological control of adults by certain predatory species of flies, *e.g.* tachinids. This has not yet been investigated but is of very doubtful value.

2. *Destruction of the larval stages.*—(a) Disposal of manure.—This is impracticable under veld conditions, where dung is dropped over large areas. It may be of some value in small dairy herds where the cattle are confined to a relatively small paddock.

(b) Biological control.—It is impossible to make any definite statement in this connection, as far as South Africa is concerned, until the natural parasites of *Lyperosia* in this country have been studied. In view of the experiences in Australia the prospects are not encouraging, nor can eradication of the fly be hoped for by this method.

3. *Rendering conditions unsuitable or unattractive for the adult and larval stages.*—So far as the adults are concerned, the only possibility appears to be the use of repellants. Up to the present no repellant has been discovered which is sufficiently lasting in effect either to mask the attractive odours of the host or directly to repel the fly. In the case of the larvæ the mere disturbance of the fæces in such a way as to allow air to enter and rapidly dry the pad is sufficient to destroy them. This method is not, however, practicable because the fæces are spread over such a wide area.

4. *Prevention of spread.*—In Australia buffer zones devoid of stock were contemplated, but in view of the spread of the fly to Mornington Island in the Gulf of Carpentaria, a distance of 20 miles over sea, it has been estimated that such zones would have to be from 40 to 50 miles wide, and this was considered impossible. The data collected in connection with the spread of the tsetse fly in this country lead one to believe that the spread of the fly by its own powers of flight, as assumed by the Australian workers, is probably not correct. It seems inconceivable that an attractive odour or stimulus could operate effectively over a distance of 20 miles of water on an insect not provided with any special sense organs, so far as is known. The probable explanation is that the flies were carried over on human beings, who are known to be attacked on occasion, and who frequently cross this stretch of water by launch. In defence of the Australian conception it must be stated that strong gales occasionally blow from the mainland and that odd flies might have been blown over; but this is doubtful.

Buffer zones may constitute a feasible means of preventing spread, provided they could be kept entirely free from all animals, and that control

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could be exerted on the movement of man in such zones. In the areas in question, however, it seems unlikely that such zones would operate satisfactorily on account of the density of population, and of the excessive amount of movement of man and animals, which would be almost impossible to control.

One other means of controlling the fly consists of trapping the adults. It has been observed that the flies remain in close association with their hosts until these enter a dipping tank when the flies leave them at the entrance, just prior to the animals' entering the actual solution. A trap has been devised which is designed to trap the flies just as they leave the animals and, if successful, should have considerable application in areas where regular dipping of livestock is practised.

A Note on Paralysis in Lambs caused apparently by *Rhipicephalus evertsi.*

By RICHARD CLARK, B.V.Sc., Bethlehem, O.F.S.

Introduction.

During September and October, 1937, many farmers reported that lambs were becoming paralysed in the hind-quarters. These reports originated in the Ficksburg district and the southern and eastern portions of Bethlehem. All these parts are mountainous. Later a report was received from the hilly eastern part of the Vrede district. Three of these outbreaks were investigated personally, the result being as follows :—

Case I (22.9.37). Three lambs and a calf were affected. Many ticks were removed from all these animals. The ticks were not identified, but were mainly *R. evertsi.* All animals subsequently recovered from the paralysis but the calf died two weeks later of what was considered to be gallsickness.

Case II (14.10.37). A paralysed lamb was brought in. All the ticks that could be found were removed and sent to Onderstepoort where they were identified as *R. evertsi.* The lamb recovered without further treatment. Fourteen similar cases were reported on this date and all recovered when the ticks were removed.

On 5.11.37 I visited the farm in question. No further outbreak had occurred since 14.10.37, but I took ticks from sheep, cattle and horses. The only species found on the sheep was *R. evertsi. B. decoloratus* and *A. megnini* were also found on the cattle. No Ixodes could be found.

Case III (5.11.37). Two partially recovered animals were seen. The owner had, on my advice, removed the ticks the previous day, when the lambs had been almost completely paralysed. All the ticks removed had been kept and were again identified at Onderstepoort as *R. evertsi*. Two other affected lambs had been left as controls and could not be found when I visited the farm. They were later reported to have been found, completely paralysed, in the veld, but recovered on being cleaned. Unfortunately the ticks gathered from these two lambs were lost.

Climatic Conditions.

The first report was received early in September and no cases have been heard of since 5.11.37. During this period it was exceedingly dry but isolated showers fell, followed by cool to cold days. Generally speaking the outbreaks appeared after these rains.

Incidence.

As previously stated, the only reports received were from hilly areas and there was never a large number of lambs affected. As a rule, three or four animals were involved in an outbreak, fourteen cases being the greatest number reported. The condition always appeared suddenly and disappeared rapidly. The period over which new cases occurred on any particular farm was never longer than five days.

R. evertsi was very prevalent on all the sheep in the affected flocks, and yet the incidence of paralysis was very low. Only sheep nine months to one year old have so far been seen suffering from this condition.

Symptoms.

The first recognizable symptom was an unsteady slow gait, with the hind feet wide apart as if to gain better balance. After about twenty-four hours the lamb would be completely paralysed in the hind-quarters. One animal, seen by me, dragged itself about its forelegs. It was interesting to note that all animals showed the paralysis of the hind-quarters, although in many cases, ticks were removed from under the shoulders as well as from the thighs, anus, etc. The day following the removal of the ticks the animals could walk, though unsteadily, and on the following day they were normal.

Post Mortem.

No deaths were reported and no post-portem examinations have been performed.

SUMMARY.

1. During September, October and November, 1937, isolated outbreaks of paralysis in lambs occurred in the districts of Ficksburg and Bethlehem. These lambs were infested with ticks.

2. The lambs recovered as soon as the ticks were removed.

3. In two cases, all the ticks removed were identified as *R. evertsi.*

4. Ixodes is not known to occur in these areas and investigation failed to reveal its presence.

Conclusion.

It is suggested that under certain conditions, *R. evertsi* may cause paralysis in young lambs. Experimental work is required to prove or disprove this idea, and will be undertaken if fresh cases can be found.

REVIEW.

The Thermometer in Veterinary Medicine.

It would appear inherent in most of us to take things for granted; we accept things as they are—our motor cars, refrigerators, microscopes and radios—and seldom, if ever, give a thought to their origin or originators. Doubtless this is as it should be. To bless MacIntosh every time we chew a toffee or don a raincoat, or to blast Dunlop every time we have a tyre puncture would be tedious. But we must not become too blasé; it is not out of place to offer up a silent prayer for the repose of Leeuwenhoek's soul when, at last, we come across that long-searched-for red cell, or to erect a mental monument to the memory of MacAdam when we strike that beautiful stretch of road after some 200 miles of corrugations and pot-holes, nor is it out of place to review a book * published 48 years ago on an instrument without which a veterinarian is as a ship without a compass.

To us, the chief interest in the book lies in the fact that between 1865 (the beginning of author's observations) and 1890 (the date of publication of the book) so little attention was paid to accurate temperature recording and so little score was placed on its significance that Armatage felt compelled to point out the use and merits of the thermometer. Although there is not a great deal that is new to us in the publication, it may keep us scientifically humble to read of the observations and struggles of a pioneer.

Armatage, a member of the R.C.V.S., first saw the thermometer used in 1865 by Gamgee who apparently was the first British veterinary surgeon to employ it on domestic animals, although it is recorded that

* *The Thermometer as an aid to diagnosis in Veterinary Medicine* by George Armatage. Second edition, revised and enlarged. Frederic Warne & Co., London and New York, 1890.

continental veterinary surgeons were measuring temperatures by this means as far back as the middle of the eighteenth century. Two types are described, the first ten inches long and protected, except at the bulb end, by a metal case, and the second five or seven inches in length, but unprotected; in both, the mercury in the bulb was separated from that in the stem (the index) by a column of air. The instrument was left in the rectum for from three to five minutes before a reading was made.

The author states that, to avoid error, various collateral agencies which have an influence on the temperature of healthy animals should be borne in mind, *e.g.* active exercise increases the temperature in direct proportion to the exertion made by the animal, exposure to cold, with inactivity, decreases it, a full meal produces a decline followed by a rise as digestion proceeds, and sudden transition from a cold to a hot place brings about an increase. The average temperature of the domestic animals, confined and at work or at liberty respectively is given—sheep 102.5, 104.5; lambs, not determined, 104.9; pigs 101.6, 103.4; bovines 100.8, 101.8; calves 100.9, 101.9; dogs 99.3, 101.2; horses 99.2, 100.6.

In disease, an abnormal elevation of temperature is invariably accompanied by an acceleration of the pulse rate and in general, an increase of about 10 pulsations is noticed for every degree rise above 98° F. (Presumably this applies to the horse.) In rinderpest, pleuro-pneumonia and foot-and-mouth disease, the thermometer records not only the intensity of the febrile action but also the *approach* of the disease. Armatage, on a visit to a farmer friend discussed the thermometer and demonstrated its use on six bovines. These proved to have temperatures between 103° and 104° F. and on this the author suggested that they were in the incubative stage of foot-and-mouth disease, prevalent at that moment in the district. On the next day, vesicles appeared on the mouth and on the teats. A horse, affected with tetanus, registered from 100° to 102° F, a dog with rabies 104.8° F. (the only record taken), a rabid calf 103° F. and a rabid ram 107° F. The temperature of a dog suffering from strychnine poisoning rose to 103° F, that of a distemper-infected dog to 104.4° F, and in parasitic pneumonia of calves 105° F was recorded. During œstrum a rise of one or two degrees takes place and an elevation is noticed for some considerable time after parturition.

In health, the natural tendency is for the temperature to rise towards evening so that a similar phenomenon is to be expected in disease. If the morning temperature is the same as that of the evening it may be inferred that the patient is growing worse. The transition from disease to health may occur slowly (recovery by lysis) or rapidly (recovery by crisis). Convalescence takes place in a regular and continuous defervescence, the earliest moment being detected by the thermometer, thus obviating the need of other and less reliable investigations. A slightly elevated temperature after apparent convalescence may be regarded as a sure sign of

incomplete recovery, although no other indication is present. Very high readings usually precede fatal issues, although just before death a considerable drop may be noticed.

The author ends his chapter on " The thermometer and prognosis " in the following strain. When once the veterinarian has learned the use of the thermometer, when he knows the " temperature form " of various diseases, he possesses a power of safe and sound judgment unequalled by, or unknown in any other means. The thermometer is not a scientific toy, but an instrument of precision, an essential companion of the practitioner, without which no clinical observation is reliable or complete.

The last chapter of the book is devoted to post-mortem temperature, local temperature and the different recording scales (Fahrenheit, centigrade and Reamur). It is interesting to note that Armatage advises the use of a special spirally shaped thermometer to record the temperature of locally inflamed areas, rather than to rely on the feeling produced when the hand is applied to the spot.

J. H. M.

OBITUARY.

Maurice Crowther Hall, B.S., A.M., Ph.D., D.V.M.

On May 1 last Dr. Maurice C. Hall died at the age of 57 following an operation for gastric ulcer. In 1936 Dr. Hall was appointed chief of the Zoological Division, National Institute of Health, U.S. Public Health Service, and before that was chief zoologist, Bureau of Animal Industry, U.S. Department of Agriculture, to which post he was promoted in 1925 following the death of Dr. B. H. Ransom.

Dr. Hall graduated in 1905 and then spent a year as post-graduate student in parasitology under Prof. H. B. Ward of the University of Nebraska. He then joined the staff of the Zoological Division of the U.S. Bureau of Agriculture and in 1915 received the Ph.D. degree of the George Washington University. His thesis dealt with the nematode parasites of rodents and is a work which today is still considered to be one of the most valuable contributions towards our knowledge of the internal parasites of this group of mammals. While preparing his thesis he also studied veterinary science and in 1916 obtained the D.V.M. degree from the same university.

While Dr. Hall's interest in parasites was at first purely that of a zoologist, he later became interested in them from a veterinary standpoint and turned his attention to researches in their treatment, prevention and

eradication. In 1921 he made the discovery for which he is best known, namely that carbon tetrachloride is an effective drug for the removal of hookworms in dogs. He was not satisfied, however, but continued his investigations on anthelminthics with the result that in 1925 he was able to announce that tetrachlorethylene was just as effective for the removal of hookworms, but had a very much higher margin of safety. First carbon tetrachloride, and later tetrachlorethylene, have become the recognized drugs for the removal of hookworms in both man and animals; millions of doses of the latter drug have already been administered to humans, and as far as is known no fatalities have occurred.

On his appointment to the U.S. Public Health Service he turned his attention to human parasites and during his last years was carrying out investigations on pinworms and trichinosis and drew attention to their widespread occurrence in the United States.

Dr. Hall was well known to parasitologists the world over and his views always merited serious consideration. Parasitologists have now lost a most valued colleague; but his memory will live in his epoch-making works.

R. J. O.
H. O. M.

NEWS.

The Association is greatly indebted to Mrs. M. Henning, wife of the late Dr. Otto Henning, for another valuable donation to our library in the form of bound volumes of old Cape of Good Hope Agricultural Journals dating from 1890 to 1895, and also of some of the earliest reports of the Veterinary Bacteriological Laboratories of the Transvaal.

The S.A.V.M.A. museum has acquired a very valuable addition as the result of a generous gesture by Mr. A. E. F. George of the Veterinary Laboratory, Grahamstown.

Mr. George has donated to the Association a costly silver plaque presented by the farmers of Albany and surrounding districts to the late Mr. William Robertson, M.R.C.V.S., in appreciation of his services rendered to that area, and also a set of photographs of the late Messrs. Robertson, Louis Pasteur, J. B. Simmonds, Duncan Hutcheon, William Hunting and of Sir John McFadyean, the first three being autographed.

These were given to Mr. George by the executors of the late Mr. Robertson, and his unselfish action in handing them over to the S.A.V.M.A. is greatly appreciated.

Drs. P. J. du Toit and J. I. Quin are at present visiting Great Britain and the Continent. They represented the Association at the Thirteenth International Veterinary Congress in Switzerland and at the Annual Congress of the National Veterinary Medical Association of Great Britain and Ireland.

Messrs. G. McIntyre and S. J. van der Walt left the Union in June to assist in the inoculation of cattle against pleuro-pneumonia in the Caprivi.

Mr. F. J. Dunning, formerly of Stellenbosch, has started private practice in Port Elizabeth.

Mr. O. T. de Villiers of the School of Agriculture, Stellenbosch, has been awarded the D.V.Sc. degree by the University of South Africa for his thesis on "The Blood of the Ostrich."

REPRINTS.—Reprints of the paper "Tick-borne Diseases" can be obtained from the Editor at 2/6 each.

The Third International Congress of Tropical Medicine and Malaria will be held at Amsterdam from the 24th September to the 1st October, 1938. Full membership will be £1. Associate membership, which entitles the holder to attend meetings and discussions, but not to receive printed reports, will be 10/-. An exceedingly interesting programme has been arranged under the chairmanship of Professor Grijns and Professor Swellengrebel. Further information can be obtained from the General Secretary, Dr. Ch. W. F. Winckel, Mauritzkade 57, Amsterdam O., Holland.

THE ASSOCIATION.

Secretary's Report for the Year ending 31st March, 1938.

Council.—Four meetings of Council were held. According to the revised Constitution ordinary members of Council will serve two years instead of one in future.

Membership.—The membership on the 31st March, 1938, was 173; an increase of ten over the previous year. Two members resigned and two were removed from the roll during the year. Fourteen new members were enrolled.

According to their occupation members may be classified as follows .

In the Union Government Service	105
,, Private Practice	34
,, Municipal Service	11
,, S.W. Africa Administration	9
,, the Protectorates	6
,, S. Rhodesia and African Colonies	5
Abroad	3
Total	173

Activities.—The first conviction under the Veterinary Act (Act No. 16 of 1933) was obtained during the year under review, and due credit must be given to one of our members for bringing the culprit to book. It is proposed to review the Act during the coming year with a view to getting certain amendments made which will render its evasion by the unscrupulous more difficult.

