Further report on the tsetse fly disease or nagana in Zululand, by Surgeon-Major David Bruce, A.M.S.

Wellcome Collection
183 Euston Road
London NW1 2BE UK
T +44 (0)20 7611 8722
E library@wellcomecollection.org
https://wellcomecollection.org

NAGANA DISEASE IN ZULULAND

MAJ. DAVID BRUCE.

1896 & 1903.

Further Report

ON THE

TSETSE FLY DISEASE OR NAGANA,

IN ZULULAND.

BY

SURGEON-MAJOR DAVID BRUCE, A.M.S.

UBOMBO, ZULULAND,
29th MAY, 1896.

London:
HARRISON AND SONS, ST. MARTIN'S LANE,
PRINTERS IN ORDINARY TO HER MAJESTY.

To His Excellency

The Hon. Sir Walter Hely-Hutchinson, K.C.M.G.,

Governor of Natal and Zululand, &c., &c.

Your Excellency,—I have the honour to inform you that in accordance with instructions received from you, I left Pietermaritzburg on the 21st August, 1895, and arrived at Ubombo, Zululand, on the 8th September, 1895, for the purpose of investigating the Tsetse Fly Disease, or Nagana, as it occurs in Zululand.

I had the honour to submit to you a Preliminary Report, containing a statement of the results of the investigation up to the beginning of December 1895, and I now beg to forward a Further Report.

This Further Report contains:—

1.—A description of the Tsetse Fly, with experiments designed to show the part this Fly takes in the causation of the disease.

2.—A description of the Hæmatozoon or Blood Parasite, which is the cause of the disease.

3.—The results of experiments having for their object proof of the connection which exists between the Big Game and the spread of the disease.

4.—A description of the disease as it affects domestic animals, with illustrative cases.

5.—Inoculation and feeding experiments to show the communicability of the disease from affected to healthy animals.

6.—Treatment of the disease, prophylactic and curative.

I have the honour to be,
Sir,
Your most obedient Servant,

DAVID BRUCE,
Surgeon-Major A.M.S.

Ubombo,
Zululand,
29th May, 1896.

CONTENTS.

LIST OF ILLUSTRATIONS.

Tsetse Fly Disease or Nagana.

1.—DEFINITION.

The Fly Disease or Nagana is a specific disease which occurs in the horse, mule, donkey, ox, dog, cat and many other animals, and varies in duration from a few days or weeks to many months. It is invariably fatal in the horse, donkey and dog, but a small percentage of cattle recover. It is characterised by fever, infiltration of coagulable lymph into the subcutaneous tissue of the neck, abdomen or extremities, giving rise to swelling in these regions, by a more or less rapid destruction of the red blood corpuscles, extreme emaciation, often blindness, and the constant occurrence in the blood of an infusorial parasite, either identical with or closely resembling the Trypanosoma Evansi found in Surra, a disease of India and Burmah. On *post mortem* examination the following changes are noticed: deposition of a yellow jelly like material in the subcutaneous tissue, inter-muscular layers, and under the serous covering of the heart; with purplish stains or ecchymoses in various regions, as on the inner aspect of the skin, the serous membrane covering the lungs, and outer and inner surfaces of the heart; enlargement and softening of spleen and congestion and fatty degeneration of the various organs.

2.—NOMENCLATURE.

This disease in South Africa may generally be said to have been called the "Fly Disease" by European travellers and hunters, and Nagana by the natives and those white settlers in Zululand who have come much in contact with the natives. The term "Fly Disease" has of course been given on the supposition that the disease is caused by the bite of the Tsetse Fly, and the term Nagana from the symptoms presented by the animals suffering from the disease, the word Nagana meaning in Zulu to be low or depressed in spirits. In past times the disease was known by the name Injoko, and at the present in some parts of the country, as in the valley of the Black Umfulosi, it is called Munca, from the sucked out appearance of the diseased animals.

M. Scloss, a Belgian Engineer, who came from the Congo to the Selati Railway in 1894, recognised the disease as being the same as "la mouche" in the Congo State.

3.—DISTRIBUTION IN ZULULAND.

For the purpose of future reference it would be well to give as fully as possible in this Report the distribution of the disease in Zululand, and to this end I intend to address the various Resident Magistrates in their several districts asking for information to enable me to prepare a map showing the localities where the disease is endemic.

A map of this kind will be more useful than a mere list of places.

Broadly it may be stated that the disease is limited to certain tracts, the physical conditions of which imply heat and moisture. These tracts in Zululand are situated in the level coast plain which extends some 50 miles inland, and in the river valleys which enter or debouch on this plain. From Ubombo, situated on the summit of one of the hills forming the chain of the Lebombo Mountains, a good view is obtained of this level coast plain, stretching from the base of the mountains to the sea some 60 miles off. It looks as level as a billiard table, and is covered as far as the eye can reach with a dense thorny scrub of mimosa, which at this time of the year, and at this distance, is olive-green in colour. Streaking the level expanse are numerous open spaces or glades covered with grasses, vivid green in colour. This huge plain stretches as far as the eye can reach to the north and south and merges in the blue of distance and of the sea to the eastward. A few miles to the south, the River Mkusi can be seen winding across the plain, having just passed through the Lebombo range by a deep cañon or poort, to fall into St. Lucia Lake, its course being marked by the denser vegetation along its banks; and some dozen miles to the north, another river, the Pongola, runs out into the plain in the same manner, to turn northward to Delagoa Bay. The strip of country opposite, lying between the two rivers and extending some 15 miles out, is "Fly Country," the home of Nagana and malaria, and uninhabited except by wild animals.

4.—HISTORICAL ACCOUNT.

In a fuller report it may be interesting to trace the history of this disease as it has occurred in Zululand during the last half century. How it has broken out in certain districts in certain years where it was before unknown, or disappeared in others where in previous years it had existed. Suffice it to say in this Report that the disease has evidently followed the laws of other infectious diseases, and spread some years to a greater extent than in others, when conditions favourable to it existed, or breaking out in epidemics and carrying off large numbers of domestic animals, where before only stray cases had occurred. The evidence, moreover, goes to show that the disease has existed in the lower tracts of the country time out of date, and is in no sense a new disease.

5.—ETIOLOGY.

The opinions of the Europeans settled in Zululand and of the natives themselves are so conflicting that little or no good will be gained by entering fully into their evidence.

Two theories are held.

A.—That the disease is caused by the bite of the Tsetse Fly. This is the European theory, and as everyone knows has been popularly prevalent ever since white men first landed in South Africa.

B.—That the disease is caused by the presence of large game, the wild animals in some way contaminating the grass or drinking water by their saliva or excretions. This may be called the native theory.

A.—THE TSETSE FLY.

In this Report I think it well to begin with the consideration of the Fly itself, not only on account of its historical value, but also because I shall try to show that the Tsetse Fly plays a most important part in the propagation of the disease.

Be it at once stated that I have not the slightest belief in the notion popularly prevalent up to the present that the fly causes the disease by the injection of a poison elaborated by itself, after the manner of a leech, which injects a fluid to prevent the coagulation of the blood, or the snake for the purpose of securing its prey or for defence, but that the Tsetse acts as a carrier of a living virus, an infinitely small parasite, from one animal to another, which entering into the blood stream of the animal bitten or pricked, there propagates and so gives rise to the disease.

1.—DESCRIPTION OF THE TSETSE FLY.

As the question often arises when a fly has been caught as to whether it is the real Tsetse or not, I think it may be found useful for the purposes of this Report if I describe the fly, as known to me, somewhat fully, and give a few drawings of its appearance.

The Tsetse Fly, Glossina morsitans (Westwood), is about 11 mm. or seven-sixteenths of an inch in length. The head is buff coloured, darker on the summit, with two minute dark brown spots on either side, and is distinct from the thorax; the eyes are dark, the thorax is gray with a slight tinge of green, streaked longitudinally with brown, and covered with minute dark dots marking the site of short black hairs. The abdomen is pale yellowish-white or buff coloured, and consists of six segments, the central four of which have large oval shaped dark brown spots on either side of the middle line, giving the appearance of a narrow longitudinal yellow medium line, crossed by transverse yellow bands, of which four are well marked and one is indistinct. These yellow lines on the upper surface of the abdomen, the one down the centre, and the four crossing it, are as clear cut and distinct as if marked or drawn by a pen and ruler. The legs are buff coloured touched with yellow, and the tarsal hooks are bifid. The transparent wings are about 10 mm. long and 4 mm. broad and are smoky in colour. When the Tsetse is at rest the wings fold over one another like the blades of a pair of scissors, and give the fly an elongated appearance (Plate I, figs. 1 and 4).

The Tsetse Fly does not lay eggs as do the majority of the Diptera, but extrudes a yellow coloured larva nearly as large as the abdomen of the mother. This larva is furnished with a black hood at one pole and two minute spikes at the other. It is annulated and consists of ten segments. Immediately on being born the larva creeps about with a good deal of activity, evidently searching for some cover or hole in which to hide. Having found a resting place it immediately begins to change colour, and after a few hours has turned into a jet black hard pupa or nympha (Plate I, figs. 2 and 3).

If these pupal cases are placed in a perfectly dry place, as in a wooden box, the perfect insect hatches out in about six weeks. From this it would appear that the life history of this species of fly is very simple, it only being necessary for the female

PLATE I

West, Newman chromo.

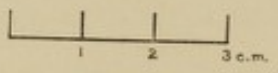
TSETSE FLY.

1 Fly at rest (natural size).
2 Larva (natural size).
3 Puparium (natural size).
4 Fly at rest (×2).
5 Fly with expanded wings (×2).
6 Fly before feeding (×2).
7 Fly after feeding (×2).

PLATE II.

1

2



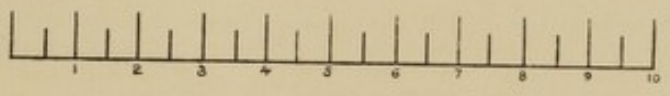
3

4

5

6

7



8

TSETSE FLY.

1, 2. Fly with expanded wings.
3. Fly at rest.
4. Fly pregnant with larva.
5. Fly during parturition.
6. Pupa.
7, 8. Flies in various positions.

insect to deposit the larva on the surface of the soil or in the grass, when the larva creeps into the nearest shelter, in a few hours becomes hard and black, and in five or six weeks hatches out into the fully developed Tsetse Fly.

It has often been surmised that this fly is bred in buffalo dung, but from a consideration of the foregoing facts it is evident that nothing is wanted except any moderately dry place.

A representation of the Tsetse Fly and its larva is given on Plate I.

Fig. 1 represents the fly in its natural size, with wings folded over each other scissor-fashion.

Fig. 2 represents the larva immediately after extrusion, yellow, segmented, with black hood and two minute spikes.

Fig. 3 shows the same larva a few hours afterwards, when it has become black and hard. Both these figures are also drawn natural size.

Figures 4, 5, 6 and 7 represent the fly magnified two diameters, in various positions, and before and after feeding.

Photographs reproduced in collotype will be found on Plate II.

It is astonishing with what rapidity the flies fill themselves; in as small a space of time as twenty or thirty seconds a fly will become swollen out like a balloon with bright red blood.

On entering "Fly Country," one is not left long in ignorance of the presence of the Tsetse. The natives may be seen slapping their naked legs, the dogs bite round, and the horses kick. The Tsetse, however, may be said to be somewhat local in its distribution in the Fly Country, being only met with now and then and in few numbers, until you enter some glade or clear space in the thorns, when suddenly the slapping, biting, and kicking go on with tenfold energy, and you can catch 30 or 40 flies in a few minutes.

The Tsetse has a direct flight, flopping, if I may use the term, suddenly on the animal attacked, and is very pertinacious. It affects in horses the legs, especially in the region of the fetlock, and on being detached from its position by the vigorous kick the horse gives, will only rise a few inches and again settle near the same spot. After the initial prick there is no irritation, and the animal will stand quietly while the fly has its feed.

In man the burying of the proboscis in the skin is accompanied by a sharp prick, which draws one's attention suddenly to the spot; but the pain is, as a rule, trivial and the subsequent redness and irritation very slight indeed,—in fact, less than that caused by the mosquito.

But under some circumstances the Tsetse may become almost unbearable. In January, when camping in the Fly Country close to the River Mkusi, these flies were very numerous, and in conjunction with the intense heat—the thermometer often registering 100° F. to 106° F. in the shade of a double tent—almost drove one mad. One can easily imagine the effect of the constant worrying of these pests by day, followed by mosquitoes at night, and minute ticks at all times, on any nervous system except the most stable. The horses fared no better, and could often be seen trotting about trying to escape from their tormentors.

About sunset seemed a favourite feeding time, and then the poor creatures would be surrounded by a perfect cloud of the flies, while some hundreds of them would be settled on them at the same moment.

I have not noticed them biting during the night, but that excellent sportsman and observer, Selous, states that by moonlight in some places they bite just as furiously as in the daytime.

The fly makes a loud buzzing sound when flying, but after its feed and at rest it emits a peculiar sharp shrill note, probably caused by an action of the wings.

In horses, dogs, and cattle I have usually failed to find any swelling or symptoms of irritation following the bite, but on several occasions a well marked soft swelling under the skin of the part bitten, as large as a hazel nut, was observed, and this persisted for some time.

The Tsetse is not at all easy to catch with the hand, especially during the day, being nimble and quick of movement, but at early morning or sunset they become more lethargic, and are then more readily secured. When I wanted living specimens for any purpose I provided myself with a butterfly net, with which it is easy enough to catch them unhurt.

The Tsetse differs from the mosquito in that both sexes are blood-sucking flies.

It is said that the fly follows the large game, but I never found them in any number in places in which a few minutes before large herds of buffalo or wildebeeste had been standing. Of course, the fly may have literally followed the game. One day, after killing a wildebeeste, I certainly found a large number of Tsetse on the dead animal, and again on a dead buffalo I counted as many as 40 flies feeding on it at the same time.

2.—EXPERIMENTS WITH THE TSETSE FLY.

These experiments may be divided into five series:—

(*a.*) Is the Tsetse Fly capable of giving rise *per se* to any local or general disease in susceptible animals?

(*b.*) Can the Tsetse Fly convey the disease from an affected animal to a healthy one?

(*c.*) How long does the Tsetse Fly retain this infective power?

(*d.*) How long does the blood of an affected animal remain capable of giving rise to the disease in a dried condition?

(*e.*) How long does the blood of an affected animal remain capable of giving rise to the disease if kept in a natural condition?

(*f.*) Does the Tsetse Fly under natural conditions convey the disease from animal to animal?

(*g.*) Is the Tsetse Fly capable of giving rise to the disease if taken out of the Fly Country into a healthy locality?

(*a.*) Is the Tsetse Fly capable of giving rise *per se* to any local or general disease in susceptible animals?

This question must be answered experimentally to satisfy those who believe that the Fly Disease is caused by an injection of a poison elaborated by the Tsetse itself. As I hope to prove later on that this is not so, but that the disease is caused by a blood parasite conveyed in some way from affected to healthy animals, a difficulty arises in the mode of experimentation. If the Tsetse Flies are brought direct from the Fly Country and placed on a susceptible animal they may themselves convey the blood parasite and so give rise to the disease. To get rid of this fallacy as far as possible I kept a certain number of flies in captivity at Ubombo for some days, in order that they might lose this infective power, if they ever had it, and then fed the same flies on the same animal for some time. The flies were kept in cages made with muslin sides to admit air, and a glass sliding door for light and to observe them feeding, and sometimes lived for several weeks under these circumstances if given a meal once in two to four days. They were fed by pressing one of the muslin sides of the cage against the skin of a suitable animal.

The following cases will sufficiently illustrate the manner and results of this experiment.

Exp. 210. Dog, English.

September 25th.—Placed five Tsetse Flies on the abdomen of a small dog after carefully shaving the part. The flies bit readily, and a small drop of blood oozed out on the surface of the dog's abdomen from each bite. The dog did not seem to be hurt much on being punctured. At no time was any swelling or irritation to be seen where the flies had settled.

These flies were fed on the same dog every two days, until the 28th November, without causing any local or general disease, when the experiment was stopped, and the dog was noted as being perfectly healthy three months afterwards.

But it may be objected that the number of flies used was too small, and that more positive results might have been obtained by using a greater number. In fact it might be argued that by using so few flies merely a method of immunising the dog to the poison was carried out. Let us see then the effect of larger numbers.

Exp. 242. Dog, Pointer.

January 4th.—Fed thirty Tsetse Flies on this dog.

January 14th.—Fed the same flies once a day since the 4th on the same dog.

February 29th.—This dog has remained perfectly healthy up to the present date.

Exp. 232A. Dog, Native.

December 5th.—Fed fifty Tsetse Flies, which have been up here for some time, on this dog.

December 19th.—Fed the same flies daily since the last date.

January 8th.—Dog in good health.

The above experiments then prove, I trust satisfactorily, that the Tsetse Fly is not capable of giving rise *per se* to any local or general disease in susceptible animals.

(*b.*) Can the Tsetse Fly convey the disease from an affected to a healthy animal?

That all blood-sucking flies are not capable of transferring the Fly Disease from affected to healthy animals is, I think, shown by the fact that up here at Ubombo, where we have several species of these pests, no single instance of the disease arising spontaneously has occurred, although healthy horses, cattle, and dogs have been constantly and closely associated with those suffering from the disease. Why this should be so is at present a mystery, and it is to be hoped that some point may be discovered which will throw light on the subject. There may be some anatomical peculiarity in the Tsetse which enables it to act as carrier, or there may be some undiscovered fact in the life history of the parasite associating it with this particular species of fly.

One fact, however, must be borne in mind, and that is the enormously greater number of Tsetse Flies in the Fly Country than any other species of blood-sucking fly met with there. I have seen as many as 200 Tsetse Flies on a horse at the same time; and when it is stated that I have often caught and put into a cage singly some 50 Tsetses in half an hour, some conception may be gained of the enormous number of Tsetse Flies which may visit a horse during its sojourn for one day in the "Fly."

The act of feeding is remarkably quick. From the moment of settling on the animal until the fly is fully blown out with blood is often as little as 20 to 30 seconds. But still this does not explain why none of the healthy animals up here on the top of the Ubombo have become infected by their diseased neighbours, because we have many small stinging flies—some species probably of Stomoxys—which cause drops of blood to exude from the legs and ears of our animals. But this I can assert, and it is an important point, that in no single case, as far as I am aware, has any case of the disease occurred up here due to infection from the diseased to the healthy.

That the Tsetse Fly can act as a carrier of the parasite is, I think, shown by the following experiments.

Exp. 228. Dog, Native.

November 21st.—Fed eight flies on this dog immediately after they had fed for a short time on a dog affected by Fly Disease.

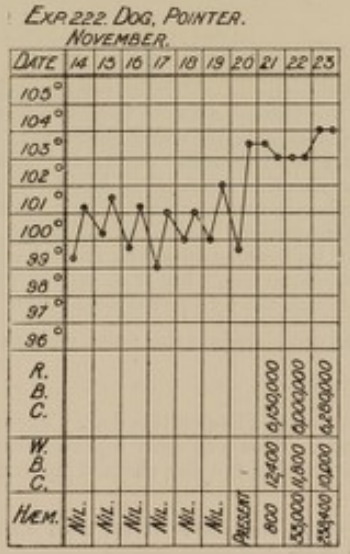
November 23rd, 25th, and 29th.—Repeated the above procedure.

December 5th.—Dog found by microscopical examination of his blood to be suffering from the disease.

Exp. 222. Dog, Pointer.

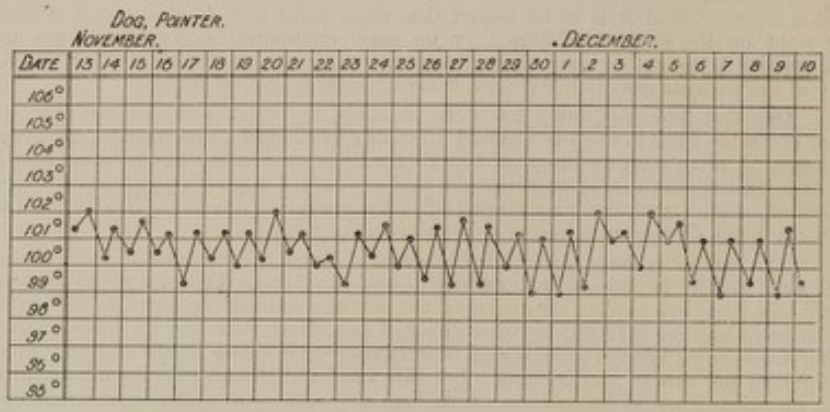
November 12th.—To-day I placed eight flies for a short time on a dog affected by the Fly Disease, and immediately thereafter on the above healthy dog. On the 13th and 14th the experiment was repeated, and on the 20th the dog was found to be suffering from the disease.

The following chart represents the onset of the fever.



A word of explanation is necessary in regard to the foregoing chart.

The normal temperature of a dog may be said as a rule to range between 99° F. and 102° F., although these limits may be somewhat exceeded without any departure from health. In order to illustrate this I give the following chart, which shows the temperature of a perfectly healthy dog taken per rectum at about 8 a.m. and 5 p.m.



The chart illustrating Exp. 222 is thus seen to range between normal limits for five days after the 14th November, the last date on which the dog was subjected to the bite or stab of the flies. This is the period of incubation, when the parasites are too few in numbers to be demonstrated in the blood by microscopical examination, and before they have been able to exercise any deleterious influence on the system as shown by a rise of temperature, loss of appetite, &c. On the evening of the sixth day, however, the temperature suddenly rises, and the hæmatozoa are found to be present in the blood.* The number of red blood corpuscles is taken in the usual way by means of a Gower's hæmacytometer, counting the corpuscles in the diluted blood in 10 squares and multiplying by 10,000, the white blood corpuscles by counting their number in 250 squares and multiplying by 400, and the hæmatozoa in the same way and at the same time as the white blood corpuscles. Should no hæmatozoa be seen during the counting of the white blood corpuscles, then two cover-glass preparations of undiluted blood are prepared and examined microscopically. If the parasites are found in the cover-glass preparation and not in the hæmacytometer, the word "present" is used instead of a definite number. Of course the hæmatozoa are frequently found to be present, although in too few numbers to be counted by the hæmacytometer.

Up to the present then from these two series of experiments it is seen in *the first place that the fly* per se *does not give rise to any local or general disease*, and this is further borne out by other experiments in which I placed minced up flies under the skin of dogs without any results, *and secondly it proves that the fly can act readily as a carrier of the Fly Disease from affected to healthy animals.*

(c.) How long does the Tsetse Fly retain this infective power?

Now as one of the main objects of this investigation is to establish the part, if any, that the wild animals play in the dissemination of this disease, and as my working hypothesis at present is that some species of animals living in the Fly Country harbour the Nagana parasite in their blood, and that the Tsetse Fly carries the infection from affected to healthy animals, much in the same way as the vaccinating needle carries the infection of vaccinia from child to child, it will evidently further this object, if it can be discovered how long the Tsetse Fly retains its infective power. In passing through Fly Country of course it is possible that a Tsetse Fly may be feeding on a wild animal one minute and the next have transferred itself to the horse, ox, or dog; but if it can be proved to retain the power of infection say for twenty-four hours, the fly's power to do harm is evidently much widened.

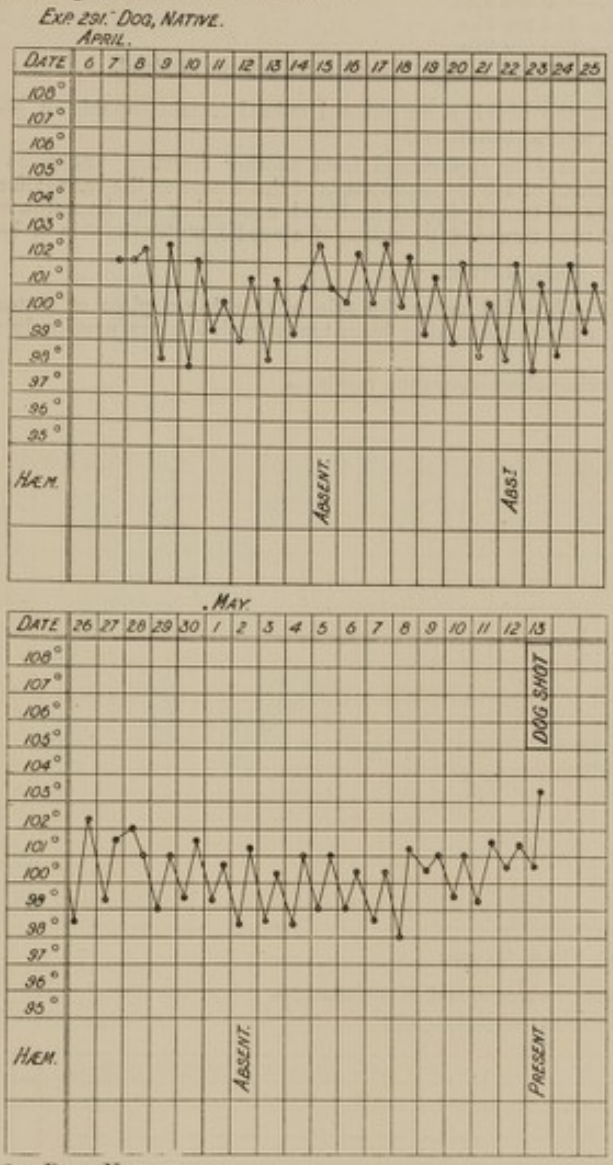
The experiments made to elucidate this point were the following:—

Exp. 291. Dog, Native.

April 6th.—Fed nine Tsetse Flies on this dog. These flies had fed on an affected animal twelve hours previously. This was daily repeated, care being taken to prevent any other method of infection by keeping the dog chained up constantly at some distance from the other affected animals.

* The three lowest spaces give the number of red blood corpuscles, white blood corpuscles, and hæmatozoa in a cubic millimetre of blood.

The following chart shows the result:—



Exp. 232. Dog, Native.

December 7th.—Fed twelve Tsetse Flies on this dog. These flies had been fed on an affected animal twenty-hours previously. This procedure was repeated, that is to say the same flies were fed alternately on an affected animal and on this dog, every twenty-four hours until the 14th January, when the healthy animal was found to be infected by the disease. This experiment lasted thirty-eight days before it was successful, so that it would appear that although the Tsetse can retain its infective power for twenty-four hours, this only exceptionally occurs.

I have also successfully tried an experiment, giving an interval of forty-eight hours.

Exp. 317. Dog, Native.

Fed Tsetse Flies on this dog forty-eight hours after they had been fed on an animal affected by Nagana.

June 26th, 1896.—Dog healthy; no hæmatozoa in blood.
„ 27th.—Fed twelve flies.
July 1st.—Fed ten flies.
„ 5th.—Fed six flies.
„ 9th.—Fed six flies.
„ 13th.—Fed six flies.

July 17th.—Fed ten flies.
" 23rd.—Fed ten flies.
" 27th.—Fed ten flies.
" 28th.—Dog off his feed. Hæmatozoa present in blood.

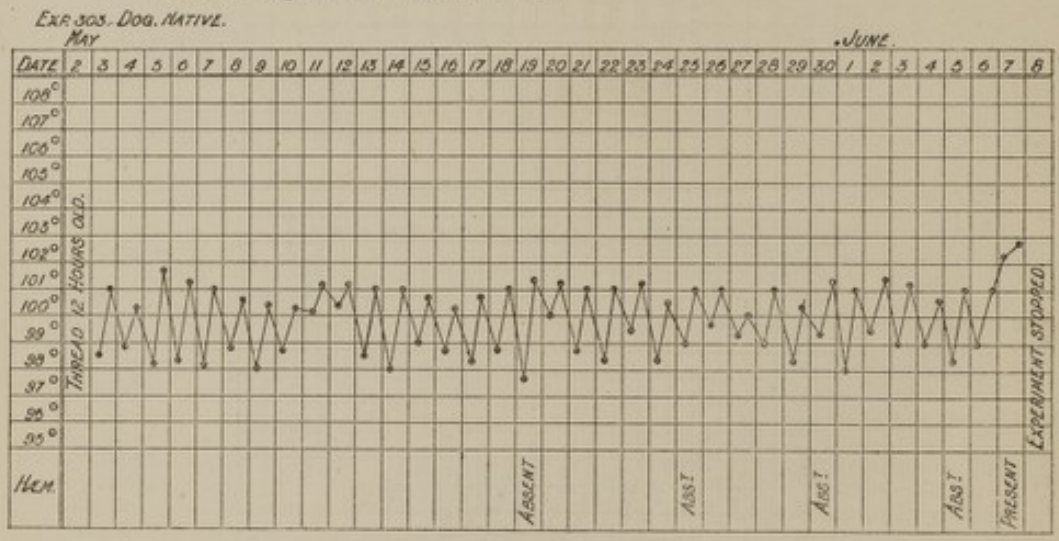
(*d.*) How long does the blood of an affected animal remain capable of giving rise to the disease in a dried condition?

This point may be approached from another direction and some control placed on the results. Instead of using the proboscis of the Tsetse as the conveying instrument, let us use short pieces of thread dipped in blood containing the hæmatozoa, dried, and at varying periods of time threaded, by means of a needle, under the skin of healthy dogs.

Exp. 303. Dog, Native.

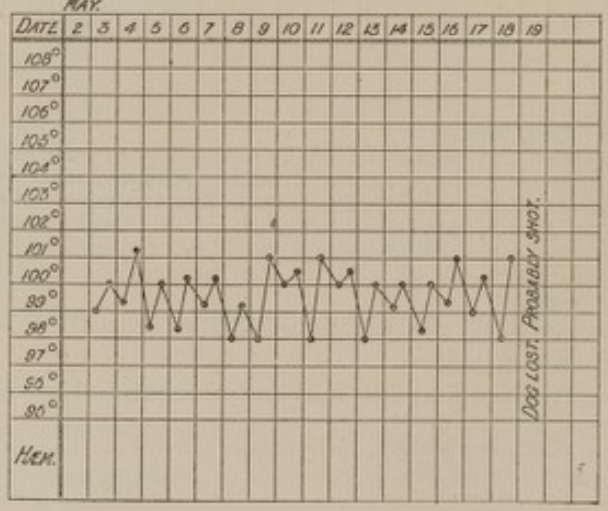
May 2nd.—Threaded under the skin of this dog a piece of cotton thread, which had been dipped in blood containing hæmatozoa twelve hours previously and then dried in the air.

The following chart shows the result:—



Exp. 304. Dog, Native.

