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NOTE ON THE CO-RELATION OF SEVERAL DISEASES OCCURRING AMONG ANIMALS IN SOUTH AFRICA.

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IN South Africa one finds quite a number of ailments occurring in the domesticated animals, and while the onset, symptoms, and morbid anatomy of those affected show the ailments to be peculiar to Africa, the names given to the maladies are unknown in any other country.

Horses and mules are liable to the enormously fatal malady known as Horse-sickness. The mode of onset, symptoms, and morbid anatomy of this disease have been already fully described[1].

High-bred goats and sheep are exceedingly liable in certain areas of Cape Colony to a disease known as Heart-water. The areas within which this disease exists begin at the coast in the Eastern Province and extend inland irregularly for some distance.

In the animals which die of this disease it is common to find a considerable quantity of pale yellow serous effusion in the pericardium. The epi, myo and endocardium commonly show little or no departure from the normal. The pleural cavity may contain some yellow serous fluid or may be empty. The lungs may be almost normal or there may be some exudation found in the interlobular tissue. While the lungs as a whole may be quite pale in colour there may be found irregular, sharply defined, chocolate-coloured patches of congestion. There may also be found more or less of gastro-enteritis, and in cases running to the full term the gall-bladder may be distended with more or less inspissated bile, which, while usually of a deep bottle-green, may be sometimes brown in colour.

[1] *Proc. Royal Society*, Vol. 67. *Reports of the Director of the Col. Bact. Inst.*, 1894 and 1899.

The incubation period after the intravenous inoculation of a clean goat with 5 c.c. to 30 c.c. of the blood of an animal dying of the malady is as a rule about ten days. This period, however, may be greatly diminished or extended; from a few days up to nearly three weeks in some cases.

From the point of view of experimental infection, the results which I have obtained from experiments conducted on nearly five hundred goats are paradoxical in the extreme. When I first began my investigations I used goats which were born and reared on Mr Palmer's farm, near Grahamstown, which was believed to be outside of the area infested with the disease. On infested farms the mortality during the summer season is very high, but no unnatural mortality had occurred on this farm during the past ten years at least.

The material used for purposes of infection was the blood taken from animals dying of the disease at Koonap and at Somerset East. The blood was either simply defibrinated or mixed with a small quantity of a solution of neutral citrate of potash. I was unable to find that either had any advantage over the other as an infecting agent. Subcutaneous injection of doses as large as 40 c.c. almost always failed to produce death, although some oscillation of the temperature of the inoculated animals was observed. Intravenous inoculations of doses up to 30 c.c. were uncertain. Where the animals inoculated in this way developed the disease and died there was no certainty that their blood would produce the virulent disease in others. Failures have occurred even with the injection of 100 c.c. into the jugular vein. In some cases blood which was drawn from inoculated animals, which did not themselves die, proved capable of setting up the virulent disease in others.

As further indicating the paradoxical nature of the malady I may add that in by far the greater number of these goats which had resisted inoculation it was proved that an inoculation of even a much smaller dose of blood at a later date, or exposure of the animals in an infested veld, was attended with the production of the malady, followed by death.

I felt, therefore, that these goats, which largely resisted infection although being not immune, had acquired what one might term a modified resistance or acclimatization.

This is the more probable from the following circumstance. Mr Thomas Hoole, a well-known breeder at Highlands, made a purchase of a considerable number of goats from a district of Somerset where

the disease is known to be absent. After purchasing he sold part to Mr L. White, whose farm lies many miles distant. After these goats had been placed on the respective farms they began to die of heart-water, while contrarily the animals belonging to the place did not die. On both farms heart-water occurs.

I may further add that the ordinary Boer goat is practically insusceptible to the malady, and that the pure bred Persian goats possess a high degree of insusceptibility to natural infection.

In some parts where the disease only occurs to a slight extent I have had it reported to me by the farmer as being gall-sickness; thus called as the gall-bladder is often very much distended with bile.

Since that time I have imported all goats by train from a clean area in Somerset, and in these animals I have found it much easier to keep up a strain of infection from animal to animal. Still, however, inoculation frequently fails, and I felt constrained to report to my Government that goats evidently were not the proper animal host for the contagium of this malady. In sheep the conditions are practically the same.