Council was recently reluctantly compelled to advise members against applying for a new post since the scale of salary was considered inadequate. It is, however, pleasing to state that, as the result of representations made by the Association, the salary scale was appreciably increased and the post was re-advertised. It is very gratifying to report that we received the fullest support from the National Veterinary Medical Association of Great Britain, and from the Associations in the African Colonies. Without their full co-operation success would probably not have been obtained.

Our Association on the other hand was called upon to assist the other African Associations in their endeavour to get certain anomalies in the conditions of the Colonial Service removed. They were accorded our full support, and it is hoped that their endeavours will meet with success.

The admission of animals to Onderstepoort for treatment was considered by Council, and the matter was taken up with the authorities concerned. While no finality has yet been reached, we have the assurance that this question is receiving very serious attention and it is anticipated that it will be solved in a manner which will afford ample safeguard for the interests of both private practitioners and the Faculty of Veterinary Science.

General Meeting.—Our greatest progress in the past year is in the manner of conducting the General Meeting. The improvements were the extension of the meeting to three days, the providing of members, prior to the meeting, with synopses of the papers to be read, and the appointment of openers for the discussion. The result was a thorough and interesting discussion on every paper.

Finance.—The books were duly audited at the close of the financial year and the various statements are appended.

According to the cash statement our income exceeded expenditure by £37 : 10 : 3.

To comply with Income Tax requirements our holdings in Union Loan Certificates had to be reduced to 800 units. This involved withdrawing £410 : 5 : 8 (£382 : 8 capital and £27 : 17 : 8 interest) from Union Loan Certificates. Of this sum £250 was placed on fixed deposit with the United Building Society and the balance of £160 : 5 : 8 was transferred to the Benevolent Fund.

The Benevolent Fund increased from £118 : 8 to £330 : 1 : 4, having benefitted by the transfer of the sum referred to above, the allocation from the General Fund, the profits derived from the Group Insurance Fund, and by donations and interest. No calls were made on this fund during the year, but several deserving cases will have to be considered in the near future and the need for further strengthening of the fund is as urgent as ever.

The Group Insurance Fund showed a profit of £14 : 17 : 8, an increase of £4 : 13 : 1 over the previous year. This profit will be transferred to the Benevolent Fund. Members contemplating taking out Endowment Insurance policies with the S.A. Mutual are again reminded of the facilities offered by this scheme, which ensure an appreciable saving to the member and at the same time helps to swell the Benevolent Fund.

Thanks and appreciation are due to the President, Vice-President, and members of Council, and to the various Committees for their guidance and help during the year; and particularly to Messrs. van der Wath and De Boom, who have devoted a considerable portion of their leisure time to the administration of the Group Insurance and Book Funds respectively.

S. W. J. van Rensburg,
HON. SECRETARY-TREASURER, S.A.V.M.A.

Report of the Finance Committee for 1937–38.

Members of Committee : C. J. van Heerden, B. S. Parkin, S. W. J. van Rensburg, J. G. van der Wath.

A. Cash Statement of Income and Expenditure, 1.4.37–31.3.38.

Receipts.				*Expenditure.*			
Credit Balance on 1.4.37	£53	12	7	Printing and Stationery	£275	5	3
Subscriptions	361	3	9	To Benevolent Fund	201	16	8
Donations : Benevolent Fund	1	1	6	Placed on fixed deposit :			
Union Loan Certificates	382	8	0	U.B.S.	250	0	0
Interest on U.L.C.	27	17	8	Book Fund	75	13	2
Advertising	37	17	8	Clerical Assistance	32	10	0
Sale of Journals	14	1	7	Petty Cash	25	0	0
Sale of Reprints	0	19	10	Annual Dinner	31	8	0
Donation towards Publications	10	0	0	Lunches	6	16	6
Annual Dinner	30	19	6	Sundries, General Meeting	4	3	0
Lunches	8	18	6	Presentation	5	2	6
Insurance Account	0	9	0	Auditing	2	2	0
Book Fund	80	1	2	Natal Branch	5	0	0
Erroneous Credit	3	4	10	Wreath	1	0	0
				Legal Advice	0	10	6
				Bank Charges	5	3	8
				Credit Balance on 31.3.38	91	2	10
	£1,012	15	7		£1,012	15	7

B. Balance Sheet, 1937-38.

Assets.				*Liabilities.*			
Union Loan Certificates	£800	0	0	Publishers (Journal)	£45	8	0
United Building Society	250	0	0	Reprints	17	10	0
Arrear Subscriptions	93	4	0	Natal Branch	15	9	6
Cr. Balance in Bank	91	2	10	Book Fund	4	8	0
Cash in hand	2	10	2	Insurance Account	3	4	10
				Credit Balance	1,150	16	8
	£1,236	17	0		£1,236	17	0

C. Benevolent Fund, 1937-38.

Credit Balance on 1.4.37	£115	8	0	Expenses	NIL		
From Cash Account	201	16	8	Credit Balance, 31.3.38	£330	1	4
From Insurance Account	10	4	7				
Interest	2	12	1				
	£330	1	4		£330	1	4

D. Group Endowment Insurance Account, 1937-38.

Receipts.				*Expenditure.*			
Credit Balance, 1.4.37	£15	9	3	Premiums Paid to S.A. Mutual	£385	5	7
Premiums Collected	397	13	6	To General Account	0	9	0
				To Benevolent Fund	10	4	7
				Bank Charges	2	5	11
				Credit Balance on 31.3.38	14	17	8
	£413	2	9		£413	2	9

S. W. J. van Rensburg,
Hon. Secretary-Treasurer, S.A.V.M.A.

Editorial Report for 1937.

Members of Editorial Committee : P. J. du Toit, A. D. Thomas, J. H. Mason, C. Jackson, M. Sterne.

Statement.

To Advertisements	£37	17	8	By printing charges No. 1	£31	10	0
,, Sale of Journals	14	1	7	,, ,, ,, No. 2	72	7	6
,, Sale of Reprints	0	19	10	,, ,, ,, No. 3	65	2	0
,, Donation	10	0	0	,, ,, ,, No. 4	39	18	0
				,, Reprints	29	2	6
				,, Blocks	19	8	6
				,, Envelopes	4	10	0
				,, Postage	6	0	0
					£267	18	6
				Revenue	62	19	1
	£62	19	1	Actual charges on Association funds	£204	19	5

Circulation.

Copies.		*Sundry Copies.*	
Members	687	Current and Back Nos.	48
Cadet Members	92	Advertisers' Vouchers	33
Exchange	240	Specimen and Complimentary	56
Subscribers	164	Book Review Vouchers	1
Free Mailing List	32		
	1,215		138

The gross expenditure on the Journal was more than in 1936, owing mainly to the cost of blocks for Dr. Curzon's history of the Army Veterinary Service in South Africa. The grant from the Association Fund was not, however, exceeded, because of the generosity of certain members who contributed to the cost of reproducing the many very interesting photographs collected by Dr. Curson.

Dr. Jackson, to the great regret of the Editorial Committee, resigned the Editorship at the end of the year. However, he consented to serve on the Committee where his assistance and advice have been of inestimable value.

For the Editorial Committee,

(Sgd.) *M. Sterne,*
EDITOR, *Jl. S.A.V.M.A.*

Report of the Hon. Librarian for the year ending 31st March, 1938.

The Librarian has nothing special to report.

(Sgd.) *Cecil Jackson,*
HON. LIBRARIAN.

The Veterinary Services and Animal Husbandry in the Phillipines.*

By Dr. ESTEFANO C. FARINAS, Superintendent, Alabong Stock Farm, Bureau of Animal Industry, Philippine Islands.

In discussing the subject of the Veterinary Services and Animal Husbandry in the Philippine Islands it will be convenient to do so under several headings.

THE PHILIPPINE BUREAU OF ANIMAL HUSBANDRY.

This body is one of the several bureaux composing the Philippine Department of Agriculture and Commerce. It was organized in 1930 from the veterinary and animal husbandry divisions of the defunct Bureau of Agriculture when it was converted into two bureaux. The chiefs of Bureaux are called directors. The Bureau of Animal Industry comprises five divisions, namely, Animal Disease Control Division, Veterinary Research Division, Animal Husbandry Division, Animal Products Division and Administration Division. The 1938 budget included an appropriation for the organization of an Extension Division which may by this time make the sixth division.

Research Work.

One of the main functions of the Bureau of Animal Industry is research. In fact, we much depend upon the results of such research in furthering our work and for the basis of information given to the public.

The Division of Animal Products undertakes the study of the properties of meat, milk, eggs and other animal products, and the different methods of processing them and conserving them for human food. Thus hams, bacon, sausages, cheese, butter, pickled meats, canned cooked meats, condensed, dried and evaporated milk, and other meat and milk products are prepared. Exhibits and sale of these products are made. When perfected, publications on the methods of manufacture are issued for the benefit of the public.

The Veterinary Research Division is in charge of the study of infections and parasitic diseases, their methods of control or eradication, and

* This paper by Dr. Farinas formed the subject of an address given by him to the students at Onderstepoort on the evening of April 26th, 1937, at the Students' Hostel. At my request he was kind enough to give me the script in full and it is published as written by him with a few minor corrections. We are deeply indebted to Dr. Farinas for the trouble he has gone to in writing this paper, which gives us a very good insight into the valuable work done by the veterinarian in the Philippine Islands.

E. M. R.

other matters which fall under veterinary science. It began as a small rinderpest vaccine laboratory and quarantine station not many years ago; it has grown rapidly in the last few years and today it is composed of a parasitology section, a serum and vaccine section, a serology and immunology section, a virus and bacteriological section, and a laboratory diagnosis section. More of course is to be desired, yet this laboratory is efficiently solving our immediate veterinary problems. Hand in hand with the animal disease control division we have been able to put under satisfactory control outbreaks of animal pests.

The Animal Husbandry Division undertakes breeding, feeding, dairy, poultry, ranching, and other animal mangement researches, aside from raising pure-bred animals for stud purposes. It also studies the adaptability of foreign breeds introduced into the country, and so with exotic grasses and forage.

Veterinary Services.

The veterinary services are undertaken by the veterinary research laboratory and the animal disease control division together. The laboratory makes studies of the nature, prevention, control or eradication measures of all new or uncommon animal pests; prepares vaccines or sera for prophylactic or curative purposes, and maintains a laboratory diagnosis service for both private persons and the field personnel. It also makes trips to infected areas to study the field behaviour of new, uncommon or atypical outbreaks. The animal disease control division maintains a large staff of veterinarians composed of a chief, an assistant chief, supervising veterinarians, assistant veterinarians, and numerous livestock inspectors. The country is divided into 15 veterinary districts, each district embracing two or more provinces; there being 48 provinces. The duties of the division are (1) to perform meat and dairy inspection, (2) to investigate, control, or eradicate infectious diseases, (3) to prevent the entrance, from overseas, and the spread of animal diseases by regulating the traffic of livestock, and (4) to help farmers by treating their sick animals, service free, where no private practitioners are stationed and by giving them advice as to the proper breeding, feeding, keeping and management of livestock, including the selection and castration of undesireable bulls. The field veterinarians are therefore required not only to have a good knowledge of infectious diseases, but also to be well armed with clinical skill, and solidly posted in animal husbandry.

A new graduate veterinarian who receives an appointment in the animal disease control division is first given practical training, in meat inspection, in laboratory technique, and in animal husbandry work, under experienced men before he is sent out to the field; unless he is urgently needed in the suppression of a severe outbreak, when he is sent to the field outright and receives further training later. It is also our practice

to bring to Manila field men, by turns, to break the monotony of provincial life, to inform them of new policies, and to acquaint them with the latest practices in veterinary science and animal husbandry. Each field veterinarian or personnel is like a soldier who must be always ready for a change of station at a moment's notice. Therefore he is expressly expected to have cash on hand to defray all bills when he leaves his station, and to finance his trips to a new assignment. He must not be encumbered by many household effects. This military-like organization is necessary to be able to combat animal diseases with strategy. The long fight we had against rinderpest gave us a great deal of experience, and sharpened out wits, and enabled us to deal successfully with the aristocrats as well as with the ignorant and yet elusive population who neither realized the good nor believed in the measures we instituted. We have also realized the un-uniform and sometimes queer behaviour of epizootics and have learnt the best way of using crude vaccines, and of enforcing the strict quarantine rules we had at first. These difficulties stimulated the thought for organization, discipline, and direction of men suitable for such work. In making recent appointments, we not only took into consideration their scholastic records and civil service ratings, but also sized up the men as to their physical fitness, their temperament, and their possible endurance to such privations as they might meet in the field in prolonged campaigns. The veterinarian fit for service in the animal disease control division must then be intelligent, tactful, skilful, sociable, strong, brave, hardy, and must possess ability to meet all classes of people and to treat poor and rich alike. Philippine conditions are most varied — both the people and the country!

1. *Meat Inspection.*

This service has not been well organized due to the absence of laws to enforce it. Meat inspection, except in the city of Manila and the large capitals, was in the hands of the Bureau of Health personnel, who based their judgment on simple rules and regulations furnished by the Bureau of Animal Industry. Bureau of Animal Industry veterinarians and inspectors did the work in Manila and supervised it in some of the cities and larger municipalities; in these cases the U.S. Federal Meat Inspection regulations are followed. Last year a law was passed which requires cities and municipalities to employ qualified veterinarians or livestock inspectors to conduct meat inspection therein. Such personnel are to be paid from funds obtained from a meat levy on carcasses of animals slaughtered in the respective municipalities or cities, and are to work under the control and supervision of the meat inspection section of the animal disease control division. As this service is being organized, a set of rules and regulations with standards similar to those of the United States is being formulated. After the carcasses are passed and stamped, regulation of such meat falls under the Board of Food Inspection and the Bureau of Health.

With regard to the meat trade, I may state that cattle-men and other livestock owners depend upon Manila as their principal market. Due to the manipulation by a ring of butchers, prices of cattle or hogs on the hoof can be so low as to give the farmer little or no profit. In order to solve this sordid state of affairs, the Bureau of Animal Industry is pushing a scheme in the form of livestock exchange in which livestock are received direct from the farmers and therein slaughtered. Carcasses are stored in refrigerator rooms and released, either at fixed prices depending upon grade or class, or by auction on a cash basis. This will do away also with bad debts, and the livestock raiser will get exactly what his animal is worth. The by-products of the establishment will be converted into useful commodities such as meat-meal, bone-meal, dripping, glue, neatsfoot oil, etc., instead of cremating them as at present.

2. *Enforcement of the Pure Food Law.*

In the United States Federal Service, this matter is in the hands of the Pure Food Administration, while in the U.S. Army, food inspection is in the hands of the veterinary corps of the medical department. While the hygiene and sanitation of foods offered for sale lies within the jurisdiction of the Bureau of Health, the pure foods law is enforced by a Board of Food Inspection, the membership of which is composed of one representative from the Bureau of Customs, one from the Bureau of Internal Revenue, one from the Bureau of Science, one from the Bureau of Animal Industry and one, the Chairman, from the Bureau of Health. Samples from all imported foods, food colours, liquors, wines and spirits, or articles that go into the preparation of food, are collected by agents of the Bureau of Health for analysis for purity in the Bureau of Science. Offenders against the pure food law are summoned to a hearing by the board meeting once a week, and those who, in the opinion of the board, are guilty are prosecuted in court. Warnings and instructions are given to many offenders.

3. *Dairy Inspection.*

While milk sold falls under the board of food inspection, the certification of health of the dairy animals depends upon Bureau veterinarians who are the only ones qualified to perform such biological tests as are necessary to eliminate animals suffering from dangerous communicable diseases. Tuberculin testing is compulsory in all dairy establishments. All milk from dairy buffaloes in rural districts where T.B. testing is not performed when offered for sale is first boiled at Bureau of Health milk stations, then bottled, and bears the Government seal.