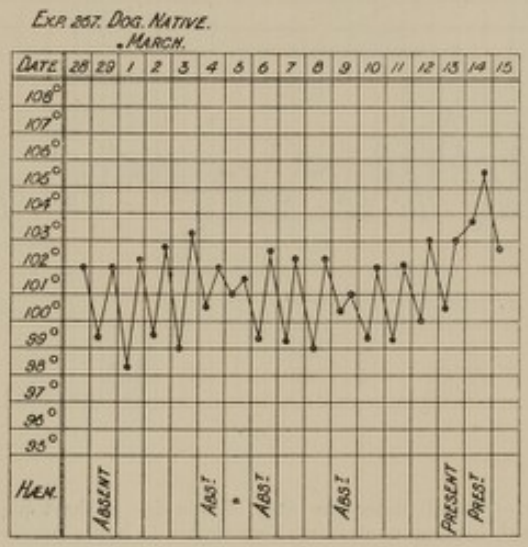
Same procedure as in last experiment:—



Exp. 267. Dog, Native.

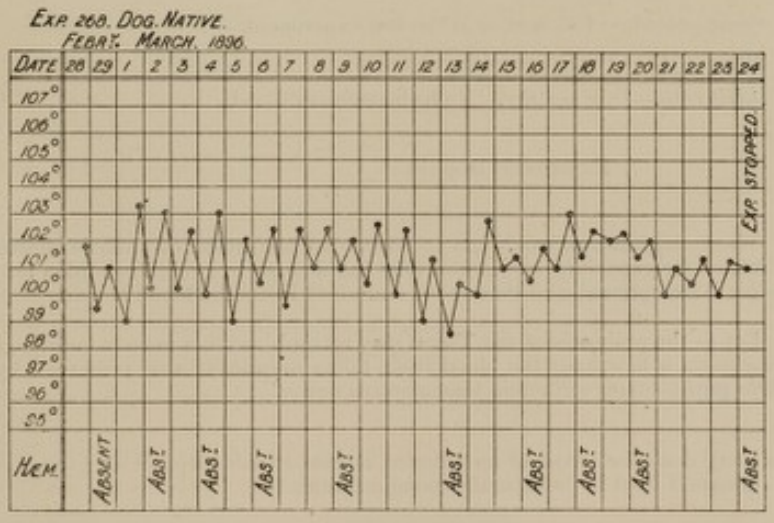
February 28th.—Passed under the skin of this dog, by means of a needle, a short piece of cotton thread, which had been dipped in blood containing the hæmatozoa 24 hours previously, dried in the air, and placed in a test-tube to preserve from dust.

The following chart shows the result:—



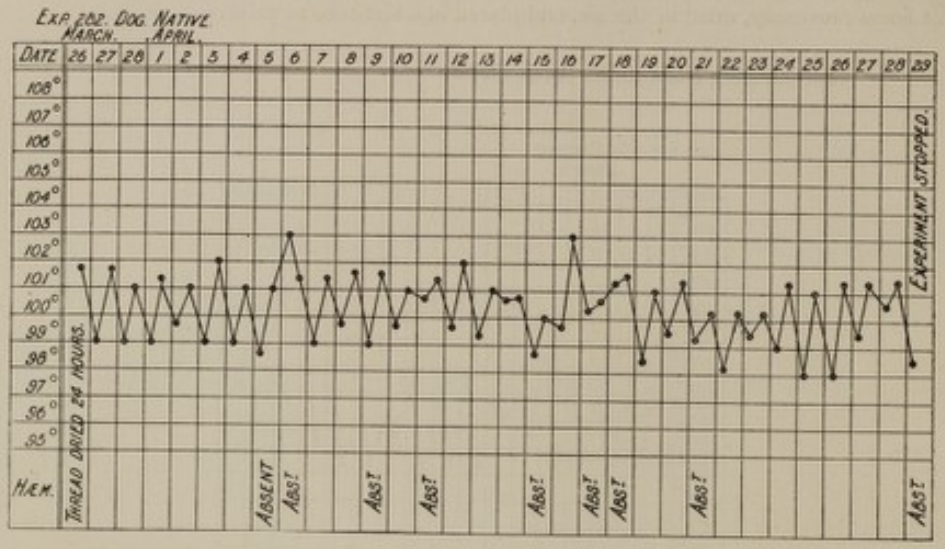
Exp. 268. Dog, Native.

February 28th.—Same procedure followed as in last experiment.



Exp. 282. Dog, Native.

March 26th.—Same procedure as in last two experiments.



Exp. 285.

March 27th.—Passed under the skin of this dog a short piece of cotton thread, which had been dipped in blood containing hæmatozoa 48 hours previously and then dried.

April 28th.—Dog healthy. No hæmatozoa in blood.

Experiment stopped.

Exp. 286.

Same procedure followed as in the last experiment.

March 27th.—Thread passed under skin.

April 28th.—Dog healthy. No hæmatozoa in blood.

Experiment stopped.

These experiments would go to show that the blood of animals affected by Fly Disease retains its capability of transmitting the disease in a dried condition for 24 hours, but that this is exceptional, and that at the end of 48 hours the blood is inert.

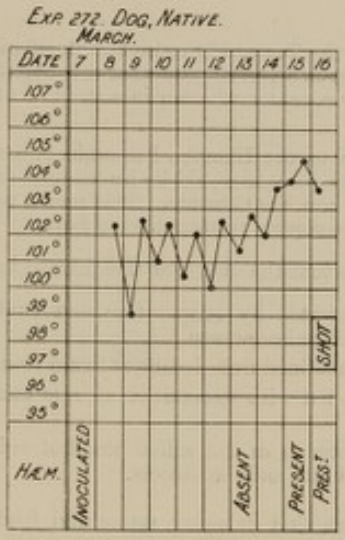
But as the proboscis of the Tsetse may retain blood in a moist condition, it will be interesting to show how long the blood itself, taken aseptically and preserved by suitable means from putrefaction, will retain this infective power.

(*c.*) How long does the blood of an affected animal remain capable of giving rise to the disease if kept in its naturally moist condition?

Exp. 272. Dog, Native.

March 7th.—To-day I inoculated this dog with 1 c.cm. of blood taken exactly 24 hours previously from a donkey suffering from Nagana.

The following chart shows the result:—



Exp. 273. Dog, Native.

March 8th.—To-day I inoculated this dog with 2 c.cm. of the same blood used in the last experiment, but now 48 hours since its removal from the affected animal.

March 19th.—Hæmatozoa present in blood.

Exp. 288. Dog, Native.

March 30th.—Inoculated with blood 72 hours old.

April 7th.—Hæmatozoa present in blood.

Exp. 289. Dog, Native.

March 30th.—Inoculated with blood 72 hours old.

April 7th.—Hæmatozoa present in blood.

Exp. 293. Dog, Native.

April 12th.—Inoculated with blood 96 hours old.

April 22nd.—Hæmatozoa absent from blood.

April 23rd.—Dog killed by accident.

Exp. 294. Dog, Native.

April 12th.—Inoculated with blood 96 hours old.

May 2nd.—Hæmatozoa present in blood.

Exp. 306. Dog, Native.

May 9th.—Inoculated with blood 7 days old.

May 25th.—Hæmatozoa absent from blood.

Dog escaped and was shot by some person unknown.

Exp. 307. Dog, English. Pointer.

May 9th.—Injected 5 c.cm. blood 7 days old.

May 22nd.—Hæmatozoa absent from blood of dog.

May 29th.—Hæmatozoa absent from blood of dog.

These experiments point to the fact that the blood of animals suffering from Fly Disease can retain its infective power for 4 days but not for 7.

(*f.*) Does the Tsetse Fly under natural conditions convey the disease from animal to animal?

Having seen that the Tsetse Fly cannot of itself give rise to disease; that, on the other hand, it can readily act as a carrier, and that it is able to retain its infective power for at least 24 hours, let us now consider whether in a state of nature it really does convey the disease.

When I first came to the Ubombo I thought it most probable that the ordinary mode of infection of the Fly Disease would be by feeding on the herbage or drinking the water of stagnant pools in the Fly Country. This seems to be the opinion held in India regarding the etiology of the closely related disease Surra.

Dr. Lingard* states that the causes of this disease in animals are:—

(*a.*) Drinking water concentrated by evaporation and contaminated with the infusorian towards the end of the hot season.

(*b.*) Eating soiled herbage obtained from localities liable to inundation.

(*c.*) Ingestion with corn soiled with excrement of rats and bandicoots affected with the hæmatozoon.

(*d.*) In hounds by eating carrion or the soiled blood of animals hunted; in other dogs by destroying affected rats and bandicoots.

From this it is seen that drinking water and food are given the first places as causes of infection.

In Zululand also some of the most experienced residents are of this opinion, and consider they can pass with impunity through Fly Country, if only they prevent their animals eating or drinking while there.

I therefore tried the experiment of taking susceptible animals into the Fly Country for a few hours, there permitting them to be bitten freely by the fly, but not allowing them to eat or drink until their return to the top of the hill. For this purpose I used five perfectly healthy horses, and the following five cases will show that under the stated conditions animals become readily affected by the disease.

It may be mentioned here that the horses were prevented from even snatching a mouthful of grass by having a stout nose-bag of network fitted over their bits. A nose-bag containing a feed of crushed mealies was also taken in order to give them a feed when off-saddled for the purpose of catching flies.

Exp. 205. HORSE.—In good condition.

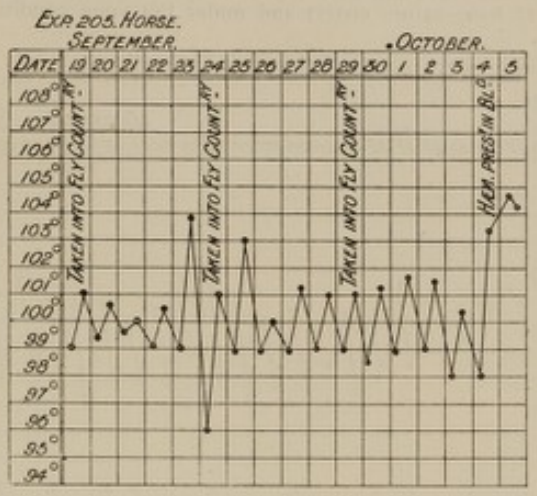
On the 19th, 24th and 29th September this horse was taken by me into the "Fly," and remained there on each occasion from about 10 a.m. to 4 p.m.

During this time the horse was not allowed to graze, and it was noted that a number of Tsetse Flies settled on him during the day.

On the 4th October this horse was seen to be looking out of sorts, his hair was staring, and a slight watery discharge ran from the eyes and nose. His temperature had risen considerably on the previous evening, and still remained high. On microscopical examination of his blood he was found to be suffering from the Fly Diesase.

* Summary of Further Report on Surra, p. 7, by A. Lingard, M.B., C.M., Bombay, 1894.

The following chart shows the onset of the fever.

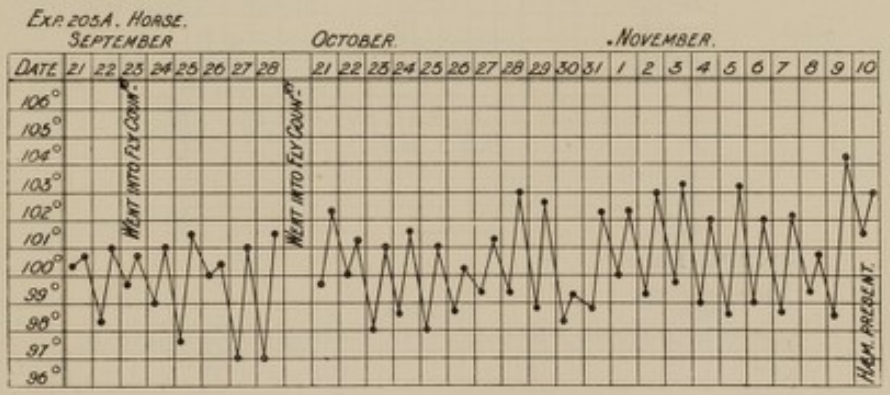


Exp. 205A. HORSE.—In good condition.

This horse was also taken into the Fly Country on the same days and under the same conditions as Exp. 205, that is to say he spent three days from about 10 a.m. until 4 p.m. in the Fly Country, and was not allowed to graze or drink.

In this case the disease manifested itself on the 9th November, although he was noticed to have lost condition as early as the 22nd October. And it is probable that he contracted the disease earlier than this date, but on account of the savageness of the animal, who had to be cast when a specimen of his blood was required, these examinations were undertaken rather hesitatingly, until it was found that a twitch brought him to submission.

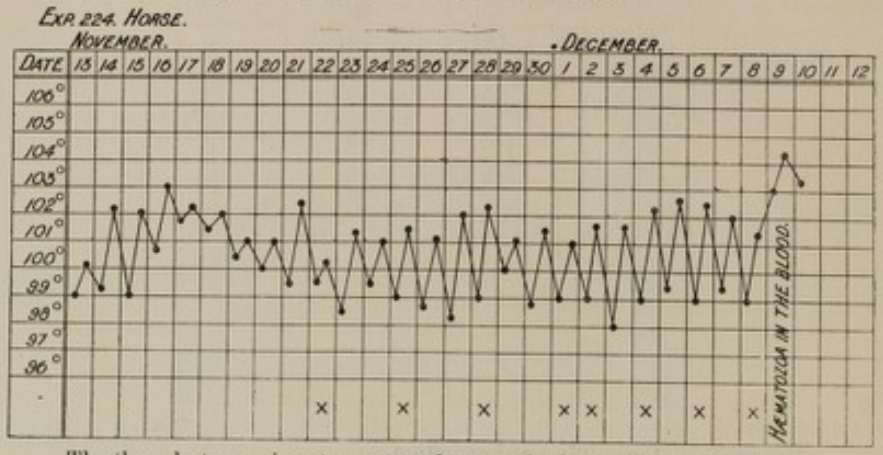
The following chart, which has been curtailed for economy of space, shows the course of the temperature. It is probable that the disease began on or about the 21st October, but no examination of his blood took place until the 10th November. The chart is not continued after this date, as the horse was at once put on large doses of arsenic, which, as will afterwards be seen, modifies the temperature curve and course of the disease.



Exp. 224. Horse.

This horse was taken into the Fly Country on several occasions (marked by the crosses on the next temperature chart) and under the same conditions as the last two experiments.

The following chart represents the onset of the disease.



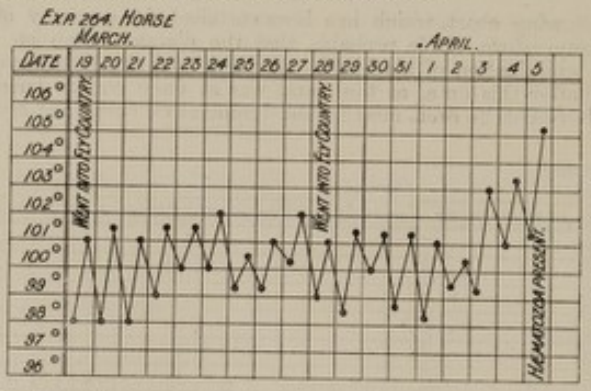
The three last experiments were made towards the end of the dry season, no rain had fallen for several months, the herbage in the Fly Country was burnt as dry as tinder, and there were no stagnant pools of water. It is difficult to imagine horses taking the disease under these circumstances, even although they had been allowed to feed.

The next cases occurred during the rainy season, when the low country is covered with green herbage, and numerous pools of water are found scattered throughout.

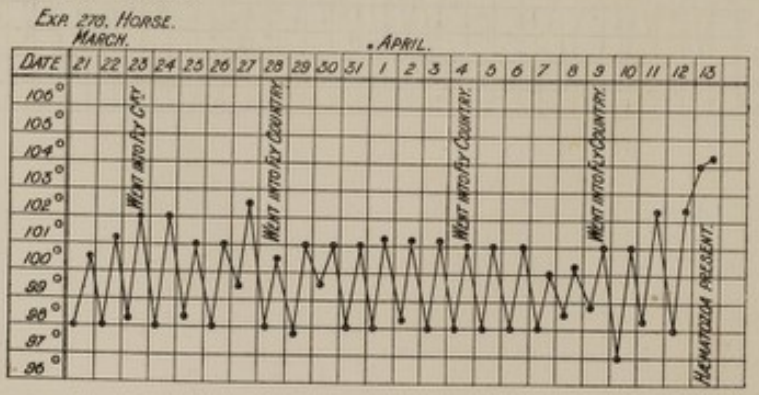
Exp. 264. Horse.

This horse was taken into the Fly Country on two occasions, and was not allowed to feed or drink while there.

The following chart shows the onset of the disease.



Exp. 278. Horse.



These five experiments show abundantly *that horses cannot be taken with impunity for a few hours into the Fly Country, even although they are not allowed to eat or drink there*, and they afford a strong presumptive proof that the disease is carried to them by the Tsetse Fly.

This last series of experiments, however, proves less than at first sight it might be supposed. It only proves that susceptible animals cannot be taken into the Fly Country, even although they are not allowed to eat or drink there. It does not absolutely prove that the disease is carried to them by the Tsetse Fly. There may be other ways of taking the disease, for example by inhalation. The disease called Ague or Malarial Fever in man is of all diseases probably the one most nearly related to the Fly Disease in animals. They are both caused by blood parasites belonging to the Protozoa, and they are both found under similar physical conditions. In the case of the much studied and familiar Malarial Fever none have up to the present had the courage to assert that man would be immune by merely taking care what to eat or drink while in a malarious district, or in other words only to eat cooked food and drink boiled water while there. On the contrary it is asserted by the latest authorities that merely breathing the air of malarious districts is sufficient to set up the disease; in other words, that the parasite can obtain entrance to the system from the air. This is a hard thing to understand, and in order to make it possible the parasite of the Fly Disease must be able to exist in some other form than that in which it is found in the blood. The supposition would be that it forms a resting stage or spore form in which it can exist as a dry impalpable dust. For my part I have much difficulty in believing that animals are infected as a rule with Fly Disease by inhaling the *materies morbi*, and until I find animals still susceptible to the disease which are protected in some way or other both from feeding and the fly, I shall continue to be sceptical.

On account then of the lack of absolute proof furnished by the last experiments, I set myself on the 22nd November to try to infect susceptible animals with the disease by having them bitten by flies brought up daily from the low country and straightway placed on the animals.

(*g.*) Is the Tsetse Fly capable of giving rise to the disease if taken out of the Fly Country into a healthy locality?

The method of carrying out this experiment was to go down to the Fly Country in the early morning, catch the flies, return to the top of the Ubombo and straightway place them on the animal under experiment.

The greatest care was taken that the flies were caught on a perfectly healthy animal, as to have allowed them to puncture one already affected by the disease would naturally vitiate the experiment.

The time which elapsed between catching the flies and placing them on the animal under experiment varied from four to seven hours.

The following experiments give the dates, the number of flies used, and the result:—

Exp. 225. Horse.—In good condition.

November 22nd.—Brought up ten Tsetse Flies from the low country and placed them on this horse.

November 28th.—Ten fresh flies.

„ 30th.—Nine fresh flies.

December 1st.—Five fresh flies.

„ 2nd.—Thirteen fresh flies.

„ 4th.—Twenty fresh flies.

„ 6th.—Seven fresh flies.

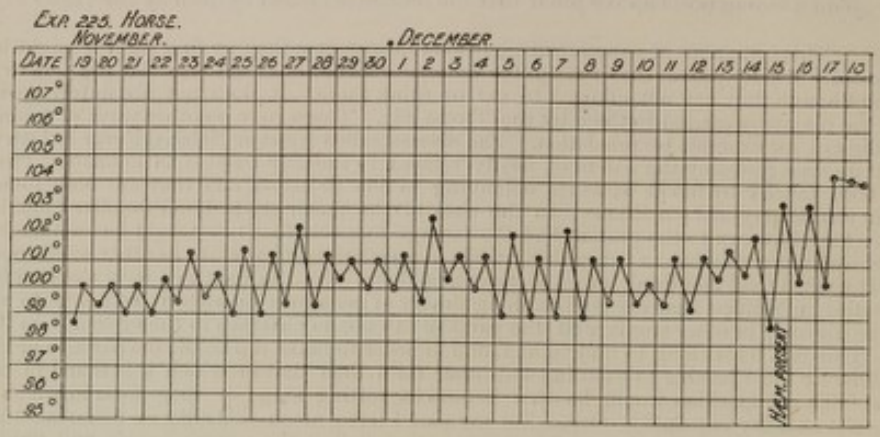
„ 8th.—Thirty fresh flies.

„ 11th.—Eleven fresh flies.

„ 14th.—Fourteen fresh flies.

„ 15th.—To-day this horse is seen to be out of sorts, his temperature has risen and his blood contains the hæmatozoa of Fly Disease.

The following chart represents the onset of the disease.



Exp. 236. Dog, Pointer.—In good condition.

December 16th.—Brought up eighteen Tsetse Flies from the low country and placed them on this dog.

December 18th.—Twenty fresh flies.

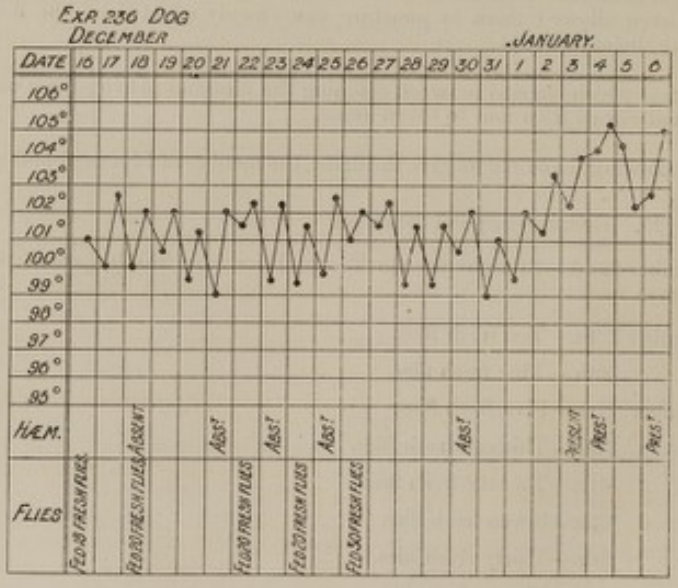
" 22nd.—Twenty fresh flies.

" 24th.—Twenty fresh flies.

" 26th.—Thirty fresh flies.

January 3rd.—To-day the blood of this dog contains the hæmatozoa of Fly Disease.

The following chart represents the onset of the disease :—



As these experiments have had a successful result, I now consider it proved that the Tsetse Fly does commonly, in a state of nature, convey the disease from animal to animal, and that on the other hand there is no proof that the drinking of water or the eating of soiled herbage plays any *rôle* in the process.

3. THE HÆMATOZOON OR BLOOD PARASITE OF THE "FLY DISEASE."

We have seen from previous experiments that there is a something in the blood of animals affected by this disease which is capable of conveying the disease to healthy animals.

At this point then I think it will be convenient to give a definite description of the parasite discovered by me in 1894 in the blood of animals affected by Nagana, and to bring forward my reasons for considering it to be the proximate exciting cause of the disease. For the present I shall call it the Hæmatozoon or Blood Parasite of Fly Disease, although on further knowledge it may be found to be identical with the hæmatozoon of Surra, which is called Trypanosoma Evansi, or at least a species belonging to that genus. For the purpose of having this point of identity settled I have sent specimens of blood containing the parasite from the horse, donkey, ox and dog to Dr. Lingard, Bacteriologist to the Indian Government, who has been investigating Surra since 1890. I have received one communication from Dr. Lingard, who doubts the identity of the two diseases, since the Indian disease does not affect cattle,* whereas here in Zululand Nagana, according to common experience, is almost invariably fatal in these animals.

Since then, however, Dr. Lingard writes that he has carefully examined microscopically the specimens sent, and has come to the conclusion that there is absolutely no difference between the Indian and South African hæmatozoa. In regard to the statement in his Report that, as far as he had been able to ascertain, Surra was not fatal to bovines in India, he says that during December, 1895, he has seen deaths in cattle from the disease in the Punjaub during cold weather, and he thinks that the fact of the weather having been cold may have had a great deal to do with this result.

As the hæmatozoon is identical in form in the two diseases, and as the symptoms of the disease are also very similar, we may consider that the widely spread Surra of India and the Fly Disease of Africa are either one and the same, or so very closely allied as to be practically the same. One difference strikes me as curious, and that is the mode of infection. In Africa the Tsetse Fly has always been popularly credited with playing a notable *rôle* in this, and the results of my experiments would go far to show that it is not necessary to assume any other mode of infection. In India on the other hand an insect go-between is seldom hinted at, and a first place is given to an infection by the eating of soiled herbage, or the drinking of contaminated water.

This hæmatozoon (Trypanosoma Evansi) is one of the most curious of all the known disease producing parasites. It was first discovered, I believe, by the late Surgeon-Major Timothy Lewis, F.R.S., a most accurate and patient observer, in the early part of 1877, in the blood of apparently healthy rats in Calcutta, Bombay and Simla. In 1880 they were discovered in the blood of horses suffering from Surra by Dr. Griffith Evans, the chief of the veterinary department in Madras, and named after him by Lewis. In 1881 Wittich described similar organisms in the blood of hamsters in Germany, and in the same year a like observation was made by Robert Koch. Both Wittich and Koch suggest that the parasites found by them in the blood of hamsters are in all probability identical with those found by Lewis in India.

Crookshank, to whom specimens of Surra blood had been sent, found that 25 per cent. of rats taken from the London sewers contained an identical organism. Dr. Lingard has proved that the blood of the rat containing these hæmatozoa is capable of giving rise to Surra in horses. Here then we have an organism which can live for an indefinite time in the blood of certain animals without disturbance to health, but on being transferred to certain other species at once sets up a severe and in some an invariably fatal disease.

That this parasite is the true cause of Fly Disease is rendered almost certain from the following considerations:—

1. It is found in the blood of every animal suffering from this disease, and is absent from the blood of all healthy horses, cattle or dogs.

2. The onset of the disease is marked by a rise in temperature, and this corresponds with the first appearance of the hæmatozoa in the blood.

3. As the disease progresses, *pari passu* with the destruction of the red blood corpuscles the parasites tend to become more numerous, sometimes reaching the enormous number of 5, 10, or 15 millions in every drop of blood.

4. The transference of the smallest quantity of blood from an affected to a healthy animal sets up the disease in the latter, as I have shown above; even the very small quantity of blood conveyed by the probosces of a few Tsetse Flies is sufficient to carry the disease from animal to animal.

As it is evident that the discovery of the hæmatozoon in the blood of an affected

* This statement by Lingard is however contradicted by his own published reports and also by those of others, as, for instance, Evans.

animal is the surest way of arriving at a correct diagnosis of the disease, I shall describe briefly the method of examining the blood. To obtain a specimen of blood from horses, cattle or dogs for microscopical examination, it is most convenient to take it from the outer surface of the ear. The ear is first carefully shaved, and then thoroughly cleaned and dried. By pricking a small vein with the point of a sharp scalpel a drop of blood wells up. It is best to remove the first drop, as it may contain a quantity of *débris* in the shape of epithelial scales or other foreign matter. The second drop is now touched by a thin slip of glass or coverglass, and this is placed face downwards on a glass slide. The blood of course flows out into a very thin layer between the two glasses, and this may at once be examined under a microscope.

As this specimen is unstained, and as the hæmatozoon is a transparent body, it will of course be necessary to examine the preparation with as small a diaphragm as will allow sufficient light to pass through.

On looking at such a specimen by a microscopical power, say of 500 diameters, the red blood corpuscles are seen as small faintly yellow discs, and among them and causing much commotion among them can be seen transparent elongated bodies in active movement, wriggling about like tiny eels and swimming from corpuscle to corpuscle, which they seem to seize upon and worry. They appear to be about a quarter of the diameter of a red blood corpuscle in thickness, and two, three or more times the diameter of a corpuscle in length. They are pointed or somewhat blunt at one end, and the other extremity is seen to be prolonged into a very fine lash, which is in constant whip-like motion. Running along the cylindrical body between the two extremities can be seen a transparent delicate longitudinal membrane or fin, which is also constantly in wave-like motion.

The hæmatozoa vary among themselves a good deal in size and shape, and seem to take on slightly different forms in different species of animals. But as a written description of such things is tedious, and after all gives very little information, I refer the reader of this Report to the various figures given on the accompanying plate (Plate III) to illustrate the parasite as it appears in the dog, horse, &c..

These parasites evidently belong to a very low form of animal life, namely the infusoria, and simply consist of a small mass of vacuolated protoplasm surrounded by a limiting membrane, and without any differentiation of structure, except in so far as the membrane is prolonged to form the longitudinal fin and flagellum. Reproduction is by simple division.

We have seen by the thread experiment that the hæmatozoa retained their vitality only on one occasion for 24 hours in a dried condition, and for less than 7 days in a moist condition, and that after this they were no longer capable of giving rise to the disease. This proves that under these conditions there had been no spore or resting-stage formation.

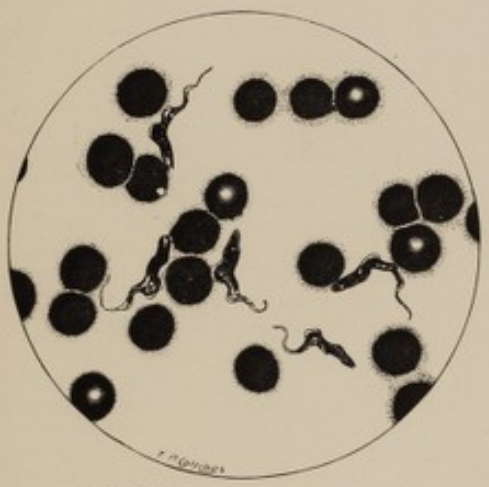
A much easier method, however, of demonstrating the presence of the hæmatozoon in the blood of animals affected by the Fly Disease is by means of stained preparations of blood.

A drop of blood is placed in the centre of a coverglass, another coverglass is placed on it, and the two gently squeezed together in order to spread the blood out into as thin a layer as possible. The two coverglasses are now slid apart, and the film of blood remaining on each is allowed to dry. The blood is then fixed on the glass by holding it for a few seconds over the open mouth of a bottle containing a few crystals of osmic acid. After washing the preparation in water it is stained for a few seconds in an aniline stain such as carbolic fuchsin, again washed, allowed to dry, and finally mounted in Canada balsam. As the hæmatozoa are now stained a bright red among the more faintly stained blood corpuscles and are now motionless, they can be more easily seen and more thoroughly examined.