In cattle a number of names are applied to diseases by the farmers, which have given me immense trouble in the attempt to identify. The names with which I shall now deal are:

1. Imapunga (Kafir: "Lung").
2. Boschziekte (Bush-sickness).
3. Gall-sickness.
4. Veld-sickness.
5. Black Lung-sickness.
6. Rivierziekte (River sickness).

There is no official work which enables anyone to identify the maladies above named, but since my own work has been completed I have obtained, a few weeks ago, a copy of the "Report of the Commission appointed to inquire into Disease in Cattle in the Colony," dated 1877. Among the numerous minutes of evidence I desire in particular to refer to the very valuable and strikingly accurate observations of the late Mr J. Webb, who owned then a farm near Grahamstown, consisting of sour veld as contrasted with the sweet veld of the Karoo.

"Question 2705. Have you noticed any change in the veld during the last few years?

"Yes, stock of all kinds have been doing badly, and sheep and goats it is now impossible to keep on farms which at one time were considered to be the best grazing farms in this neighbourhood.

"2706. What do you think is the cause of it?

"My opinion is we have a tick which made its appearance in the last eight or nine years, I suffered from them then, a bonte-tick, small, like a ladybird. I was farming on a farm without ticks; directly this tick appeared all my stock did badly, calves died of gall-sickness, boschsickness, one man lost 2000 or 3000 sheep and goats, I believe the tick caused it. I have also shot bush-bucks suffering from the same causes, this was at Southey's Poort, Fish River. As this tick increases so diseases increase, for wherever the tick is found there are the same diseases; the tick has now travelled over 60 miles.

"2710. Did you open and examine them? A few sheep, not often.

"2711. What did you notice? The heart-bag and chest full of water.

"2716. You have had large experience in cattle? Yes.

"2717. What do they die of? Below Grahamstown of gall-sickness. North very few die compared to the south. We have three sicknesses here, called by the farmers: gall-sickness, bosch-sickness, and sweet veld-sickness, I believe they are all the same."

It is generally known to farmers that if Karoo cattle are brought down to the coast areas of the Eastern Province the greater number will die. For restocking the northern territories large numbers of cattle have been bought, of which great part are Karoo animals. Of these many have been grazing on the same farm which Mr Webb spoke of, and of the Karoo cattle a very large number are already dead.

Horse-sickness co-related to Veld-sickness.

During the earlier part of my work in this colony I endeavoured without success to transfer the disease known as horse-sickness from the horse to cattle. The cattle used in these experiments were of the class known as Zuurveld (sour veld) cattle.

It has been known during several generations of farmers that if cattle living in sweet veld areas are brought to Zuurveld areas they are exceedingly liable to die very soon after their arrival. Owing to this the Zuurveld cattle, sold on the Grahamstown market, fetch higher prices than those from sweet veld, and, indeed, most farmers in this area refuse to purchase sweet veld cattle at any price owing to the area being a Zuurveld one. If then sweet veld cattle die when transferred to sour veld, what is the nature of the disease produced in them?

After inoculation for Rinderpest had been well advanced in the

Eastern Province it was found necessary to be exceedingly careful of the kind of animals used to produce virulent blood, and a large number of animals were conveyed from sweet veld areas to a camp at Waai Nek, about two miles from this Institute. Of these considerable numbers died, but the cause of their death was not understood, and the enormous pressure of work connected with Rinderpest prevented definite investigations being taken up for this purpose. We had to content ourselves with attempting, by treatment, to save as many as possible.

Most of the deaths were reported to me by Mr Robertson as belonging to Steynsburg cattle and he emphasized the fact of their being sweet veld cattle while our veld was Zuurveld.

While this condition of things was in progress, a Bechuana boy (a herd brought from Taungs who had worked with me there) living at the camp, came in and reported to my veterinary assistant, Mr William Robertson, M.R.C.V.S. (now assistant to the Colonial Veterinary Surgeon), that one of the cows had died of "Paardeziekte." As a result of this report Mr Robertson rode to the camp and returning almost immediately stated to me that an animal had just died and that it had a cloud of white foam lying around the nostrils and mouth. I immediately proceeded with him to the camp and saw the animal lying dead. It had a large quantity of white foam lying around the nose and mouth, exactly as one sees so frequently in the cases of horses which have died of horse-sickness.