Tuberculin testing of dairy cows was begun in 1927, and naturally opposition was offered in some quarters but then the Government made a determined effort to protect the health of milk consumers, and we had to perform the tests. Reactors were very common; so bad was the infection

in two dairy farms in Manila that practically all animals had to be destroyed, and thus automatically effected the closing of these dairies. During the first years, tuberculin testing was done twice a year until reactors were nil, when testing was done once a year. Heavily infected farms took several years to clear out. No Government compensation is given to owners of animals so destroyed, but slaughter in the abattoirs is allowed, and carcasses are passed or condemned in accordance with meat inspection regulations.

4. *Suppression and eradication of animal diseases.*

In order that you may have an idea of the livestock wealth of the Philippine Islands which needs veterinary protection, the report of the of the Division of Statistics, Department of Agriculture and Commerce, for the year 1935 is quoted as follows : Caraboos (water buffaloes), 2,272,319; cattle, 1,480,260; hogs, 3,018,758; horses, 400,250; goats, 518,813; and sheep, 140,041. Ducks, turkeys, geese and poultry number millions.

a. *Rinderpest.*—The eradication of rinderpest from the Philippines without resorting to such drastic measures as slaughtering affected and exposed animals is to our mind the greatest veterinary achievement of my country. Rinderpest was found by the Americans when they first landed in the Philippines in 1898. It must have been introduced from China or French Indo-China, where the pest has been in existence for centuries. For many years it decimated our work animals to the extent of seriously affecting agriculture and industry. We had to import both work and beef cattle to supply our needs. It is on account of this disease that the Bureau of Agriculture was organized; also a veterinary college of the University of the Philippines was established to prepare young men to fight the disease. I must not omit to mention that the first veterinarians who fought the scourge were Americans and we are grateful for what they have done. From them we received our training.

The first fight against rinderpest was a great national romance. It was a difficult, expensive job, and it taxed not only our financial resources, but the brain and genius of the government as well. Battalions of soldiers were employed and a kind of terror on the part of the people was created. So unpopular were the veterinarians that they were chased with *bolos* by the people who wanted to use their animals, but could not on account of the rigid quarantine rules. We had to counteract the suspicion of the ignorant people who charged the government personnel with spreading the disease to maintain their jobs. This ill-feeling stayed in their hearts until rinderpest was no more. Then confidence in the Government prevailed. During the last few years, liberal moral and financial support was given the Bureau of Animal Industry by the Philippine Legislature and the National Assembly.

But in order to win we had to make superhuman efforts — we had to clean the lowlands first, and when the disease could not move coastward against a solid wall of immune animals, it had to advance to the wild interior. We had to chase the infection, encircle it and smother it if we could, only to find it leak out somehow from our control. We chased it further in. To do this we had to cross swollen and crocodile infested rivers, work among wild and fierce tribes, get lost in deep forests, and at the same time carry the vaccine and ice on our shoulders. We had to live on roots and mealies and wild game, camping as we did in the interior. Thus we carried on — and, when the diseases could hardly be found, we continued stationing scouting parties in and around the infected and suspicious areas for years, until we were satisfied that the disease was gone for good.

We are now practically through with the disease, and we attribute our success to the continuous improvement of the rinderpest vaccine, to the proper application of the imperfect product, to the tact and genius of the men who directed the campaigns, to liberal financial support, and to the endurance, sacrifice, loyalty, perseverance and skill of the field forces at the fronts of combat. The rinderpest vaccine was perfected by greatly reducing the dosage, by increasing its potency and keeping qualities, and by doing away with ice. Another factor which contributed to its eradication was the banning of importation of live animals, except for breeding purposes, and the rigid control of animal movements.

b. *Foot-and-Mouth Disease.*—This malady was introduced into the Philippines through work and beef animals from the neighbouring countries. There was a time when the disease was rampant throughout the islands. It is now localized in a few provinces, but has apparently become endemic in the cattle province of Masbate in the Visayan Group and in Bukidmon in the island of Mindanao. The disease is so mild in its manifestation that ordinarily it could not be recognized in infected herds by casual inspections. We usually discover outbreaks in the abattoirs, in the form of very insignificant lesions found in the tongue, lips and feet. When such a case is encountered, a thorough scouting is then made in the province of origin of the first cases, and this invariably results in the discovery of mild infection. At once such a province is placed under quarantine, and in that case under no circumstances are cattle allowed to be removed from that province unless accompanied by permits issued by authorized Bureau representatives. Slaughter-out methods have not been resorted to. We just allow the disease to burn itself out and keep the infected area under strict surveillance. Under the system we have kept the outbreaks within safe limits.

We consider foot-and-mouth disease important although it is not a fatal disease, because it hits the business aspect of dairying tremendously, and curtails agricultural operations by laying off affected animals.

c. *Anthrax.*—Anthrax is endemic in certain localities. In 1923, a very severe outbreak of anthrax swept Central Lyzan, killing thousands and thousands of livestock and involving people. Control used to be done by the simultaneous inoculation of serum and vaccine. Recently our veterinary research laboratory put out a spore vaccine, first used intracutaneously, but later on subcutaneously. Result of vaccination has been very satisfactory.

d. *Rabies.*—Rabies is common throughout the islands. Dogs, cattle, horses, caraboos and hogs are affected. Rabies vaccine, manufactured with phenol, has been employed, effectively, in the control of field outbreaks and for the prophylactic treatment of dogs in the cities.

e. *Tuberculosis.*—Tuberculosis is common in human beings and I am sorry to state the toll from this plague in normal years outnumbers that from all other diseases combined. Abattoir findings with regard to range cattle show that T.B. is rare in this species, but very high in hogs. Research on T.B. in hogs in the Philippines disclosed that T.B. in hogs is the human type.

f. *Contagious abortion.*—It is a recent discovery. First it was found in our hog project in the Alabang Stock Farm, and later in established dairy farms of imported stock. Of late serologically positive cases of undulant fever have been reported among human beings in hospital; these patients have been closely associated with the handling and slaughter of hogs.

g. *Pasteurellosis.*—Hæmorrhagic septicemia is one of the most important pests affecting caraboos, cattle, horses, pigs, and poultry in the Philippine Islands. It is a disease which sometimes behaves like rinderpest and sometimes like anthrax. The causal organism is not easy to isolate from an affected carcass — one has to make massive cultures of heart blood in broth to isolate the micro-organism for purposes of diagnosis. In poultry it is not difficult to isolate the organism. Control is done by the use of vaccines prepared from killed broth cultures.

h. *Surra.*—Surra, a trypanosome disease, is the most serious malady affecting horses in the Philippines. Caraboos, cattle, and dogs are also affected, but in the first two animals, infection is normally mild and spontaneous recovery occurs, except when they are over-worked in which case they also succumb. We lose hundreds of horses from this disease every year. Affected horses, when found, are destroyed.

Recent work by our Bureau showed that the infection can be produced by the mechanical injection of a single trypanosome, so this decided the point that no intermediate development in hosts is necessary for transmission. Medical treatment of artificial cases has been tried extensively in the Philippines, using Naganol, ethersenol, tartar emetic, antimony, atoxyl, and their combinations, by intravenous, intrathecal and other

routes. Encouraging results were obtained. At present natural cases have been successfully cured, as tested by complement fixation and inoculation. What success will be achieved I cannot tell at the moment.

i. *Hog Cholera.*—Swine fever kills more hogs in the Philippines than all other diseases combined. There have been no campaigns for its eradication, but it is in the list of those to be eliminated. Pork, hams and sausages form such an important part of the Philippine diet that swine raising has to be encouraged to the fullest extent. Control is carried out by the simultaneous injection of hog cholera serum and virus. The production of vaccine has been tried for a number of years and the last batches made are very encouraging.

j. *Fowl diseases.*—Avian pest (Newcastle disease), infectious roup, and fowl cholera are the most important. Avian pest was introduced in 1927. First discovered in Manila, it has spread as far north as the Cajayan valley and south to the Visayan Islands. Localization of the disease has not been possible on account of the lack of control of the movements of table poultry and fighting cocks. Research has been going on for the last ten years in search of a vaccine, but success has not been forthcoming. Field control of outbreaks is however simple, due to the fragile nature of the virus.

Animal Husbandry.

For many years, the animal husbandry work of the Government was not given proper attention. Although valuable livestock has been imported time and again, breeding methods have not been systematically done, probably due to lack of adequate knowledge, shortage of experience, and absence of the right men to organize work. Rinderpest control had been so absorbing of men and resources that not much could be spared for livestock improvement. Further it was argued : " Of what use would be good animals if rinderpest would just wipe them out ? "

When rinderpest subsided, impetus in animal improvement was keenly felt and, as a result, a reorganization of the animal husbandry division was determined and promptly effected. A new chief was assigned and lavish support was given. The policy of using only pure bred animals for breeding was adhered to in all cases and a lot of culling and revision of breeding plans were carried out until the old mess was straightened out. Now we are pushing a definite program of improvement of the national herds by means of Government stock farms, breeding stations, and extensive service in animal husbandry.

Horse breeding.—A haphazard system of horse breeding was cast; in its place a definite goal was aimed at by following definite lines of breeding. We imported Arab stallions, one batch after another, and used these for grading up native mares, and mated them with the grade mares which were produced from early importations. We not only use these

Arab stallions in our stock farms and breeding stations, but have many of them distributed to the different provinces for both natural and artificial breeding. Since 1934, we practised artificial insemination and already have many offspring as a result thereof. We are now employing the most up-to-date methods of artificial insemination in horses as well as in cattle, sheep, and goats. By the use of diluting fluids we can inseminate a large number of animals per ejaculation and by it too we can maintain the potency of sperms for comparatively long periods. Clinical methods of diagnosing pregnancy have been improved and have been employed as a routine, not only in the breeding stations to aid in ensuring pregnancy, but also among privately owned mares. Of course, serological or biological methods of pregnancy tests are used as well.

Filipinos are lovers of sports. Horse racing and polo are indulged in. In Manila there are two large, costly and up-to-date race tracks and two polo clubs. We are also building up a strong Philippine Army. It is our aim to help farmers and breeders to rear the horses in the Philippines for these institutions, as all of them need good horses. To increase the size of the Arab grades, we bore in mind the use of Thoroughbreds later. There are no signs of degeneration of the high grades; blending of the Arab and the native is very satisfactory.

Cattle breeding.—Previous to 1909 the erstwhile Bureau of Agriculture imported and tested out Devons, Galloways, Aberdeen Angus, Herefords and other British beef breeds to improve the native cattle on account of their small size. These imported animals did not do well; as a matter of fact, all of them perished and their blood could not even be traced in the animals reared in the areas to which they were put as sires. In line with the cattle breeding activities of other tropical countries, the Government imported in 1909 a number of Indian Nellore (Zebu) bulls and cows—first prizes in the shows in Madras Presidency, British India. They formed the nucleus of a pure bred Nellore herd from which bulls were obtained for public breeding in the different parts of the Philippine Islands. Due to the larger size and good conformation of the first crosses, cattle men became interested and bought Nellore bulls from the Government as fast as we could produce them. In that manner, the whole country used this breed to upgrade their stock, and today all cattle in the ranches are practically all grades of the Nellore in a higher or lower degree. The Government is still maintaining a large herd of pure Nellore—the finest in the country.

The Nellore and its grades are not ideal beef cattle although they are excellent draft animals. They are rangy, deep, but flat, lack sufficient cover, and of just fair beef quality. For this reason, we are not satisfied. Yet, we cannot depend upon the European beef breeds on account of our experience in the past. So learning about the Afrikander as a good beef breed, my Government decided to put it to a trial, hence my presence in

South Africa. Of course, we are also trying the crosses between the Sussex and the Zebu, but the bulls are kept in the stables. What we want are bulls to let loose on the veld. Brown Swiss bulls on Zebu cows have given encouraging results.

For dairying, the European breeds seem to do well under stable conditions; yet, we are after dairy breeds not for commercial dairying, but for the peasants to keep at their backyards so they could have milk for their daily needs. There is so much truth in the talk of undernourishment of the Filipinos, and the Government wants to teach the masses to drink more milk to improve their diet. The Red Scindi from British India, another Zebu, has been tried in fairly large numbers and they have a great possibility for that purpose, since they are easy to handle and to keep, they are hardy, and are fair milk producers.

Since 1921, we have been trying to increase the hardiness of the Ayrshire by grading up to purity the Zebu breed. We have obtained 3/4 cows and 7/8 heifers and 15/16 calves, but, like your upgrading work in cattle, there are sure signs of degeneration from the 3/4 up. We will, however, continue the work to the finish and see what it brings in the end.

Among the native caraboos (water buffaloes) there are some outstanding milkers which produce up to about 2 gallons. The milk is very rich in butterfat, averaging 10 per cent. Plenty of caraboo milk is sold in Manila. Cheese is also manufactured by farmers from caraboos milk and it is a very rich cheese indeed! The Bureau of Animal Industry has just proceeded in selecting about 20 of the best obtainable milk caraboo strains to begin intensifying the milk producing capacity with a view to producing a caraboo dairy herd.

Sheep and goat breeding.—The Philippines is not a mutton or wool country. The few thousand sheep we have, have been introduced by the Spaniards and must be neglected merinos. I am bringing home plain-bodied, large type merinos from the "Karroo," both for improving what we have, and to try to form the nucleus of a mutton and wool industry. (This idea is mine.) As to goat raising, the Bureau has a waiting list of about a metre long of orders for milk goats. We cannot supply the demand. I am therefore bringing home with me about ten British Alpine and Saanens for this purpose. We are raising Anglo-Nubians and Indian goats.

Swine breeding.—Practically all breeds of hogs obtainable from the United States have been thoroughly tried. Western breeds of hogs could be successfully raised in the Philippines, but there are certain breeds that are outstandingly suitable and are preferred by the farmers. These are Black Berkshire, the Black and Spotted Poland China, and the Duroc Jersey, in the order named. The Government is propagating these four breeds either for pure breeding or for crossing purposes.

Poultry.—Practically all breeds of poultry have been imported from the United States and Australia. In the last analysis, we decided to keep only three of these breeds, namely, the white Leghorn, the Rhode Island Red, and the Barred Plymouth Rock, as they are the most profitable to keep. Ultimately we will also discard the Barred Plymouth Rock. We advocate raising all of these breeds pure.

THE RÔLE OF THE VETERINARIAN IN ANIMAL HUSBANDRY.

The veterinary profession is looked upon as the standby of animal husbandry in the Philippines. The Bureau of Animal Industry, as I have indicated, has drawn many veterinarians from the animal disease control division to handle the animal production phase of animal industry in the archipelago, and will employ veterinary graduates, whenever possible, to continue the Government programme of breeding animals for stud purposes for the country. Should we fail, then we may consider that it will mean the failure of the veterinary profession in the Philippines. It is up to us, therefore, as veterinarians, to prove ourselves capable of not only protecting animals from disease, but also producing pedigree stock, of raising productive animals, and of showing how to farm them.

Jl. S.A.V.M.A.
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Rickettsiosis in the Dog.

By W. O. NEITZ and A. D. THOMAS, Onderstepoort.

Introduction.

In 1932, while touring in the Kruger National Park, the writers heard from the Warden and from the Rangers that it was practically impossible to keep dogs for any length of time in the southern portion of the Park, as all died from a disease against which the usual treatment for biliary fever and distemper seemed to be of no avail. It was also suggested then that the great reduction in numbers of wild dogs (*Lycaon pictus*) noted in recent years might be caused by the same or a similar disease. Unfortunately at the time there were no cases available for examination.