The fate of the Hæmatozoa when ingested by the Tsetse Fly:—

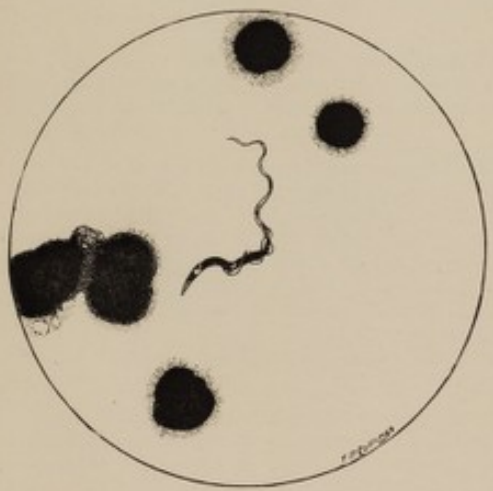
A priori one would think that the hæmatozoa on being taken into the stomach of the Tsetse Fly would soon perish on account of the processes of digestion which one imagines going on. It will therefore be interesting and bear directly on the subject if we trace the history of the hæmatozoon after it has been removed from the blood vessels of an affected animal and swallowed by the Tsetse Fly. I therefore caused the flies to feed on an animal suffering from Nagana, whose blood contained numerous hæmatozoa, and at hourly intervals subjected the proboscis, the contents of the stomach and the contents of the lower end of the intestine to microscopical examination.

Before giving the result of this examination I may in a few words describe, without any pretence to minuteness, the anatomy of the Fly. The sharp needle-like process which is seen directed forward from the head of the living Fly is composed of several pieces; it can readily be divided into three parts, the two processes called palpi and the proboscis. While feeding the Fly still keeps the two palpi directed forward, and depresses at a right



HÆMATOZOA IN THE BLOOD OF THE DOG.

HÆMATOZOA IN THE BLOOD OF THE HORSE.



HÆMATOZOA IN THE BLOOD OF THE HORSE.

HÆMATOZOA IN THE BLOOD OF THE COW.

All magnified 1,000 times.

angle to them the proboscis, by which it pierces the skin and sucks up the blood. The proboscis is formed of several pieces, some acting and looking like narrow knife blades, others acting as a tube to contain the blood.

On placing the proboscis under the microscope this tube can be readily seen lying alongside the knives, and lined with large epithelial cells, some of which are developed into hair-like processes or cilia, by the movement of which I presume the blood is conveyed to the œsophagus of the Fly. At the base of the proboscis is a relatively large brown or yellow coloured gland, from which a clear fluid can be expressed, which, on being dried, deposits very beautiful fern-like crystals. This is probably the fluid which the Fly injects into its victim and which sets up the well-known irritation and swelling.

From the mouth a straight thin tube or œsophagus leads to the stomach, which, of course, is situated in the abdominal part of the insect. The stomach when dilated with blood is about the size of a buck shot, and bright red in colour. The blood inside is always coagulated into a firm red-currant jelly-like mass, and does not change its colour until it reaches the intestine. The intestine is a blackish coloured tube, which passes from the stomach to the vent. It does not contain many blood corpuscles, but merely minute round shining crystalline bodies, the product of the disintegration of the red blood corpuscles.

This description of the anatomy of the Tsetse is only meant to describe broadly its pronounced features. The proboscis itself is a most complicated piece of apparatus, and would require a paper as long as this Report to describe it fully.

The result of my examination up to the present of the Tsetse Fly is briefly this:—

Immediately after feeding, the tube of the proboscis can be seen to be crammed full of red blood corpuscles, among which the hæmatozoa can be seen actively wriggling. Up to 46 hours after feeding I have seen living hæmatozoa and red blood corpuscles in the proboscis. After 118 hours the hæmatozoa are still very numerous and vigorously active in what remains of the blood in the stomach. After 140 hours the stomach is empty. After 25 and 70 hours I have seen many motionless hæmatozoa in the fæces, but I have never seen at any time any appearance of life in the hæmatozoa in the fæces or contents of the lower part of the intestine. The parasites appeared, however, unchanged in form, and I intend making some injection experiments with the fæces to find out if these motionless hæmatozoa have any vitality left in them.

B.—RELATION OF BIG GAME TO THE FLY DISEASE.

As we have found that the Tsetse Fly in a state of nature does act as a carrier of the hæmatozoon of Fly Disease, it is evident that it must procure the parasite somewhere, and what more natural than that it should procure it from the blood of warm blooded animals living in the Fly Country?

It is not necessary to suppose that in these animals Nagana is a fatal disease, but only that the big game harbour the parasite which causes the disease for a longer or shorter time with little or no disturbance to health. We have seen, as in the case of the heifer mentioned on page 33, that cattle may have the parasite in their blood for at least 18 months without causing death, and it is possible that some of the big game die of this disease. When in the Fly Country quite lately I chanced on a dead wildebeeste surrounded by vultures. The skin of this animal was quite intact and showed no signs of its having come to a violent death. It was somewhat emaciated and had the appearance of an animal dead of Nagana. I did not inject any blood from it into a susceptible animal, as it was in a state of decomposition.

According to Lingard two species of rat (Mus decumanus and Mus rufescens) in India harbour the Surra parasite often in large numbers without appearing to produce any noticeable symptoms in the great majority of them, although some few rats appear to succumb to the disease. Lingard examined 1,107 rats for the hæmatozoon and found it present in 421, absent 686.

He also made experiments on animals with the hæmatozoon of the rat, and as this has an important bearing on the subject of the relation of big game to the Fly Disease, I shall give his account in his own words. Dr. Lingard states (page 4, Summary of Further Report on Surra, 1894):—

"Proof was wanting that this hæmatozoon was capable of producing Surra, when blood containing "the organism was subcutaneously injected into horses and some other species of animals. As early as "November, 1890, I commenced a series of experiments for the purpose of deciding the above question, and "obtained positive results, but found that the periods of incubation were more prolonged than in Horse-Surra, "in one case being as much as 63 days. Notwithstanding this, the virulence of the disease when once developed "was intense, as shown by the short period during which the animal survived after the appearance of the "hæmatozoon in the blood; this being in three cases only 2 to 5 days.

"*Inoculation with the Soiled Blood of the Rat* (*Mus D.*).

"Of twelve horses inoculated with soiled rat's blood, four animals contracted Surra, or 33·3 per cent.

" (i)	Period of incubation lasted	18	days.	Animal lived	19	days.
" (ii)	"	"	29	"	"	"	2	"
" (iii)	"	"	13	"	"	"	2	"
" (iv)	"	"	63	"	"	"	5	"

"Although the latent period of Rat-Surra was so prolonged in the first instance when inoculated from "rat to horse (average 40·7 days), it immediately returned to the normal period (average 7 to 8 days) when a "second horse was inoculated with the blood of the first affected. The latent period however becomes still more "reduced (5 days) when a third animal is inoculated with the blood of the second.

"Horses unsuccessfully inoculated and re-inoculated subcutaneously with the Rat Hæmatozoon, when "subsequently inoculated with Horse-Surra blood, show no protection from the former, but acquire the disease "after an incubation period of 6 to 10 days.

"Animals other than horses which contracted the disease were: Bovines, monkeys and field rats; "while the rabbit, guinea pig, fowl, cat, dog and donkey proved refractory to the Rat Hæmatozoon. But all "the above animals, with the exception of the fowl, were very susceptible to the Rat Hæmatozoon when it had "first been passed through the horse."

From this we may be justified in believing that some of the wild animals in South Africa may harbour the Nagana parasite and pass it on to the domestic animals.

In regard to this relation of big game to the Fly Disease, there can be no question as to the very widely spread opinion which exists regarding the wild animals as in some way or other responsible for its propagation, and in this connection the buffalo, wildebeeste, quagga, waterbuck, and koodoo are usually named. Europeans and natives in all parts of the country are found who state most emphatically that where there is no game there is no Nagana, and example after example could be cited of the statement that the game having been driven out, the disease disappeared, and on the reappearance of the game that the cattle again became sickly. Those who hold the Tsetse Fly theory explain this by affirming that where the big game is there also is the Fly. Others, as mentioned above, are of opinion that the game contaminate the grass and drinking water and so infect the cattle.

That the presence of the wild animals in the vicinity of horses and oxen is not the only factor in the problem is shown by the fact that in the old days when the big game was numerous and roamed over the whole country, hunters and travellers never complained of the Fly until they encountered the disease in low lying tracts of country or along the large river valleys; and at the present day it is stated, on the authority of Mr. L. Peringuey, that in the Hermansdorp District of Cape Colony, where herds of buffalo are still found, the Fly Disease is unknown, nor has the Tsetse Fly itself been ascertained to occur there. Now that we know the disease to be caused by a blood parasite, and that the disease can be carried by the Tsetse from affected to healthy animals, we can see how such theories may have some foundation in fact.

At first I thought that by a careful microscopical examination of the blood of the big game some light could be thrown on the subject, and to this end I submitted the blood of buffalo, wildebeeste, koodoo, impala, bush buck, reed buck, and the smaller varieties shot in the heart of the Fly Country to a lengthened microscopical examination, but with no result.

Thinking, however, that the parasite might exist in too small numbers, as we have seen is frequently the case in cattle, to be readily discovered by direct examination, I next instituted a series of experiments by injecting a moderately large quantity of blood taken immediately after death into dogs, as it has been proved by experiment that in cases in which the parasite could not be demonstrated by the microscope, an inoculation experiment of this kind readily discovered them.

Of course it was necessary that the dogs used for experiment should be kept at the top of the Ubombo, as the results would have been useless if they had been exposed to infection in any other way than by the injection of the blood.

This meant a delay of several hours between the procuring of the blood and the injection of it into the dog, as the wild animal might be shot five or ten miles from the foot of the hills and the Ubombo itself is some 2,000 feet high.

Again, it would evidently facilitate matters if the blood could be kept liquid, as the blood coagulum might entangle in itself all the parasites contained in the blood.

I therefore made a preliminary experiment to find out if blood kept liquid by some suitable means and kept in a bottle at the temperature of the air for several hours would still retain its infective power.

The following experiment shows that this is so.

Exp. 234. Dog, Native.

December 16.—Removed 10 c.cm. of blood from a dog suffering from Nagana, mixed it with a twentieth of its bulk of a ·5 per cent. citrate of potash solution, and kept the blood in a bottle at the temperature of the air (80° F.) for 7 hours. The blood was now injected into the above healthy native dog.

December 21.—To-day the blood of this dog was found to contain the hæmatozoa of Fly Disease, and he died of the disease 12 days later.

It was evident then that we might expect successful results by injecting blood procured in the Fly Country and kept liquid by the above mentioned means into native dogs kept on the top of the Ubombo.

The following experiments were carried out in this manner:—

Exp. 227. Dog, Native.

November 22nd.—Injected 20 c.cm. blood from wildebeeste. Shot 11 a.m. Blood coagulated. Injected liquid part 5 p.m.

February 23rd.—Dog healthy.

Exp. 229. Dog, Native.

November 25th.—Koodoo shot 9 a.m. Blood coagulated. Injected 20 c.cm. 3 p.m.

February 6th.—Hæmatozoa present in blood.

February 19th.—Dog died of Fly Disease.

Exp. 230. Dog, Native.

November 30th.—Koodoo shot 1 p.m. Blood coagulated. Injected 20 c.cm. 7. p.m.

January 29th.—No hæmatozoa in blood.

February 20th.—Died. Cause unknown.

Exp. 239. Dog, Native.

January 2nd.—Wildebeeste shot 10 a.m. Blood liquid. Injected 10 c.cm.

February 23rd.—Dog healthy.

Exp. 240. Dog, Native.

January 3rd.—Waterbuck shot 4.45 p.m. Blood liquid. Injected 5 c.cm. 8 p.m.

February 3rd.—Hæmatozoa absent from blood. Dog shot, as he was in a low state of health.

Exp. 241. Dog, Native.

January 3rd.—Waterbuck shot 4.50 p.m. Blood liquid. Injected 5 c.cm. 8 p.m.

February 23rd.—Dog healthy.

Exp. 243. Dog, Native.

January 5th.—Buffalo shot 1.30 p.m. Blood coagulated. Injected 5 c.cm. 4.15 p.m.

February 3rd.—Dog healthy.

Exp. 233. Dog, Native.

January 8th.—Buffalo shot 11 a.m. Blood liquid. Injected 5 c.cm. 5 p.m.

January 14th.—Blood swarming with hæmatozoa.

January 29th.—Dog died of Fly Disease.

Exp. 245. Dog, Native.

January 9th.—Bush Buck shot in Fly Country 11 a.m. Blood liquid. Injected 5 c.cm.

January 20th.—Hæmatozoa in blood.

February 1st.—Dog died of Fly Disease.

Exp. 227A. DOG, NATIVE.

January 10th.—Wart hog shot. Blood liquid. Injected 5 c.cm.
February 23rd.—Dog healthy.

Exp. 244. DOG, NATIVE.

January 11th.—Wildebeeste shot. Blood liquid. Injected 5 c.cm.
January 25th.—Hæmatozoa present in blood.
January 26th.—Dog died of Fly Disease.

Exp. 246. DOG, NATIVE.

January 14th,—Wildebeeste shot. Blood liquid. Injected 10 c.cm.
January 21st.—Dog found dead. Had disappeared for two days. Cause of death unknown.

Exp. 247. DOG, NATIVE.

January 15th.—Impala shot. Blood liquid. Injected 10 c.cm.
February 23rd.—No hæmatozoa in blood. Dog shot.

Exp. 248. DOG, NATIVE.

January 17th.—Impala shot 8 a.m. Blood liquid. Injected 5 c.cm. 2.30 p.m.
February 23rd.—Dog healthy.

Exp. 249. DOG, NATIVE.

January 18th.—Buffalo shot 1 p.m.
January 19th.—Blood coagulated. Injected 5 c.cm. 10 a.m.
February 2nd.—Found dead. Cause unknown.

Exp. 250. DOG, NATIVE.

January 20th.—Buffalo shot 10 a.m. Blood liquid. Injected 5 c.cm. 1 p.m.
February 10th.—Found dead. Cause unknown.

Exp. 251. DOG, NATIVE.

January 20th.—Buffalo shot 10.15 a.m. Blood liquid. Injected 5 c.cm. 1 p.m.
January 29th.—Found dead. Cause unknown.

Exp. 252. DOG, NATIVE.

January 20th.—Buffalo shot 11.5 a.m. Blood liquid. Injected 5 c.cm. 2.30 p.m.
February 23rd.—Dog healthy.

Exp. 254. DOG, NATIVE.

February 2nd.—Koodoo shot 4 p.m. Blood coagulated. Injected 5 c.cm. 9 p.m.
February 20th.—Hæmatozoa present in blood.

Exp. 281. DOG, NATIVE.

March 24th.—Wildebeeste shot 10.15 a.m. Blood coagulated.
March 25th.—Injected 5 c.cm. 10.30 a.m.
May 2.—Dog healthy.

Exp. 287. DOG, NATIVE.

March 28th.—Wildebeeste shot 11 a.m. Blood coagulated. Injected 5 c.cm. 5.30 p.m.
April 21st.—Dog died. No hæmatozoa in blood.

Exp. 292. Dog, Native.

April 8th.—Wildebeeste shot 10 a.m. Blood coagulated. Injected 5 c.cm. 5 p.m.
May 2nd, 13th, 19th.—Absent.

Exp. 297. Dog, Native.

April 21st.—Shot koodoo 10.45 a.m. Blood coagulated. Injected 5 c.cm. 6.15 p.m.
May 2nd.—Hæmatozoa present in blood.
May 3rd.—Dog shot.

Exp. 298. Dog, Native.

April 21st.—Wildebeeste shot 4 p.m. 3 c.cm. blood injected 6.30 p.m.
May 19th.—Hæmatozoa present in blood.

Exp. 299. Dog, Native.

April 22nd.—Wildebeeste shot 1 p.m. Blood, 5 c.cm., injected 7 p.m.
May 19th.—Hæmatozoa absent.

Exp. 300. Dog, Native.

April 25th.—Wildebeeste shot 12 noon. Not coagulated. Injected 5 c.cm. 6 p.m.
May 13th.—Hæmatozoa present in blood.

Exp. 301. Dog, Native.

April 29th.—Buffalo shot 10 a.m. Blood, 5 c.cm., injected 9 p.m.
May 19th.—Absent.

Exp. 302. Dog, Native.

April 29th.—Wild pig shot 12 a.m. Injected 5 c.cm. 9 p.m.
May 19th.—Absent.

Exp. 308. Dog, Native.

May 15th.—Buffalo shot 9 a.m. Blood coagulated. 5 c.cm. injected 4 p.m.
July 3rd.—Hæmatozoa absent.

Exp. 309. Dog, Native.

May 15th.—Stein buck shot 5 p.m.
May 16th.—Blood, 3 c.cm., injected 11 a.m.
July 3rd.—Hæmatozoa absent.

Exp. 310. Dog, Native.

May 16th.—Burchell's zebra shot 1 p.m. Blood, 5 c.cm., injected 6.15 p.m.
July 3rd.—Hæmatozoa absent.

Exp. 311. Dog, Native.

May 16th.—Wildebeeste shot 2.30 p.m. Blood, 5 c.cm., injected 6.15 p.m.
July 3rd.—Hæmatozoa absent.

Exp. 312. Dog, Native.

May 17th.—Wildebeeste shot 1.30 p.m. Blood injected 7.30 p.m.
June 19th.—Hæmatozoa absent.

Exp. 313. Dog, Native.

May 17th.—Wildebeeste shot 1.30 p.m. Blood injected 7.30 p.m.
July 3rd.—Hæmatozoa absent.

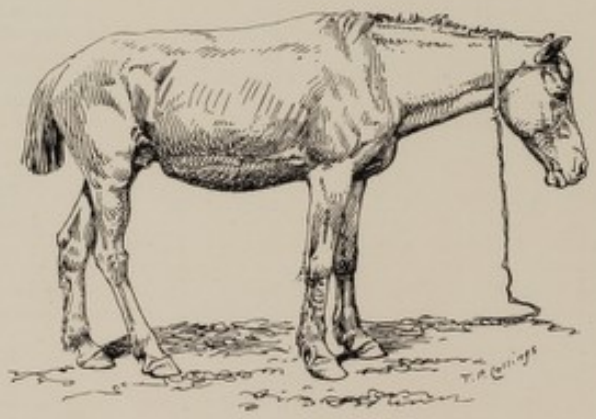
The following table shows the results of the above and other experiments, the various species of wild animal being tabulated together:—

INJECTION OF BLOOD FROM WILD ANIMALS INTO DOGS.

Exp.	Species of Wild Animal.	Blood Injected.	Hæmatozoa Present.	Hæmatozoa Absent.	Remarks.
243	Buffalo	5.1.96	—	3.2.96	Dog healthy 23.2.96
233	*Buffalo*	8.1.96	14.1.96	—	Dog died 29.1.96
249	Buffalo	19.1.96	—	1.2.96	Dog died 2.2.96
250	Buffalo	20.1.96	—	9.2.96	Dog died 10.2.96
251	Buffalo	20.1.96	—	25.1.96	Dog died 29.1.96
252	Buffalo	20.1.96	—	23.2.96	Dog healthy 23.2.96
301	Buffalo	29.4.96	—	12.6.96	Dog healthy 12.6.96
308	Buffalo	15.5.96	—	3.7.96	Dog healthy 3.7.96
227	Wildebeeste	22.11.95	—	10.1.95	Dog healthy 23.2.96
239	Wildebeeste	2.1.96	—	23.2.96	Dog healthy 23.2.96
244	*Wildebeeste*	11.1.96	25.1 96	—	Dog died 26.1.96
246	Wildebeeste	14.1.96	—	21.1.96	Dog died 21.1.96
281	Wildebeeste	24.3.96	—	2.5.96	Dog healthy 2.5.96
287	Wildebeeste	28.3.96	—	21.4.96	Dog died 21.4.96
292	Wildebeeste	8.4.96	—	12.6.96	Dog healthy 12.6.96
298	*Wildebeeste*	21.4.96	19.5.96	—	
299	Wildebeeste	22.4.96	—	8.6.96	Dog shot
300	*Wildebeeste*	25.4.96	13.5.96	—	Dog shot
311	Wildebeeste	16 5.96	—	3.7.96	Dog healthy 3.7.96
312	Wildebeeste	17.5.96	—	19.6.96	Dog lost
313	Wildebeeste	17.5.96	—	3.7.96	Exp. stopped
229	*Koodoo*	25.11.95	6.2.96	—	Dog died 19.2.96
230	Koodoo	30.11.95	—	20.2.96	Dog died 20.2.96
254	*Koodoo*	2.2.96	20.2.96	—	
297	*Koodoo*	21.4.96	2.5.96	—	Dog shot
240	Waterbuck	3.1.96	—	3.2 96	Dog shot
241	Waterbuck	3.1.96	—	23.2.96	Dog healthy 23.2.96
247	Impala	15.1.96	—	23.2.96	Dog shot 23.2.96
248	Impala	17.1.96	—	23.2.96	Dog healthy 23.2.96
310	Zebra, Burchell's ..	16.5.96	—	3.7.96	Dog healthy 3.7.96
245	*Bush buck*	9.1.96	20.1.96	—	Dog died 1.2.96
309	Stein buck	16.5.96	—	3.7.96	Dog healthy 3.7.96
227A	Wart hog	10.1.96	—	23.2.96	
302	Wild pig	29.4.96	—	12.6.96	Dog shot 12.6.96
316	*Hyæna*	2.6.96	13.6 96	—	

These experiments I think prove that several species of wild animals inhabiting the Fly Country harbour the Nagana parasite, and the links in the chain of the causation of this disease connecting the game through the Tsetse Fly with the domestic animals are complete.

In regard to other points relating to the etiology of this disease, such as seasonal prevalence, sex, age, breed, &c., I may dismiss them in this Interim Report by the remark that, according to my present knowledge, neither season, sex, age nor breed has any predisposing or protective influence in this disease.



HORSE SUFFERING FROM FLY DISEASE OR NAGANA.

6.—DESCRIPTION OF THE FLY DISEASE OR NAGANA AS IT OCCURS IN THE DOMESTIC ANIMALS.

In a Report of this kind and at this stage in the investigation, which has only lasted a few months, and with the small number of cases at my disposal, it would be premature to generalize too freely on the symptoms, course, and duration of this disease.

All diseases are protean in character, and something new can be learnt from each new case. It will be more suitable then to give a very brief description of the disease as it broadly manifests itself in the different animals, and to give as far as possible a typical case or cases, with temperature charts, fluctuations in the number of the blood corpuscles and parasites, and a short description of the principal changes found on *post mortem* examination.

A.—FLY DISEASE OR NAGANA IN THE HORSE.

The first appearance of a horse being affected by Nagana is that his coat stares, and there is a watery discharge from his eyes and nose.

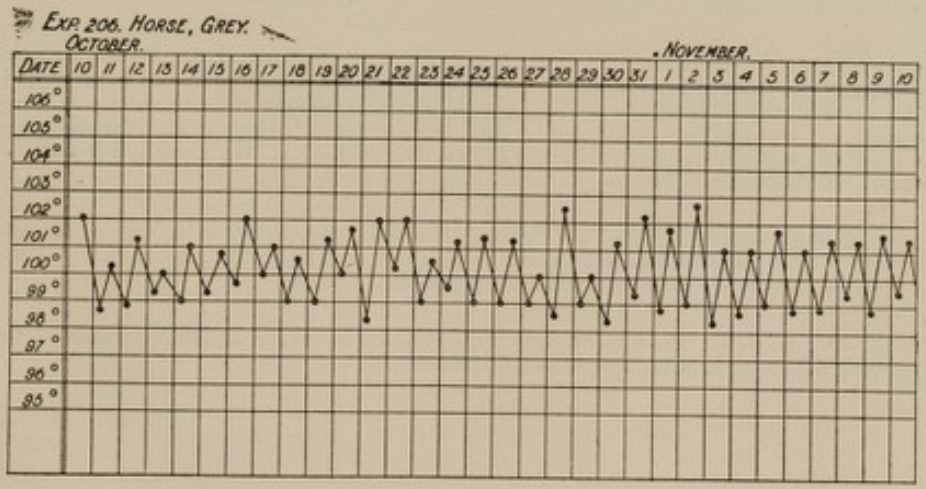
Shortly afterwards a slight swelling under the belly or a puffiness of the sheath may be noticed, and the animal falls off in condition.

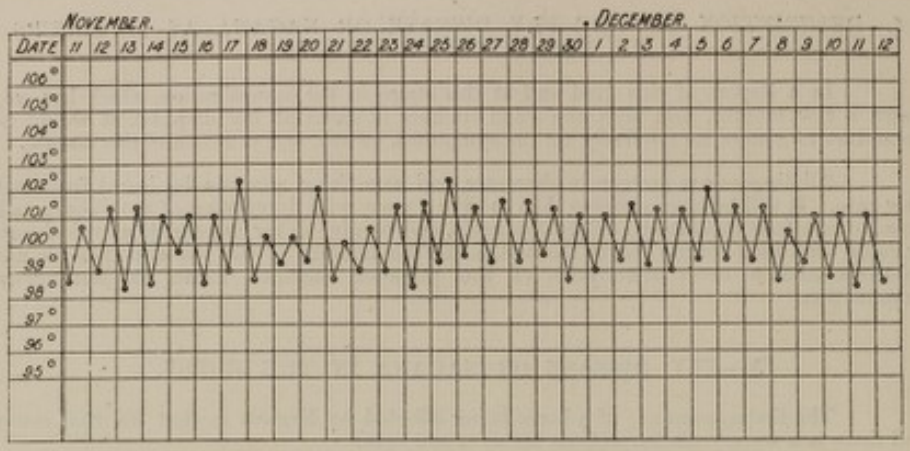
The hind extremities also tend to become swollen; and these various swellings fluctuate, one day being marked, another day being less marked or having disappeared. During this time the animal is becoming more and more emaciated, he looks dull and hangs his head, his coat still stares, becoming harsh and thin in places; the mucous membranes of the eyes and gums are pale, and probably a slight milkiness of the cornea of the eyes is observable. In severe cases and in the last stages a horse presents a miserable appearance. He is a mere scarecrow covered with rough harsh hair, which has fallen off in places. His hind extremities and sheath may be more or less swollen, sometimes to a great extent, and he may have become quite blind. At last he falls down unable to rise, his breathing becomes shallower and shallower, and he dies of exhaustion. During his illness he has shown no symptoms of pain, and up to the last day has had a fairly good appetite. Plate III represents such a case and shows a huge swelling under the belly.

Before proceeding to give illustrative cases of the disease in the horse I shall give, for the purpose of comparison, the temperature chart of a healthy horse.

Exp. 206. Horse, Grey.—This horse arrived at Ubombo on the 8th September, 1895, and was kept at the top of the hill as a control experiment. During the day he was constantly with affected animals, and for several months was stabled alongside horses suffering from the disease.

As will be seen from the chart the temperature varies between 98·4° F. and 102° F., sometimes, however, rising a fraction of a degree above.





The following two cases, with their accompanying charts, illustrate the disease as it occurs ordinarily in the horse:—

Exp. 212. Horse, Bay.

September 27th, 1895.—This horse arrived at Ubombo to-day from Nongoma, and is in poor condition, but otherwise healthy. No hæmatozoa in blood.

October 6th, 1895.—Taken down to camp in Fly Country.

October 20th, 1895.—Returned to Ubombo.

October 21st, 1895.—On examining this animal to-day for the first time since his leaving for low country, he is found to have a slight watery discharge from his nostrils and eyes, there is a slight swelling of the off hind leg, his temperature is raised, and the blood contains numerous hæmatozoa.

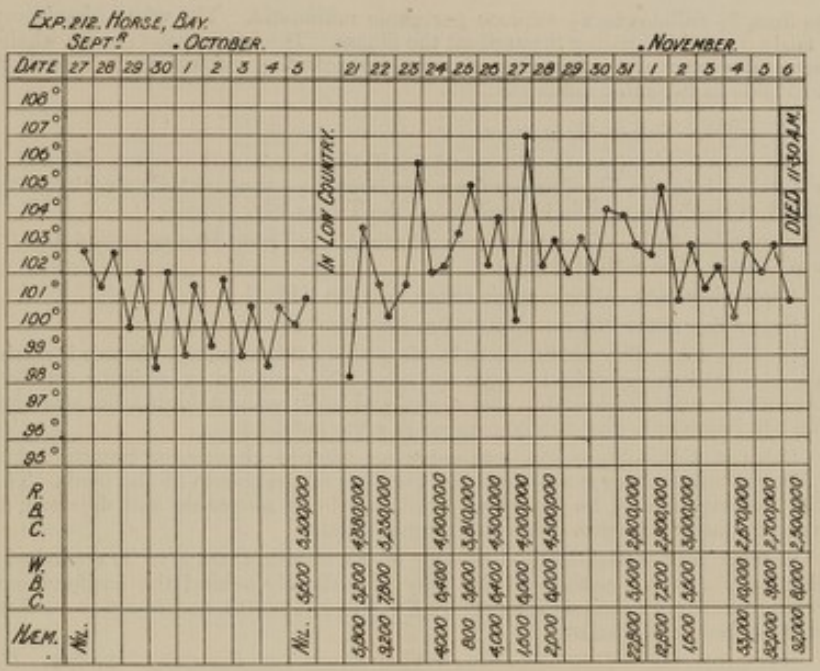
October 24th, 1895.—Since the last entry, the condition of this animal has remained unaltered. This morning he is rather worse, his coat is staring, he has a dull, heavy, depressed look, there is a faint milky opacity of both corneæ, the watery discharge still continues from eyes and nostrils, and he is rapidly growing thinner.

October 28th, 1895.—Condition much the same as on the 24th, with the addition of a well marked œdematous swelling of the sheath.

November 5th, 1895.—This animal is much worse this morning. He is very weak and emaciated. There is still marked swelling of the sheath, the milkiness of the corneæ is more manifest, although not to such an extent as to interfere much with vision; the conjunctival mucous membrane is marked by dark red blotches or petechiæ, and the discharge from nostrils and eyes still continues. Except the swelling of the sheath there are no external visible swellings, and no eruption of any kind on the skin.

November 6th, 1895.—This morning this horse was seen to be very weak, and stood for some time with legs wide apart as if afraid of falling. At 11 a.m. he lay down, his respiration became hurried and shallow, and he died at 11.30.

The following chart represents the course of the fever and the number of the blood corpuscles and hæmatozoa per cubic millimetre.



Autopsy one hour after death.

Body extremely emaciated. The corneæ of both eyes are opaque, and there are petechiæ on both conjunctivæ. There is no apparent swelling of any of the extremities, nor is there any eruption present on the skin.