On making a post-mortem examination the similarity to horse-sickness was extended, since we found the following conditions:

The lungs showed an exceedingly well-marked interlobular yellow serous exudation. This was so characteristic, that, had the lungs only been shown to me, I should have believed they had been taken from a horse that had died from horse-sickness. In another case of the same sort which had been dead some hours before the post-mortem examination was made, there was in addition some emphysema of the apices and free edges of the lungs. The pericardium contained an excessive amount of yellow serous fluid. No abnormal condition was seen in the abdominal cavity except the spleens, which were slightly enlarged, the liver, which was in both cases congested and friable, and some exudation of serous material into the omentum and mesentery. No micro-organisms of any kind were found in the blood.

This occurrence was somewhat surprising to us both and I thereupon determined upon attempting once more the infection of clean cattle

with horse-sickness from the horse. Accordingly I took several animals of a new consignment to the Institute and then under careful conditions carried out the experiments.

On the 4th February, 1898, Mr Robertson and I inoculated a clean young ox with 30 c.c. of fresh horse-sickness blood which we injected into the jugular vein. Some reaction occurred during the first few days after which the temperature fell, but on the 16th day it rose. The temperature maxima were as follows till the moment of death.

16th day ...	106·4° Fahr.	19th day ...	107·2° Fahr.
17th „ ...	106·6° „	20th „ ...	106·2° „
18th „ ...	107·0° „	21st „ ...	died.

"The post-mortem was of interest inasmuch as nearly every symptom of horse-sickness was reproduced, the interlobular pulmonary effusion, the pleural and pericardial effusions[1]."

Ten c.c. of the blood of this ox was used to inoculate by subcutaneous injection horse No. 122. After an incubation period of eight days the temperature rose, and the animal died, on the 13th day, of typical horse-sickness.

Ten c.c. of the blood of this ox was also used, by intravenous injection, to inoculate a second ox, in which the temperature rose to 106·4 on the 11th day, to 107·4 on the 12th, and which we killed when dying of the disease on the 16th day.

At that time, having succeeded so completely in transferring the disease to cattle, I tried also to infect goats. The goats, however, were born in this area and are more insusceptible than goats taken from other areas. Of the several goats inoculated none died, but most had severe reactions. One of these goats, which had been inoculated with 10 c.c. subcutaneously of preserved horse-sickness blood, and developed a high temperature as a result, was bled on the 10th day after inoculation.

A young ox was inoculated with 30 c.c. of this blood by intravenous inoculation on the 18th February, 1898. During the first few days the temperature was irregular and then took a normal course, but thereafter the following temperatures were recorded.

10th day ...	104·6° Fahr.	14th day ...	107·4° Fahr.
11th „ ...	106·6° „	15th „ ...	107·4° „
12th „ ...	106·6° „	16th „ ...	105·4° „
13th „ ...	107·0° „	17th „ ...	96·8° death.

[1] *Vide Report of the Director of the Col. Bact. Inst.* 1898.

In this case the post-mortem examination showed very well-marked symptoms of heart-water.

These experiments therefore showed:

(1) In a most definite fashion, that cattle, from sweet veld areas are more or less susceptible to horse-sickness.

(2) That the disease so produced was indistinguishable from that which had occurred spontaneously in our camp.

Still, however, I was unable to identify the disease, although I learned that it was well-known to the Kafirs under the name of Imapunga.

Shortly after this a number of deaths were occurring among young calves on the farm of Mr Hyde, and Mr Robertson and I who proceeded there obtained a post-mortem examination which enabled us to determine that it was the same disease which we had already seen in our camp.

During the past two years a very large mortality has occurred among young calves from this disease, but it is to be remarked that the old animals are either insusceptible or well-protected, since very few of the old animals, which have been accustomed to the veld, die of it.

In the case of animals, however, which are brought from sweet veld areas it is the rule rather than the exception for them all to die. I have had to import a considerable number of calves from other areas for use in the Institute, and among these a fairly heavy mortality has occurred from this cause when they have been allowed to run in the veld.

During the war a large number of fine trek oxen were sent to Grahamstown by the military authorities, and to the best of my knowledge almost all of these coming from De Aar, Naauwpoort, and Cradock eventually died. The following will show the results in two lots, coming respectively from Naauwpoort and Cradock.