Mention is made in the Warden's annual reports for 1933, and subsequent years, of the unaccountable disappearance of most of the wild dogs from sections south of the Olifants River. The following interesting remarks referring to wild dogs are quoted from the report for 1937 :—

> 'With the exception of a small troop which appears from time to time along the Crocodile River, there seem to be no Wild Dogs in the Park between it and the Olifants, and relatively few north of the latter. It is now over two years since the last one was seen at Skukuza, where at the time of the impala lambing season they used to be constantly in evidence. The type of sickness to which they have certainly fallen victims during the last 6—7 years, and which I believe is still attacking them and becoming endemic, is probably akin or identical with the very fatal and novel type of dog disease which killed nearly all the domestic dogs some ten years ago, when it appeared in epidemic form and which is still endemic amongst them. I have not personally seen a wild dog dead of sickness, but natives who say they have, describe the symptoms as similar to those which the domestic dogs showed.'

It was not until the beginning of this year (February, 1938) that an opportunity of examining such a case and subinoculating blood from it presented itself.

Two dogs (a bitch and her pup) were take down from Onderstepoort to Skukuza and received intravenously and subcutaneously 20 cc. and 10 cc. of citrated blood respectively.

The ailing dog, a bull terrier-ridgeback cross, two years old, was very emaciated, although its appetite was said to be good. Rectal temperature was 101.6° F; the appearance was dejected and listless. The eyes were sunken, conjunctiva dirty pink with a sticky muco-purulent discharge. The mouth had a fœtid odour, the teeth being covered with a brownish, slimy substance. There were numerous sores on the skin of

the neck, back, and cheeks partly healing and covered with scabs. As it was desired to attempt further treatment, no post-mortem examination was possible. The subinoculated dogs were brought back to Onderstepoort the same day and, after an incubation period of nine days, both developed a high fever which was not due to *Piroplasma canis*. After another ten days, during which the animals were visibly loosing condition, a second rise in temperature was found to be accompanied by *Piroplasma canis* in the blood. Immediate treatment with acaprin caused the parasites to disappear from the blood but did not prevent the dogs from dying on the 30th and the 31st day respectively. In the organ smears from these animals the presence of a rickettsia corresponding to the description of *Rickettsia canis*, Donatien and Lestoquard (1935) was recognized. This observation was reported to the Biological Society of Pretoria on the 30th June, 1938.

Aetiology.

Two distinct species of *Rickettsiæ* have been described in the dog in Africa. In the Mediterranean region a disease in man caused by *R. conori* was described by Conor and Brugh (1910) under the name "fièvre boutonneuse." Durand (1932) set up an inapparent disease in the dog with "fièvre boutonneuse" virus and recovered the virus from the blood ten days later. The second species *R. canis* was decribed by Donatien and Lestoquard (1935) in dogs exposed to tick infestation in Algeria. The cross-immunity experiments carried out by Donatien and Lestoquard (1936) have shown that these parasites are distinct and different. Whereas *R. conori* produces a mild febrile reaction in dogs, *R. canis* causes a severe febrile reaction and clinical symptoms which frequently result in the death of the animals. Both diseases are transmitted by the dog tick *Rhipicephalus sanguineus*. A further important difference between these parasites is that *R. conori* develops in the endothelial cells of the blood vessels and peritoneum, while *R. canis* parasitizes the monocytes and neutrophiles. The latter parasites, when stained with Giemsa, appear as a collection or colony of purple-coloured coccoid granules. A parasitized cell may contain 1–12 colonies varying from 2–10μ in size. The individual granules vary from 0.5–1.5μ. Some colonies consist of small granules only, others of large granules, and then again large and small granules may be found in one colony. It is somewhat difficult to demonstrate the organisms in blood smears, but relatively easier to find the parasitized cells in organ smears.

Transmission.

Infection may be transmitted artificially from dog to dog by the subcutaneous or intravenous subinoculation of blood or emulsified organs.

Little is known about the natural mode of transmission of *R. canis*, but Donatien and Lestoquard (1935, 1936) were able to transmit the disease with adults and larvæ of *Rh. sanguineus* bred from infected adults.

Pathogenicity.

All breeds of dogs are apparently equally susceptible. Donatien and Lestoquard (1935, 1936) were able to infect monkeys (Macacus). The only wild carnivorous animal, a jackal (*Thos mesomelas*), available to us was infected by intravenous injection, but developed no symptoms, although it was shown by subsequent subinoculations to harbour the parasite for a long period.

Pathogenesis.

Examination of the blood has shown that anæmia is produced. A detailed study of the blood was not attempted, because biliary fever proved such a common complicating factor. Our preliminary observations are insufficient to enable us even to suggest what damage is produced and how death is brought about.

SYMPTOMS.

In dogs exposed to tick infestation, Donatien and Lestoquard (1935) record that death may occur as early as the 15th day. In our experience the period of incubation following artificial infection is 8–16 days. The first symptom noticed is a distinct febrile reaction which may be continuous for 4–8 days, or remittent. The temperature may rise to 107° F, but usually it does not exceed 106° F. After the primary reaction there may be febrile exacerbations and remissions at intervals of 2–3 days, or complete absence of hyperthermia until death. *P. canis* infection often accounts for the secondary rises.

The appearance of parasites in the blood smears has been observed 3–5 days after the first rise in temperature, and their presence may be demonstrated until the death of the animal which as a rule takes place about 4–5 weeks after infection.

During the initial rise in temperature one hardly notices a change in the behaviour of the animal. Later, however, one notices inappetence and listlessness. Dogs lie down in a crouched position and move only if they are forced to do so. On several occasions it was observed that when such dogs are handled they snap. There is a progressive emaciation, the eyes are sunken, anæmia is present. The respirations are slow, and the pulse weak and slightly accelerated. Prior to death the animal may lie in coma for two to three days.

In many of the dogs used, *P. canis* appeared either as a result of a relapse or as a result of the infective blood of the donor. In these complicated cases the course of the disease is very much shorter and also more severe. Many dogs show a discharge from the eyes and nostrils, and broncho-pneumonia not infrequently hastens the fatal issue. In some cases excoriation of the skin is seen.

Course and Prognosis.

The duration of the disease may be one to four weeks or longer. At times an apparent improvement may be noticed, but in all our cases the disease has terminated fatally. There is no doubt that the biliary fever which often occurs at the same time, either from a latent or recent infection, influences the course of the disease adversely, in spite of the timely administration of trypan blue or acaprin.

Pathological anatomical changes.

In the majority of cases the post-mortem changes are typical of the superimposed (a) biliary fever, *i.e.* marked anæmia, icterus, tumour splenis, and trypan-blue staining where the drug was used, or (b) broncho-pneumonia.

In all cases emaciation is pronounced and skin excoriations and decubital sores are common, and in the few that were uncomplicated or killed the following changes were also noted, *viz.* pulmonary oedema, oedema of the hind limbs, gastro-intestinal catarrh, ascites, hydrothorax, and buccal ulcerations and erosions.

Histologically it has been possible to demonstrate the rickettsiæ in Giemsa-stained sections of the lung, but no specific changes have so far been recognized.

Diagnosis.

R. canis infection may be expected in any locality in which *Rh. sanguineus* or other dog ticks occur. Clinically it is extremely difficult to make a diagnosis. Not only are there several diseases which may give rise to similar symptoms, but some actually exist in the same animal. In the living animal, the only reliable method of diagnosis is the microscopical examination of blood, and lung-puncture smears. The demonstration of the parasites in organ smears, the lung particularly, is comparatively easy in dead animals.

In preparing blood smears it is even more important than in biliary fever to make smears from the first drop of blood that exudes from a small puncture or cut in the ear, because in this drop many more leucocytes are present than in subsequent drops.

For the preparation of lung smears from the living animal, the following procedure should be adopted. The hair over the middle of the body at the 5th and 6th intercostal spaces should be clipped and the skin disinfected. A hypodermic needle three to four inches in length is passed into the lung. By applying suction with a syringe a small quantity of the fluid of the lung is drawn into the needle. It is necessary to remove the needle together with the syringe in order to prevent the contents of the needle escaping. From the contents of the needle smears are prepared

and stained in the usual way with Giemsa. Provided this operation is carried out aseptically, no danger of bacterial infection need be feared.

Differential Diagnosis.

From the differential point of view the following diseases have to be considered.

1. Piroplasmosis.
2. Babesiosis.
3. Hepatozoonosis.
4. Trypanosomiasis.
5. Weil's disease (Spirochætosis).
6. Stuttgart disease.
7. Leishmaniasis.
8. Distemper.
9. Dumb form of rabies.
10. Verminosis.
11. Malnutrition due to mineral deficiency, avitaminosis, etc.

Since it is practically impossible to differentiate clinically, a definite diagnosis can only be made microscopically. It is therefore necessary to be able to distinguish *R. canis* from other blood protozoa and structures such as :—

1. *Leishmania donovani.*
2. *Hepatozoon canis.*
3. Phagocytozed *P. canis* and *Babesia gibsoni.*
4. Phagocytozed bacteria.
5. Clusters of blood platelets.
6. Stain deposit and artefacts superimposed on the leucocytes.

Immunity.

Up to the present all the dogs, about 30 in number, have died from the disease in 15 to 70 days after infection, so that from our observations nothing can be said about the nature of the immunity. Donatien and Lestoquard (1936) state that recovered dogs harbour a latent infection, and that splenectomy in such dogs produces a relapse. In the artificially infected jackal mentioned above, parasites were still demonstrable, by subinoculation of blood into dogs, after a period of 112 days.

Treatment.

Several drugs have been tried but so far none can be said to cure or even influence the course of the disease. In dogs suffering from a mixed infection of *P. canis* and *R. canis,* trypan blue and acaprin injections have caused the former parasite to disappear, but have not affected the latter at all.

Prophylaxis.

Theoretically, if ticks are the only vectors, it should be possible to prevent this disease by keeping dogs tick free. In practice it is extremely difficult to do so. Regular dipping and the application of substances like pulvex and odylen are indicated, but unless the procedure is kept up continuously and conscientiously it may not have the desired effect. Attempts should also be made to destroy breeding ticks in kennels by spraying, burning, etc. A measure of far greater importance is to avoid the introduction of dogs into localities known to be infected, or the moving of dogs from an infected locality into clean areas. Once the disease has been diagnosed, it is best to destroy the animal, which, even though it may recover, can still act as a reservoir and so continue to infect ticks in its neighbourhood.

Acknowledgment.

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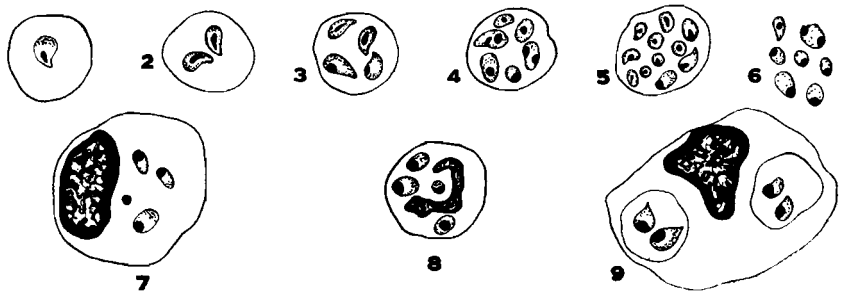
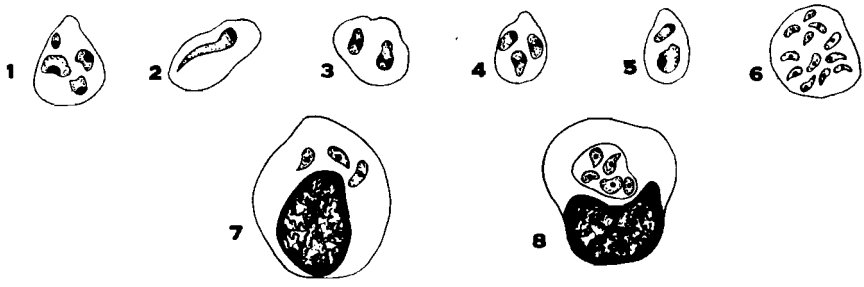
Blood parasites of the Dog.

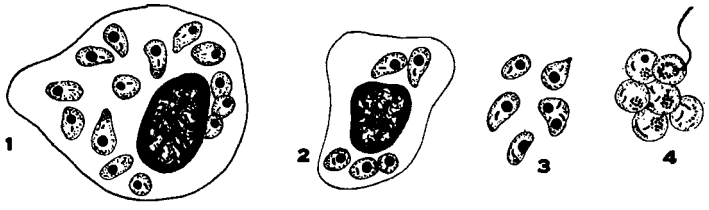
PIROPLASMA CANIS



BABESIA GIBSONI

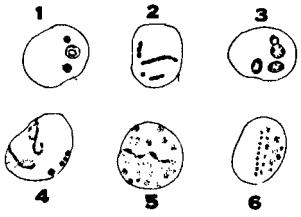


LEISHMANIA DONOVANI

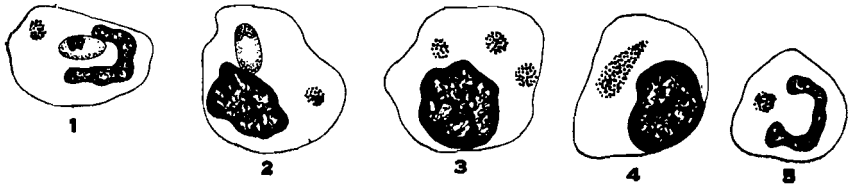


HEPATAZOON CANIS

BARTONELLA CANIS



RICKETTSIA CANIS



BLOOD PARASITES OF THE DOG.

Piroplasma canis.

Fig. 1 – 5 : Parasites in the erythrocytes.
Fig. 6 : Extracellular parasites.
Fig. 7 : Phagocytozed parasites in a monocyte.
Fig. 8 : Phagocytozed parasites in a neutrophile.
Fig. 9 : Phagocytozed infected erythrocytes in a monocyte.

Babesia gibsoni.

Fig. 1 – 6 : Parasites in the erythrocytes.
Fig. 7 : Phagocytozed parasites in a monocyte.
Fig. 8 : Phagocytozed parasites in a monocyte.

Leishmania donovani.

Fig. 1 and 2 : Parasites in monocytes.
Fig. 3 and 4 : Extracellular parasites.

Hepatozoon canis.

Fig. 1 – 3 : Parasites in neutrophiles.

Bartonella canis.

Fig. 1 – 6 : Parasites in the erythrocytes.

Rickettsia canis.

Fig. 1 : Mixed infection of *Rickettsia canis* and *Hepatozoon canis* in a neutrophile.
Fig. 2 : Mixed infection of *Rickettsia canis* and *Hepatozoon canis* in a monocyte.
Figs. 3 and 4: *R. canis* in monocytes.
Fig 5 : *R. canis* in a neutrophile.

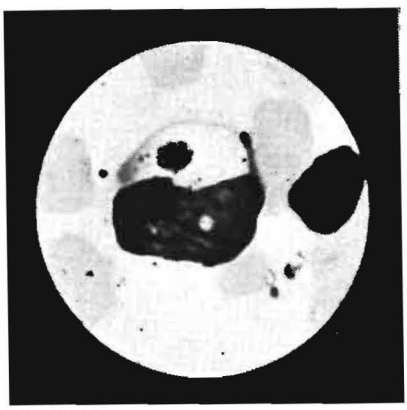


Fig. 1.

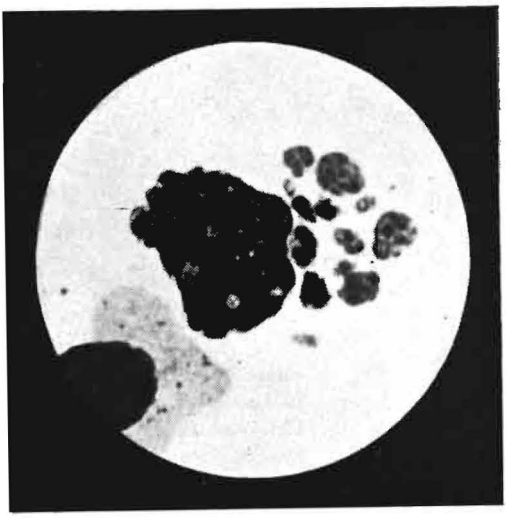


Fig. 2.