On making an incision along the middle line of the body and reflecting the skin, the lymphatic glands in the groin on both sides are seen to be enlarged to the size of a small orange. These glands on section are found to be œdematous, and the subcutaneous tissue in this region is infiltrated with a yellow jelly-like substance. On opening the abdominal cavity only a few ounces of clear straw-coloured serum are found in the peritoneum, the intestinal coils are exceedingly pale, and the spleen is much enlarged.

After sawing through the sternum and exposing the cavity of the chest, both lungs are seen to be healthy in colour, and there is no fluid in either pleural cavity.

The pericardium contains 48 ounces of clear straw coloured serum. The heart, which is now exposed, is seen to be bright red in colour, due to the crowding together of patches of extravasated blood or petechiæ under the epicardium. Along the course of the coronary arteries huge masses of yellow jelly-like material are seen, most prominent at the base of the heart, where the rounded masses are quite two inches in thickness. The heart is greatly distended with blood. On cutting into the ventricles the lining membrane of both is seen to be extensively marked by petechiæ. The muscular substance is very pale, and the valves normal.

The lungs are both healthy except for some emphysema at the apices, and a slightly œdematous condition of the lung substance.

The liver is enlarged, of a dark olive-green colour, and extremely friable, breaking readily across, and leaving a rough ragged surface.

The spleen measures 16 inches in length and 8 inches in greatest breadth. The capsule is bluish-white in colour. On section the tissue is moderately firm.

The kidneys on section are seen to be very pale yellowish in colour, especially in the region of the cortex.

Remarks.—This is a case of spontaneous Nagana in the horse, the infection having been caught between the 6th and 20th October, when the animal was in the low country. The horse was weakly and in low condition on arrival from Nongoma, and probably on this account did not resist the disease as long as a stronger horse would have done. On looking at the temperature chart, the temperature curve is seen to be regular and normal during the week or so which elapsed before the horse was taken into the "Fly." On his return on the 21st October the temperature is raised and the blood contains numerous hæmatozoa. The fever curve is rather irregular, but generally shows a marked rise in the afternoon, on one occasion registering as high as 107° F. The red blood corpuscles rapidly decrease in

numbers from 5½ millions to 2½ millions per cubic millimetre. The white blood corpuscles remain fairly normal in number throughout the illness. The hæmatozoa on the whole range high throughout, and are extremely numerous towards the end, rising on the morning of death to 92,000 in the cubic millimetre.

Exp. 205. Horse, Galloway.

Motive of experiment. To ascertain if this horse will become affected by Fly Disease if taken into the Fly Country for a few hours at a time, not allowed to graze there, but exposed as much as possible to the bite of the Tsetse Fly.

September 19th.—Took this horse into the "Fly" from 10 a.m. until 4 p.m. A good number of Tsetse Flies settled on him.

September 24th.—Horse again taken into the "Fly."

September 29th.—Horse again taken into the "Fly."

October 4th.—The Galloway is looking seedy this morning, his hair is staring, and there is a watery discharge from his eyes and nose. No eruption or swelling can be noted. His temperature rose considerably last night, and still remains high. On examining his blood the Fly Parasite is found to be present in some numbers.

October 25th.—Since the last entry no marked change in condition has been noted. This morning there is a pronounced swelling of the sheath of the penis. The horse is rapidly growing thinner, he hangs his head, and looks miserable and dejected, and the mucous membrane of his eyes, gums and tongue is pale.

October 26th.—Besides the swelling of the sheath, there is to be remarked to-day a swelling on the under surface of the belly immediately behind the ensiform cartilage. The swelling measures 6 inches in length by 3 inches in breadth, and is not more than three quarters of an inch in thickness.

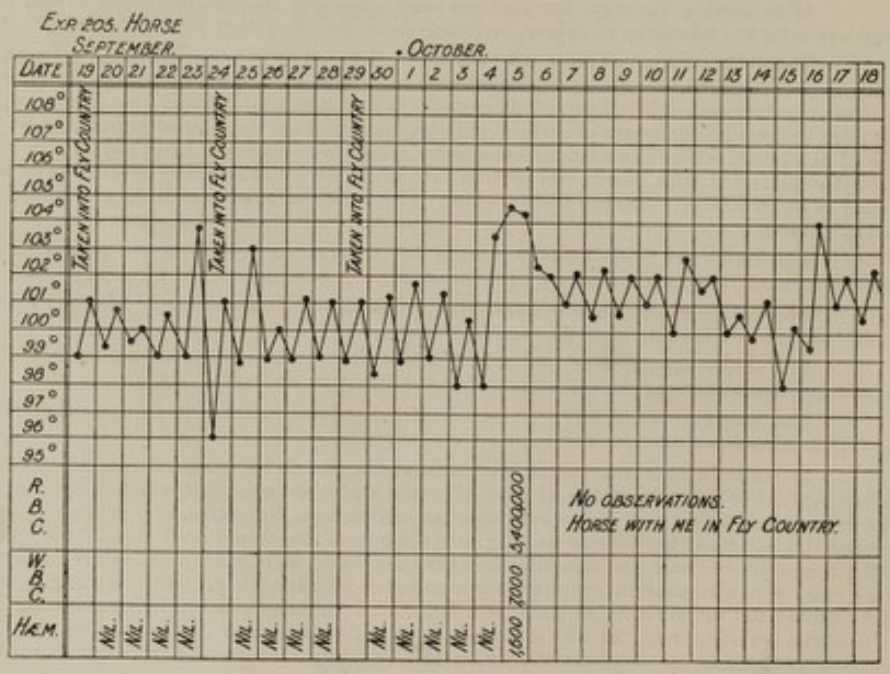
November 6th.—This horse was ridden down into the "Fly" to-day. He went very badly, and had to be led most of the way up hill in the evening. When near the top of the hill he fell, and was with difficulty got on his legs again.

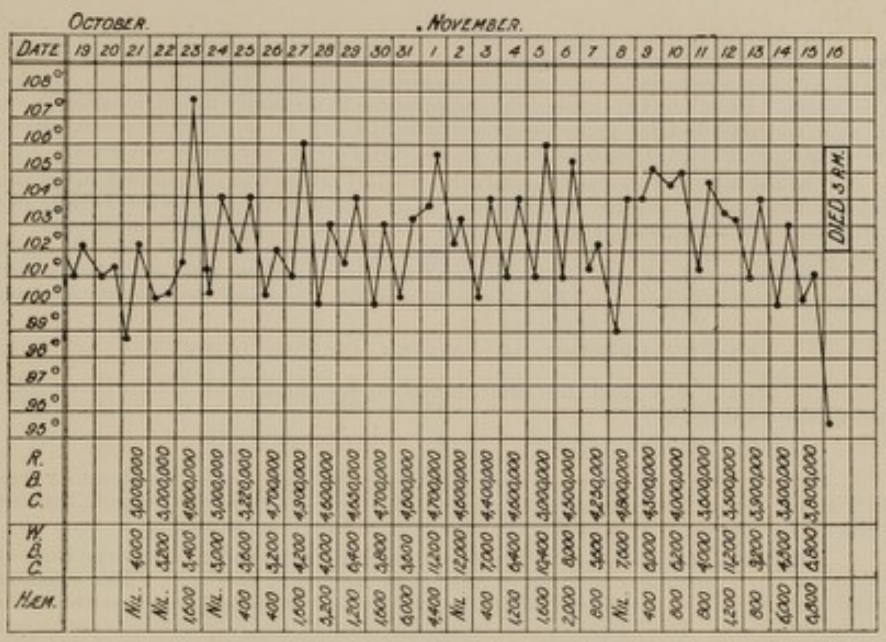
November 9th.—This morning a slight milky opacity of both corneæ is noted. The animal looks very low.

November 15th.—A herpetic eruption has appeared over the lips and nostrils.

November 16th.—The Galloway is dying. He is too weak to get on his feet this morning, and is lying down with his chin resting on the ground. Breathing shallow, pulse imperceptible. 3 p.m. shot.

The following chart represents the course of the disease:—





Autopsy.—One hour after death.

The body is extremely emaciated, there is no visible swelling of the extremities or under surface of the abdomen, and both corneæ are opaque.

On making an incision along the middle line of the body, a deposit or infiltration of a yellow coloured jelly-like material is seen in the subcutaneous tissue, especially about the chest and axillary regions, and also between the layers of muscle in various parts of the body. On opening the abdomen no fluid is found in the peritoneal cavity, the coils of intestine are pale, the spleen is enlarged and the liver dark in colour. On sawing through the sternum and opening the thorax the lungs are found to be collapsed, pink in colour and healthy in external appearance. There is no fluid in either pleural cavity

The pericardium contains six ounces of straw-coloured serum. The heart is distended with blood. The exposed surface of the heart is dark red in colour, especially so in the region of the coronary vessels, caused by the presence of numerous petechiæ under the epicardium. There is further a large deposit of yellow jelly-like material at the base of the heart and along the course of the coronary vessels, this deposit being nearly an inch in thickness at the base. On opening the ventricles the lining membrane of both is found to be marbled with petechiæ throughout; the auricles are normal in appearance, the valves are healthy, and the muscular tissue is pale in colour and flabby in consistence.

Both lungs are healthy.

The spleen measures 18 inches in length and 11 inches in greatest breadth. The capsule is bluish-white in colour, and many minute extravasations of blood are seen under the capsule. On section the spleen pulp is dark red in colour and much softened.

The liver is enlarged, the tissue is dark olive-green in colour and very friable.

The capsule of the kidneys strips off readily, exposing well marked venæ stellatæ. On section the kidney substance is pale in colour as if from fatty degeneration.

The stomach contains a few bot-fly larvæ, otherwise it is healthy, as also are the intestines.

Remarks.—This case is interesting as showing how readily a horse is affected by this disease on his being taken into the "Fly." On the 19th, 24th and 29th September he was ridden into the Fly Country. A supply of crushed mealies was taken, and the horse not permitted to eat a mouthful of grass while in the unhealthy district. On each occasion he was seen to be bitten many times by the Tsetse Fly. On the evening of the 4th October, fifteen days after his first visit to the low country, his temperature rises two degrees above normal, and next day he is found to be suffering from Nagana, and his blood contains the hæmatozoon. He gradually becomes weaker, and dies on the 16th November, forty-three days after the first appearance of the parasite in his blood.

On examining the temperature chart, the temperature is seen to range very fairly between normal limits from 19th September until the 4th October, except on the 23rd September, where a remarkable rise and corresponding fall take place, for which no reason can be given. From the 4th October the temperature is distinctly raised, sometimes very high, and characterised by great irregularity, until a day or two before death, when it shows a tendency to sink, falling suddenly on the day of death far below the normal.

The red blood corpuscles fall gradually from 5½ millions to 3½ millions in the cubic millimetre, with many irregular fluctuations. The white blood corpuscles remain fairly normal in number throughout, never being more than 12,000 per cubic millimetre. The hæmatozoa never rise to any astonishing number, 6,800 per cubic millimetre being the highest number registered, and generally speaking their numbers tend to point to some connection between the presence of the hæmatozoa in the blood and the temperature curve.

B.—FLY DISEASE OR NAGANA IN THE DONKEY.

The disease runs much the same course in the donkey as in the horse, as the following cases will show.

Exp. 219. Donkey, Mare.

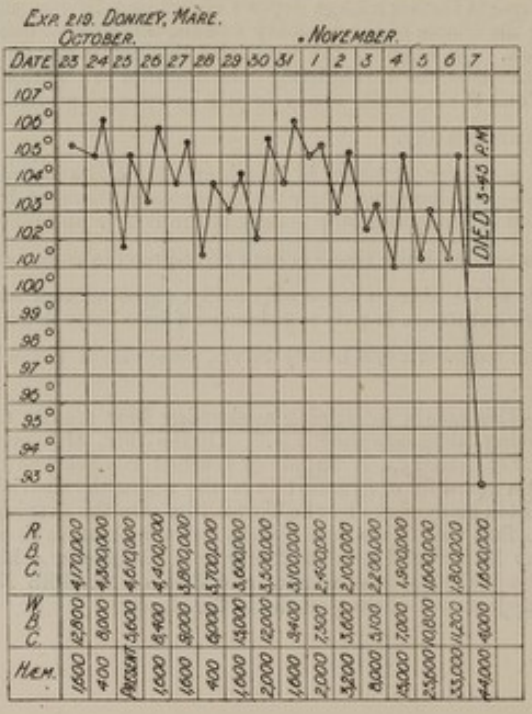
October 23rd.—This donkey was sent to me this morning by the Resident Magistrate, with the history that she had been down in the low country at the Pongola Drift from the 16th to the 19th October, and that she was now evidently out of health.

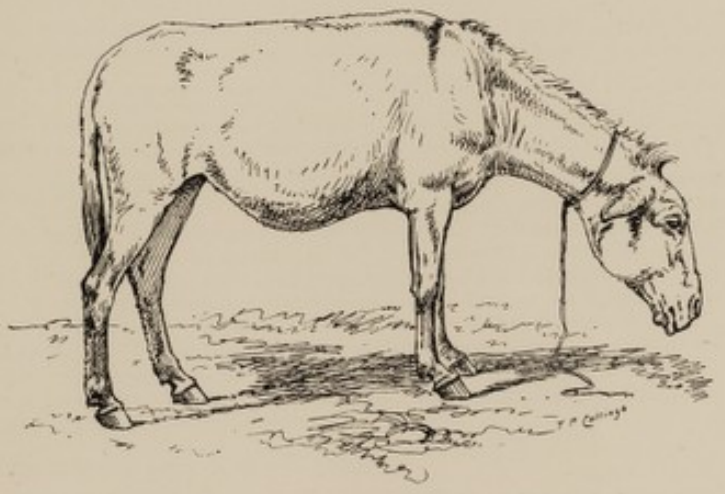
Her present condition is: hair harsh and staring, slight watery discharge from eyes and nostrils, and a prominent swelling on the under surface of the belly. This swelling is firm and painless to the touch, and measures quite a foot in length and breadth and two inches in thickness. The temperature is 105·4° F., pulse 90, and respirations 20 per minute. On examining a coverglass preparation of the blood the hæmatozoon of Nagana is seen.

November 2nd.—The condition of this donkey has been gradually growing worse day by day, but no prominent symptoms beyond those noted above have supervened. This morning she is looking very ill indeed, her head hangs, and she has generally the aspect of utter apathy and dejection. Her bloodlessness is extreme, the visible mucous membranes being almost white in colour, and blood drawn from an ear vein is almost colourless. She however continues to graze at intervals. Plate V shows the donkey in this condition.

November 7th.—This animal is in the last stage of debility, and reduced to mere skin and bone. She is nearly blind, as both corneæ have become cloudy during the last few days. The tongue and gums are white, and the conjunctival membrane is the seat of numerous petechiæ. The temperature has fallen 12 degrees since last night and is now 93° F. The pulse is almost imperceptible, and beats 64 to the minute, while the respirations have fallen to nine per minute. 1 p.m.—Has fallen down. 3.45 p.m.—Died.

The following chart represents the course of the disease.





Donkey suffering from Fly Disease or Nagana.

Autopsy.—Immediately after death.

The body is extremely emaciated, there is a large swelling on the under surface of the abdomen, there is no apparent swelling of the extremities, both corneæ are opaque, and the conjunctivæ petechial.

On cutting through the integument a thick layer of subcutaneous tissue infiltrated with a pale yellow jelly-like substance is seen extending from the ensiform cartilage backwards for some 18 inches, and corresponding in position to the swelling observed during life. The tissues are pale but not jaundiced. On opening the abdominal cavity 15 ounces of turbid yellowish fluid are found in the peritoneal cavity. The coils of large intestine are pale, the small intestine is somewhat injected, and the spleen is much enlarged. On cutting through the sternum and opening the thorax no fluid is found in either pleural cavity, the lungs are collapsed and healthy in appearance.

The pericardial sac contains 20 ounces of clear straw-coloured serum. The heart is pale in colour, and there is a slight deposition of yellow jelly-like material at the base. On removing the heart and opening the ventricles, their walls appear to be thinned, and the muscular tissue is seen to be extremely pale and softened. There are no petechiæ of the internal or external serous membrane visible, and the valves are healthy.

On removing the lungs and cutting into them, save for some emphysema of both apices and a few petechiæ showing through the visceral pleural membrane, they are found to be healthy.

The liver is enlarged, contains a large quantity of blood; the tissue is firm, pale chocolate in colour, and apparently moderately healthy.

The spleen measures 18 inches in length and 12 inches in breadth. The splenic pulp is dark in colour and softened, so that it can be readily scraped away by the knife.

The kidneys are very pale on section, but otherwise appear to be normal.

The stomach contains many larvæ of the bot fly, otherwise its lining membrane and that of the intestine present nothing markedly abnormal.

Remarks.—This is a case of spontaneous Nagana in the donkey, and is interesting as showing the rapidity and extent of blood destruction which may take place in this disease. The animal went down to the low country in good condition on the 16th October, returned on the 19th, was discovered to be ill on the 23rd, and died fifteen days later. During these fifteen days the red blood corpuscles fell from 4,170,000 to 1,600,000 per cubic millimetre, a condition of the most extreme anæmia. On examining the temperature chart the temperature is seen to run high until the day of death, when a sudden fall of twelve degrees takes place. The white blood corpuscles do not show any marked increase, 15,000 per cubic millimetre being the greatest number registered. The hæmatozoa, on the other hand, show a marked tendency to increase in numbers, and during the last days of the illness are present in very large numbers

Exps. 269 and 270. Donkeys, Geldings.

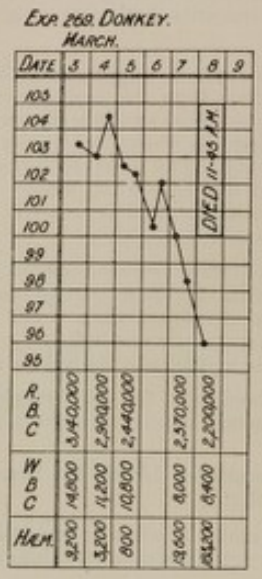
March 3rd.—These donkeys have of late been employed between Ubombo and Nongoma, and have undoubtedly become infected by Nagana while crossing the Segane. To-day while at work they were noticed to be out of sorts and were sent to me for examination. There is a watery discharge from the eyes and nose, a swelling under the belly of both, and their blood contains numerous hæmatozoa.

March 6th.—Exp. 270, died to-day at noon. Before death he showed a marked swelling of the sheath and a slight swelling under the belly.

March 8th.—Exp. 269, died at noon. He had the usual swelling under the belly, otherwise there were no marked symptoms.

On *post-mortem* examination these two donkeys were alike, and showed nothing very noteworthy. There was an infiltration of the subcutaneous tissue along the line of incision, no fluid in the peritoneal and pleural cavities, a slight deposit of the yellow, jelly-like material at the base of the heart; the lungs were healthy, the spleen enlarged, and the various organs congested.

The following charts represent the course of the disease :—



Remarks.—These are cases of spontaneous Nagana, and illustrate the rapidity with which these animals can be carried off by this disease. In all probability they were suffering from the disease for a week or ten days before being brought to me, as the number of their red blood corpuscles on the 3rd March would seem to indicate. During the time they were under observation they received no treatment whatever. The marked anæmia and the extraordinary number of the hæmatozoa are noteworthy.

Exp. 271.—Donkey, Gelding.

March 3rd.—This is a similar case to the two last, and presented the usual symptoms of running from the eyes and nose and a slight swelling under the belly.

March 14th.—Died, 5 a.m.

Autopsy.—Eight hours after death.

The body is emaciated, there is a slight swelling in the abdominal region, and both corneæ are clear. On opening the body a small quantity of clear serum is found in the peritoneal space, the stomach is small and contracted, the spleen small, the liver not enlarged, and the intestinal coils pale.

On sawing through the sternum and exposing the thoracic cavity both lungs appear healthy and normally collapsed, both pleural cavities contain about one ounce of clear serum.

The pericardial sac contains four ounces of clear straw coloured serum. There is a marked deposit of coagulated lymph at the base of the heart and along the course of the coronary vessels. The muscular substance of the heart is pale and glistening, otherwise the organ is healthy.

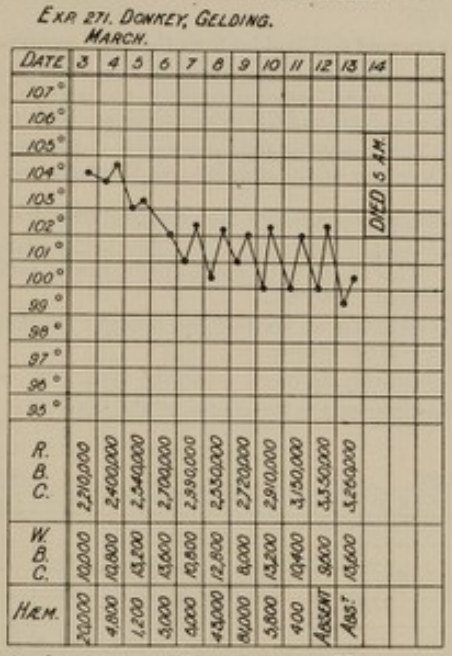
The liver is dark green in colour on section, very friable, and the lobules show marked fatty degeneration and chronic congestion.

The spleen on section is dark in colour and firm in consistence.

The kidneys are extremely pale in the cortical portion, and also show signs of congestion.

The stomach is quite empty except for a small quantity of greenish coloured fluid. One half the mucous membrane appears white in colour and healthy in appearance, the remainder has disappeared apparently by *post-mortem* digestion. There are no signs of arsenical irritation either in the stomach or intestines.

The following chart shows the course of the disease :—



Remarks.—This is also a case of spontaneous Nagana contracted in the Segana Valley, and only differs from the two previous ones by the fact that some treatment was attempted during the last five days. This accounts for the lessening in number and disappearance of the hæmatozoa during the last two days of the illness. Cause of death was probably failure of the heart's action, due to debility brought on by anæmia and want of nourishment, as shown by the empty condition of the stomach.

C.—FLY DISEASE OR NAGANA IN CATTLE.

This disease as it occurs in cattle may be of a much slower and more chronic nature than in the horse or the dog. When I visited Ubombo in November, 1894, I found a heifer on the hill, a mere hairless hidebound skeleton, which had had Nagana for months. On coming back a year later I found the same heifer still alive, and in fact much improved in appearance, although the blood still contained the hæmatozoa. But there is a great difference in the duration of the disease in cattle, a few will die within a week of taking the disease, many die within a month, and others linger on for six months or longer. The general opinion among the traders and natives in Zululand is that only a very small percentage recover.

The general symptoms in cattle are less marked than in horses or dogs. They gradually waste away, the hair, at first harsh and staring, tends to fall off, there is the same trickling of a watery fluid from the eyes and nose, and a tendency to diarrhœa, which, however, I have never found marked. In many cases the dew-lap becomes swollen and baggy, but I have not found the same tendency to the swelling of the under surface of the belly or the extremities as in the other animals, nor have I ever seen blindness occur in cattle. The hæmatozoa are also in my experience much less numerous in the blood of cattle than in that of horses and dogs, and often require to be looked for on several days in succession before they can be demonstrated.

In cattle, therefore, it would be rash to affirm that the disease from which they were suffering was not Nagana until the blood had been subjected to daily microscopical examination for at least a week.

The following two cases, with their accompanying charts, will sufficiently illustrate the course of the disease in cattle :—

Exp. 213. Black and White Heifer, 3 years. In good condition and healthy.

September 27th, 1895.—This heifer was procured from a healthy locality, and arrived at Ubombo to-day.

October 6th.—Up to the present this beast has appeared healthy and well nourished, and the temperature has remained regular.

To-day she has gone down to the camp in the Fly Country.

October 13th.—During the past week this animal has been lost, having strayed away on the night of the 6th.

October 20th.—Returned to Ubombo from the Fly Country.

October 21st.—On examining the blood a few hæmatozoa are seen, and the temperature has risen. The animal appears healthy, there are no swellings visible; the visible mucous membranes are clear, and the evacuations are healthy.

November 2nd.—The animal is falling away in condition, otherwise there are no noteworthy symptoms, no swelling of the dew-lap, no diarrhœa, and the animal appears to eat well.

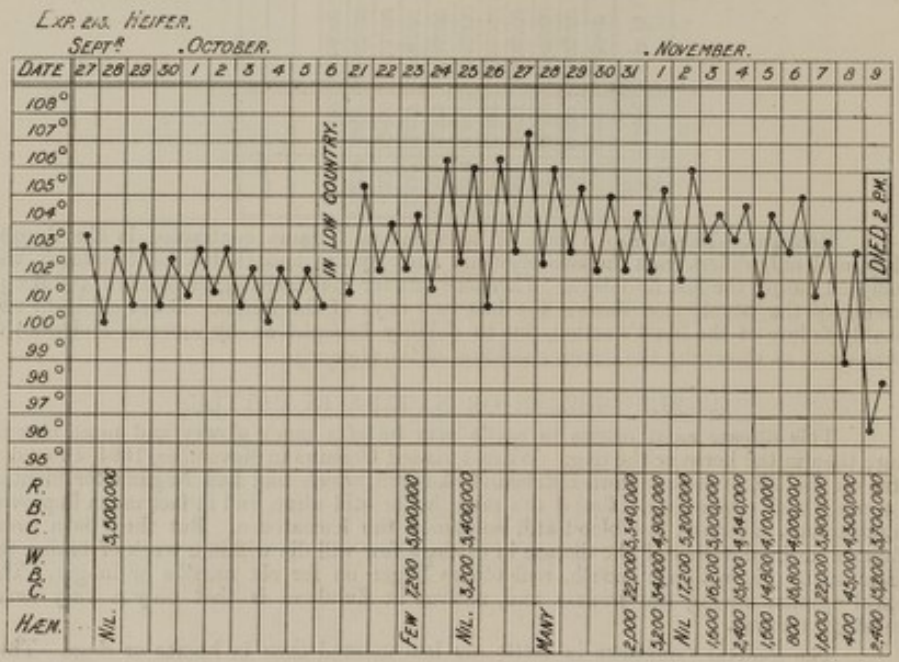
November 6th.—There was a heavy thunderstorm this afternoon, with much rain.

November 7th.—Weather wet and cold. The heifer was found lying down this afternoon unable to rise. This seems to be due to the cold and rain acting on a debilitated animal, rather than any inherent paralysis.

November 8th.—Got heifer on her feet and brought her down to kraal. Gave her hot coffee. There are no swellings visible.

November 9th.—Animal lying down evidently dying. 2 p.m. dead.

The following chart represents the course of the disease:—



Post mortem.—One hour after death.

The body is emaciated. Externally there is no swelling of the dew-lap or extremities. The lips and tongue are pale, as is also the mucous membrane lining the eyelids. On removing the skin a large number of patches of a dark red colour are seen in the transparent membrane or fascia covering the flesh. These are particularly numerous over the left side of the neck, and are due to extravasations of blood similar to what would be seen under a bruise, but in this case caused by an altered condition of the blood. This fascia, especially in the abdominal region, is orange-yellow in colour, and there is a deposition of a jelly-like material of the same colour in the region of the groin. In the loose tissue between the skin and the flesh, and between the muscular layers at various places, and especially about the region of the shoulders, a good deal of a yellowish fluid and thin jelly-like substance is also seen. This is the lymph or fluid part of the blood in a liquid or semi-coagulated condition which has escaped from the blood vessels, and is also due, like the patches of extravasation, to the altered condition of the blood.

On opening the abdominal cavity, a small quantity, not more than six ounces, of yellow-coloured serum is found; the viscera appear healthy, the spleen is enlarged, and the gall bladder is seen to be distended with bile to about the size of an orange.

On cutting through the sternum and opening the thorax no fluid is found in either pleural cavity, the lungs are collapsed, and healthy in appearance.

On opening the pericardial sac it is found to contain six ounces of clear serum.

The exposed surface of the heart is blotched over with patches of a dark red colour, some as large as half-a-crown, due to extravasation of blood under the membrane covering the heart. The patches or petechiæ are found all over the heart, especially numerous over the right auricle, and at the base of the heart there is a collection of the yellow jelly-like material. On opening the heart both ventricles are found to be distended with blood clot. The muscular tissue is pale and glistening, and the lining membrane is normal, except in the right auricle, where it is covered with petechiæ.

The right lung is emphysematous at the apex, the pleural membrane covering it is smooth, and there are no petechiæ. On section the lung tissue is pink in colour, and a small quantity of frothy blood exudes on pressure; the lung is quite healthy except for this slight œdema. The left lung is in the same condition as the right.

Liver.—The capsule is smooth, and dark purple in colour. On section the organ is found to contain much blood, there is some congestion, otherwise the tissue appears healthy. The gall bladder contains eight ounces of dark chocolate-coloured fluid bile.

The spleen is enlarged, measuring 20 inches in length and 7 inches in breadth. There are a few extravasations of blood under the capsule. On section the tissue is dark in colour, congested, and somewhat softened.

The kidneys are somewhat congested, but otherwise appear healthy.

In the stomach and intestines nothing pathological is to be noted.

Remarks.—This is a case of spontaneous Nagana set up by the animal grazing for a few days in the "Fly Country." The temperature chart shows that the fever had already begun on the return of the animal to the hill on the 21st October, and remained 3 or 4 degrees above normal for some seventeen days. On the 8th and 9th November there is a gradual sinking of the temperature, and a sudden fall on the day of death to several degrees below normal. The red blood corpuscles diminish rapidly in numbers during the course of the disease, falling from 5,500,000 to 3,700,000 per cubic millimetre. The white blood corpuscles show an increase in numbers. The hæmatozoa are never very numerous in the blood, and show no marked relation to the temperature or the course of the disease. The only symptoms in this case are emaciation and anæmia, and the death was no doubt hastened by the sudden onset of cold and rain.

Exp. 214. Cow.

September 27th, 1895.—This cow arrived to-day from Nongoma. Temperature 103° F. In good condition.

October 6th.—Up to the present this animal has appeared healthy and in good condition. No hæmatozoa in blood.

To-day she has been driven down to the camp in the "Fly Country."

October 13th.—During the last week this beast has been lost, having strayed away on the night of the 6th.

October 20th.—Returned to Ubombo from the Fly Country.

October 21st.—On examining the blood a few hæmatozoa are seen, and the temperature has risen. The animal appears healthy, there are no swellings visible, the mucous membranes are clear, and the evacuations are healthy.