(1) Oxen from Naauwpoort (16) which arrived at Grahamstown on the 23rd August, 1901.

1 died on	Sept. 25th	1 died on	Oct. 8th
1 „	„ 27th	1 „	Nov. 15th
1 „	Oct. 3rd	1 „	„ 24th

(2) Oxen from Cradock (14) arrived at Grahamstown on 2nd December, 1901.

1 died on	Dec. 24th	1 died on	Jan. 7th, 1902
1 „	„ 25th	1 „	„ 11th, „
1 „	„ 30th	6 „	„ 12th, „
1 „	Jan. 2nd, 1902	1 „	„ 13th, „
1 „	„ 3rd, „		

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I had an opportunity of examining some of these animals and was able to determine the identity of this disease with that which I had formerly seen occurring spontaneously and with that which I had produced by the inoculation of clean animals with horse-sickness blood.

While the Kafirs call this by the term Imapunga, I have found, by consulting transport riders whose experience extended over many years, that this disease is known to them under the name of Veld-sickness or Veldziekte.

The principal lesion is an exudation of a yellow serous fluid into the following structures:

(1) Subcutaneous: in and along the lines of the intermuscular fasciae.

(2) Sometimes but not always in the pleural cavity.

(3) Commonly into the interlobular tissue of the lungs. Sometimes it is present to an exceedingly slight degree here, and it is necessary to examine carefully to determine where the normal becomes abnormal since the interlobular tissue in ruminants is more lax than in the equids. In very many cases, however, one finds the interlobular infiltration forming bands from one-eighth to a quarter of an inch in thickness.

(4) Into the pericardium. The amount found in this situation varies within wide limits: in some cases it is but little in excess while in others the pericardial sac is filled. A variation is to be found also in horse-sickness: I have found in some horses only a few ounces of fluid while in others more than half a gallon was found in the sac.

(5) Around the base of the heart.

(6) In the anterior mediastinum.

(7) Between the lower border of the pleura and the diaphragm. I have several times found the exudation to form here a solidified layer nearly half an inch in thickness.

(8) Into the tissue of the omentum and mesentery.

(9) Into the submucosa of the intestines.

Secondary lesions:—(1) Collapse of lobules of the lung with a corresponding traumatic emphysema of the adjoining lobules.

(2) In cases which live for a day or two longer than the more highly susceptible animals it is common to find congested lobules of a dark, almost black colour. These lobules are sharply defined from those immediately adjoining, and from their somewhat superficial resemblance to the appearance seen in pleuro-pneumonia such cases are called by the farmers black lung-sickness.

(3) In some cases one finds extravasations of blood below the endocardium of the left ventricle, especially in relation to the attachment of the chordae tendineae.

(4) The liver is commonly congested and enlarged, and in the last stages the gall-bladder is distended. The bile is of a deep green colour as a rule, but in some cases is brown. When the quantity of bile is very small it may be of a somewhat syrupy consistence, but never shows the peculiar tenacious mucous character so well known in Texas fever.

(5) The small stomach is frequently the seat of patches of congestion, more or less of a red colour, which may even have gone on to active inflammation.

(6) The conditions seen in the small stomach may be found frequently in the intestines, and a general gastro-enteritis may even be set up.

(7) The spleen may be slightly enlarged but is firm in consistence. The malpighian bodies are more prominent than in the normal condition.

(8) A slight amount of yellow serous exudation may be found sometimes in the pelvis of the kidney: otherwise the organ is normal.

(9) In even the best marked cases the urine and the bladder are commonly absolutely normal.

(10) In cases which have been dead for some time and exposed to a hot sun there may be some patches of emphysema in the lungs.

On examination of the blood and of smears from the kidneys and liver no micro-organisms are to be found except in animals which have been dead for some hours, when a large putrefactive bacillus is frequently to be found. The blood is always of a good colour and the rapidity of coagulation is always increased.

The fever in these cases is commonly very high. A remarkable feature in the malady is the fact that animals may seem in perfect health, yet when the temperature is taken it may be found to be over 106° F. It is common to find animals showing symptoms of illness only a few hours before death.

As this disease is well defined in cattle and runs on parallel lines with horse-sickness in horses, I suggest that it should be denominated "South African cattle-sickness."