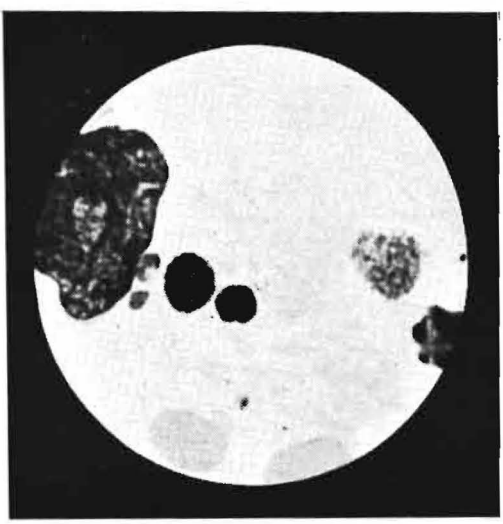


Fig. 3.

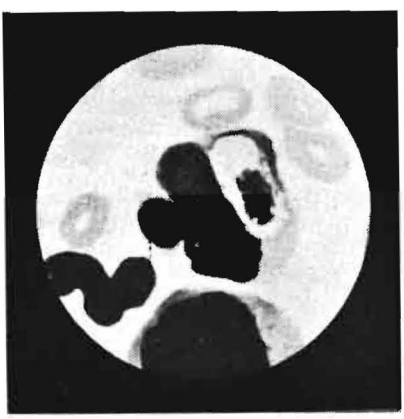


Fig. 4.

Fig. 1 : One colony of *Rickettsia canis* in a monocyte. 1,500 ×.

Fig. 2 : Twelve colonies of *Rickettsia canis* in a monocyte. 1,500 ×.

Fig. 3 : Four colonies of *Rickettsia canis* in a monocyte. To the right is a group of blood-platelets which resembles a colony of Rickettsia. 1,500 ×.

Fig. 4 : Two colonies of *Rickettsia canis* and a *Hepatozoon canis* lying in a neutrophile. 1,500 ×.

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Rickettsiosis in a Dog.

By D. A. LAWRENCE, Veterinary Research Dept., S. Rhodesia.

The object of this note is twofold :—

(1) To record the occurrence of a rickettsia infection in a dog in Southern Rhodesia.

(2) To draw attention to the advisability of resorting to confirmatory diagnosis even in a disease so clinically characteristic as biliary fever.

So far as can be ascertained no canine rickettsia has ever been recorded in Central or Southern Africa and reference to the available literature has not proved very fruitful. Wenyon in his " Protozoology " does not mention the occurrence of any rickettsia in canines. The occurrence of this parasite has, however, been described by Donatien and Lestoquard (1935, 1936) in Algeria, and for it they have suggested the name " Rickettsia canis."

It has not been possible to consult their original articles, but from two abstracts appearing in the " Veterinary Bulletin " and an annual report of the Pasteur Institute of Algiers there is every reason to believe that the rickettsia encountered in S. Rhodesia is identical with the one described by them.

In the annual report of the Pasteur Institute of Algiers for the year 1936 it is stated that canine rickettsiosis due to *R. canis* occurs not only in Algeria, where it was first detected in 1935, but also on the French littoral of the Mediterranean. Its detection in S. Rhodesia, in the circumstances to be described, would suggest that this infection may also be present in other parts of Africa. In this connection it might be mentioned that during a visit to the Veterinary Laboratory, Kabete, Kenya, the author was shown a blood film from a dog, in which one or more bodies very similar to, if not identical with, those subsequently encountered in this country were present.

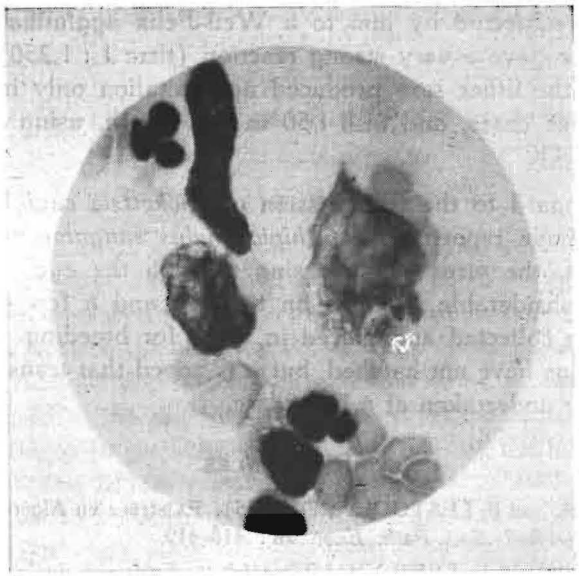
The probable explanation for *R. canis* not having been recorded more frequently is that private practitioners rarely resort to confirmatory diagnosis in cases of canine biliary fever and that, therefore, this disease is sometimes diagnosed in the rare cases where the cause is a rickettsia.

The subject, a three months' old English Setter owned by Mrs. A., had been " off colour " for a few days, and, when seen by the writer had completely lost its appetite. In addition, the gums were pale and the owner felt that treatment for biliary fever was essential. Because of the

history, elevation of temperature, pallor of conjunctiva and oral mucous membranes, and the characteristic throbbing pulse, the owner's diagnosis was confirmed; but following the usual practice, blood films were prepared and examined before proceeding with treatment. Prolonged search of these films failed to reveal *Babesia canis,* although there was definite evidence of anæmia—anisocytosis and fairly well marked polychromasia. A further and more detailed clinical examination was then made, but apart from the slight gastritis nothing further could be found. The owner was advised to give the animal a dose of syrup of buckthorn and castor oil and to improve the diet.

Two days later, however, she returned as the dog was definitely worse. It was now extremely weak, with respiration rapid and somewhat distressed, temperature elevated to approximately 105°, pulse very fast and weak, and visible mucous membranes almost white. Blood films were again examined, and it was thought that *B. canis* would be found with ease. Such, however, was not the case, and prolonged search failed to reveal a single parasite. Anæmic changes were now very pronounced and it was noticed that some of the very numerous leucocytes present in the tail-end of the film contained granular clumps. Attention was therefore concentrated on these, and in view of their close resemblance to *R. ruminantium* clumps they were identified as members of this family. These clumps occurred both intra- and extra-cellularly; the intracellular forms were found in mononuclears of the large lymphocyte type and the granules appeared to be more densely packed than in the extracellular type. Sometimes two distinct clumps were present in the cytoplasm of the same cell. The clumps varied in shape from true spherical or oval to irregular elongated bodies. The granules were extremely small and mainly coccus-like, but in some cases they appeared to be irregularly cocco-bacillary. The size of the clumps and the number of granules varied considerably, the smallest being only about 2 μ in diameter and with 6–7 granules, and the largest up to 13 x 8 μ and containing between 60 and 80 granules. Photomicrographs, kindly taken by Mr. Carlisle of the Public Health Laboratory, are shown on facing page, and clearly indicate the variations described.

In the absence of any known treatment for this disease and in view of its clinical resemblance to biliary fever, a subcutaneous injection of Akiron was given. Six days later the dog was again examined. It was still very listless and although the owner considered that improvement had set in, the temperature was 104° F, conjunctiva less pale, but slightly icteric, fæces soft and very offensive, spleen distinctly palpable and enlarged, pulse unchanged, appetite still very poor. Blood films on this occasion showed only extremely rare and rather indefinite rickettsia clumps. From this stage onwards the dog made a slow recovery.



× 810.
Rickettsia canis, dog, Southern Rhodesia.

At the suggestion of Mr. Alves, of the Public Health Laboratory, serum samples were collected from this case and from four normal dogs whose history indicated that they could never have been similarly affected. These were subjected by him to a Weil-Felix agglutination test. The rickettsia case gave a very strong reaction (titre 1 : 1,250) with *Proteus* OX_2, while the other sera produced agglutination only in a dilution of 1 : 25 in three cases, and in 1 : 50 in the fourth, using *Proteus* OX_{19}, OX_2, and OXK.

With regard to the transmission of *Rickettsia canis*, Donatien and Lestoquard have reported that *Rhipicephalus sanguineus* is infective in all its stages, the virus even passing through the egg. This tick was present in considerable numbers on the dog and a few engorged adult females were collected and placed in tubes for breeding. Unfortunately as yet the eggs have not hatched, but it is hoped that transmission experiments will be undertaken at a later date.

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Purulent Arthritis in a Foal due to *Salmonella Typhi-murium* var. *Copenhagen* (Storrs).

By M. W. HENNING and R. CLARK, Onderstepoort.

History.

Recently, one of us was consulted about a three-week-old foal suffering from an extensive swelling of the near stifle. This animal was bred near Bethlehem on a stud farm, which was recently acquired by the present owner for the purpose of horse-breeding. The existing premises had not been used previously for stabling horses; but some of the calves which had been housed in the building by the previous owner had developed a disease thought to be paratyphoid. At the time of the consultation, the hygienic conditions of the premises and the general management of the horses appeared to be beyond reproach.

Prior to the appearance of the swelling on the stifle joint the foal was thought to be in perfect health. There was no evidence or history of infection of the umbilicus. At the time of birth the cord was ligated and the stump painted with tincture of iodine.

Symptoms.

The swelling was diffuse, hot and painful; and, as it was thought to be the result of an injury, cold applications were tried. After a few days, however, both the knees, the off elbow, and the near shoulder, became affected in turn. All the affected joints became enormously enlarged, hot, and painful. After applying hot fomentations to the swellings fluctuation occurred, and a large amount of white fluid pus was evacuated. Some of this pus was streaked on to agar slants and yielded an apparently pure growth of a Gram-negative bacterium. The characters of this organism are described below.

The temperature remained approximately 103° F during the greater part of the course of the disease. The foal became progressively weaker until it was unable to stand up; but could suck from its dam when supported. After about three weeks' illness the animal suddenly went into convulsions and died within 12 hours. The owner, fearing that the infection might spread to the rest of his foals, burned the carcass, so that a post-mortem examination was not possible.

In addition to fomentations being applied to the joints, omnadin was administered parenterally and sodium salicylate given with the drinking water.

Some of the growth obtained from the pus was seeded on to MacConkey's bile-salt agar in Mason tubes. After 24 hours' incubation numerous pale, translucent, non-lactose-fermenting colonies were observed. A few of these were picked and tested against various " O " type, and group sera of different strains of Salmonella. Rapid flocculation occurred with the " O " sera of members of group B of the Kauffmann-White schema, with *typhi-murium* type serum, and with group sera. This suggested that the organism (labelled strain 478) was closely related to *typhi-murium*. It also proved to be diphasic. An antiserum was prepared and agglutination and absorption tests were performed. The technique employed was that described by Henning (1938).

Absorption tests showed that *typhi-murium* removed all " O," type, and group agglutinins from 478 serum as well as from the homologous serum; whereas strain 478, although completely exhausting the type, and group agglutinins from *typhi-murium* serum, removed only part of the " O " agglutinins from this serum, reducing its titre from 800 to 200; but completely exhausted its own serum.

The results of these tests showed that strain 478 contained the same " H "-antigens, type and group, as *typhi-murium,* but that it lacked some of the " O " factors contained in the latter. Agglutination and absorption tests were then performed with the IV-variants of *typhi-murium,* viz. varieties *Copenhagen* and *Storrs,* which lacked " O " factor V. According to these tests the antigenic structure of strain 478 is identical with that of the *Copenhagen* and *Storrs* variants—strain 478 completely exhausted both *Copenhagen* and *Storrs* sera, while either *Copenhagen* or *Storrs* removed all the agglutinins from 478 serum. Strain 478 should, therefore, be regarded as a IV-variant of *typhi-murium.*

Its biochemical characters were not quite the same. Like *Copenhagen,* it was Bitter, Stern, and d-tartrate positive, and fermented maltose. Unlike *Copenhagen,* it fermented inosite and failed to ferment arabinose. It resembled *Storrs* by giving positive Bitter, and d-tartrate tests and by fermenting inosite; but is different from this variant by giving a positive reaction with maltose, and Stern's fuchsin-broth, and by failing to ferment arabinose.

Strains of *typhi-murium* devoid of " O " factor V have been described by a number of workers. Thus, Landsteiner and Levine (1932) found it present in the *Binns* strain of Schutze. Kauffmann (1935) studied 16 such variants (his *Copenhagen* strains), while Edwards (1935), Hoffman and Edwards (1937), and Hohn and Hermann (1936) described it in pigeons and rabbits. But all these variants have been obtained from man, pigeons,

and rabbits. The organism described by us is apparently the first record of a IV-variant of *typhi-murium* isolated from a horse.

For further particulars about this organism reference can be made to another paper by one of us (Henning, 1938).

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An Investigation into the Nature and Cause of a Disease in New-born Merino Lambs affecting the Thyroids and Nervous Systems.

By J. G. WILLIAMS, B.V.Sc., Bloemfontein;
DOUW G. STEYN, B.Sc., Dr. Met. Vet., D.V.Sc., Onderstepoort;
and J. W. GROENEWALD, D.Sc., Onderstepoort.

INTRODUCTION.

A disease in new-born lambs, of which the most prominent symptoms were enlargement of the thyroids and peculiar forms of ataxy, was reported by one of us (J.G.W.) and J. M. Fourie (Government Veterinary Officer, Hoopstad, Orange Free State). As far as we could ascertain the disease made its appearance on four farms only, namely on Fairfield in Thaba 'Nchu district, and on Saaibult, Hebron, and Middelpan in the Brandfort district, Orange Free State.

Apart from publications on ordinary congenital goitre in the young of animals, we failed to find references to a disease which coincided exactly, especially as far as the symptoms were concerned, with the disease noted here. The authors would welcome information on whether a disease similar to the one described here has been known to occur in any other part of the world. Uotila (1938) refers to changes in the thyroids in cases of vitamin deficiency (A, B, C and D). He describes the following changes in the thyroid glands in cases of vitamin D deficiency : enlargement of the glands associated with degenerative changes and an increase in height of the epithelium, together with vacuole degeneration and colloid hyperplasia.

INVESTIGATIONS.

Symptoms of the Disease.

A small percentage of the lambs were still-born, at full term. The majority of the lambs (in some cases almost up to one hundred per cent. of the entire crop) showed the following symptoms at birth.

Some were born paralysed and unable to rise. When assisted on to their feet, they would stagger about, exhibiting muscular tremors of the body muscles, and eventually fall down again. Others exhibited symptoms of paresis and partial paralysis. They were able to struggle on to their feet unaided. When standing they showed varying degrees of muscular tremor. Some managed to lift the fore quarters only and moved about dragging the hind quarters. A large number of lambs showed either a pronounced spastic contraction (a form of tetany) or a slackness of the flexors affecting either the fore limbs or all four legs. In the majority of

cases only the fore limbs showed contraction of the flexors. The result was a most peculiar form of ataxy. The head was usually carried low and the front legs were moved forward and outward in a semicircle, and lifted very high. There was also marked inco-ordination of the movements of the hind legs. An outstanding feature of the disease, exhibited by practically all the lambs, was marked enlargement of the thyroids, up to approximately ten times their normal size. In some lambs the carpal joints appeared to be enlarged. The affected lambs were lively and, when put to the ewes, sucked readily. They were, however, unable to suck by themselves, as weakness or stiffness of the neck prevented them from lifting the head to reach the teat. The pronounced dark-pinkish colour of the gums and tongue reminded one of vitamin A deficiency.

The mortality among the affected lambs was almost one hundred per cent. and they died from within a few hours to a few days after birth. In those lambs which recovered, symptoms of ataxy were still detectable up to three weeks after birth.

Some of the ewes were in a fair condition and had sufficient milk for their lambs, although the majority were poor and had very little milk. The owners complained that they had to assist many ewes to deliver their lambs.

Post-Mortem Appearances.

The most outstanding feature was the enlarged and hyperæmic thyroids. In some cases they were of normal consistence whilst in others they were flabby and, on section, a thick, turbid, light-brownish fluid substance could be squeezed from the cut surface. In some cases the liver was yellowish (icterus neonatorum ?) and soft in consistence. One of the lambs upon which an autopsy was conducted showed œdema of, and hæmorrhages in, the lungs. There was hyperæmia of the pia and dura mater.