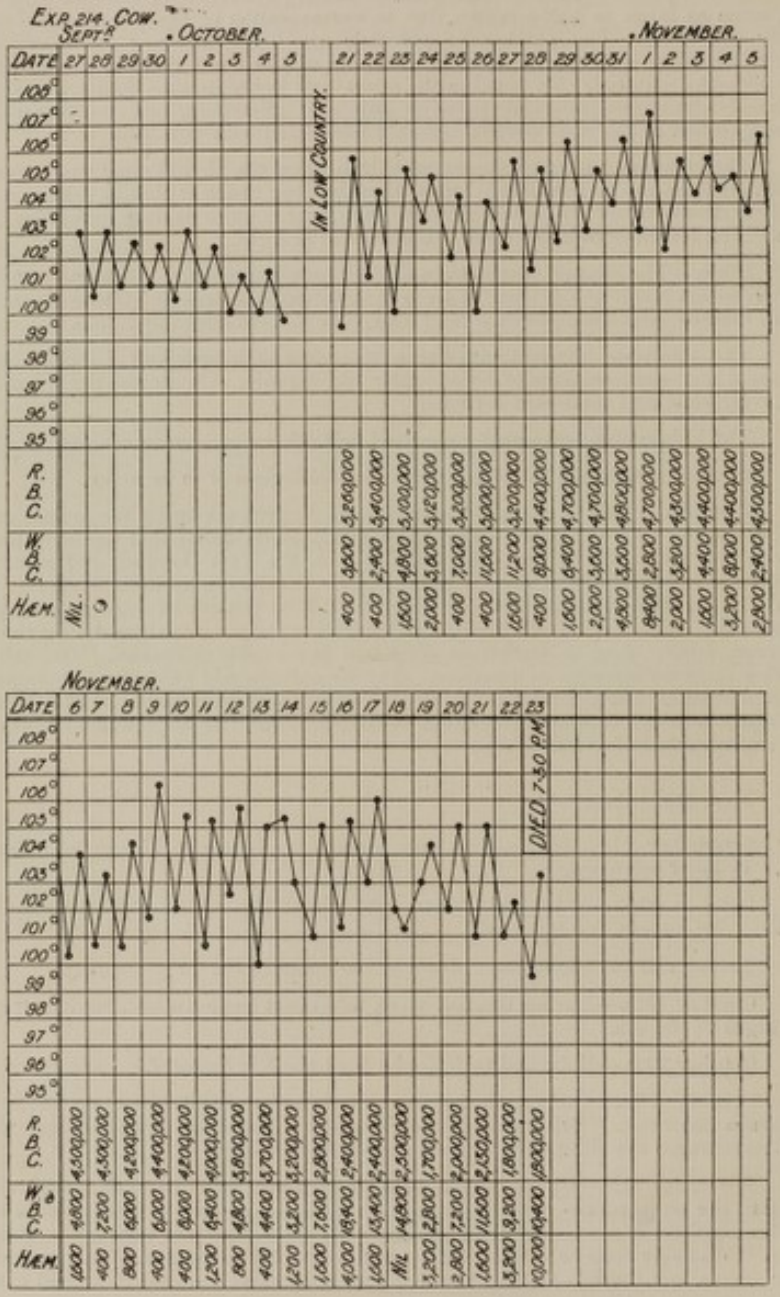
November 6th.—Up to the present there have been no noteworthy symptoms, except that the temperature has remained high, and the animal is falling off in condition. This morning there is a well marked swelling of the dew-lap. On taking the mass in the hand the fold of skin feels as if filled with a soft semi-solid substance. The animal is thin, the hair stares, and there is a slight running of watery fluid from the nose and eyes, otherwise there are no visible signs of disease.

November 13th.—This animal is much emaciated, and the hair is becoming thin, especially over the region of the neck. The swelling of the dew-lap has disappeared. There is no corneal opacity, no petechiæ on the conjunctiva, and the animal feeds well. There is a tendency to looseness of the bowels, but nothing which could be properly termed diarrhœa, much less profuse diarrhœa.

November 23rd.—This animal has become exceedingly weak, and is now lying down unable to rise.

7.30 p.m.—Died.

The following chart shows the course of the fever, number of parasites, &c.



Autopsy.—Thirteen hours after death.

The body is extremely emaciated, the hair thin and harsh in appearance, no visible swelling of the dew-lap, body or extremities. The corneæ are transparent, and the visible mucous membranes white. On making an incision along the ventral aspect of the body and reflecting the skin, the subcutaneous tissue is found destitute of fat. In various regions there is an infiltration of the subcutaneous tissue with a pale yellow jelly-like material. This is especially developed under the chin, in the axillary region, over the shoulders, on the inner aspect of the thigh, and in the intermuscular spaces of the neck and back.

On opening the abdominal cavity about 40 ounces of fluid are found in the peritoneal space, and the viscera appear pale but otherwise healthy. On sawing through the sternum and opening up the thoracic cavity, both lungs are seen to have collapsed normally, and there are a few ounces of clear serum in each pleural cavity. Over the exposed surface of the pericardium there is a marked deposit of jelly-like matter. The pericardial sac contains ten ounces of serum. The exposed surface of the heart is bright red in colour, from the presence of numerous extravasations of blood under the epicardium. Both ventricles are distended with blood.

On removing the heart the whole of the left ventricle is seen to be thickly blotched over with petechiæ, giving it a bright blood-red appearance. The anterior aspect of the right ventricle is also stained a bright red. The left auricle is also dotted over with petechiæ, while the right auricle is almost black in colour throughout its whole extent, as if it had been the seat of a violent bruise, from the same cause. At the base, and running to some extent along the coronary vessels, there is a large deposit of yellow jelly-like material, which at places is quite an inch in thickness.

On opening the left ventricle the muscular tissue is found to be pale in colour; it has a greasy feeling, and is somewhat more friable than normal, as the point of the finger can be pushed into it with some ease. The lining membrane of the left ventricle is pale, blotched with yellow, and having here and there a few small petechiæ. The interior of the left auricle is studded all over with bright red petechiæ. The lining membrane of the right ventricle is also exceedingly pale and streaked with yellow, and a few petechiæ are seen. The interior of the right auricle is stained dark red from the same cause.

The Right Lung.—The pleural membrane is smooth, there is a little emphysema at the apex. Here and there are patches of dark purple on the light red surface. On section the general colour is pink, with a few darker patches where the lung tissue appears to be collapsed, airless, and carnified. By far the greater part of the lung, however, is healthy, although somewhat œdematous.

Left Lung.—Pale throughout. On section the tissue appears to be healthy.

Liver.—This organ is enlarged, the gall-bladder contains a quantity of yellow coloured fluid bile. On section the liver substance is found to be in an advanced condition of chronic congestion and fatty degeneration.

Spleen—Is enlarged, and measures 23 inches in length and 8 inches in breadth. The capsule is bluish-white in colour, and is dotted over with minute petechiæ. On section the tissue is dark in colour and extremely soft.

Right Kidney.—The capsule strips off easily, leaving the surface underneath dotted all over with dark red points. On section the cortex is found to be exceedingly pale, with parallel lines of congested vessels. The left kidney is in the same condition as the right.

Stomach.—Contains a large quantity of grass. Nothing of a marked pathological character is noted in the stomach or intestines.

Remarks.—This is a companion case to the last. The temperature chart shows that the fever had begun before the animal's return to Ubombo on the 21st October, and remained some degrees above normal until the fatal termination of the disease. The red blood corpuscles fell from 5,260,000 to 1,800,000 per cubic millimetre. The white blood corpuscles remained within fairly normal limits. The hæmatozoa were never very numerous, although they were found to be present every day except one, and showed a tendency to rise in numbers towards the end.

D.—THE FLY DISEASE OR NAGANA IN DOGS.

This disease is in my experience rapid, and invariably fatal. The chief symptoms are extreme emaciation, swelling of the extremities, eruption over the body with the formation of blebs and pustules containing more or less purulent matter, and finally milky opacity of the corneæ, giving rise to blindness.

The following two cases illustrate the disease as it occurs spontaneously in dogs, and a perusal of them and an examination of the attached charts will show what a serious disease Nagana is in the species.

Exp. 189. Dog.—Breed, Pointer.

This dog was taken into the "Fly" on the 12th December, and remained there until the 18th, when he returned to Ubombo. On the 12th and following two days he accompanied me into the thorns. He stayed in camp during the remaining three days. The camp was situated on a ridge some distance from the thorns, but was not altogether free from the Tsetse Fly. During the time the dog was in the low country he appeared in excellent health and his temperature remained normal.

December 21st.—This morning the dog is seedy, lies about in a listless way, refuses to eat, and his near hind leg is somewhat swollen. His temperature has gone up the last two days, and on examining his blood the hæmatozoa are found to be present.

December 23rd.—The near hind leg is now puffy and swollen to a marked extent; below the knee joint the sound limb measures 7·2 inches in circumference, the unsound 9·5 inches.

December 24th.—The leg is not more swollen, but is this morning the seat of an eruption of largish yellow-coloured blebs or blisters containing purulent fluid. There are no other prominent symptoms, except that the dog looks ill and his coat is harsh and staring.

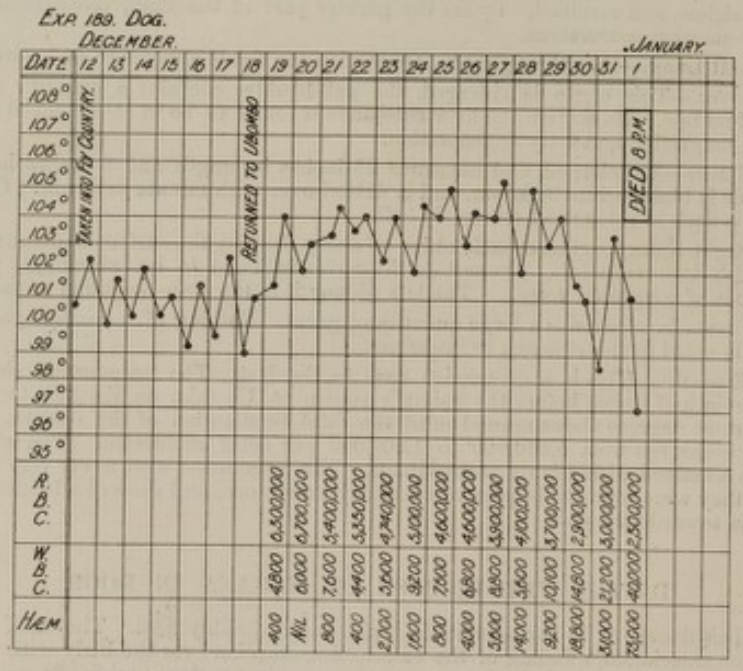
December 31st.—The dog looks very bad indeed. He has become greatly emaciated, and is covered with a pustular eruption which gives him a mangy look. His left hind leg is still puffy and swollen, and further the tissues of the scrotum have become infiltrated and enlarged, the swelling having a boggy feeling to the touch. His eyes have become affected, both corneæ being opaque and milky in appearance, and more especially the right. He walks with difficulty, as if he were blind, lies about all day, and eats very little.

January 1st.—He is unable to stand this morning. The left hind leg is much swollen, and wet by the transudation of moisture from the œdematous limb. The left fore leg is also swollen this morning. His hair is coming off in patches. He is blind.

Plate VI represents this dog some hours before death. The swelling of the extremities and the general emaciation are well shown.

8 p.m.—Died.

The following chart represents the course of the disease.



Autopsy.—Twelve hours after death.

The body is extremely emaciated. *Rigor mortis* is absent. The skin over the abdominal region is yellow in colour. The conjunctivæ are yellow, the gums pale yellow. The left hind leg is much swollen and pits on pressure. The abdominal region is retracted. The whole of the skin is covered with a pustular eruption.

On making an incision from the chin to the pubic region the subcutaneous tissue is seen to be distinctly yellow in colour. On reflecting the skin the subcutaneous tissue, especially in the region of the left groin and left hind leg, is found to be infiltrated with yellowish jelly-like matter, from which a quantity of pale yellow fluid exudes. The right groin is affected in the same way, but to a less extent. The axillary glands on both sides are swollen, some being as large as a walnut. On cutting into these a purulent fluid escapes.

DOG SUFFERING FROM FLY DISEASE OR NAGANA.

On opening the abdomen, 4 ounces of blood-stained fluid are found in the peritoneal cavity. The spleen is enormously enlarged and the stomach and intestines appear healthy. On cutting through the costal cartilages and opening the thorax, no fluid is found in either pleural cavity; the lungs are collapsed and appear healthy.

The pericardial sac contains a small quantity of bile stained serum. The heart is distended with blood. On section the muscular tissue is pale and softened, otherwise no pathological lesion can be noted.

Both lungs are healthy.

The liver is somewhat enlarged, in colour dark and jaundiced. On section the tissue is found to be congested and extremely friable.

The spleen measures 11 inches in length and 4 inches in breadth, it is purplish in colour, and on section is found to be excessively soft and pulpy.

Kidneys.—The capsules strip off readily. On section the organs are pale, the cortical part especially being pale yellow in colour.

Stomach and small intestines.—Empty and healthy.

Large intestine.—On its mucous surface one small patch of ulceration about the size of a sixpence is seen.

In connection with the wall of the œsophagus two tumours of *Spiroptera Sanguinolenta* are found, one as large as a walnut, the other the size of a French bean. These tumours contain many worms.

The lymphatic glands in the left groin are much enlarged, and on section a purulent fluid escapes. An abscess some 5 inches in length and 1½ inches broad lies along the left iliac vessels on the brim of the pelvis. This abscess appears to have been formed in enlarged lymphatic glands, and contains a quantity of pus. The corresponding glands on the right side are much swollen, but have only broken down here and there into pus.

Remarks.—This is a case of spontaneous Nagana in the dog. The animal was exposed to the influence of the "Fly Country" on the 12th December and following days. Eight days after exposure, that is on the 19th, the temperature rose and the hæmatozoa were found in the blood. The disease ran a rapid course, and the dog died thirteen days after the first appearance of the parasite. The principal symptoms were rapid emaciation, swelling of the extremities and blindness. On examining the temperature chart, the principal things to be noted are the continuous fever, never rising very high, and the tendency to fall below normal at the last. The red blood corpuscles fall rapidly from 6½ millions to 2½ millions per cubic millimetre. The white blood corpuscles tend to increase greatly in numbers during the last three days. The hæmatozoa show a like tendency to rise in numbers as the disease progresses, and on the day of death are seen to number as many as 73,000 per cubic millimetre.

Exp. 190. Dog. Breed, Pointer.

This dog was taken into the "Fly" on the 12th December, and remained there until the 18th, when he returned to Ubombo. On the 12th and following two days he accompanied me into the thorns. He stayed in camp during the remaining three days. The camp was situated on a ridge some distance from the thorns, but was not altogether free from the Tsetse.

During the time the dog was in the low country he appeared in excellent health, and his temperature remained normal.

December 23rd.—As this dog's temperature rose suddenly last night and remains high this morning, I have examined his blood and find hæmatozoa present.

December 31st.—This dog has been off his feed the last few days, is falling off in condition rapidly, and is already very thin. His coat is harsh and staring. Yesterday his left hind leg was noticed to be swollen, but this swelling has almost disappeared to-day.

January 2nd.—Is much swollen around the eyes.

January 3rd.—The whole of the face is much swollen this morning. The corneæ of both eyes are opaque and milky. The left leg and scrotum are swollen. The hair is harsh. There is a pustular eruption on the skin causing the hair to be reversed and giving the coat a rough appearance.

January 4th.—The dog is quite blind this morning, otherwise his condition is much the same.

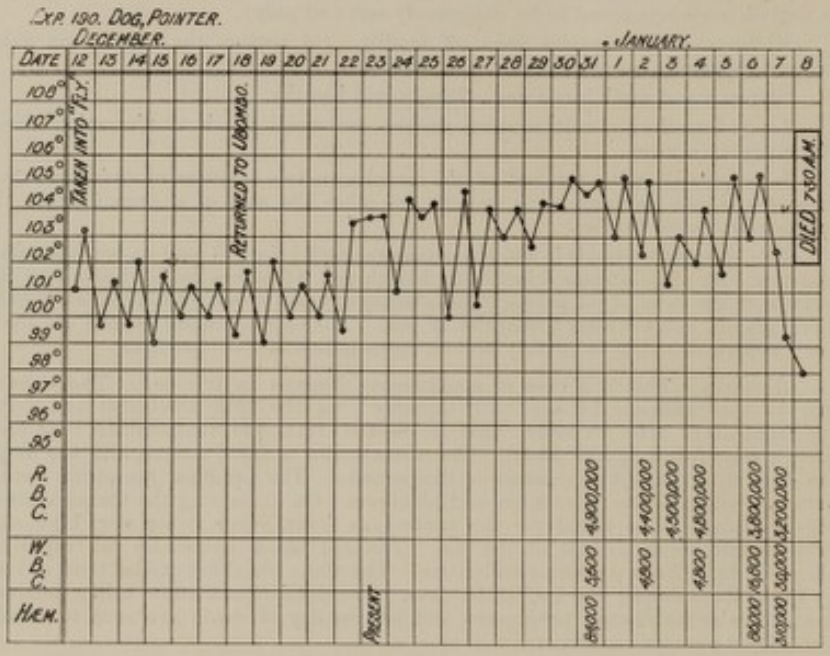
January 6th.—Both hind legs are much swollen this morning. He is quite blind and growing weaker.

January 7th.—The dog can barely walk this morning. Both hind legs, the left front leg and face are much swollen. The scrotum is as large as an orange. The whole body is covered with a pustular eruption. Here and there are flat collections of pus under the skin as large as a shilling, and showing greenish-yellow through the hair. He is still quite blind. He lies on his side, breathing deeply about twenty times to the minute. Is quite conscious, and wags his tail if spoken to. Still eats a little meat, but refuses biscuit.

1 p.m. Is very weak, and cannot walk without assistance. 11 p.m., still alive. Ate a little meat.

January 8th.—Dead and stiff at 7.30 a.m.

The following chart shows the course of the disease.



Autopsy.—8.30 a.m.

Rigor mortis present. The body is extremely emaciated, and the abdomen retracted. The whole of the body from the nose to the tip of the tail is covered with a pustular eruption. The hair over these pustules is matted together by the discharge. Many of the pustules are large and freely discharge pus on pressure. On removing the crusts, the skin underneath is found to be ulcerated. The nose is much swollen. The mucous membrane of the lips and gums extremely anæmic. Both corneæ are milky and opaque, so that the condition of the pupils cannot be seen through them. The left fore leg is slightly swollen. The left hind leg is much swollen and pits on pressure. The right hind leg is somewhat enlarged in its lower half. The scrotum is much enlarged, and also pits on pressure.

On making an incision from the chin to the vent, the subcutaneous tissue is not jaundiced as in the last case. The subcutaneous tissue of both hind extremities is infiltrated with a pale yellow jelly-like substance, from which transparent watery fluid exudes. The glands on both sides of the groin are much swollen, and measure 2½ inches in length. On cutting into them the glandular tissue is found to be œdematous, brown in colour, but there are no signs of pus formation. The glands in the axillary region are also enlarged, but none show any signs of pus formation.

On opening the abdomen a few ounces of clear serum is found in the peritoneal cavity. The spleen is enormously enlarged, the liver and other organs present nothing noteworthy. On opening the thorax, both lungs are seen to be collapsed, and there is little or no fluid in the pleural cavities. The pericardium is infiltrated on its anterior aspect with a pale yellowish jelly-like substance, and the pericardial cavity contains a small quantity of clear straw coloured serum having many pale coloured flocculi suspended in it.

The heart is distended with blood. The external surface is smooth and pale. On cutting into the heart the lining membrane is seen to be smooth, the valves are healthy, and the muscular substance pale and somewhat softened.

On slitting up the œsophagus, a swelling is seen in its walls about 3 inches above the cardiac end of the stomach. This swelling is about the size of a small walnut and communicates with the interior of the œsophagus by two openings, through which, on pressure, purulent matter exudes. On cutting into this tumour four worms are found similar to those found in Exp. 189.

Lungs.—Healthy.

Liver.—On section the tissue is seen to be in a condition of fatty degeneration and chronic congestion.

Spleen—Measures 13 inches in length and 3 inches in greatest breadth. The tissue is soft and pulpy.

Kidneys.—Capsules strip off readily. Cortical portions pale, with congestion of the vessels.

Stomach.—Contains a mass of straw, hair and undigested food.

Small and large intestines—Anæmic, otherwise present no pathological condition.

Eyes.—On opening the anterior chamber, an opaque colourless film is found lying between the cornea and the lens. The cornea itself is perfectly transparent, as is also the lens. The opacity during life is evidently due to this condition of the fluid in the anterior chamber and not to any inherent opacity of the cornea.

Remarks.—This case is the companion case to the last (Exp. 189) and is so similar that the remarks made in it would apply equally to this. The rapidity of onset is somewhat less, being twelve days instead of eight, and death is also slightly delayed, being sixteen days after the first appearance of the hæmatozoa instead of thirteen. Otherwise the course and symptoms of the disease are almost identical. The rapid emaciation, swellings and blindness are found in this case equally with the last.

The red blood corpuscles rapidly diminish in numbers, the white increase towards the end, and the hæmatozoa show the enormous number of 310,000 per cubic millimetre on the last day of the disease.

7.—INOCULATION OF BLOOD FROM AFFECTED TO HEALTHY ANIMALS.

A.—The following experiments were made in order to discover if the Fly Disease can be transmitted to native dogs by the inoculation of blood from affected animals:—

A.—INOCULATION OF BLOOD IN WHICH THE HÆMATOZOA CAN BE DEMONSTRATED BY THE MICROSCOPE.

Exp. 220.—Dog inoculated from horse. Contracted disease five days later.

Exp. 221.—Dog inoculated from horse. Contracted disease four days later.

Exp. 204.—Dog inoculated from cow. Contracted disease twenty-three days later.

From these experiments it will be seen that inoculations into native dogs of blood from wild animals would prove successful if the blood of these wild animals contains the hæmatozoon of the Fly Disease.

Exp. 220. Dog, Native.

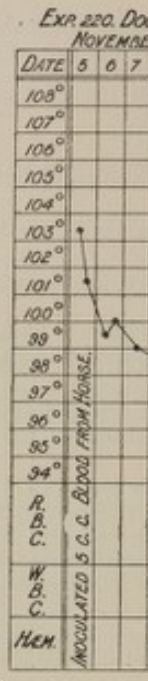
Experiment.—To try the effect of inoculating a native dog with blood from a horse suffering from Nagana.

November 5th, 1895.—Removed 5 c.c. of blood from the jugular vein of Exp. 212, Bay Horse, and at once injected it under the skin of native dog.

November 10th.—As the temperature of this dog rose to 104°·2 F. at 5 p.m. yesterday, I have examined his blood this morning, and find it contains very numerous hæmatozoa.

November 15th.—This dog died this morning. Except progressive emaciation and debility the case has shown no external symptoms. There never was any swelling at the site of inoculation. No swelling of the extremities or of any part of the body occurred. No corneal opacity, nor eruption of any kind.

On examining the temperature chart, the rapid onset of fever, the rapid destruction of the red blood corpuscles, the increase in numbers of the white blood corpuscles, the very large number of hæmatozoa which suddenly appeared in the blood and increased to almost 100,000 per cubic millimetre before death, are the most interesting points to be noted.



Exp. 221. DOG, NATIVE.

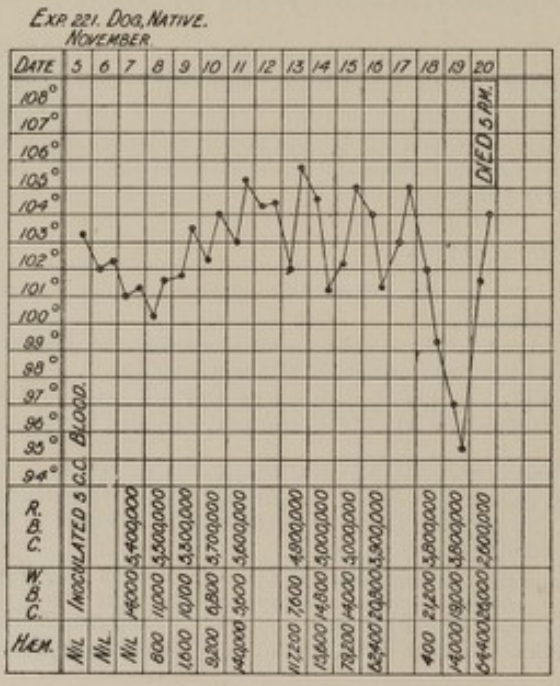
November 5th, 1895.—Injected 5 c.c. of blood from the jugular vein of horse, Exp. 212, under the skin of this dog in the abdominal region.

November 9th.—To-day the temperature shows a tendency to rise, and on examination of the blood a few hæmatozoa are found.

November 20th.—This case has run a rapid course, and has been characterised by rapid emaciation and anæmia, without any other noteworthy symptoms.

Died 5 p.m.

The following chart shows the course of the disease:—



Autopsy.—Immediately after death.

The body is that of a small native dog, and is in the last stage of emaciation, being mere skin and bone. *Rigor mortis* is absent. Both corneæ are slightly opaque. There is no visible external swelling of the body or extremities. On making an incision from the chin to the pubes and reflecting the skin, the subcutaneous tissue is found to be infiltrated to some extent with a pale watery jelly-like substance, especially over the region of the chest, groin, and thigh on the left side.

On opening the abdominal cavity no fluid is found, the intestinal coils appear healthy, the spleen is enlarged to more than double its usual size, and the liver is also enlarged. The abdominal lymphatic glands near the bifurcation of the aorta are enlarged, and on cutting into them a quantity of slightly opaque purulent fluid escapes from them. On cutting through the costal cartilages and opening the thorax, 6 to 8 ounces of opaque purulent serum are found in both pleural cavities. The lungs are collapsed. The pleural membrane lining the chest wall on the left side is red in colour, the blood vessels are injected, and the condition is evidently one of commencing acute pleurisy.

On opening the pericardium a small quantity of clear serum is found. The heart is distended with blood, the blood vessels on its surface are injected, but there are no petechiæ on its surface or jelly-like material at the base. On removing the heart and opening into the ventricles the lining membrane is found to be free from petechiæ and the valves are healthy.

Both lungs are healthy.

In connection with the œsophagus there is a large tumour containing very numerous nematode worms, probably *Spiroptera Sanguinolenta.*

The liver is enlarged, has many yellow coloured concretions on its surface and in its substance. The liver tissue contains much blood, is pale in colour, and is in a state of chronic congestion, with fatty degeneration.

The spleen measures 6 inches in length and 3 inches in its greatest breadth. On section the splenic pulp is pale in colour and softened.

The left kidney.—The capsule strips off readily. On section the organ is extremely pale, especially the cortical portion, which is of a light yellow colour, and evidently in a state of fatty degeneration.

The right kidney.—Several small cysts, containing a clear fluid, appear on the surface of the organ under the capsule. The capsule strips off readily. On section the kidney substance is in a state similar to that of the left.

Remarks.—This case is very similar to its companion, Exp. 220. Like it, it is remarkable for the rapid destruction of the blood, the increase in number of the white blood corpuscles, and the extraordinary number of hæmatozoa present in the blood throughout, rising on the 12th November to as many as 140,000 per cubic millimetre. The temperature curve shows also the same tendency to fall far below the normal as the case approaches its fatal termination.

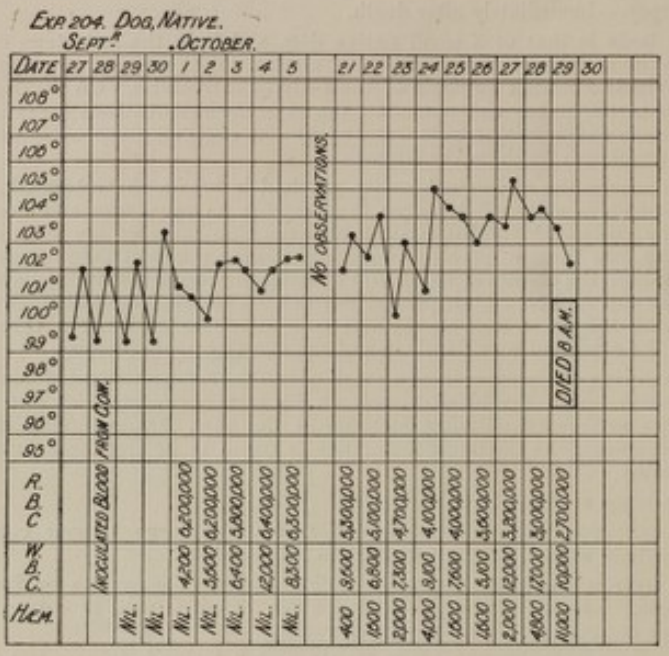
Exp. 204. Dog, Native.

September 28th.—Injected 4 c.c. of blood containing a few hæmatozoa from Exp. 207, Cow, under skin of healthy native dog.

October 21st.—On my return from the low country yesterday I found this dog evidently in good health, but on examination of his blood this morning a few hæmatozoa are found to be present.

October 30th, 1895.—This dog has been listless for the last few days, lying about and taking no interest in his surroundings. This morning I found him in convulsions, and he died at 8.30 a.m.

The following chart shows the course of the disease:—



Autopsy.—One hour after death.

This dog is about the size of a large English Fox Terrier, and is not markedly emaciated. There is no swelling of the extremities or of the body to be seen by external examination. Both eyes appear normal.

On making an incision from the chin to the pubes and reflecting the skin, a small quantity of subcutaneous fat is found, showing the dog to be well nourished. There is no infiltration of lymph or jelly like material into the subcutaneous tissue in any region, nor are the axillary or inguinal glands enlarged.

On opening the abdominal cavity no fluid is found, the gall-bladder is partially distended with bile, the spleen is enormously enlarged, the coils of the intestines appear quite healthy. On opening the thorax no fluid is found in either pleural cavity, both lungs are collapsed, the right lung appears perfectly healthy, but there is an effusion of lymph on the posterior aspect of the left lung, showing some inflammatory action. This lymph appears to be quite recent in its formation, is red in colour, and readily detachable. This inflammation extends along the left side of the œsophagus. On cutting into the pericardial sac no fluid is found in this cavity.

The heart does not present any marked pathological change, either externally or internally.

The lungs are both healthy, except for the patch of pleurisy on the posterior aspect of the left lung where it comes in contact with the œsophagus. In the wall of the œsophagus in this region there is a tumour as large as a walnut, and containing many nematode worms (*Spiroptera Sanguinolenta*). This tumour has ruptured and given rise to the localised pleurisy mentioned above. There is also another tumour of the same kind, but smaller in size, in connection with the wall of the aorta.