While the blood of the first ox proved virulent to an ox I found after three transferences through oxen that it becomes relatively virulent to the ox but may fail to produce virulent disease in the horse even when used in the fresh state.

Heart-water.

In my Annual Report for 1896 observations were made which at a later date were communicated to the Royal Society (Vol. 65) showing that the germs of Red-water or Texas fever may remain latent in the blood of cattle for long periods of time after their recovery from an attack of the malady, and that cattle born on red-water veld although they may not have been affected by this malady yet can and do carry infection, in a latent form, in their blood.

During my investigations into heart-water I began to have suspicions that something of the same nature was concerned in regard to the latter malady.

The following experiments show in how far these suspicions were verified.

Experiment 1: To prove that the contagium of heart-water may be communicated to a susceptible animal in a non-virulent form and passed in succession through several others, eventually being raised to full virulence in the passage: I obtained a number of clean goats by train from a clean area and enclosed them in a court-yard which, in turn, was bounded by stone walls. Along one side of this yard galvanized iron enclosures were erected into which the animals were placed while under experiment. The most rigorous care was exercised in regard to cleanliness of the place, each shed being at frequent intervals thoroughly cleaned and disinfected.

While I have found that horse-sickness blood can be preserved, by the addition of an equal volume of glycerine and water containing 1 per 1000 of phenol, so that it retains its virulence for at least three years, the blood of goats dying of heart-water when so treated loses its virulence in a few days' time. Such preserved blood does, however, almost always set up a slight oscillation of the temperature in animals inoculated with it.

Goat No. 252 was inoculated with 10 c.c. of glycerinated blood taken from an animal which had died of heart-water. The injection of this material was made subcutaneously on September 5, 1901. No febrile change was observed until September 21st. On this day the temperature rose to 107° F. in the middle of the day but regained the normal on the following day.

Goat No. 258 was now inoculated with 100 c.c. of the blood of No. 252. Some irregularity of temperature was produced but no very

definite reaction, and on October 9th (being the fourteenth day after inoculation) it was bled and Goat 265 was inoculated subcutaneously with 100 c.c. of its blood and with 25 c.c. injected into the jugular vein.

On the following day there was a sudden elevation of temperature to 106·4°, and on the fifth day the animal died of characteristic heart-water.

The two previously inoculated animals remained meanwhile in seemingly good health.

Experiment 2: To prove that Goats Nos. 252 and 258 through which the virus had been transmitted while in a non-virulent form were in no degree protected thereby against subsequent inoculation with virulent virus.

Goat No. 266 was inoculated on October 10th with 100 c.c. of the blood of 265 by subcutaneous injection and with 20 c.c. injected intravenously.

On the 8th day the temperature ran up to 105·4° Fahr.
„ 11th „ „ „ 104·8° „
„ 12th „ „ „ 106·4° „
„ 13th „ „ „ 104·4° „

when it died of heart-water.

(*Note.* During the progress of these experiments clean goats were always kept with the experimental ones, and, at the close of the experiments they were all inoculated with virulent blood and all died of heart-water.)

Goat No. 252, which had been inoculated as already seen with non-virulent blood, was now inoculated on October 23rd with 30 c.c. of the blood of No. 266, by intravenous injection.

On the 6th day the temperature rose to 104·4° Fahr.
„ 7th „ „ „ 107·6° „
„ 8th „ „ „ 107·4° „
„ 9th „ „ „ 106·6° „

when it died of typical heart-water.

Goat No. 258, which also had been already inoculated 28 days previously with non-virulent blood, was now inoculated on October 23rd with 30 c.c. of the blood of No. 266 by intravenous injection.

On the 7th day the temperature rose to 107·4° Fahr.
„ 8th „ „ „ 103·6° „

when the animal died of typical heart-water.

Goats Nos. 278 and 279 were each inoculated in the same manner as controls and in both cases the temperature began to rise on the eighth day afterward and death occurred on the eleventh.

In the above experiment, therefore, it is seen that the virus, which had originally passed through Goats Nos. 252 and 258 and was by that means raised to virulence, actually killed these animals when re-inoculated into them after its accession to virulence had been achieved.

Hence heart-water virus of which the virulence has been lowered does not necessarily afford protection to animals which have been inoculated with it.

Experiment 3: To prove, in such cases as those of animals Nos. 252 and 258, that an inoculation with weakened virus actually predisposes to subsequent infection with virulent blood.