Histology.—The following histological findings described by Dr. A. D. Thomas, Section of Pathology, Onderstepoort, are based on the microscopic examination of material from some eight lambs which suffered from this condition.

Although in these lambs the striking feature was a greater or lesser increase in size of the thyroid, the microscopic examination of this organ did not reveal a constant or uniform change to explain this enlargement.

In three of the eight cases submitted, the structure of the thyroid could not be distinguished from the normal. In the others there were various types and grades of changes, some or all of which may have given rise to the enlargement of the organ. For instance, in three other cases there was hyperæmia with marked dilation of the interstitial blood vessels. The acini were irregular in size, contained colloid which stained unevenly

or very badly and showed numerous vacuoles. The epithelium was usually low cubical, but in parts papilliferous proliferation was visible and desquamation of cells into the lumen was not infrequent.

Of the two remaining lambs showing the most conspicuous alterations of the thyroid, one appeared to be suffering from a straightforward *struma parenchymatosa, i.e.* colloid was practically absent, the acinal lumina being small and rather irregular, and the whole organ having a dense, glandular appearance due to the proliferation of the epithelium and the elongation of the acinal cells.

The other lamb suffered from a severe form of hæmorrhagic thyroiditis. In this case the acini were larger than usual and angular in section. The colloid substance was profusely vacuolated and admixed with a considerable amount of desquamated lining-epithelial cells. The interstitial blood-vessels were dilated and congested and in many places there was severe extravasation of blood into the interstitial spaces and capsule as well as into the acini. In the latter instances, admixture with variable amounts of fibrin was common. In this lamb there was also a decided hæmorrhagic infiltration of the adrenals. Other organs, including the brain and endocrine glands, were also examined, but, except for a noticeable hyperæmia of the brain in two cases, nothing of significance could be recorded.

Bacteriology.—Dr. E. M. Robinson, Head of the Section of Bacteriology, Onderstepoort, examined specimens of the blood and of the intestine of affected lambs, but was unable to find organisms which could be held responsible for the condition.

INVESTIGATION OF CONDITIONS UNDER WHICH THE DISEASE OCCURRED.

In the Brandfort district the disease made its appearance on the three farms mentioned above. The owners of approximately fifteen farms which adjoin the three affected farms, and on which no cases of the disease had occurred, were interviewed and their flocks inspected.

All the farmers, both on affected and unaffected farms, complained that the grazing, which is purely grass veld, was inferior, owing to the heavy rains during October, November and December, 1936, followed by months of drought. The very quick and rank growth of the grass resulted later in pasture which consisted mainly of stalks. This state of affairs was rendered worse by early and extremely severe frosts. As a result of the very poor quality of grazing lambing was delayed until the middle of June, 1937, and even later; while in previous years, lambing commenced in February and was over by the end of April.

We have summarized, below, the results of our investigations in the field.

(a) Owing to the abnormal climatic conditions, lambing was very

late, and on the affected farms the pregnant ewes had been running on dry and very poor (mature) grass pastures for at least three to five months before lambing. *On all the farms which adjoin the affected farms, where the pregnant ewes were allowed to graze on green wheat, barley, or oat lands for at least one to two months before lambing commenced, not a single case of the disease occurred.* It is true that on the farm Fairfield the pregnant ewes were brought on to a green oat-land, but not until the disease had already become serious. It is obvious that the ill-effects of very poor grazing exerted upon animals over a period of months cannot be rectified in the course of a few days or weeks, especially as far as the effects upon fœtuses are concerned.

(b) The affected flocks had for years been receiving very poor attention from their owners. Apart from heavy overstocking there was ample evidence that the stock on the affected farms were in dire need of phosphatic licks. The worm problem is a very serious one in this area, but, apart from dosing their sheep once or twice a year with Cooper's Dip, the farmers concerned did nothing to fight this serious menace to their sheep.

Every one of the more intelligent and progressive farmers interviewed agreed that healthy lambs could not be raised on dry and mature grass veld, unless the pregnant ewes were grazed for a few hours daily on green lands (oats, barley, wheat) for at least one to two months before lambing.

(c) We were able to exclude the possibility of the " wild gooseberry " (*Physalis minima L.*) being the cause of the trouble, as none of the flocks concerned had had access to the plant. It is a common belief among sheep farmers that the "wild gooseberry" causes abortion in ewes, and paralysis in new-born lambs, although experiments conducted at Onderstepoort upon pregnant ewes have failed to confirm this suspicion.

(d) The possibility of poisoning by some plant or other agent was also excluded by a careful examination of the grazing and diet of the animals.

(e) Mr. J. H. R. Bisschop, Professor of Zootechnics, Onderstepoort, was able to exclude the possibility of the condition being inherited.

(f) It was definitely established that the drinking water was not responsible for the disease.

Laboratory Experiments.

Ten pregnant ewes and five affected lambs from the flock on the farm Fairfield were selected and sent to Onderstepoort for observation and experiment. Unfortunately five of the ten pregnant ewes either lambed or aborted in the truck on their way to Onderstepoort and all the lambs were dead on arrival, having either been still-born, or having died

soon after birth. Although the unreliability of results of experiments conducted upon such a small number of animals was fully realized, attempts were made to elucidate the cause of the disease. Two of the pregnant ewes were given green feed (barley) *ad lib.* and cod liver oil was administered to another two. Although the lambs from these four treated ewes appeared less affected than those of the untreated ewes, it would be unwise to attempt to draw any definite conclusions.

The five affected lambs brought to Onderstepoort were treated by subcutaneous injections of potassium iodide and calcium lactate. Calcium lactate was administered in order to exclude the possibility of calcium tetany. The injections, however, had no effect on the spasms.

All the lambs died two days after arriving at Onderstepoort. Thus this experiment was also inconclusive.

The inorganic blood phosphorus content of the ten ewes averaged 4.9 mg. per 100 cc. of blood shortly after their arrival. The calcium content of the blood averaged 7.3 mg. and, like phosphorous, was higher than would have been the case had the sheep been suffering from acute phosphorus deficiency. A test for carotene on the liver of one sheep revealed no vitamin A deficiency. As the diet which these animals received in transit was not known, they were kept on a known phosphorus and vitamin A deficient diet for a further period of six months. At the conclusion of this period the blood chemical figures were rather higher than they were expected to be; the inorganic blood phosphorus decreased most and now was 3.7 mg. per 100 cc. blood. Notwithstanding these blood figures, a sheep destroyed at the end of six months showed marked osteoporosis.

Unfortunately we were unable to conduct an intensive investigation into the cause of the disease, as in all but one case the farmers reported the disease when no more cases were available for investigation.

Discussion and Conclusions.

From our investigations it appears that the disease described in newborn lambs is a deficiency disease caused by the prolonged grazing of pregnant ewes on very poor, dry, mature, grass pastures. Very poor treatment of the flocks concerned, in previous years and during the lambing season, 1937, appeared to be contributory factor.

The information collected by us seems to indicate that the disease is due to prolonged deficiency, not only of one, but of several food substances which are of vital importance to the maintenance of health and normal development; for example vitamin A, protein, phosphorus, iodine. The animals which were affected grazed on very little more than cellulose (long, dry, seeded, lanky grass stalks with very few leaves).

It may be mentioned here that during the course of an experiment

conducted some years ago in which one of us (J.W.G.) was a collaborator, a similar condition was produced in new-born lambs whose mothers received a ration deficient in phosphorus and vitamin A. In groups which received the same ration but to which iodine was added, the condition developed to a lesser extent, although a larger number of abortions occurred.

In practice the disease can probably be prevented as follows :—

(a) Overstocking should be avoided.

(b) The animals should be regularly treated for worms and the steps necessary to prevent re-infection taken.

(c) Pregnant animals should be provided with good summer grazing and provision made for pregnant animals to have access to green feed (oat, barley, wheat lands) during autumn and winter months, for at least one to two months before lambing.

If no green feed is available, cow-pea hay (with pods) and lucerne hay may be fed to the pregnant animals.

(d) Stock, especially those which run on grass pastures, should be provided with phosphatic and protein licks during autumn and winter months. This is of vital importance to pregnant and to lactating animals.

Summary.

A disease in lambs, of which the characteristic signs are enlarged thyroids and nervous symptoms, is described. As far as could be ascertained the disease was the result of the prolonged grazing of the pregnant ewes on very poor, dry, mature, grass pastures deficient in substances (vitamin A, phosphorus, protein, etc.) of vital importance for normal growth and the maintenance of health.

Very poor treatment of the affected flocks was a contributory factor.

Methods of preventing the disease are suggested.

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Rinderpest: the Immunizing Value of Frozen Desiccated Goat Spleen.

By G. PFAFF, Insein, Burma.

In his excellent résumé of recent progress in East Africa, Daubney (1937) mentions that the chief apparent disadvantage of goat virus is the variability of goat spleens in respect of virus content. He goes on to say: "This difficulty can be countered by the pooling of several spleens and the testing of a bulked batch of suspension, provided a satisfactory method of preservation can be worked out, and we propose to attempt the storage of frozen desiccated tissue." Work on these lines was started by me in 1934 and mentioned in the Annual Reports on the Veterinary Department, Burma. (Mitchell, 1935, 1936, 1937.) The investigations were also briefly referred to at the Animal Husbandry Research Workers' Conference at Delhi in 1936. (Pfaff, 1937.)

Experimental work and field results have been fully described in an article entitled "Immunization against Rinderpest, with special reference to the use of Dried Goat Spleen," which is appearing in the October, 1938, issue of the Onderstepoort Journal. (As this will not be available for some time, this short note has been prepared to acquaint those interested with what has been done.)

Vaccine prepared from dried goat spleen was first used in field trials in October, 1935. 59,583 cattle and 4,785 buffaloes were inoculated in March, 1938, and the total number inoculated in the thirty months ending the 31st March, 1938, was 759,792 cattle and 62,474 buffaloes. The total mortality up to 30 days after inoculation was 944 cattle and 1,385 buffaloes, which represents a percentage of 0.124 in cattle and 2.216 in buffaloes. This vaccine, which is now the only protective inoculation against rinderpest employed in Burma, is used in actual outbreaks as well as in clean areas threatened with the disease.

Essential features in the preparation of the vaccine are rapid drying of the spleen pulp and storage at a temperature in the region of 42° F. Briefly described, the process of manufacture is as follows: goats are killed on the fourth day after infection with goat adapted rinderpest virus. The spleens are removed aseptically, finely ground in a latapie pulper and dried *in vacuo* over calcium chloride. The material, which is not tested for sterility or potency, is stored in the laboratory at 42° F and despatched to the field in a thermos jar of ice. The ice is replenished every third day, and inoculators are allowed to use the vaccine up to 30 days after receipt. Immediately before use the spleen powder is emulsified

in normal saline and used in doses of one gram for 400 cattle, and one gram for 2,400 buffaloes.

All experimental work was done on the pooled material from not less than three spleens. Four of the more important findings may be mentioned here.

1. *The viability.* The immunizing value of dried spleen is unaffected by four months at 42° F or by three days at 97° F. It rapidly deteriorates after storage for more than three days at 97° F.

2. *The immunizing dose.* 0.0005 gram of dried spleen never failed to immunize experimental animals. In practice, however, the vaccine is used in doses of 0.0025 gram.

3. *The time taken to confer immunity.* An immunity, which is serviceable in 24 hours and absolute in 48 hours, is conferred by 0.00025 gram of dried spleen, which is one-tenth the vaccine dose now used.

4. *The duration of immunity.* The experiment to determine this is still in progress. It has so far shown that the immunity conferred lasts at least twenty-five months.

The cost of the vaccine, even in the dosage now used, including the cost of goats, apparatus, packing and freight, amounts to about four shillings per thousand doses. Of 759,792 cattle inoculated, 0.124% are reported to have died. This probably includes many deaths due to other causes. No breakdowns in immunity have been reported and field experience is confirming laboratory findings on the rapid onset of immunity. It is no exaggeration to say that the vaccine is cheap, safe and effective.

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Jl. S.A.V.M.A.
IX(4): 190 – 191,
1938.

An Outbreak of an Unusual Type of Anthrax in Cattle in the Eastern Transvaal.

By G. C. v. DRIMMELEN, B.V.Sc., Ermelo.

The diagnosis of anthrax in the field is usually made by examining blood or spleen smears microscopically. Sometimes the fresh unopened carcass is seen shortly after death and may show changes which lead to a reasonable suspicion of anthrax. In the majority of cases of the " sudden death " or septicæmic type of this disease the smear reveals the presence of anthrax bacilli in large numbers.

Recently, a farmer living in the Ermelo district, reported that a number of his cattle were suffering from swollen throats. They had difficulty in breathing and swallowing and could barely lower their heads to drink. I visited this farm and found that a number of these animals showed a marked œdema of the throat and intermandibular space accompanied by a high temperature. The swellings were tense and gave the impression that they contained a gelatinous exudate.

An attempt at tracheotomy was made on one animal which showed signs of asphyxia. Unfortunately this beast died of suffocation during the operation. A post-mortem examination revealed nothing except a marked gelatinous exudate in the intermandibular space. This exudate was slightly yellowish in colour and looked clean. No disease-producing organisms could be seen in bloodsmears or in the smears from the exudate.

A week later a second animal died. At post-mortem all internal organs appeared healthy, but the lymph glands in the vicinity of the gelatinous exudate round the throat were enlarged, moist and somewhat hæmorrhagic. Smears from these glands showed large numbers of anthrax bacilli.

A third death occurred a day or so later and a bloodsmear showed the presence of anthrax bacilli.

The farm on which these cases occurred was a small one and carried 96 head of cattle. Anthrax was only diagnosed nine days after the first death. Three days later inoculation with saponin spore vaccine was carried out and after another fourteen days deaths ceased. The farmer claimed to have saved seventeen head by making multiple small incisions into the swelling around the neck and painting these with tincture of iodine. Five recoveries can be vouched for.

No records of this type of local anthrax in South Africa are available and I gained the impression that it was only seen in abattoirs.

Mr. A. S. Canham, in a personal communication, stated that this condition is not uncommon in parts of the Western Transvaal and north-

eastern Free State. The distribution is patchy and the disease usually appears on a group of farms in an area. Animals may live for ten days after the first appearance of the œdema, but recoveries are rare. The possibility of anthrax should always be considered when swellings are seen in the throat region of cattle, pigs and horses. Swellings in the vicinity of the perineal or sternal regions of donkeys, horses and mules should also be regarded as possible anthrax lesions. A peracute hæmorrhagic abomasitis in cattle may be the only evidence, at post-mortem, of an anthrax infection. It is unwise to make a diagnosis of enzootic icterus in sheep unless a smear has been taken after death. An enlarged spleen is seen in only 20 per cent. of sheep dying of anthrax.

Viljoen *et al* (1928) state that fatal cases of anthrax are "always of a typically septicæmic character."

Hutyra and Marek mention that anthrax in pigs is often reflected by a marked œdema of the throat region, and that anthrax in cattle may be confined to a single lymph gland near the intestinal canal.

It has been shown by overseas workers that neo-salvarsan injections frequently exercise a beneficial effect in cases of localised anthrax.

The object of this report is to draw the attention of my colleagues to a form of anthrax which may be more usual than is commonly supposed.

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Jl. S.A.V.M.A.
IX(4): 191 – 196,
1938.

A Clinical Report on Diabetes Mellitus in Dogs.

By B. S. PARKIN and H. GRAF, Onderstepoort.

Diabetes mellitus is a disease in which the metabolism of carbohydrates, proteins and fats is disturbed and in which sugar and acetone bodies are found in the urine. The object of this article is, primarily, to record the clinical examination of two cases of the disease in Dobermann Pinschers.

It would appear, if judgment be based on the numbers of cases recorded in the literature, that the disease in dogs is rare. Possibly, however, many cases may be overlooked, because the examination necessary for its diagnosis has not been instituted.