The liver is enlarged and the gall-bladder somewhat distended with dark coloured viscid bile. On section the organ contains much blood, otherwise the tissue appears normal.

The spleen is enormously enlarged, measuring 12 inches in length and 4 inches in its greatest breadth. On section the tissue of the spleen is much softened, the splenic pulp being easily scraped away by a knife.

Right kidney.—The capsule strips off readily. On section the organ is pale, especially the cortical portion, which is of a pale yellow colour.

Left kidney.—Same condition as right.

The stomach and intestines present nothing abnormal.

On examining a coverglass preparation of spleen pulp no hæmatozoa can be seen either in a stained or unstained preparation. On the other hand in the blood from an internal vessel examined in the same way, very numerous living hæmatozoa are seen and also many motionless ones, which by their coarsely granular appearance appear to be dead. In a stained preparation of blood large aggregations of these granular partly disintegrated hæmatozoa are seen scattered all over the field, so that the hæmatozoa in this blood may be described as countless.

Remarks.—Death in this case occurred suddenly, and was probably hastened or indeed caused by the rupture of the Spiroptera tumour into the left pleural cavity, and subsequent septic pleuritis. The temperature chart shows the usual rise in temperature, the destruction of the red blood corpuscles, and the occurrence of the hæmatozoa in the blood.

B.—INOCULATION OF BLOOD IN WHICH THE HÆMATOZOA CANNOT BE DEMONSTRATED BY THE MICROSCOPE.

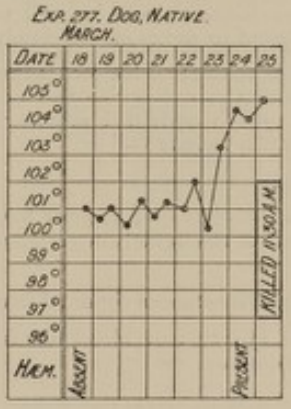
These experiments have a bearing on the inoculation of blood from wild animals into dogs, as will be seen from the experiments, under these circumstances dogs sometimes do and sometimes do not take the disease. It is evident then that many of the wild animals whose blood injected into dogs did not produce the disease, may have been harbouring the parasite in some form or other or in very small numbers, and were in fact suffering from the disease.

Exp. 277. Dog, Native.

The donkey (Exp. 258) under xii grs. arsenic daily, has had no hæmatozoa in his blood for 35 days as recorded by the microscope; will his blood give rise to the disease in a healthy dog?

March 18th.—Inoculated sub-cutaneously 5 c.c. blood from Exp. 258.

The following chart shows the result.



Exp. 279. Dog, Native.

The donkey (Exp. 257), under xii grs. arsenic daily, has had no hæmatozoa in his blood for 32 days as recorded by the microscope; will his blood give rise to the disease in a healthy dog?

March 19th.—Inoculated 1 c.cm. blood from Exp. 257.

April 21st.—Dog healthy. No hæmatozoa in blood. The hæmatozoa again appeared in blood of the donkey (Exp. 257) on April 23rd.

Exp. 280. Dog, Native.

The horse (Exp. 256), under xii grs. arsenic daily, has had no hæmatozoa in his blood for 36 days.

March 20th.—Inoculated 4 c.cm. blood from Exp. 256.

April 21st.—Dog healthy. No hæmatozoa in blood. The horse (Exp. 256) died of Nagana on March 31st.

8.--FEEDING HEALTHY ANIMALS ON TISSUES FROM ANIMALS AFFECTED BY NAGANA.

The following experiment was made in order to discover if this disease can be transmitted by feeding a healthy dog on the raw tissues of an animal dead of Nagana.

Exp. 223. Black and White Dog, Native.

November 9th, 1895.—This dog ate a piece of coagulated blood from the heart of heifer, Exp. 216.

November 15th.—This dog has a large swelling under the jaw and throat, he is rapidly growing thinner, and there are numerous hæmatozoa in his blood.

November 18th.—Large swelling under throat.

November 21st.—Blood contains many hæmatozoa. This animal is suffering from Nagana.

In the Segane Valley many of the dogs belonging to one of the kraals fed on the raw flesh of a Quagga, and I am informed that several of these dogs subsequently died of Nagana.

Mr. B. G. Lloyd also states that in the winter of 1892, Mr. Saunderson, of Spitz Kop, Z.A.R., took some oxen into the "Fly" hunting and lost them all, though one of them died after returning to the farm. Some dogs got at the dead bullock and ate a lot of the meat raw. They all died within three months, showing all the signs of "Fly," whereas some pups which were kept shut up were fed on the same meat, but cooked, for several days, yet none of them showed any sign of being affected.

9.—MEDICINAL TREATMENT.

On account of the very great similarity which exists between the parasites of Nagana and Surra, and also in the symptoms of the two diseases, it may be assumed that a medicinal treatment which affects the one will affect the other. Now as a most painstaking investigation has been proceeding for several years in India under Dr. A. Lingard, whose reports, entitled "Report on Horse Surra, 1893," and "Summary of Further Report on Surra, 1894," have already been issued, it will be well to give here a brief account of what has been done in the treatment by drugs of the closely allied or identical disease.

Dr. Lingard prefaces his account of his own experimental inquiry into the treatment of Surra, by the statement that few observers had tried any systematic treatment in this disease, and those few had unfortunately had only negative results. He then proceeds to detail his own experiments, which include:—

(*a.*) Treatment by drugs.

(*b.*) The subcutaneous inoculation of animals by serum derived from blood containing swarms of Surra Hæmatozoa, the latter being removed by passing the serum through a porcelain filter.

(*c.*) The injection of solid substances obtained from Surra blood.

(*d.*) The subcutaneous injection of blood obtained from an animal which had recovered from an attack of the disease.

The drugs experimented with were the following, and they were chosen on account of their proved power of destroying low animal and vegetable parasites:—

Perchloride of mercury.
Iodine and iodide of potassium.
Bichromate of potash.
Iodoform.
Turpentine.
Carbolic acid and iodine.
Hydrate of potash.
Quinine
Arsenic.

It may briefly be stated that none of these drugs, with the exception of arsenic, had any beneficial influence on the course of the disease. In regard to arsenic, however, Dr. Lingard found that after 2 to 8 days' administration of the drug the hæmatozoa disappeared from the blood and remained absent for long periods. In his first report all the animals subjected to this treatment died, but in his "Summary to Further Report," he gives a case which has evidently recovered. As this is an important point, his account of this case may be given in full.

"The animal contracted Surra by gastric ingestion of soiled blood, after a latent period of 6 days. "Treatment was commenced at once, consisting of arsenic, as liq. arsenicalis, in 4 gr. doses, increasing rapidly to "12 grs. daily. After a paroxysm of 5 days, during which time the hæmatozoon was very numerous, it disap- "peared from the blood. The arsenical treatment was continued for 65 days, 454 grs. of arsenic being "administered. At this period the animal showed an aversion to the arsenic in this form. For this reason, "and as previous experience pointed to the fact that arsenic by itself did not, in some cases, destroy the resting "or immature form of the hæmatozoon, the double iodide of arsenic and mercury was substituted, as it was "known that the iodide of mercury does not form an albuminate, and consequently the whole of the salt would "be available. The iodides were continued for a period of 106 days in order to ensure the absolute destruction "of the immature form. In all, 288 grs. each of the tri-iodide of arsenic and bin-iodide of mercury were given. "Since the medicine was discontinued about 8 weeks have elapsed, and the animal is in good health and con- "dition, and no hæmatozoon has appeared in the circulation for 223 days, or over 7 months. For 6½ months "during this period the animal has averaged 4½ miles walking exercise daily. As it did not thrive on grain, "barley (boiled) was given in its place, with the most satisfactory results, the animal rapidly putting on flesh."

I have not heard whether this horse has remained healthy up to the present or not. From Dr. Lingard's own cases it is apparent that the cure of the disease by arsenic is the exception and not the rule, even when the treatment has been carried out under the most favourable circumstances. I confess I am at a loss to understand what is meant by "the "resting or immature form of the hæmatozoon" as I have never seen any evidence of the existence of such a form. Probably Dr. Lingard will throw more light on this point in his next report.

A.—ARSENIC AS A CURATIVE AGENT.

(1.) Treatment in Horses.

Since I have been here at Ubombo I have tried the arsenical treatment on several horses, donkeys, and dogs, but in this interim report I would rather not make any general statement concerning the usefulness or otherwise of the drug, leaving rather the reader to draw his own conclusions from the experiments themselves. Only in two horses was the disease allowed to run its course without any treatment. These were:—

Exp. 212. Horse, Bay, page 26. Number of days from beginning of disease until death, about 22 days.

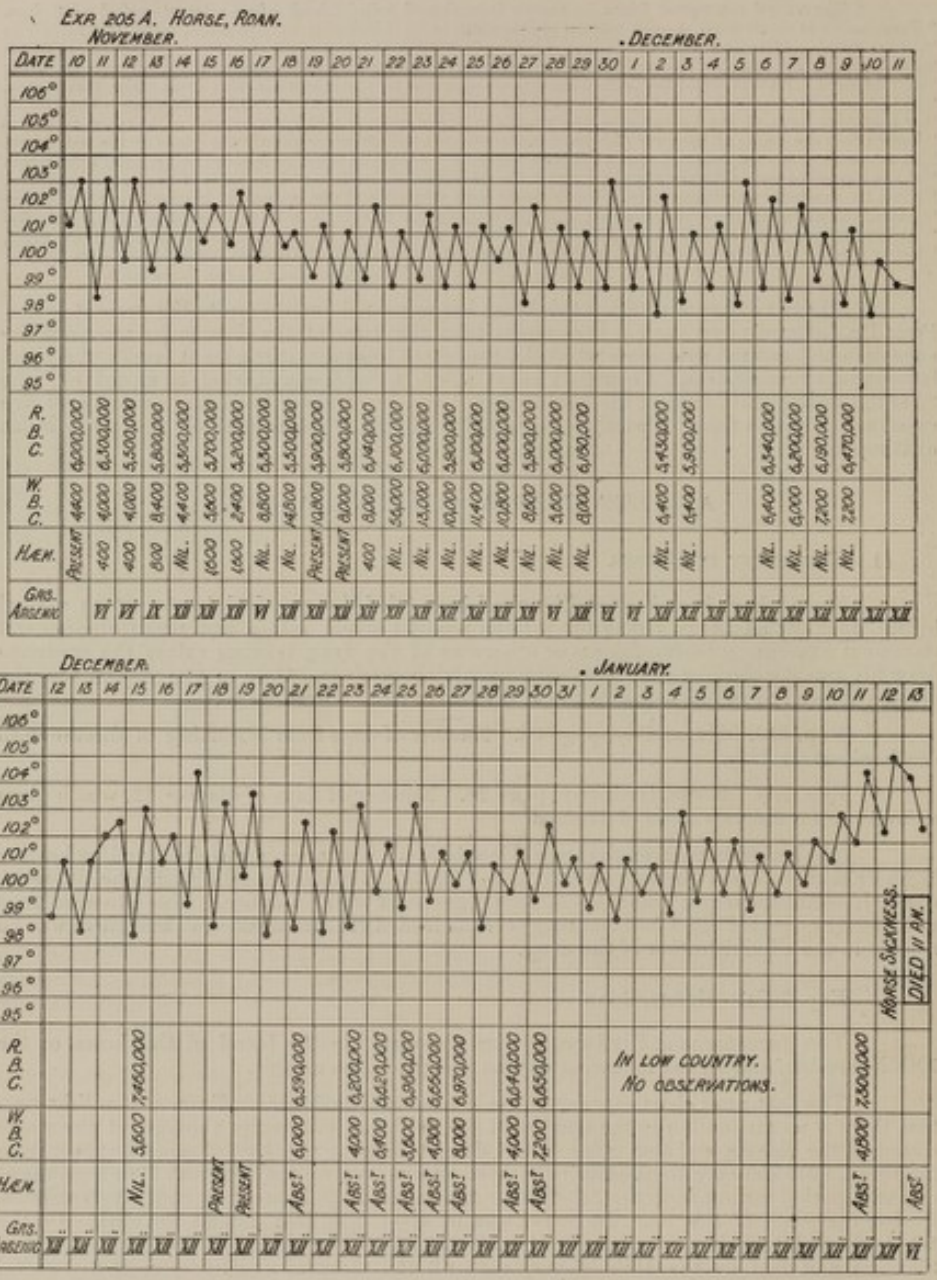
Exp. 205. Horse, Galloway, page 28. Number of days of disease 42, which gives an average for the two cases of 32 days.

The horses were given the arsenic in solution. The arsenic was dissolved by heating in water with an equal quantity of carbonate of soda. I made the solution in the following strength:—Arsenic 6 grs., carbonate of soda 6 grs., and water 1 ounce. The ounce of fluid was merely sprinkled night and morning over his feed of crushed mealies, so that there was no trouble in the administration of the drug.

Exp. 205A. Horse, Roan.

The Hæmatozoa of Fly Disease were discovered in the blood of this horse on the 10th November, and on the following day he was placed on arsenic.

The following chart represents the course of the disease. From it it will be seen that there is no diminution in the number of the red blood corpuscles throughout the whole course of the disease, and that after a few days' treatment the hæmatozoa disappear from the blood, only showing themselves in very small numbers on the 18th and 19th December; during a period lasting from the 22nd November to the 13th January, or nearly two months. During the whole of this time the horse was in good condition and fit for work. On the 1st January I took him with me to the Fly Country where he unfortunately took Horse Sickness, and died on the 13th January with all the classical symptoms of that dreadful scourge. I commend this and the following chart to the attention of those who recommend arsenic as a prophylactic in Horse Sickness. The large number of red blood corpuscles present two days before death I imagine to be due either to the action of the arsenic, or to the great loss of fluid from the blood which obtains in this disease.

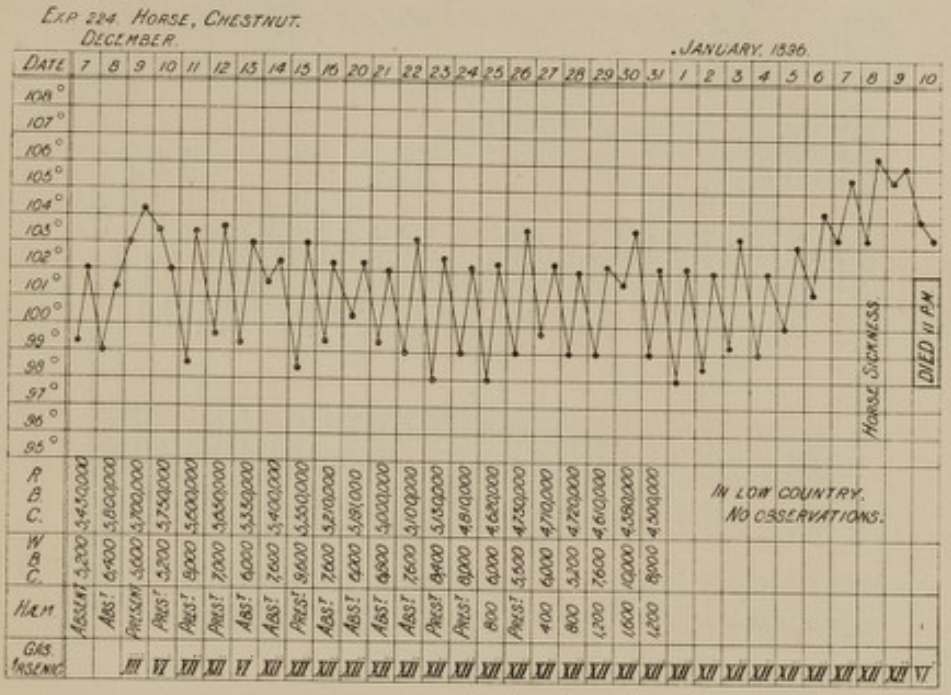


Remarks.—This horse remained alive sixty-five days from the commencement of the disease, and then only succumbed to Horse Sickness. While under arsenic he very fairly retained his strength, as may be proved by the fact that he very often carried me (riding over 14 stone) down to the Fly Country and back when I went down to collect flies. This means being on the march from 5 a.m. until 5 p.m., with a climb in the afternoon of the Ubombo, which is some 2,000 feet above the plain.

Exp. 224. HORSE, CHESTNUT. "The Unicorn."

This is a companion case to the last (Exp. 205A). The disease was contracted spontaneously in the Fly Country, and the hæmatozoa discovered in the blood on the 9th December. The horse was at once treated by arsenic in the same way and by the same dose as the last case. He was also taken by me to the Fly Country on the 1st January where he also contracted Horse Sickness, and died on the 10th of the same month.

The following chart shows the course of the disease :—



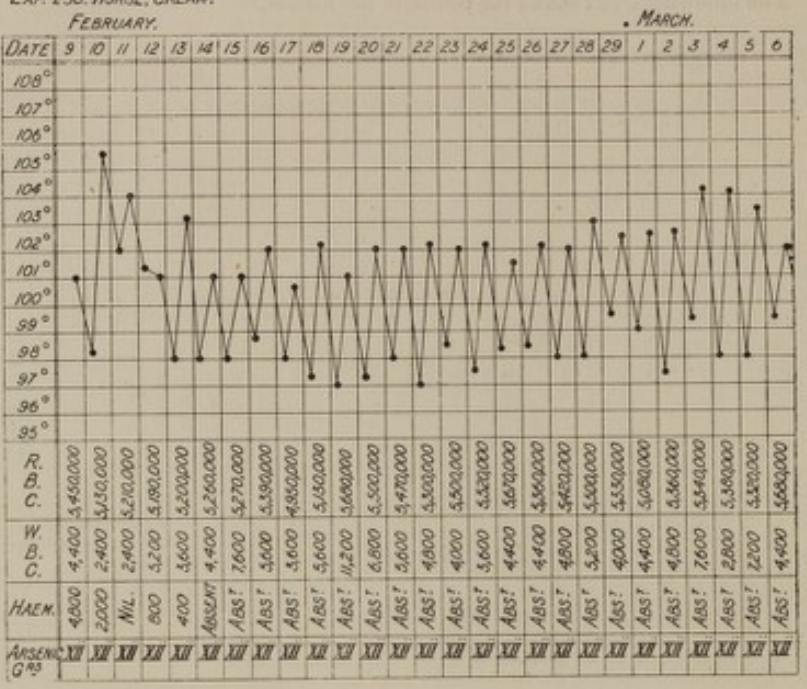
Remarks.—It is evident that the arsenic had much less effect than in the previous case (Exp. 205A). The hæmatozoa are seldom quite absent from the blood, and the red blood corpuscles show a tendency to fall slowly in numbers. On the other hand the horse remained in fairly good condition and was capable of doing a moderate day's work. The rise of temperature at the end is due to Horse Sickness.

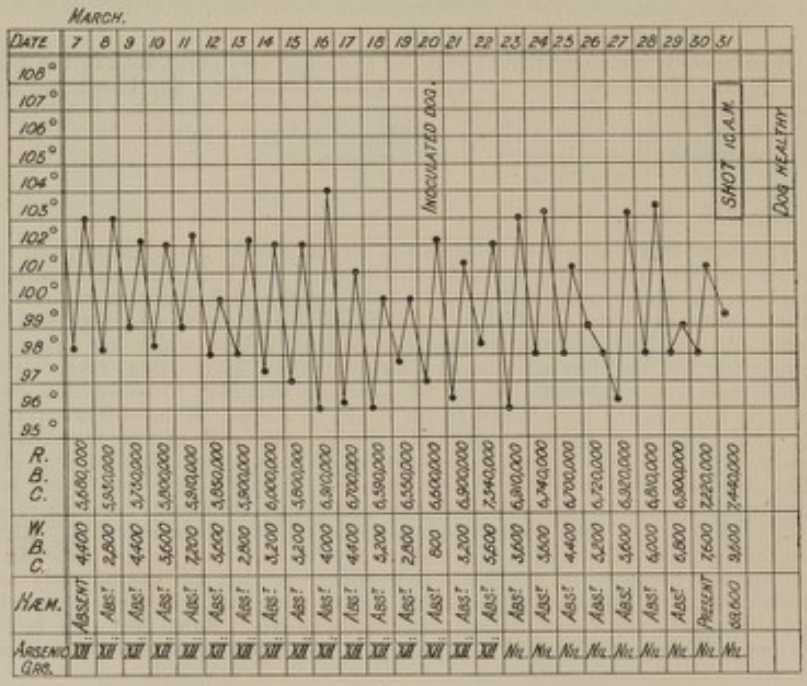
Exp. 256. Horse, Cream.

This horse was ridden from Nongoma to Ulundi on the 23rd January, and back to Nongoma on the 26th, off-saddling at the Black Umpalozi drift both going and coming. He was brought across the Segane Valley to Ubombo on the 29th, but was not off-saddled during the journey, nor were any flies seen. Before this journey he was in perfect health. He probably got the disease while crossing the Segane Valley, which has become very unhealthy during the last few months, and numerous flies have been seen, especially at the Mkusi drift. On February 9th his off hind leg was seen to be swollen, and the hæmatozoa of Fly Disease were found in his blood. February 10th.—Off hind leg still markedly swollen, also a slight swelling of the fetlock joint of the near hind leg. Besides a running from the eyes and nose there are no other symptoms. February 25th.—The swelling of the hind leg has disappeared for some days, but there is now a copious eruption of small pustules over the same leg. The horse has lost condition very much and is quite unfit to be ridden. March 30th.—This animal is extremely emaciated, being mere skin and bone, and to-day he is found to be suffering from paresis or partial paralysis of the hind extremities, so that he can scarcely balance himself on his feet. He eats his mealies readily and tries to graze. In the afternoon he fell down, and although he made many efforts was unable to rise. March 31st.—As he is still unable to get on his legs, and as his condition gave no hope of amendment, he was shot at 10 a.m.

The following chart shows the course of the disease, the number or blood corpuscles and hæmatozoa, and the dose of arsenic given daily.

EXP. 256. HORSE. CREAM.





Autopsy.—Immediately after death.

The body is extremely emaciated, there are no external swellings visible, and both corneæ are clear. There is a herpetic eruption of skin over the nose.

On making an incision along the ventral aspect of the body and reflecting the skin, the subcutaneous tissue is found to be free from infiltration. On opening the abdominal cavity a few ounces of fluid are found, the coils of intestine appear pale and atrophied, the spleen and liver are small, and the stomach almost empty of food.

On sawing through the sternum and opening the thoracic cavity a small quantity of fluid is found in each pleural cavity, the lungs are normally collapsed and appear healthy. The pericardial sac contains one ounce of clear straw coloured serum. The surface of the heart is smooth and free from petechiæ, but along the auriculo-ventricular groove and following the course of the coronary vessels is seen a huge deposit of a yellow jelly-like material, in some places quite one inch in thickness and three inches in width. This coagulated serum is also seen in large masses about the beginning of the aorta and pulmonary vessels. On opening the ventricles the lining membrane is seen to be pale and yellowish in colour, there are no petechiæ, and the muscular substance of the wall of the heart is firm in texture, pale and glistening in colour. Weight of heart 5 lbs.

The lungs contain much blood, are somewhat congested, otherwise present nothing abnormal.

The liver is dark in colour, and is dotted over with minute pearly white tubercles, about the size of a pin's head, which are slightly raised above the surface. These tubercles are extremely hard and are probably calcified concretions resulting from irritation set up by some minute parasite. On cutting into the substance of the liver the tissue is olive-green in colour, and the lobules are seen to be in a state of chronic congestion and fatty degeneration. The liver weighs 6 lbs. 11 oz.

The spleen weighs 2½ lbs. On section the tissue is firm and leathery, and the fibrous tissue of the organ appears to be increased.

The kidneys weigh 1 lb. 2 oz. and 1 lb. 3 oz. respectively. The capsule strips off readily, disclosing dilated capillaries. On section the cortex is seen dotted over with dark red enlarged Malpighian bodies, standing out on the pale cortical background.

In the wall of the stomach are seen three larvæ of the bot fly and a tumour as large as a walnut, containing nematode parasites; otherwise the lining membrane is healthy.

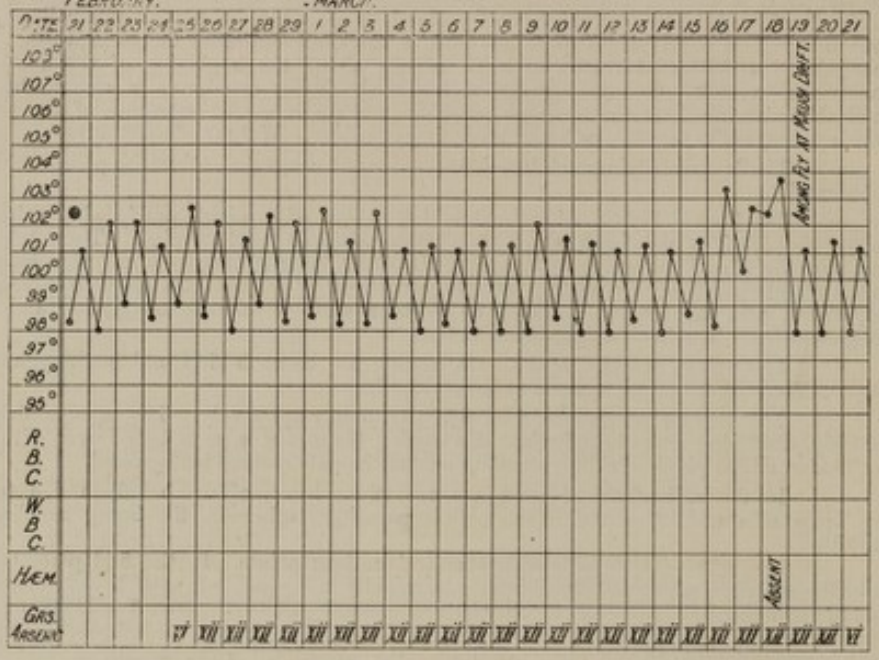
Remarks.—This case presents several curious points. In the first place the red blood corpuscles, which usually disappear so rapidly in Nagana, are seen here to increase in numbers during the course of the disease from about 5½ millions to over 7 millions per cubic millimetre. This must be due to the action of the arsenic. In the second place, in spite of the conservation of the red blood corpuscles, the animal fell away steadily in condition, at no time during his illness was it fit for work, and at the end presented an appearance of extreme emaciation, and this in spite of the fact that the arsenic had caused the hæmatozoa to disappear from the blood after five days' administration, and that they remained absent until eight days after the cessation of the drug. The arsenic was stopped on the 23rd March, as it was thought it might be the cause of the steadily increasing debility. Another curious point is that a dog inoculated with a large quantity (5 c.cm.) of blood from this horse on the 20th March, up to the present (May 29th) shows no signs of the disease, and yet nine days after stopping the arsenic the hæmatozoa are again found in the blood of the horse in very large numbers. This must mean either that the hæmatozoa were in such small numbers that even 5 cubic centimetres of blood could be drawn off without containing a single hæmatozoon, or that by the action of the arsenic they had been driven out of the blood and only existed in some internal organ. It is thus seen that the inoculation of a susceptible animal with a large quantity of blood from an affected animal not giving rise to the disease is no proof that the latter has recovered completely from the disease.

Exp. 264. Horse, Bay.—"M.D."

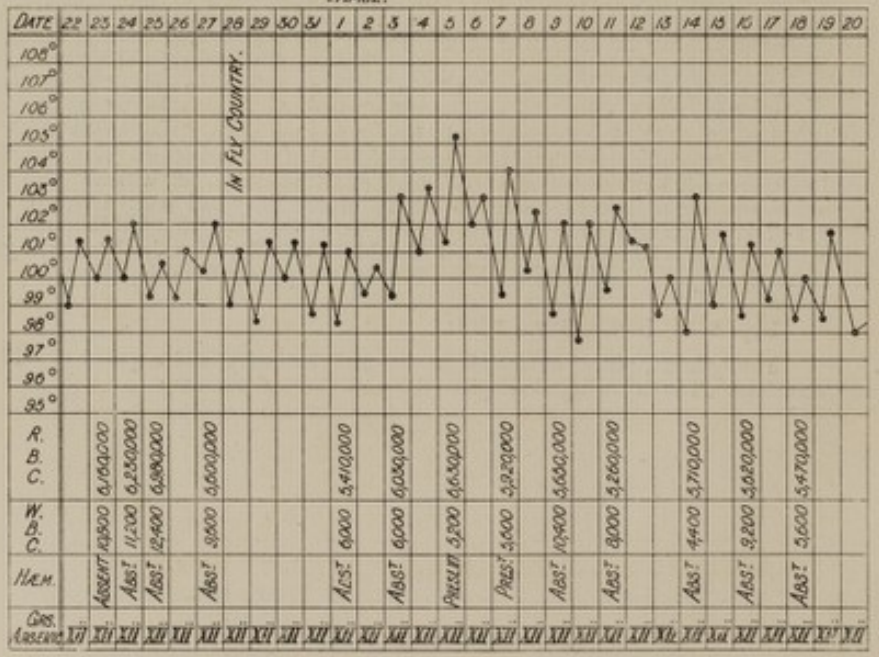
Young horse, placed on arsenic on the 25th February, and exposed to "Fly" on two occasions. The following chart gives the main facts of the case:—

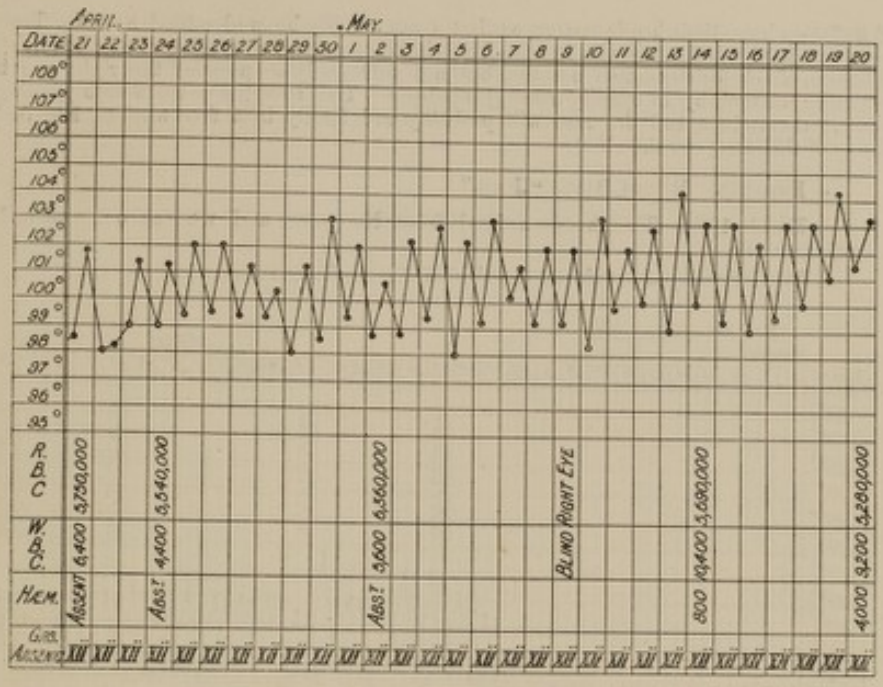
Exh. 204. Horse, Bay. "M.D"

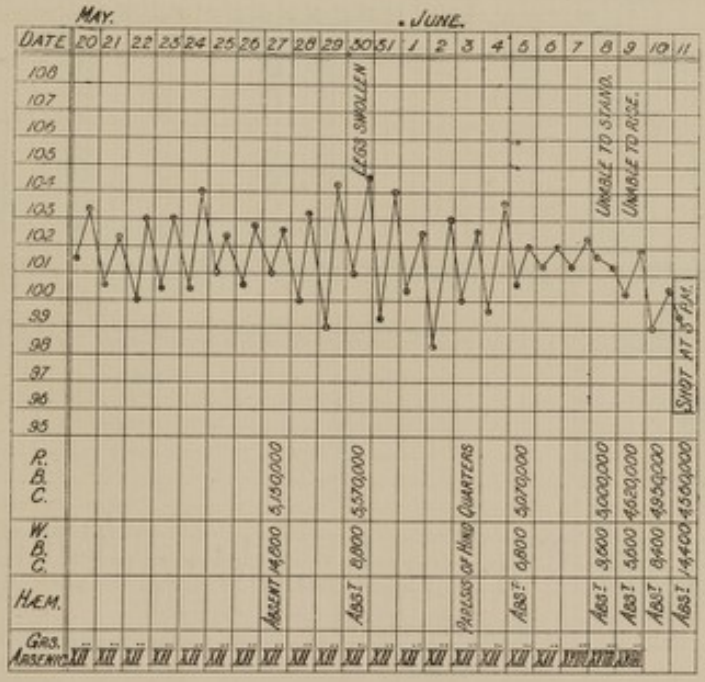
FEBRUARY. MARCH.