In both of the above cases it is to be noticed that the incubation period was shortened as compared with the "control" animals, and I have further to add that this observation has been abundantly confirmed in a vast number of other cases.

Transmission of heart-water from goats to cattle.

Experiment 4: A clean ox from a sweet veld area was inoculated by the injection of 5 c.c. subcutaneously and 5 c.c. intravenously of blood from a goat which was dying of heart-water. On the 15th day after inoculation the temperature began to rise, reaching during the evening to 106·4°. During the five days subsequent it was maintained about 105° F. without remission, but the following morning it fell to 101·8° and on the same evening ascended to 107·2°. It died two days later.

On making a post-mortem examination I found the pericardium filled with fluid; there was some interlobular pulmonary infiltration and indeed there were produced conditions similar to those we are accustomed to find in goats dying of heart-water.

On the 16th day of the disease it was bled, and after defibrination of the blood, a goat was inoculated by the injection of 10 c.c. subcutaneously and 10 c.c. intravenously.

This goat died seventeen days later of typical heart-water.

The type of fever induced in cattle by the inoculation of horse-sickness blood is practically the same as that obtained by the inoculation of the same species of animal with heart-water blood.

The post-mortem appearances are likewise identical and agree in all particulars with those found in the endemic disease occurring in cattle

and known to the Kafirs as Imapunga, while having shown typical cases to experienced transport riders they have assured me that it is known to them as veld-sickness.

As I have already said, Karoo cattle coming to the coast areas are liable to become attacked, the coast cattle remaining in perfect health. Transport riders assure me that cattle from these coast areas can travel throughout the whole of South Africa except in the Tsetse-fly belts. I have ascertained that this disease occurs on the velds on which heart-water is known to exist.

The co-relation of veld-sickness and heart-water.

Experiment 5 was made to determine the relation of Imapunga or veld-sickness in cattle to heart-water in goats.

With blood taken from a Graaf Reinet calf dying of veld-sickness I inoculated Goat No. 312 on Dec. 16th by the intravenous injection of 30 c.c. of the blood. The temperature began to rise on the 5th day and the animal died on the 15th day of heart-water.

The post-mortem appearances were absolutely typical of heart-water occurring spontaneously among goats.

Goat No. 327 was inoculated in the same manner with the blood of Goat No. 312 on Dec. 30th and died on the 17th day of heart-water.

Goat No. 331 was inoculated in the same manner with the blood of No. 327 and died on the 15th day with similar symptoms.

I could not detect the slightest difference between those cases and cases of heart-water produced either spontaneously or by inoculation.

Experiment 6: To show that goats born and reared on a farm infected with veld-sickness are not so susceptible to that disease as are goats which have been reared on a clean veld.

Goat No. 305 from a farm on which veld-sickness exists was inoculated on Nov. 24th by intravenous injection of 20 c.c. of the blood from a calf which died of veld-sickness. A slight reaction followed immediately and soon subsided.

On Jan. 12th it received intravenously 30 c.c. of the blood of No. 327 at the same time as No. 331 of the previous experiment.

While this goat remained unaffected the clean goat No. 331 died.

Experiment 7: To show that goats reared on a farm infested with veld-sickness are relatively insusceptible but not immune.

Goat No. 315 from a veld-sickness infected farm, was inoculated

on December 14th with 10 c.c. injected subcutaneously and 10 c.c. intravenously of the blood of a calf which died of veld-sickness.

A slight febrile reaction of short duration followed. On the 12th January it received 30 c.c. intravenously of the blood of No. 327, and as a result died of the disease on the 13th day. (This result is in agreement with what we find obtaining among goats running on a heart-water veld when these are inoculated with heart-water.)

Experimental observation 8: To show that goats reared and running on a farm infested with veldziekte are relatively insusceptible to heart-water.

In my prefatory remarks I alluded to the fact that goats purchased on the farm of Mr G. Palmer (which is a veld-sickness infested farm) were relatively insusceptible to heart-water but not immune, since although they very frequently resisted the intravenous injection of heart-water blood, yet if a second inoculation was made at a later date they commonly succumbed.

Experiment 9: To prove that goats relatively insusceptible are not actually immune.