Milks and Stephenson (1937) record ten cases of diabetes mellitus in the dog. In most of their cases lesions of the pancreas were found at autopsy.

The two Dobermanns, police dogs trained to the trail and on duty at out-stations in Natal and in the Orange Free State, were presented for examination on the same day (28.6.37). They were both females and just over eight years old. Their condition was very poor. They had been treated at different times for intestinal parasites, and one of them had been examined by a Government Veterinary Officer (Mr. F. B. Wright. B.V.Sc.), who made a tentative diagnosis of diabetes mellitus. They were not related to each other and were progeny of dogs which had not suffered from a chronic debilitating disease.

The urine of both dogs had a specific gravity of over 1030, and a positive reaction for sugar and acetone bodies.

One of the dogs (2153) died a few days after admittance. It had not been treated. The blood analyses of this dog on 28.6.37 showed total nitrogen (T.N.) 3.1 gm. %, non-protein nitrogen (N.P.N.) 41.6 mg. %, urea nitrogen (U.N.) 14.8 mg. %, glucose (S.) 317.4 mg. % and cholesterol 207 mg. %. The urine showed 7.2 gm. % of sugar and albumin and acetone bodies. On the 6.7.37, the sugar content of the urine was 3.7 gm. %.

The other dog (2152) was kept under observation until it died on the 19.8.38. Insulin was used in this case, more for the purpose of obtaining information about the disease than for the production of a stabilized state, as it was considered that the dog would no longer be of value for trail work. Details of the urine and blood examinations, and of the insulin therapy are appended. The death of this dog was due to the insulin therapy being stopped.

The methods of examining the urine and blood were the following.

Analysis of Urine.

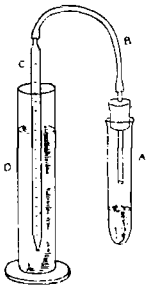
Examination for sugar.

(a) The presence of sugar in the urine was determined by means of Sheftel's reagent which consists of copper sulphate 25 gm., sodium citrate 250 gm., anhydrous sodium carbonate 150 gm., acacia (10%) 4 cc., and distilled water up to 1 litre. To 5 cc. of this reagent 0.5 cc. of urine is added. The mixture is heated in a water bath for about 5 minutes at 100° C. By comparing the colour of the mixture after it has cooled with the colours of a standard chart, an approximate estimation of the sugar content can be obtained. If the sugar content of the urine be above 1.75% only 0.12 cc. of urine is added to the reagent and the colour obtained is compared with the colours of a second colour chart. The colour changes from green through a greenish-yellow to yellow, with an increase in the percentage of sugar. This method was employed in the daily examinations and proved to be satisfactory for clinical use.

(b) A fermentation test was also employed on a number of occasions.

The advantage of this test is its ease of application and the availability of the materials even in out of the way places. The simplest form of this test is the introduction of a piece of baker's yeast (compressed tablets), about the size of a pea, into a test tube of the urine, and the inversion of the full tube into a small basin of urine. The gas produced by the fermentation collects in the test tube.

An endeavour was made to obtain a quantitative determination of the sugar content by measuring the gas produced during fermentation from a known amount of urine. (See diagram.) The apparatus, containing the urine, and the water which was later to be displaced by the gas produced by fermentation was brought to a constant temperature of 37° C. When everything was ready a pellet of yeast was introduced into the test tube containing a measured quantity of urine. The water levels of the cylinder and the graduated pipettes, in which the gas was to be measured, were balanced and a reading taken. Subsequent readings were made with the water levels balanced by raising the pipette in the cylinder. The figure finally obtained was adjusted for vapour tension and N.T.P. and the theoretical amount of sugar in the urine was calculated from



$$C_6H_{12}O_6 \rightarrow 2C_2H_5OH + 2\ CO_2.$$

The figures obtained in this way were always about 20% lower than the theoretical value. This was apparently due to factors such as retention of CO_2 by the urine and absorption of CO_2 by the water.

The number of grammes of sugar in 100 cc. of urine was calculated from

$$\frac{\text{cc. of } CO_2 \text{ under pressure } (P_1-p) \text{ and temp. of } t^\circ C}{\text{cc. of urine taken}} \times \frac{273 \times (P_1-p) \times 0.18 \times 100}{(273 + t) \times 760 \times 44.8,}$$

$$\textit{i.e.,}\ \frac{\text{cc. of } CO_2}{\text{cc. of urine}} \times 0.1443 \times \frac{(274 + t)}{(P_1-p)}$$

where P = barometric pressure; p = vapour pressure of water at temp. t° C; t = temp. of determination.

In spite of its inaccuracy this is a useful, easily applied method for determining the sugar content of urine, especially as the copper sulphate solutions used in some methods sometimes give positive reactions in the absence of glucose.

(c) Most of the determinations of the sugar content of the urine were made by Benedict's Quantitative Method.

Examinations for acetone bodies.—Rothera's nitroprusside test was used. This is done by adding a few drops of liquor ammoniæ fort. to the urine saturated with ammonium sulphate, and then adding a few drops of a fresh solution of sodium nitroprusside. A colour reaction (permanganate colour) is obtained. This is recorded, according to its intensity, as + to +++.

Examination for albumin.—Dhomme's reagent (picric acid 10 gm., trichloracetic acid 10 gm., glacial acetic acid 100 gm., water to 1 litre) was used to determine the presence or absence of albumin. A drop of the reagent was allowed to fall on to two drops of urine on a glass slide. This enabled even slight albuminuria to be detected and was better than mixing the two in a test tube. The albumen was recorded as trace (+), moderate (++) and heavy (+++).

The pH of the urine determined with the B.D.H. Universal Indicator varied between 3.5 and 5.0.

The specific gravity of the urine was determined with a small urinometer for which only 5 cc. of urine was required.

Analysis of Blood.

The tests for N.P.N., U.N., sugar, and amino-acid nitrogen (A.A.N.) were performed on Folin-Wu's laked filtrate by Folin's methods [Graf (1933)].

The T.N. was obtained by Kjeldahl combustion of whole blood, and the cholesterol was obtained colorimetrically, by the use of Bekker's modification (1938) of the method of Bernhard and Drekter (1931).

The hæmoglobin was determined by Newcomer disc method.

As can be seen from the table the dog was observed from 28.6.37 to 14.8.37. It did not receive insulin until the 13.7.37, and the initial dose was eight units per day. The maximum daily dose was 24 units. The sugar content of the urine was not held at a uniform level by the dosage of insulin employed even though it was probably sufficient during periods. This is probably owing to a somewhat marked irregulality of the diet which was due to difficulties met with in controlling the food supply and also because the daily allowance of insulin was sometimes given as one dose. Such a single large dose did not, however, produce symptoms of hypoglycæmia.

This dog ultimately died in diabetic coma. The blood, just before death, showed a thick milk white serum from which, on standing, there separated a thick "cream" layer, *i.e.* a severe lipæmia.

In addition to the abnormalities mentioned above the dogs showed cataract, keratitis, and a number of wounds on the legs.

Post-mortem Reports.

The post-mortem of dog 2152, which had been treated with insulin for six weeks and had died 72 hours after the last dose revealed only minor changes of the pancreas, of a hydropic and hyaline nature. A striking feature found in this dog was the pronounced lipæmia observed before death.

The details of the autopsies were :—

Dog 2152 (P.M. report 16110 of the Section of Pathology) : hyperæmia and œdema of lungs; hyperæmia of kidneys; regressive changes and stasis of liver; pancreas somewhat pulpy.

Microscopic appearance : pancreas showed hæmorrhages from venules, and atrophy of islets of Langerhans with the smaller nuclei denser and more irregular than normal; kidneys showed fatty changes and pigmentation; liver showed stasis, fatty changes and marked bile pigmentation; cerebral capillaries showed fat phagocytosis.

Dog 2153 (P.M. report 16052 of Section of Pathology) : regressive changes of liver; catarrhal enteritis; degeneration of lens of both eyes; pancreas apparently normal.

Microscopic appearance : kidneys showed fatty changes and induration, the liver vacuolar, hyaline and fatty degeneration (the last mentioned being peripheral in distribution), the spleen atrophy, the lungs fat phagocytosis, and the adrenals vacuolar degeneration, increased lipoid content and minor hæmorrhages in the cortex. The pancreas showed hydropic and hyaline degeneration of islets of Langerhans, hydropic degeneration of the epithelium and of the smaller interlobular ducts.

DOG 2152.
URINARY EXAMINATION.

	Specific gravity.	Albumin.	Ketones	Sugar gm.%.		Units of Insulin per day.
28 June		+	+++	6.1	28.6.37 *Blood*, T.N. 2.9 gm.%;	
29 ,,	1042		+++		N.P.N. 37.1 mg.%; U.N. 14.3	
6 July	1050	+		6.9	mg.%; sugar 363.6 mg.%;	
9 ,,	1038	+	+++	5.3	cholesterol 14.1 mg.%.	
13 ,,	1016	++	+++	2.6		8
14 ,,	1017	+++	+++	3.0		12
15 ,,	1015	+++	+++	2.1		16
16 ,,	1005	++	+	0.9		16
17 ,,	1028	+++	+++	4.3		16
19 ,,	1020	++	++	4.8		16 (also 20 units on 18th)
20 ,,	1026	++	++	5.0		16
21 ,,	1012	+	+	2.3		20
22 ,,	1027	+	++	5.7		20
23 ,,	1032	+	++	5.3		20

DOG 2152.

URINARY EXAMINATION.

(Continued from previous page.)

	Specific gravity.	Albumin.	Ketones	Sugar gm.%.		Units of Insulin per day.
24 ,,	1029	++		5.8		20 (also 20 units on 25th and 26th)
27 ,,	1043	++	++	8.6		20
28 ,,	1033	++	++	6.9		24
29 ,,	1030	+		3.0		24
30 ,,	1022	++	+			24
31 ,,	1020	+	+			22
3 Aug.	1042	+	++	6.0		20 (20 units on 1st and 2nd Aug.)
4 ,,	1038	+	+	0.1	4.8.37 *Blood*, T.N. 2.8 gm.%; N.P.N. 30 mg.%; U.N. 12.8	18
5 ,,	1018	+	+	0.3	mg.%; sugar 78.1 mg.%; cho-	16
6 ,,	1022	++	++	0.9	lesterol 2.86 mg.%; A.A.N.	16
7 ,,	1035	++	++	7.0	8.4mg.%.	16
9 ,,	1032	+	++	6.2		16 (16 units on 8th Aug.)
10 ,,	1038	+	++	6.0		16
11 ,,	1036	++	++	1.2		16
12 ,,	1030	+	++	1.6		0
13 ,,	1042	++	++	5.9	13.8.37 *Blood*, T.N. 2.7gm.%;	0
14 ,,	1031	+++	++	4.2	N.P.N 27.7 mg.%; U.N. 5.9 mg.%; A.A.N. 5.6 mg.%; sugar 268 mg.%.	Died during night.

Summary.

1. Two cases of diabetes mellitus, received for examination on the same day, are recorded. The clinical examination and treatment of one are recorded in detail.

2. The chief points of interest are hyperglycæmia (maximum 363.6 mg.%), glycosuria (maximum 8.6%), low initial cholesterol, pronounced lipæmia in blood collected just before death, persistent albuminuria (trace to heavy) and ketosis (always present), specific gravity (ranging from 1005 to 1050) and lesions of the eyes and skin.

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REVIEW.

The increasing demand for a knowledge of the ingredients incorporated in cattle-cakes and meals makes it necessary for the food analyst to pay attention to the botanical structure of various cereal-grains and weed-seeds.

The Authors * give a description of various plant ingredients commonly used in cattle-cakes, five methods of preparing samples for analysis, and give several tables whereby these ingredients may be compared by microscopical examination. The microscopical appearances of the botanical structure of the seeds or grains of cereals and weeds are very clearly shown. Although primarily intended for laboratory use, this book may be of assistance to the field veterinarian who wishes to identify certain grain particles found in the fæces of animals.

Not much space is, however, devoted to feed adulterants. A simple method for the separation of sand and grit from a sample is given. This consists of shaking up the sample with a little carbon tetrachloride instead of chloroform. Upon standing, the sand, grit, or minerals will settle to the bottom of the tube.

J. W. G.

* *The Microscopic Examination of Cattle Foods* by S. P. Parkinson and W. L. Fielding (1930), 97 pages, illustrated with 15 plates and 125 original photographs. Headley Brothers, Invicta Press, Ashford, Kent; and 18, Devonshire St., London, E.C. 2.

OBITUARY.

SAMUEL HILL EWING.

With the passing of any professional colleague, a feeling of regret and sorrow overwhelms those who knew him well. This feeling is deepened many-fold when that person was a capable professional and administrative officer and a real good sort. Such a one was Samuel Hill Ewing.

Originally of Lissen House, Cliftonville, Belfast, Ewing obtained the diploma of the Royal College of Veterinary Surgeons from the Dublin College on 22.7.1909, and entered the Union Department of Agriculture in October, 1910.

At first stationed in Zululand, he later saw service in the Orange Free State during the period when glanders was being eradicated. He told many tales of how (by handing out packets of sweets to the children) he influenced the old Boer farmers to kill glanders-infected horses. This typifies his character—kindly, generous, always ready to help, often giving away what he should have kept for himself. Later he served in the Cape Province and in Natal and at the time of his death was at Durban.

Married in June, 1930, he leaves a wife to mourn his loss.

He had suffered from heart trouble for about eight months before his death and apparently was on the way to recovery when he died in his sleep on the 27th September, 1938.

A. W. D.

JOHN ALEXANDER MAYBIN (1895 – 1938).

It is with deep regret that we record, in this issue, the death of John Alexander Maybin.

A native of County Antrim, Ireland, he was born on 3rd May, 1895, and, after serving with His Majesty's Forces during the Great War, he entered the Royal Veterinary College, Ballsbridge, Dublin, where he obtained his Membership Diploma on 24th July, 1924.

Six months later he entered the service of the South-West Africa Administration as a district veterinary officer, thus renewing an association with South Africa which had already commenced in Dublin, where he was a fellow student with a number of us. He later spent a few months at Onderstepoort on post-graduate work, and a short while on meat inspection in Durban.

He attended the recent S.A.V.M.A. meeting at Onderstepoort and it was during this conference that the illness, which led to his untimely demise on 18th November, befell him.

Of a quiet disposition, he was industrious, well conducted, and in every way a credit to the profession which now mourns his death.

To his widow and two daughters we extend our sincere sympathy in their sad bereavement.

C. J. v. H.

THE ASSOCIATION.

Council Meeting held at Polley's Hotel, Pretoria, on 24th October, 1938.

Present: S. T. Amos (President), C. J. van Heerden, H. H. Curson, A. D. Thomas, H. O. Mönnig, A. M. Diesel, R. A. Alexander, D. G. Steyn, M. Sterne, S. W. van Rensburg (Hon. Sec.-Treas.).

(1) **Minutes** of meeting held on 13th April, were taken as read and were confirmed.

(2) **Arising from these**—

(a) *Admission of Animals to Onderstepoort:* The Secretary submitted the following correspondence—

(i) Letter dated 21.10.38 from the Hon. Secretary, Faculty of Veterinary Science.

(ii) Letter dated 1.10.38 from the Acting Director of Veterinary Services to the Secretary for Agriculture.

(iii) The Secretary for Agriculture's reply dated 13.10.38 to above.

(iv) Report of the committee appointed by Faculty to investigate and report on the clinic at Onderstepoort.

(v) A letter dated 6th September, 1938, and signed by eleven private practitioners in Johannesburg.

Considerable discussion ensued, but it was generally felt that the matter could not be finally disposed of at this meeting since Faculty had not yet considered the Committee's report nor the ruling given by the Secretary for Agriculture.