APRIL.







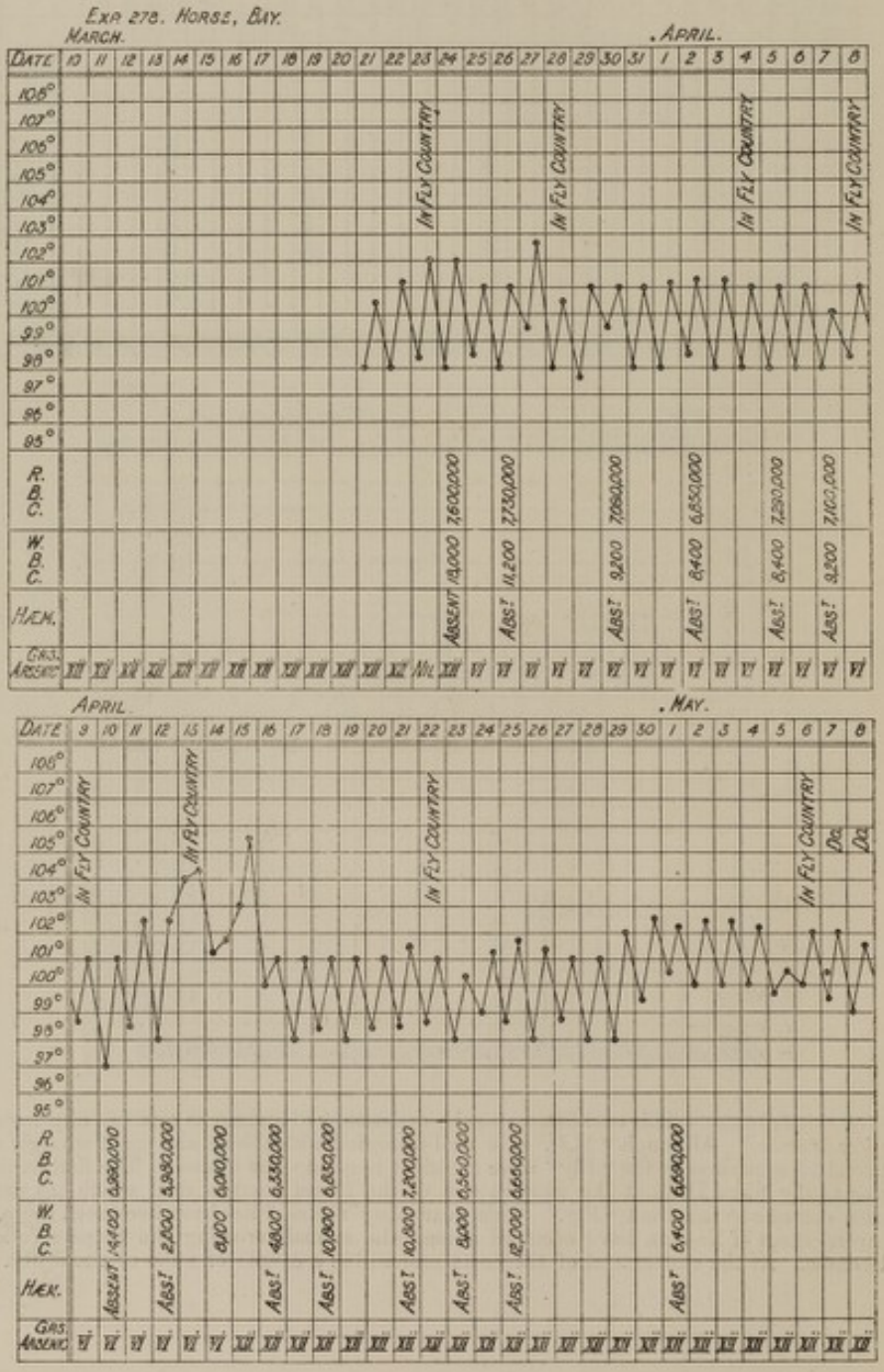
Remarks.—This case illustrates the uselessness of arsenic as a means of preventing "Fly Disease." Although "M.D." was well saturated with arsenic and smelt strongly of it, two short exposures to the "Fly" were sufficient to set up the disease. The first exposure was at the Mkusi Drift, at the western end of the Mkusi poort, a drift which has usually been considered free from "Fly." This year many Tsetse Flies have been seen there, and several cases of Nagana have occurred among the animals crossing the Segane Valley to Ubombo. At the eastern end of the poort and within a couple of miles of the drift a herd

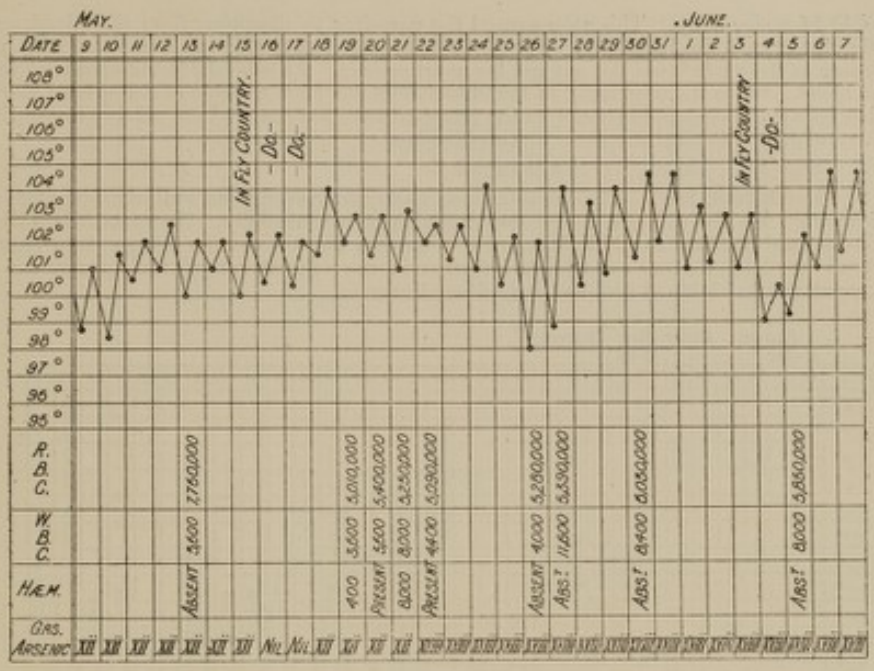
of buffaloes have their headquarters, so that it can readily be understood how the flies find their way to the drift. As will be seen from the temperature chart, the arsenic has held the disease in check for some two months, but not sufficiently to prevent the right eye from becoming affected by an opacity causing blindness. The hæmatozoa are now beginning to reassert themselves, and the case will probably end fatally in a few weeks. For further course of this case, see the chart.

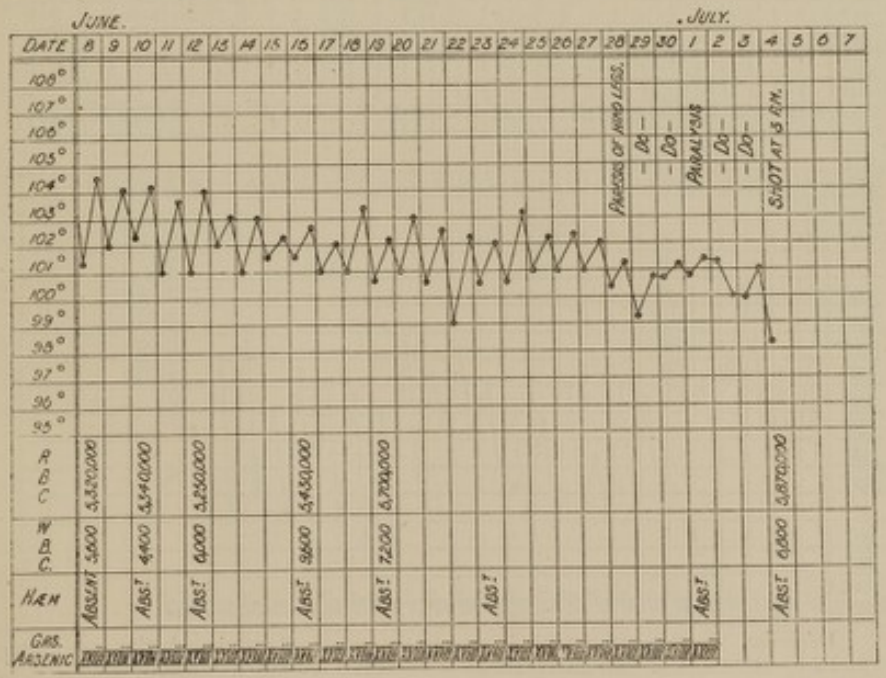
Exp. 278. Horse, Bay. "Jevu."

March 10th. This horse arrived from Nongoma and was at once placed on 12 grains arsenic.

The following chart shows the chief features of the case.







Autopsy.—Vertebral canal contains much coagulated lymph (yellow jelly-like substance), which probably had caused the paresis of the hind extremities. No intestinal worms were found in the stomach or small intestines; there are a few oxyures in the large gut.

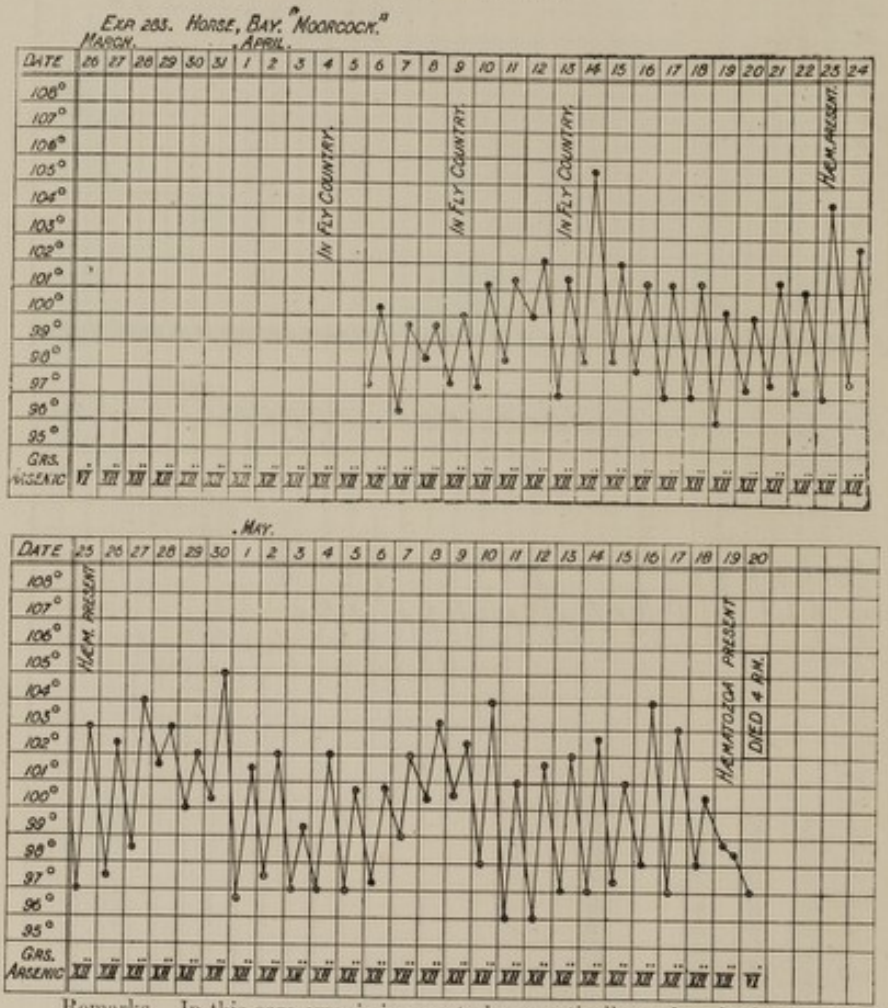
The absence of hæmatozoa was probably due to the large doses of arsenic.

Remarks.—Another case showing the futility of arsenic as a prophylactic agent. At first 12 grains daily were given, but as this caused the horse to fall off in condition the dose was reduced to 6 grains; when the hæmatozoa appeared in the blood the dose was again

raised to 12 grains daily. This soon caused the parasites to disappear from the blood, and remain absent until the 19th May. The three days previous to this the horse was with me at a camp in the "Fly" and did not get his usual arsenic. At once the hæmatozoa reappear in the blood. Since the beginning of his illness Jevu has been capable of doing hard work, and has on several occasions (noted on the temperature chart) carried me all day in the Fly Country without showing any signs of knocking up.

Exp. 283. Horse, Bay. "Moorcock."

March 26th, 1896.—This horse arrived to-day from Nongoma well nourished and in good health. He was at once placed on 12 grains of arsenic daily. He nevertheless took the disease and died on the 20th May. The following chart gives the temperature curve, the dates on which he was exposed to infection in the "Fly Country," the appearance of the hæmatozoa in his blood, and the daily dose of arsenic.



Remarks. In this case arsenic is seen to be practically useless both as a prophylactic and a curative agent. No observations were taken on the number of hæmatozoa in his blood. The temperature curve is irregular and shows a markedly remittent type. No noteworthy symptoms occurred in this case. Towards the end the head and extremities became swollen but not to any very marked extent. The eyes remained healthy to the last. No *post-mortem* examination was made.

(2.) Treatment in Donkeys.

Four donkeys were left untreated to act as controls, and they show only an average duration of the disease of nine days. It must be remembered, however, that these animals were brought to me suffering from the disease, and may have been ill for some days before being placed under observation. But this cannot have been more than a week at the outside, as the donkeys, being constantly in work, were soon noticed if out of condition.

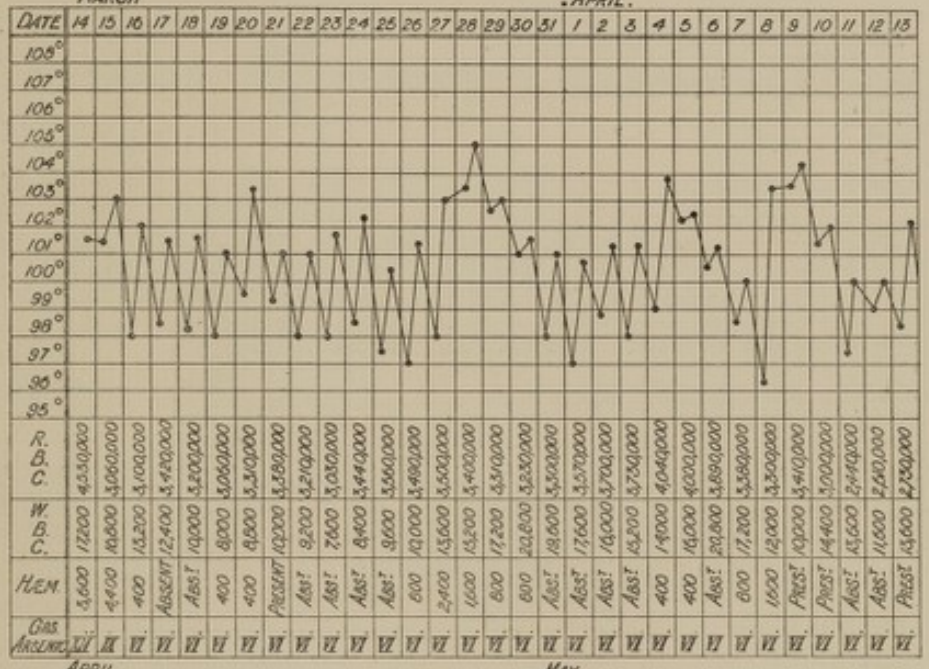
The donkeys were given arsenic in the dry condition, the mouth being opened by an assistant and the powder placed on the back of the tongue. There is no difficulty in administering it in this way.

Exp. 275. DONKEY.

March 14.—This donkey was brought to me to-day with running at the eyes and nose, staring coat, and slight swelling under the abdomen. He has been engaged of late in the cart conveying goods to Ubombo from Bagonoma Depôt at the other side of the Segane Valley. On microscopical examination his blood is seen to contain hæmatozoa.

The following chart represents the course of the disease:—

EXP 275. DONKEY

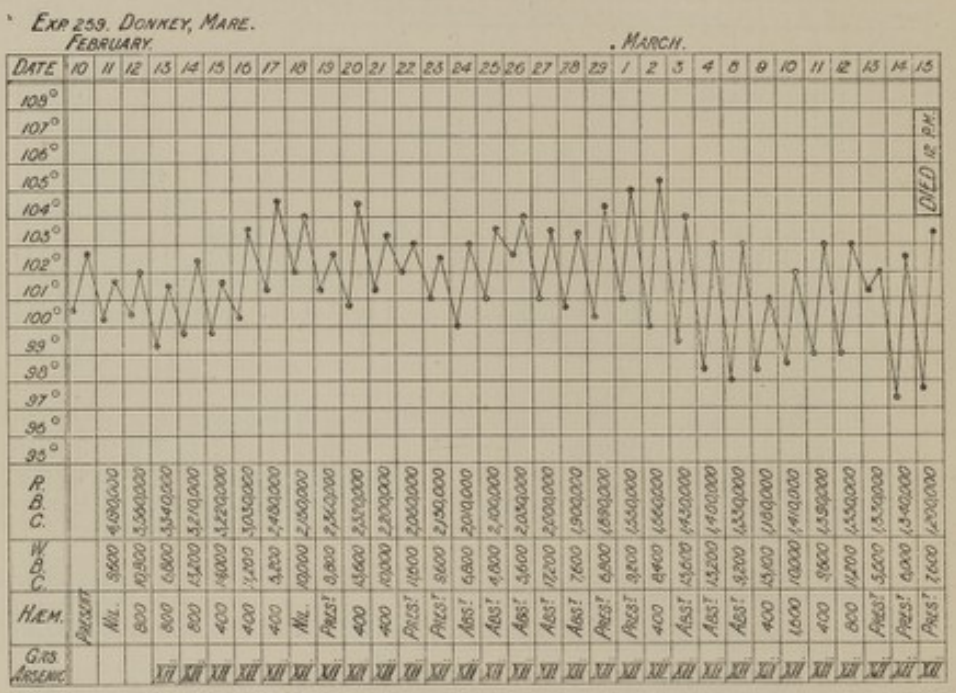


Remarks.—This donkey had been taking 5 grains arsenic daily as a prophylactic for some weeks previous to the onset of his illness. He was placed on 6 grains arsenic daily, and the effect this dose had on the course of the disease can be seen from the above chart. The hæmatozoa are seldom quite absent from the blood, and the diminution in the number of red blood corpuscles progresses steadily to the fatal termination.

Exp. 259.—Donkey, Mare.

February 10th.—This donkey was brought to me to-day with the usual symptoms of Nagana.

The following chart represents the course of the disease:—



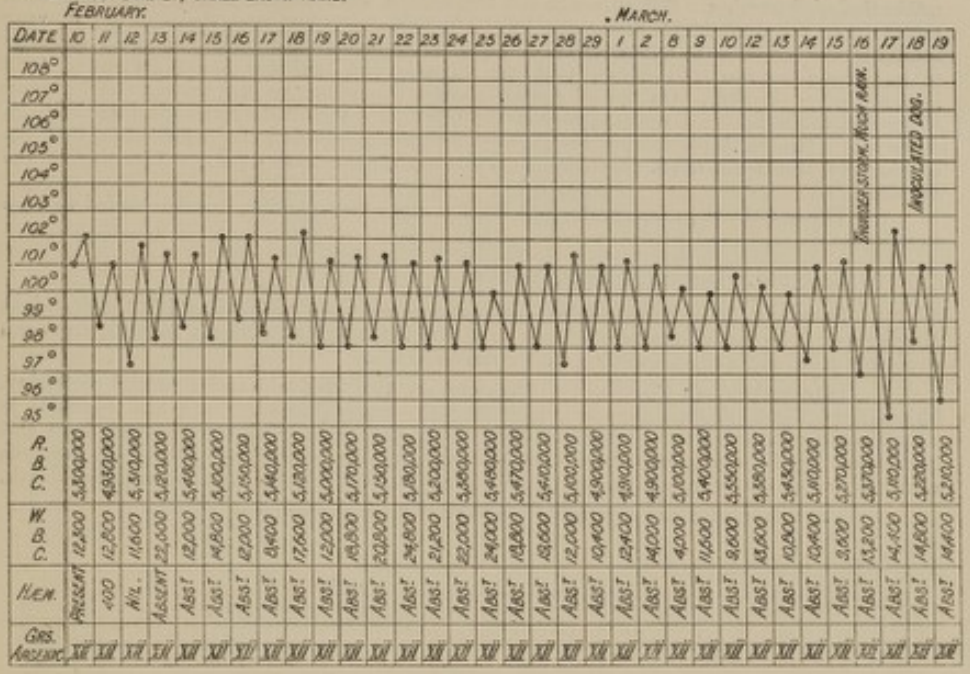
Remarks.—In spite of the persistent exhibition of 12 grains arsenic daily, the red blood corpuscles are seen to diminish steadily until they reach the very small number of 1,200,000 per cubic millimetre, and this in spite of the fact that the number of the hæmatozoa are held strictly in check by the drug. The *post mortem* appearances were very similar to those recorded in Exp. 271, except that the stomach was filled with grass.

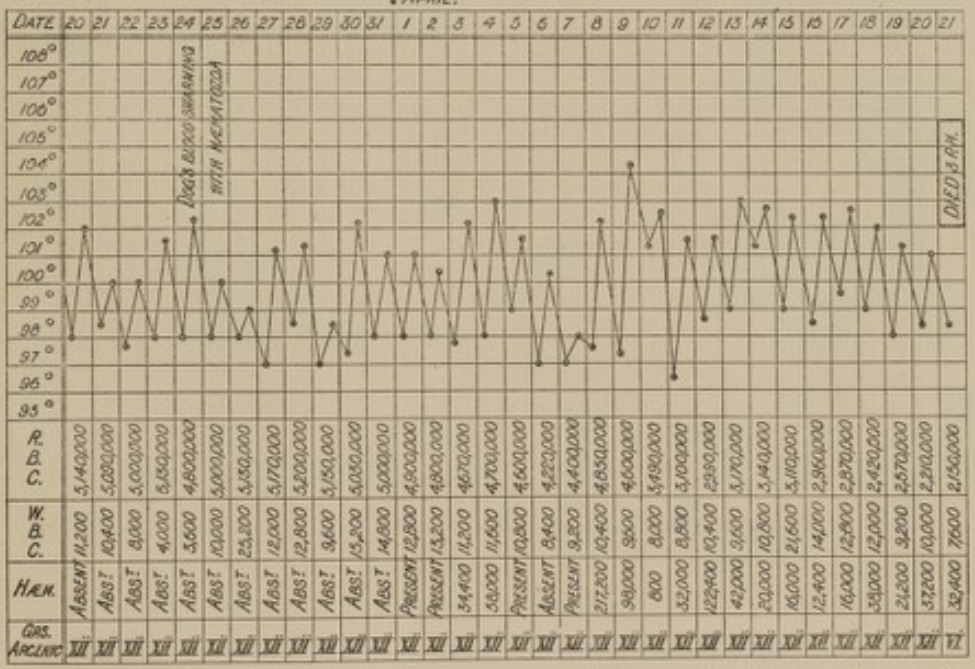
Exp. 258. Donkey. Small Brown Mare.

February 10th.—This donkey was brought to me presenting the usual symptoms of Nagana.

The following chart shows the course of the disease, the number of blood corpuscles and hæmatozoa, and the daily doses of arsenic:—

EXP 258. DONKEY, SMALL BROWN MARE.





Remarks.—This is as favourable a case for a trial of arsenic in this disease as could be desired. The donkey is placed under treatment at the very beginning, when the hæmatozoa are in very small numbers in the blood, and the red blood corpuscles have not begun to diminish. Except for the usual watery discharge from the eyes and nose, and slight swellings under the abdomen and chest, which appeared on the 18th and 20th February, the animal presented no noteworthy symptoms throughout the course of the disease. As will be seen from the chart, the hæmatozoa remain absent from the blood until the 31st March, a period of forty-nine days, and during this time the red blood corpuscles

retain their full number. But although the hæmatozoa were absent during this time from the blood as far as could be made out by the microscope, they were there either in very small numbers or in some unrecognised form, since a dog inoculated with a small quantity of her blood on the 18th March took the disease and had his blood swarming with hæmatozoa on the 24th. On the 1st April, although the donkey is still taking the full dose of arsenic, the hæmatozoa again appear in the blood, and the donkey at once begins to go down hill. The red blood corpuscles rapidly decrease in number, the hæmatozoa increase enormously, and death takes place from exhaustion on the 21st April, or 71st day of the disease.

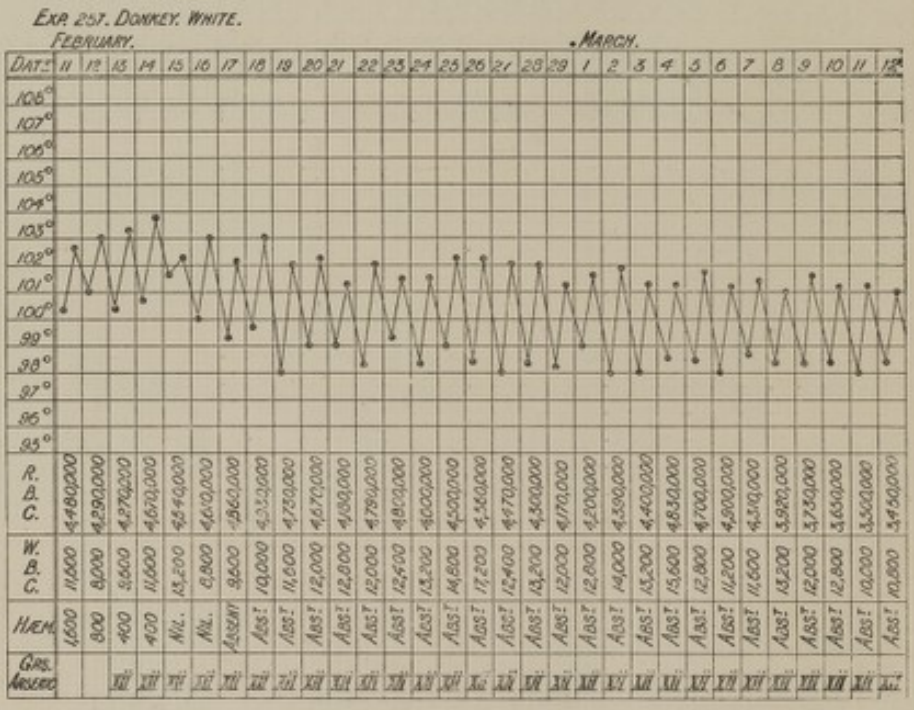
The arsenic then has prolonged life for some two months, but has failed to cure the disease.

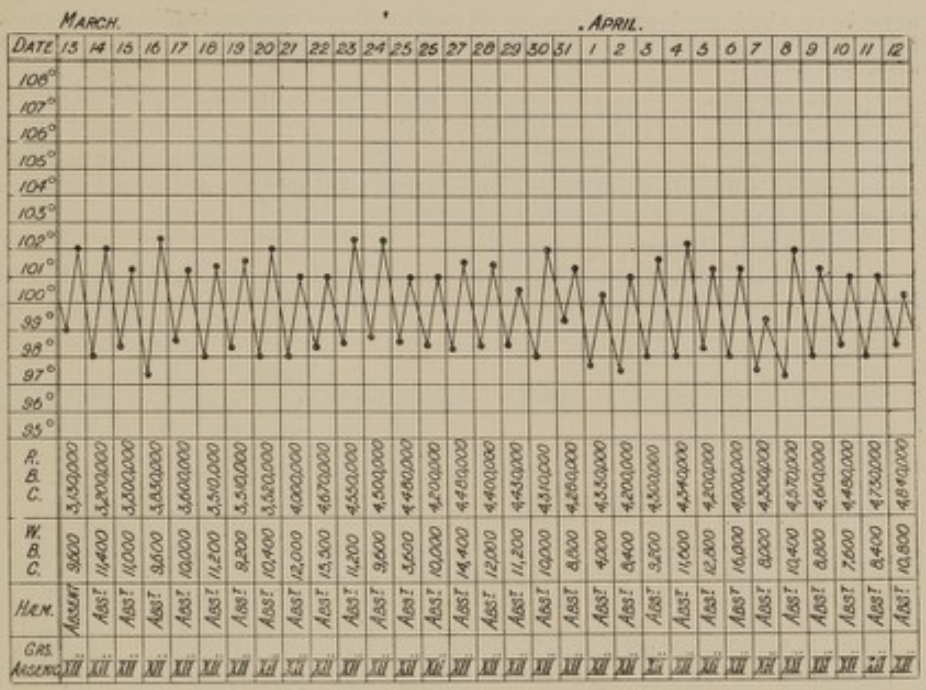
Exp. 257. Donkey. Mare, White.