In almost every case where one of the goats from Mr Palmer's farm, inoculated with virulent blood either from Somerset station, Koonap, or that obtained by me from experimental goats, have withstood the intravenous injection of virulent blood I have found:—

(1) That they have been actually infected although showing no signs of disease, since with their blood I have been able to infect susceptible goats, which in some cases have died of the virulent malady.

(2) That if, after unsuccessful inoculation, they are allowed to remain in the Institute for several weeks, a subsequent intravenous inoculation of virulent blood is almost always successful in producing the disease and death.

Experimental observation 10: To show that goats, on farms in this and adjoining areas, reared and living there, are relatively insusceptible.

I have already referred to the experiences of Messrs Hoole and White which suffices as evidence in this respect. Co-relation of horse-sickness to heart-water, to veld-sickness *in cattle and to a condition known as veld-sickness* in horses:—

If horses which have been reared in the Karoo are brought down to the coast areas it is usual to find that they fall off in condition, and in some cases die. From what I have heard and seen I am constrained to believe that this condition is that which was referred

to in the report of Lieut.-Colonel Joshua Nunn, F.R.C.V.S., A.V.D., to the Director-General of the Veterinary Department of H.M. War Office in 1888 as the biliary form of horse-sickness. Among the farmers it is, however, commonly referred to as veld-sickness.

In my communication to the Royal Society (Vol. 67) I referred to the results which I had obtained by the inoculation of donkeys with the blood of animals dying of horse-sickness and thereafter using the donkeys' blood for the inoculation of horses.

Since that communication was made I have been able to extend experiments of that class and the results may be summarized as follows:

1. The reaction produced in the donkey is no guide to the result which may follow the inoculation of its blood into a clean horse. The reaction may be slight or may be fatal.

2. If the donkey's blood is drawn at the tenth day and used to produce in a clean horse a violent reaction, then the blood of the same donkey if drawn two or three days later (without any further re-inoculation) will cause a much more violent reaction, and possibly death from virulent horse-sickness.

3. If a mild reaction is produced it may be of the nature of high temperature with remissions, or if still milder, may have a lower degree of fever with long intermissions.

In the case of animals which suffered from the last form of fever it was always noticed that they fell off in condition to a remarkable extent, becoming mere skeletons.

If killed or dying as late as the 50th day, one found evidence of horse-sickness in the form of exudation of serous material into the subcutaneous tissues, the interlobular tissue of the lungs, into the mesentery, the pleural cavity, and sometimes into the peritoneum. In the interventricular groove of the heart one always found some serous exudation, and the vessels lying here were always opaque owing to an infiltration into the vascular coats. The condition might be regarded as a sort of chronic horse-sickness.

The inoculation of 10 c.c. of heart-water blood into a clean horse produced similar phenomena, the animal dying two months after inoculation.

I have only made a few inoculations with heart-water into horses, and in some cases even a large dose (50 c.c.) has only produced a transient febrile reaction.

During the war, camps were formed for the receipt of farmers' horses, and reports reached me that horses running in some of these

camps were dying in large numbers from "poverty," "scab," etc., and as these camps are infested with veld-sickness it seemed to me that probably they were suffering from the "chronic form of horse-sickness" which I had experimentally produced.

On December 29th I proceeded to the protection camp at Thorn Park in this district accompanied by the local officers, Mr E. White and Mr Dalton. I saw no dead animals, as these had been already buried, and therefore decided to shoot any one which I might see in a poor condition. After several hundreds had galloped by I determined upon one which seemed poor enough, although it galloped quite freely. One of the officers then managed to bring it down with a rifle shot, and we at once proceeded to make a post-mortem examination, the appearances noted being as follows :—

The subcutaneous tissue was not invaded to any definite degree by exudation, although along the lines of the great vessels in the neck there was evidence that it had existed, but had coagulated and was now in process of absorption, leaving tough lines of dry exudation.—The pleural cavity contained about one gallon of a clear yellow serous fluid.—The lungs showed patches of congestion, some of which were deep liver-coloured. There was a definite amount of subpleural infiltration of serous fluid. There was also a widespread condition of interlobular infiltration of serous exudation.—The pericardium or heart-sac contained about 40 ounces of clear yellow or straw-coloured serous fluid, and some mass of coagulated gelatinous material produced by the coagulation of the fluid.—The base of the heart was surrounded by a huge gelatinous mass, and the interventricular groove was filled up by the same material.—The aorta and the larger vessels of the interventricular groove were invaded by the exudation, and the latter were rendered absolutely opaque, looking like white clay-pipe stems lying in a jelly.—Some fluid was also found in the peritoneal cavity, but no other characteristic pathological lesion was found.