It was finally decided, that a special meeting of Council be held in Johannesburg on a date to be fixed by the President, preferably in November, for further consideration of this question: that Faculty be asked to consider the relative report and correspondence and supply Council with its view; that private practitioners and other members who may desire to attend the special meeting be allowed to do so, and that a stenographer be employed to take down a verbatim report of the meeting.

(b) *Book Fund Prize*: The report of the General Purposes Committee was read and considered. It was eventually decided that a prize of £10 known as the "S.A.V.M.A. Book Fund Prize" be awarded annually to a student in the third, fourth or fifth year of the course; that the recipient be nominated by Faculty, and that in making the nomination Faculty should consider mainly the financial needs and the ability of the candidates. The award is to be made as from the beginning of 1939.

(3) **New Members:** Messrs. J. F. Fick and E. J. Pullinger were

nominated. Decided to recommend their acceptance to the General Meeting.

(4) **Resignations:** Letters of resignation from Messrs. J. Forrest and W. Orr were considered. Decided to recommend General Meeting to accept both, with regret.

(5) **Arrears:** The Secretary reported that of the three members who were three years in arrear with their subscriptions one had paid, another had promised payment at an early date, while the third was seriously ill at present. Council accepted these explanations.

(6) **Benevolent Fund:** (a) The Secretary submitted the correspondence in connection with two donations amounting to £20.10.0 made to Mrs. C., widow of a late member. The action taken by Finance Committee in this matter was approved.

(b) Council sanctioned the expenditure of £1.10.0 on the text book (Mönnig's Veterinary Helminthology) sent to a colleague in poor health. The amount to be taken from the general fund.

(7) **Complaints:** (a) By three members *re* articles published in the lay press by a veterinarian. Decided that this matter be referred to the Veterinary Board.

(b) By a member *re*:

(i) *Formation of a Cape Province Branch.*—Council was very sympathetic and decided to assist as far as possible to form a Cape branch; but found it impracticable to hold the General Meeting in Capetown next year.

(ii) *A Stock Inspector doing veterinary work.*—Decided that this be referred to the Director of Veterinary Services with a request that the complaint be investigated and suitable action be taken.

(iii) *Medical practitioner doing veterinary work.*—Seven cases were mentioned by the complainant. Since however none of these was in a town or district in which a veterinary practitioner is practising, and since medical practitioners are not debarred from doing veterinary work under Act 16 of 1933, it was decided that no action could be taken.

(iv) *Contagious abortion control.*—This referred to a scheme submitted by the member to the Minister of Agriculture. Since contagious abortion has not been withdrawn from the list of scheduled diseases, and since this matter is being dealt with by the Department of Agriculture, it was felt that the Association could not intervene.

Drs. Curson and Thomas left the meeting at this stage.

(8) **General:** (a) *Dr. Schulz:* The Secretary submitted a letter dated 18.10.38 from Dr. Schulz enclosing copies of letters dated 21.8.38

and 8.8.38 from the Secretary for Agriculture. From this corerspondence it was evident that the Department of Agriculture and Treasury have given very sympathetic consideration to Dr. Schulz's claim for compensation, and that he has been advised to proceed with his claim by means of a petition to Parliament.

After full discussion it was agreed that the relative correspondence be submitted to the General Purposes Committee with power to act.

(b) *Constitution* : It was agreed that the revised constitution, and the Veterinary Act and the Regulations, be printed in both languages, and issued to members.

(c) *Foot and Mouth Disease* : The following motion proposed by the President was passed unanimously :

" That this Council places on record its appreciation of the manner in which the administrative powers and the Department of Agriculture carried out their duties in the foot and mouth disease campaign in Natal."

The meeting closed at 11.30 p.m.

S. W. J. v. RENSBURG,
HON. SEC.-TREAS. S.A.V.M.A.

33rd General Meeting held at Onderstepoort, 25th—27th October, 1938.

Present : S. T. Amos (President), C. J. van Heerden, J. H. Mason, R. Alexander, L. L. Daly, A. S. Canham, J. A. Thorburn, M. Sterne, J. J. G. Keppel, A. M. Diesel, P. S. Snyman, D. G. Steyn, G. McIntyre, J. R. Scheuber, R. Clark, W. B. Allchurch, M. M. Neser, G. de Kock, M. J. N. Meeser, J. Quinlan, H. G. Franz, W. J. Ryksen, N. C. Starke, C. H. Flight, V. Cooper, J. H. R. Bisschop, A. Thiel, R. du Toit, N. H. Boardman, W. G. van Aswegen, H. Theiler, J. M. de Wet, H. A. Crawshaw, G. F. van der Merwe, J. A. Maybin, M. Bergh, J. G. Williams, I. P. Marais, H. P. Steyn, J. Nicol, F. Hellberg, C. W. Belonje, J. M. Fourie, J. H. B. Viljoen, J. G. Boswell, W. O. Neitz, N. T. van der Linde, L. W. Rossiter, C. Jackson, E. J. Pullinger, C. C. Wessels, L. T. Edwards, G. Martinaglia, H. H. Curson, M. W. Henning, W. Rainey, N. F. Viljoen, H. O. Mönnig, A. D. Thomas, P. J. J. Fourie, E. M. Robinson, B. S. Parkin, W. D. Malherbe, J. L. Dickson, G. Kind, C. J. Erasmus, J. Spreull, J. G. Bekker, H. Graf, M. de Lange, B. van der Vyver, J. H. N. Hobday, C. T. Nilsen, G. van der Wath, S. W. J. van Rensburg (Hon. Sec.-Treas.).

Visitors : Dr. Sheppard Cruz and Dr. V. Camara (Portuguese East Africa), Dr. Enigh (Germany), Dr. Squires (Bechuanaland), Col. J. Stevenson-Hamilton, Dr. J. P. van Zyl, and several members of the Division of Chemistry, and the Onderstepoort professional staff.

Apologies for Absence: G. T. Henderson, C. V. E. Mare, F. J. Carless, W. S. B. Clapham.

Deaths: The President referred to the loss the Association had sustained during the past year through the death of two of its oldest and most popular members, Messrs. S. H. Ewing and B. Young. He asked those present to stand in silence for a few moments as a mark of respect.

(1) **Minutes** of General Meeting held on 27th–29th October, 1937, having been published were taken as read and were confirmed.

(2) **New Members:** The following were proposed: H. A. Crawshaw, J. L. Dore, J. F. Fick, E. B. Kluge, E. J. Pullinger, J. Wakefield Rainey and L. J. F. von Maltitz.

(3) **Election of Council:** The following four were declared elected as members for the next two years: A. S. Canham, A. M. Diesel, D. G. Steyn, A. D. Thomas. The Council for the next 12 months would thus be:

President: S. T. Amos.
Vice-President: C. J. van Heerden.
Hon. Sec.-Treas.: S. W. J. van Rensburg.
Members: R. A. Alexander, A. S. Canham, H. H. Curson, A. M. Diesel, P. J. du Toit, H. O. Mönnig, D. G. Steyn, A. D. Thomas and the Editor of the Journal.

(4) **Presidential Address:** This dealt with the expansion of private practice in South Africa, the thirteenth International Veterinary Conference, the Journal of the Association, and a tribute to the officials concerned in the recent foot and mouth disease outbreak in Natal.

(5) **Resignations** were considered from Messrs. W. Orr and J. Forrest. The former left South Africa two years ago, while Mr. Forrest had retired and was not certain of his future movements. Both were accepted with regret.

(6) **Arrears:** The Secretary reported that Council had dealt with these.

(7) and (8) **The Reports** of the Secretary and Standing Committee for 1937-38, having been published in the Journal, were taken as read, and were approved.

(9) **Amendments to Constitution:**

(a) *On behalf of Council*: that rule 9 (a) be amended by the addition of: "The President, Vice-President and Secretary-Treasurer shall hold office for one year, but shall be eligible for re-election."

Carried unanimously.

(b) *By the Secretary-Treasurer*: that rule 7 (a) be amended by the addition of: "In the case of lady members the subscription shall be £1.1.0 per annum for ordinary membership and £15.15.0 for life membership."

Drs. Alexander, Sterne, and Mr. Coles opposed this on the ground that there should be no differentiation amongst members, and that once such a principle is accepted by the Association it might also lead to differentiation in salaries. The motion was lost by a large majority.

(c) *Translation of Constitution.*—The principle of having the Constitution translated into Afrikaans was approved. It was agreed that the translation be available for members to see during the meeting, and that it be considered on the last day of the meeting.

(10) **General:**

(a) *Advertisers.*—Dr. Mason appealed to members to support the firms advertising in the Journal.

(b) *Book Fund Prize.*—The President explained the origin of the Book Fund and stated that £100 had been handed over to form the nucleus of the Book Fund Prize. Council had decided that a prize of £10 would be awarded annually to a student in the 3rd, 4th or 5th year, recommended by Faculty on a basis of need and ability.

(11) **Opening:** After the tea interval the President read a letter from the Secretary for Agriculture expressing regret that the Minister was not able to address the meeting as he had not yet fully recovered from his recent indisposition. The following motion was passed unanimously:—

"The meeting expresses its thanks to the Honourable the Minister of Agriculture for consenting to open Congress, and regrets that he was prevented by illness from perfoming this ceremony. It further wishes the Honourable the Minister a speedy and complete recovery from his recent illness."

The scientific portion of the meeting next took place, and the following papers and demonstrations were given:—

11–12.15 p.m. "Water poisoning in man and animals with a note on urinary calculi" — Dr. D. G. Steyn and Mr. N. Reinach. Opener: Dr. A. I. Malan.

12.15–1 p.m. "The differential diagnosis of chronic arthritis in bovines" — Mr. J. G. v. d. Wath. Opener: Dr. A. D. Thomas.

2 – 3.15 p.m. "Suggestions towards the eradication of Cysticercosis-Taeniasis" — Dr. N. F. Viljoen. Opener: Dr. H. O. Mönnig.

3.30–4.30 p.m. Demonstration.—Rumenotomy — Dr. G. G. Kind.

Wednesday, 26th October.

9–10.45 a.m. "Some observations on dourine" — Drs. G. de Kock, E. M. Robinson, and B. S. Parkin. Opener: Maj. J. J. G. Keppel.

11–12.15 a.m. Skin conditions — Mr. J. A. Thorburn.

(a) " Eczema in dogs and cats." Opener : Dr. J. G. Bekker.

(b) " Sarcoptic mange in cattle, horses, and goats." Opener : Mr. J. Nicol.

12.15–1 p.m. Demonstration on the use of " Zysternal for bovine mastitis" — Dr. J. G. Bekker.

2–4.30 p.m. Excursion to Rietondale Pasture Research Station.

Thursday, 27th October.

9–10.45 a.m. Foot and mouth disease.

(a) Occurrence, transmission, control in South Africa — Mr. C. J. van Heerden.

(b) Pathological-anatomical aspect — Dr. G. de Kock. Opener : Dr. J. H. Mason.

Among those who took part in the discussions on foot and mouth disease were Dr. Sheppard Cruz and Mr. J. H. N. Hobday, who gave an account of the disease as it occurred, and was controlled in their territories. (Portuguese East Africa and Bechuanaland respectively.)

In view of the pressure of time it was decided that the paper on " Rickettsiosis in Dogs " be not read, but be published in the next issue of the Journal.

12 – 1 p.m. An address on " Wild Animal Life in the Kruger National Park " by Col. J. Stevenson-Hamilton.

Dr. de Kock, in thanking Col. Stevenson-Hamilton for his very interesting paper, pleaded for closer collaboration between the veterinary profession and the Park authorities, as this might provide an explanation of many of the mysterious fluctuations shown by some species.

2 – 3 p.m. " The Lungsickness Campaign in Caprivi Zipfel " — Mr. G. McIntyre. Opener : Mr. S. G. van der Walt.

3 p.m. **General**—

(a) *Constitution* : The translation by Dr. Mönnig was approved, excepting the name. The latter give rise to considerable discussion and finally Dr. de Kock proposed that a Committee consisting of Drs. Mönnig, Fourie, and Messrs. van Heerden and Coles, be appointed to investigate the matter and to report back to Council. This was agreed to.

(b) *Resolutions* : Dr. N. F. Viljoen on behalf of the Committee appointed (*viz.* Drs. Viljoen, Fourie, Mönnig, and Martinaglia) proposed that :

1. " In view of the seriousness of the problems of measles in bovines and pigs, and tapeworm infection in man in South Africa, this Congress of the S.A.V.M.A. urgently

requests the Department of Agriculture, and of Public Health, to formulate an active policy necessary for a campaign, and to take steps for its immediate execution, in order to combat this menace to human health, and to the economy of stock farming and the beef export trade."

2. Dr. de Kock suggested that next year's Conference should deal with milk and meat hygiene, and that one or two papers on this subject be also obtained from members of the medical profession. Agreed.
3. Dr. Fourie proposed that this meeting forward a letter to the Secretary for Agriculture thanking him for allowing such a number of State veterinarians to attend this meeting, which proved very instructive. Agreed.
4. Mr. van Heerden proposed a hearty votes of thanks to the President for having again given up a lot of his valuable time to come up and guide this meeting through its deliberations with his usual tact and goodwill.

The meeting closed at 4 p.m.

S.W. J. v. RENSBURG,
HON. SEC.-TREAS. S.A.V.M.A.

Minutes of the Annual General Meeting of the Natal Branch of the South African Veterinary Medical Association.

Held at Allerton Veterinary Research Laboratory, Pietermaritzburg, on 26th April, 1938.

Present:

Messrs. R. Paine, W. P. Hamblyn, J. Nicol, W. A. Dykins, Dr. H. O. Mönnig, C. M. Sharpe, A. F. Tarr, J. H. B. Viljoen, J. Zwarenstein, F. B. Wright, W. G. Barnard, L. W. Rossiter, W. F. Belonje, F. Hellberg, Z. B. Klüge, T. H. Sandrock, D .A. Haig, J. L. Mainprize, N. C. Starke, J. H. Schoeman, J. L. Doré, Dr. P. R. B. Smith, S. T. Jackson, F. J. Carless, A. M. Diesel, H. J. Franz. Visitor: Mr. Chas. Tyler.

The President, Mr. Diesel, welcomed the members and expressed his pleasure at seeing two of the older members of the profession, Messrs. W. P. Hamblyn and Chas. Tyler, present at the meeting.

The Secretary then read his report after which it was unanimously agreed to take the minutes of the last Annual General Meeting as read.

After reading the financial statement, the Secretary explained that the position was satisfactory, but that the subsidy for 17 subscriptions collected during the 1937 meeting were not included in the S.A.V.M.A. statement.

He further pointed out that one member had been struck off the list, as requested by the Parent Body. Another member's subscription for 1933-4, 1934-5 and 1935-6 could not, for some inexplicable reason, be traced, and he suggested that the matter be dropped, which was unanimously agreed to by the meeting.

The following new members were proposed, seconded and welcomed : Messrs. Hamblyn, Hellberg, Klüge, Belonje, Doré, and Sandrock. Mr. Chas. Tyler was proposed and seconded but he declined, as he did not anticipate remaining in the country for long.

The following Office Bearers were elected :—

President : R. Paine.

Vice-Presidents : A. M. Diesel, W. A. Dykins, N. C. Starke.

Secretary : L. C. Blomefield.

Committee : J. Zwarenstein, C. M. Sharpe, W. G. Barnard, J. H. Schoeman, S. T. Jackson, F. B. Wright.

Without further business the retiring President, Mr. A. M. Diesel, after an address, vacated the chair for Mr. R. Paine who, in an address, thanked the meeting for the honour conferred on him.

The following papers were then read and discussed :—

(1) *The Therapy of Parasitic Diseases*—Dr. H. O. Mönnig.

(2) *East Coast Fever* and *Single Death East Coast Fever*—Mr. A. F. Tarr.

(3) *Anaplasmosis—Its Prevention and Treatment*—J. J. Zwarenstein.

The meeting commenced at 9.30 a.m. and concluded at 4.30 p.m.

L. C. BLOMEFIELD,

HON. SECRETARY, S.A.V.M.A. (NATAL BRANCH).

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