Two days later Mr Dalton proceeded to another camp, and having shot a horse there, brought to me the heart and lungs *en masse*. The conditions found here were identical with the foregoing case, but rather more aggravated in type.

I cannot regard these and similar observations which I have made otherwise than as indicating that the condition which I produced in clean horses, by heart-water blood inoculation, and also by the injection of the blood of horse-sickness inoculated donkeys, is of the same nature as that which existed among the horses of the protection camps.

In cases where I carried my experimental inoculations so far as to produce perfect protection against horse-sickness, the animals immediately thereafter began to put on flesh.

Transmission of horse-sickness from horses to goats.

As I have already said the inoculation of even a large dose of heart-water blood into a horse may fail to be attended with any very definite result.

Conversely the inoculation of horse-sickness blood into goats was attended with uncertain results. In my first experiments, out of seventeen inoculated at different times, a febrile reaction occurred in only ten and none died. These goats, however, were obtained from Mr Palmer's farm. Since then I have used absolutely clean goats and have had further success.

On March 7th, 1902, I inoculated goat No. 381 with 20 c.c. of fresh horse-sickness blood by intravenous injection. It died three days later.

On post-mortem examination I found an enormous interlobular exudation into the lungs and pericardium. In the latter the whole exudation was absolutely solid.

This remarkable result is somewhat to be compared with experiments which I made a few years ago in inoculating a goat and a sheep with the serum of a "salted" goat which had been reinfected by inoculation a week previously.

The sheep and goat were inoculated in the forenoon and were found dead the next morning with symptoms very similar to those just recorded.

With the blood of goat No. 381 I now inoculated No. 383 by intravenous injection of 5 c.c. of defibrinated blood on March 9th.

Some fever followed, and on the 10th day it had a temperature of 106°, making however a good recovery.

On March 20th I bled this goat and inoculated No. 393 with 5 c.c. by intravenous injection.

After an incubation period of six days the temperature began to rise and the animal died on the 16th day. On making a post-mortem examination I found the usual signs of heart-water.

No. 393 was used to inoculate No. 408, which died on the 14th day.

No. 408 was used to inoculate No. 411, which died on the 13th day.

No. 411 was used to inoculate No. 419, which died on the 11th day.

This experiment which has been carried out with every care as regards the keeping of control animals in contact during the experiment and subsequently showing by inoculation that the controls were still susceptible to virulent infection, admits me to say that horse-sickness can be transferred to goats, and that, when acclimatized to the goat, it produces in this animal a virulent disease which is indistinguishable from the endemic disease of goats which is known in South Africa as heart-water.

Heart-water produced by the inoculation of the blood of *protection-camp horses:*

I was enabled to get a protection-camp horse sent into the Institute and while there I bled it and inoculated goat No. 365 with 30 c.c. intravenously and 30 c.c. subcutaneously on February 11th.

On the 7th day the temperature rose and remained high, 105° F. and slightly over, until the 11th day, when it fell to 101° and the animal then died.

On making a post-mortem examination I found oedema of a semi-transparent character at the base of the heart extending up the aorta. The lungs were pale, but the left lung had a dark patch of congestion about two inches in diameter, and being sharply circumscribed within a group of lobules. The pericardium was quite filled with a clear yellow serous fluid which quickly coagulated when transferred to a glass. The conditions found were thus absolutely typical of what one obtains in ordinary heart-water.

I therefore conclude that the contagium which causes horse-sickness in the equids of South Africa is responsible, under conditions of relative virulence, for the infection of other species of the domesticated animals.

The means by which the virulence becomes relatively altered is not entirely clear, but my colleague the Colonial Entomologist has been able to produce heart-water in goats and calves at Cape Town by means of the progeny of bont-ticks taken in the Eastern Province from infected goats. In this way therefore the very striking observation of the late Mr Webb has been proved to be correct. One is not, however, yet able to say that heart-water is not conveyed by any means other than the bont-tick.