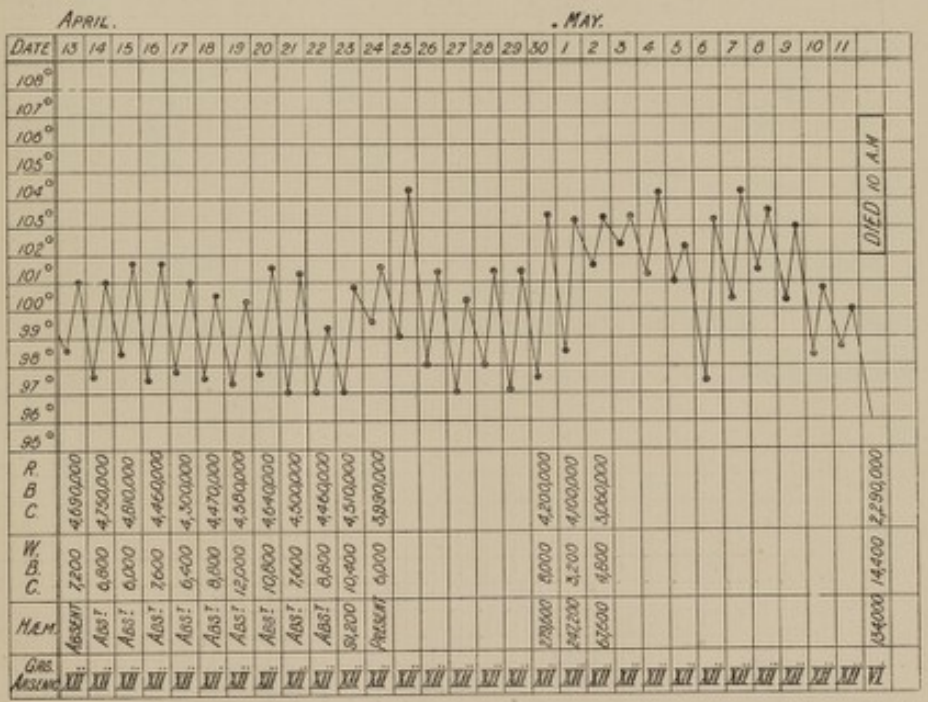
February 10th, 1896.—This donkey was sent to me to-day by the Resident Magistrate. The hæmatozoa are present in the blood, there is a slight running from the eyes and nose, and a small swelling under the abdomen.

This donkey left Ubombo for Nongoma on the 28th ult. and returned yesterday. She had not been off the hill previously since the 23rd December and has therefore in all probability incurred the disease while crossing the Segane Valley between here and Nongoma. On February 26th there was some swelling of the head and neck. On the 29th all swelling had disappeared, and she presented no other marked symptoms until her death.

The following chart represents the course of the disease:—







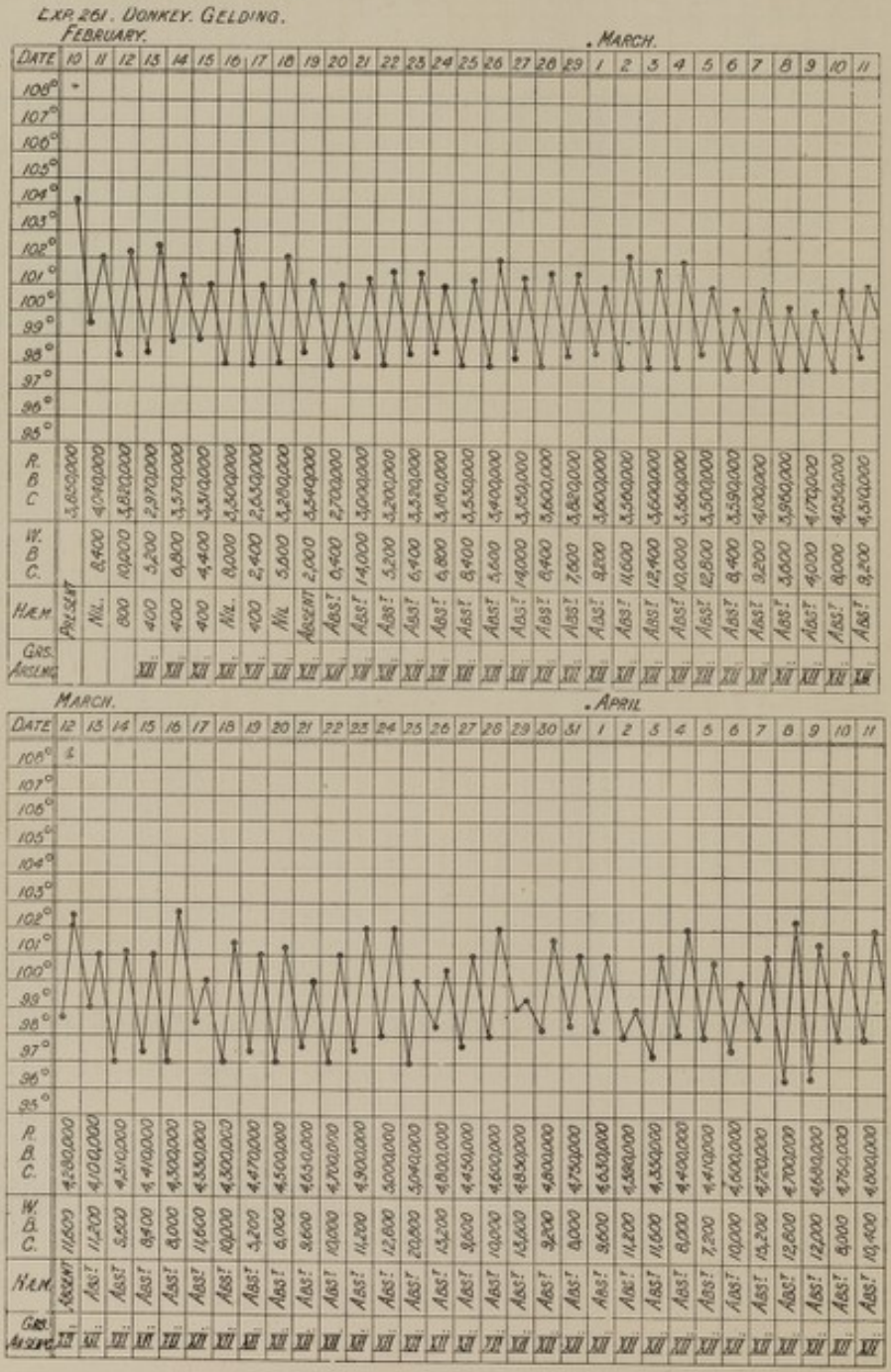
Remarks.—This case shows in a marked manner the effect of arsenic in prolonging life in this disease. The hæmatozoa, which are present for the first four days, disappear altogether from the blood for sixty-eight days, when they suddenly reappear in large numbers. From the seventy-third day of the disease until death on the ninety-second day the parasites present are in enormous numbers and the progress of the disease is rapid. Up to the 22nd April the arsenic holds in check the destruction of the red blood corpuscles which after this date and corresponding to the reappearance of the hæmatozoa in the blood, rapidly disappear. The white blood corpuscles are little affected by the disease; they are

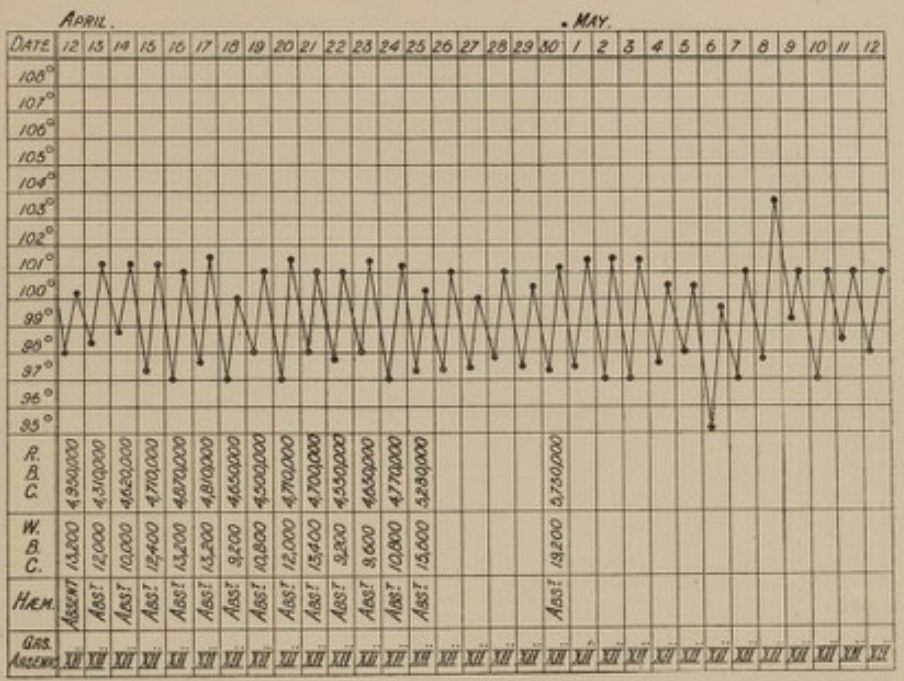
never in very high numbers, and are not affected by the presence or absence of the parasites in the blood. Up to the 22nd April this donkey was quite capable of doing a moderate amount of work.

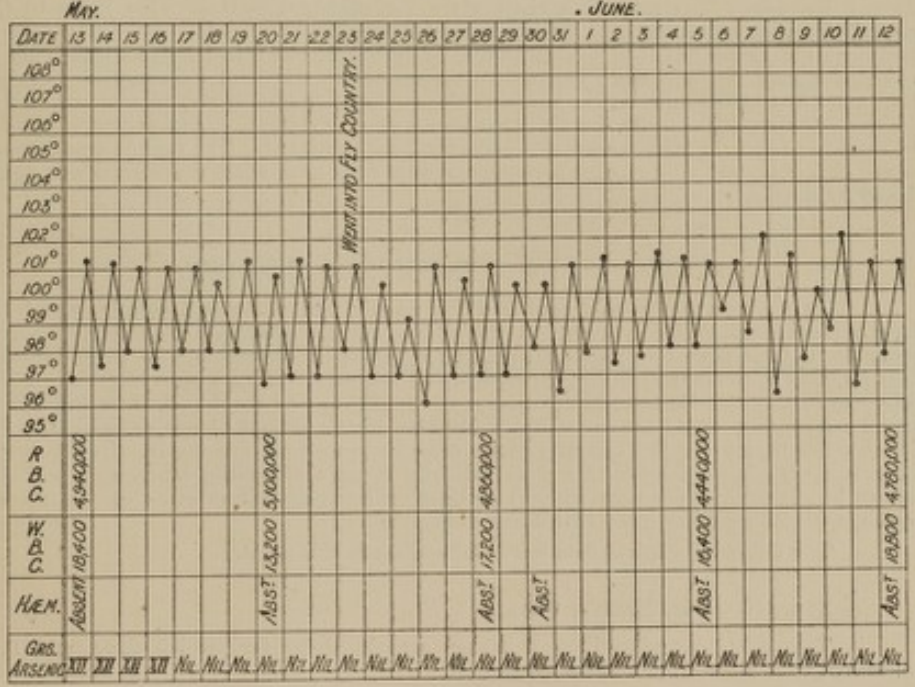
It may be noted that on March 19th, the hæmatozoa having been absent from the blood thirty-two days, a dog (Exp. 279) was inoculated with 1 c.cm. of this donkey's blood and remained unaffected by the disease up to the 21st April, when the experiment was stopped. In what part of the organism these parasites exist during their absence from the blood is a curious point. Of course the explanation may be, and probably is, that they are circulating in the blood stream but in excessively small numbers.

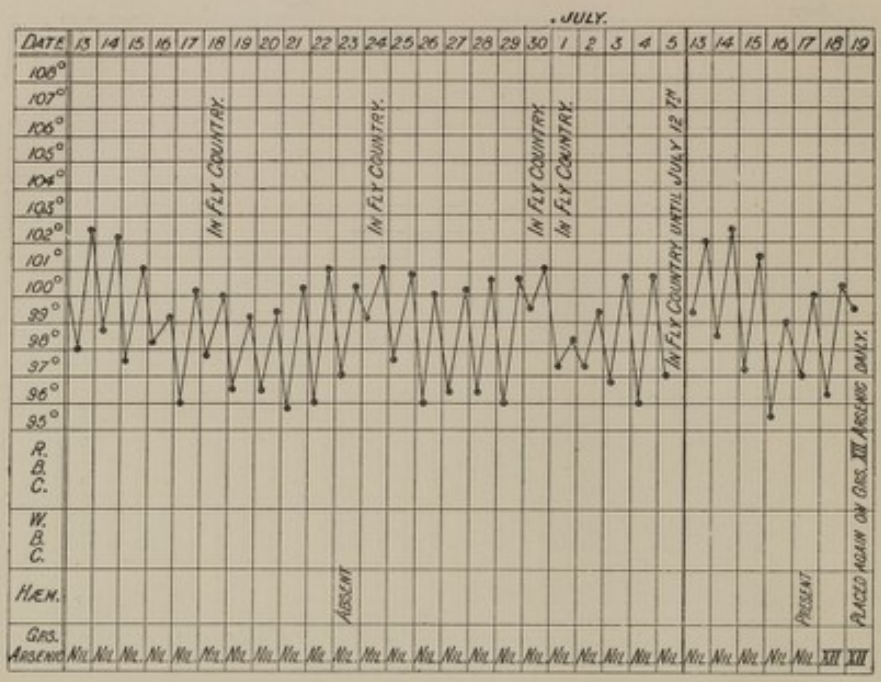
Exp. 261. Donkey, Gelding.

February 10th, 1896.—This donkey was sent to me to-day by the Resident Magistrate, Ubombo, presenting the usual symptoms of Nagana. The following chart shows the course of the disease.




APRIL.
MAY.
DATE
12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 1 2 3 4 5 6 7 8 9 10 11 12
108°
107°
106°
105°
104°
103°
102°
101°
100°
99°
98°
97°
96°
95°
R. B. C.
4,930,000 4,510,000 4,620,000 4,710,000 4,870,000 4,810,000 4,650,000 4,500,000 4,710,000 4,700,000 4,550,000 4,650,000 4,770,000 5,280,000 5,750,000
W. B. C.
13,200 12,000 10,000 12,400 13,200 13,200 9,200 10,800 12,000 13,400 9,300 8,600 10,800 15,600 13,200
HEM.
ABSENT ABS.T ABS.T ABS.T ABS.T ABS.T ABS.T ABS.T ABS.T ABS.T ABS.T ABS.T ABS.T ABS.T ABS.T
GRS. ARSENIC
XII XII XII XII XII XII XII XII XII XII XII XII XII XII XII


MAY.
JUNE.
DATE
13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31 1 2 3 4 5 6 7 8 9 10 11 12
WENT INTO FLY COUNTRY.
108°
107°
106°
105°
104°
103°
102°
101°
100°
99°
98°
97°
96°
95°
R. B. C.
4,940,000 5,100,000 4,800,000 4,440,000 4,760,000
W. B. C.
18,400 13,200 17,200 15,400 18,800
HEM.
ABSENT ABS.T ABS.T ABS.T ABS.T
GRS. ARSENIC
XII XII XII XII Nil Nil Nil Nil Nil Nil Nil Nil Nil Nil Nil Nil Nil Nil Nil Nil Nil Nil Nil Nil Nil Nil Nil Nil Nil Nil



Remarks.—The preceding chart gives the principal facts of what is up to the present the most successful case I have had of treatment by arsenic. After five days of treatment the hæmatozoa are seen to disappear from the blood and not to return for a period of almost five months. The red blood corpuscles which in February lie between three and four millions per cubic millimetre, are seen to gradually rise in numbers to five millions. Corresponding with this there has been a gradual increase in fitness, and at present the donkey has all the appearance of a perfectly healthy animal. The arsenic was stopped on the 17th May, but the hæmatozoa re-appeared on July 10th, when the animal was again placed on arsenic.

B.—ARSENIC AS A PROPHYLACTIC AGENT.

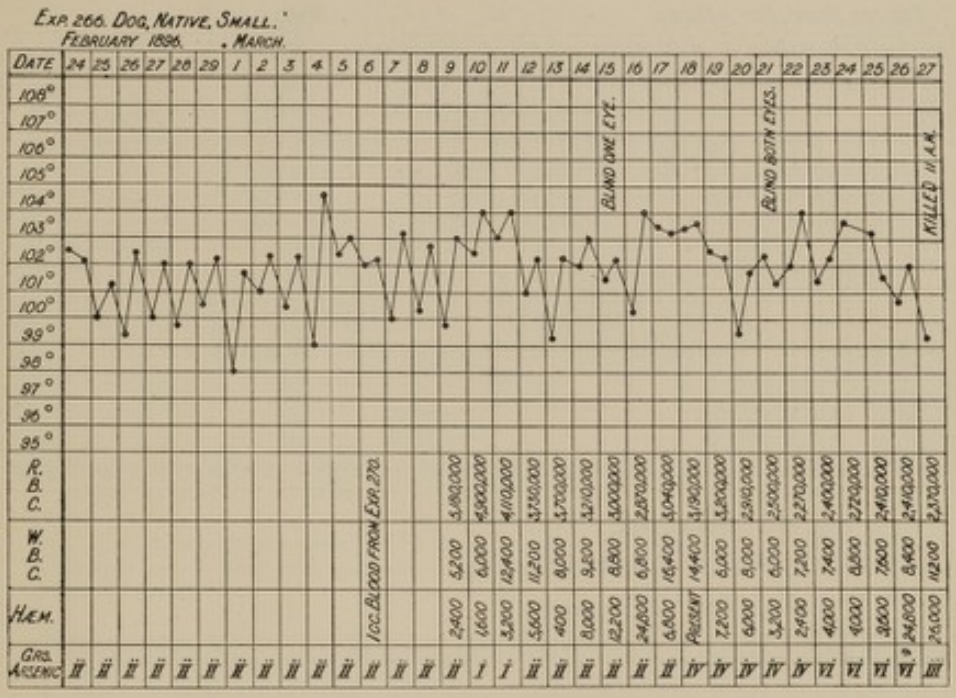
As has been seen in the cases in which arsenic was given as a curative agent, the drug undoubtedly markedly modified the course of the disease. It therefore seemed probable that the disease would be prevented altogether if the arsenic were given for some time previous to the animal being exposed to the infection. If 12 grains of arsenic given daily can cause the hæmatozoa to disappear rapidly out of the blood, it is difficult to imagine how they could appear in the first place if the animal before entering the Fly Country had been given this quantity of the drug for some time previously. But 12 grains daily is a very large dose of this powerful drug to give a healthy horse, and probably cannot be given in every case without setting up inflammation of some of the internal organs, with falling away in condition and general debility.

The following experiments were made to settle this point.

Exp. 266. Dog, Native, Small.

This dog was placed on 2 grains arsenic daily for twelve days, and then inoculated with blood from an affected animal.

The arsenic was continued in increasing doses, with the result shown by the following chart.



Remarks.—In spite of the large doses of arsenic given to this small dog, the hæmatozoa appear in the blood three days after inoculation and continue in large numbers till his death. The red blood corpuscles are seen to dwindle away in number; the animal becomes emaciated and blind. From this case it would appear that arsenic has little control over Nagana in the dog.

Exp. 290. Donkey, Large Grey.

March 14th.—Placed this donkey on 5 grains arsenic daily. Sent to Fly Country on several occasions.

April 5th.—Suffering from Nagana, many hæmatozoa in blood.

April 16th.—Died of disease.

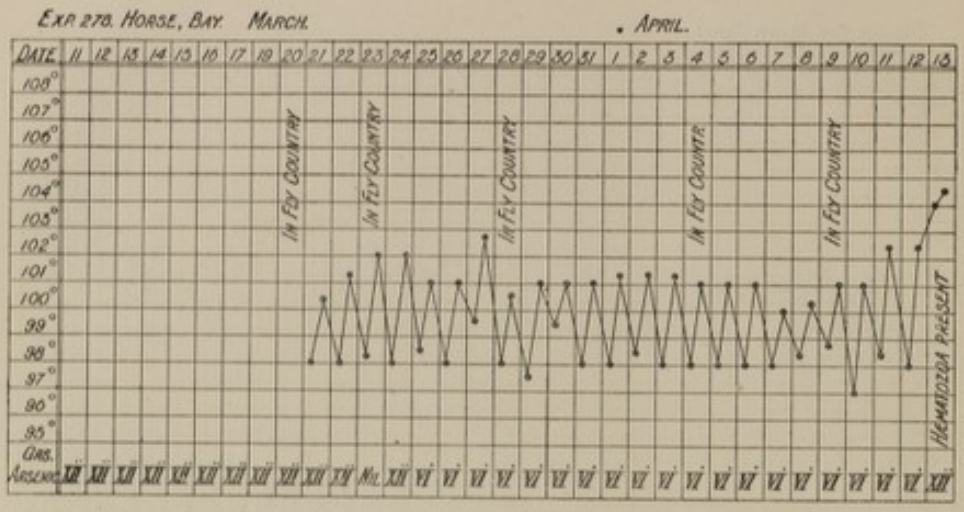
Exp. 295. Donkey, Grey.

March 14th.—Placed on 5 grains arsenic daily. Sent into Fly Country on several occasions.

April 18th.—Has contracted Nagana.

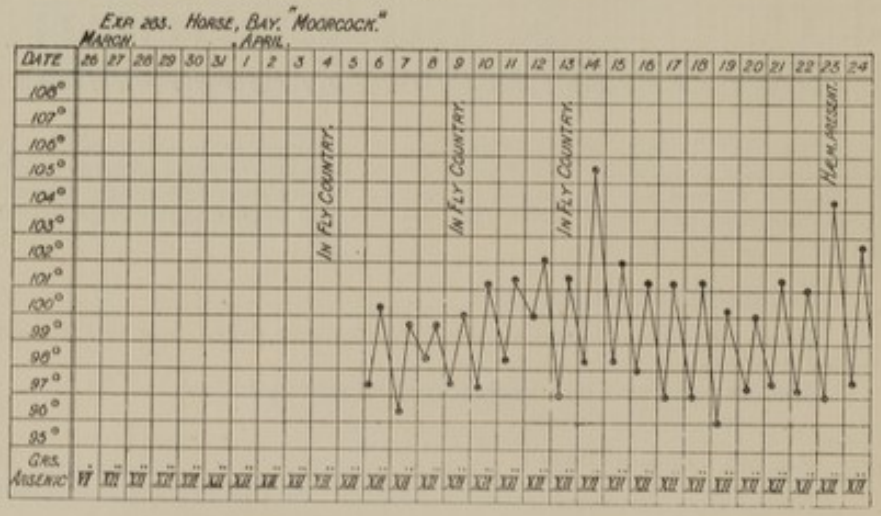
Exp. 278. Horse, Bay.

The following chart shows the result.



Exp. 283. HORSE, BAY.

The following chart shows the result:—



From these experiments it may be concluded that arsenic is quite useless as a prophylactic agent, but that it is useful in prolonging life and usefulness in the Fly Country after the disease has begun.

10.—CONCLUDING REMARKS.

The practical object to be aimed at in an investigation of an infective disease is of course how to prevent it. This can be approached in various ways, for example, by striking at the *fons et origo* of the disease, in other words by removing the cause, or by rendering animals immune by some process of vaccination or immunisation and leaving the cause alone, or lastly to discover some medicinal substance, be it serum or drug, which will cure the disease after it has been set up. In snake bites for example the snakes may be killed off, or the animals may be rendered immune to snake poison by the administration of gradually increasing doses until for instance with cobra venom rabbits become so far protected as to be able to receive in a single dose a quantity sufficient to kill fifty animals; or lastly the effects of the snake poison may be removed or neutralised, as Calmette and Fraser have shown, by the injection of serum taken from an immunised animal, that is to say, by the injection of a substance which will antagonise the action of the venom.

Before recommending any preventive measures it is necessary to know as fully as possible the etiology of the disease.

To return to Nagana, what do we know of the disease? We know that it is caused by the entrance into the blood of a minute parasite which multiplies there and causes death. That this parasite exists in the blood of many animals and that it is conveyed from animal to animal by the Tsetse Fly, or by the eating of the raw flesh of animals affected by the disease. We also know that the disease is limited to certain tracts of country having certain physical features, but that its distribution in these tracts is very variable.

We do not know how the parasite causes death, but surmise that it may do so by the poisonous action of some substance or substances elaborated or secreted by it, or by producing a progressive hæmolysis and anæmia, leading to a form of auto-intoxication. We do not know all the animals in which this parasite may exist, but judging from the number of domestic animals in which it is fatal, we may consider that it has a wide range. It may exist not only in the koodoo, wildebeeste and other big game, but it also may exist in the wild cats, rats, birds, and even fish of the Fly Country.

We do not know whether there are other species of fly which convey the disease besides the Tsetse, but this perhaps is a minor consideration. We do not know of any method at present of isolating the hypothetical poisonous substance elaborated by the hæmatozoon so as to be able to study it or to render animals immune to it by gradually increasing doses. No experiments have, as far as I am aware, been made in this country in the direction of studying the effect of the serum of animals in whose blood the hæmatozoa have been numerous. Dr. Lingard has, however, made a few with a negative result in Surra.

As treatment of disease by means of serum is very popular at present and I may be blamed for not having entered more into this aspect of the subject, I give Dr. Lingard's results *in extenso*.

"THE SUBCUTANEOUS INOCULATION OF BLOOD AND ITS DERIVATIVES IN ITS DIFFERENT "FORMS, OBTAINED FROM ANIMALS SUFFERING FROM, OR WHICH HAD LATELY "SUCCUMBED TO, SURRA.

"A series of experiments were conducted in order to test the efficacy or otherwise of the blood serum "as a protective agent against inoculated 'Surra,' or in other words to ascertain whether any substance capable "of conferring immunity against the disease was present in it when obtained from animals in whose circulation "the hæmatozoon of Surra had been swarming for some days previously. In the first series fresh blood "containing numerous hæmatozoa was drawn from the horse immediately after death into sterilised glass "vessels, and set aside for several hours in order to allow of the serum separating. The serum was then "transferred to one of M. Pasteur's filters, and forced through a porcelain cylinder. Three rabbits and two "guinea pigs were then subcutaneously injected with varying quantities during a period of three days. On the "evening of the third day each animal received subcutaneously 0·2 c.c. of blood containing numerous hæmatozoa "another animal in each case receiving a like quantity so as to act as control experiments.

"Rabbit A, which received 3·0 c.c. serum after inoculation with the soiled Surra blood, survived the "first appearance of the hæmatozoon twenty-four days.

"B received 6·0 c.c. serum; survived twenty-two days.

"C received 9·0 c.c. serum; survived forty days.

"The control animal survived forty-seven days.

"In the case of the guinea pigs—

"X received 6·0 c.c. serum, and survived the first appearance of the hæmatozoon after inoculation with "the soiled Surra blood thirty-four days.

"Y received 6·0 c.c. serum, and survived sixty-six days.

"The control animal survived the first appearance of the hæmatozoon in the blood eighty-four days."

"TABLE XIX.

Animals.	Date of Sub-cutaneous Injections.	Quantity of Horse Serum Injected.	Date of Inoculation with Blood containing the Hæmatozoa.	Quantity of Blood Inoculated.	Date of the first Appearance of the Hæmatozoa in Blood.	Date of Death.	Number of Days elapsing between Inoculation and Death.
		c.c.		c.c.	D. M. Y.	D. M. Y.	
Rabbit A	—	3·0	—	0·2	18.12.90	10.1.91	—
„ B	—	6·0	—	0·2	18.12.90	8.1.91	—
„ C	—	9·0	—	0·2	18.12.90	26.1.91	—
Control	—	—	—	0·2	—	—	—
Guinea-pig X	—	—	—	0·2	20.12.90	22.1.91	—
„ Y	—	—	—	0·2	20.12.90	23.2.91	—
„ Control ..	—	—	—	0·2	—	—	—

"In the second series blood was withdrawn from a horse immediately after death in which the hæmatozoa "of Surra had been swarming for some days, and immediately subjected to the method described by Ogata as the "one he used in isolating from the blood of dogs and fowls substances capable of conferring immunity against "anthrax and swine erysipelas. Three guinea pigs were subjected to daily injection of this fluid in varying "quantities: No. 1 received 0·5 c.c., No. 2 0·75 c.c., No. 3 1·0 c.c. daily for a period of eleven days. After "each animal had received eleven injections seven days were allowed to elapse, and on the eighth all were "inoculated with an equal quantity, viz., 0·2 c.c. of blood containing numerous hæmatozoa taken from a horse, "the subject of naturally contracted 'Surra.' At the same time a healthy animal was inoculated with a similar "quantity to act as a control experiment.

"The results were as follows:—

"No. 1 received 5·5 c.c. of the solution, and was inoculated with soiled blood on the 3rd October. The "hæmatozoon appeared on the 7th October, and death occurred on the 12th December, sixty-six days after the "discovery of the organism in the blood.

"No. 2 received 8·5 c.c. of the solution, and was inoculated with soiled blood on the 3rd October; the "hæmatozoon appeared on the 6th, and death occurred on the 25th October, nineteen days after the discovery "of the organism in the blood.

"No. 3 received 11·0 c.c. of the solution, was inoculated with soiled blood on the 3rd October; the "hæmatozoon appeared on the 7th, and death occurred on the 1st November, twenty-five days after the discovery "of the organism in the blood.

"The control animal was inoculated on the 3rd October; the hæmatozoon appeared on the 8th October, "and death occurred forty-two days after the organism appeared in the blood.

"Consequently it will be observed that the latent period in the three injected animals occupied four, "three, and four days respectively, whilst in the control animal it occupied five days. The animal which received "the smallest quantity of the above solution lived sixty-six days, and survived the other two by forty-seven and "forty-one days respectively, and the control by twenty-four days."

Up to the present then the results of experiments with serum have been negative, but I purpose if time permits to continue this line of investigation.

Another important point we are still ignorant of is whether animals can become affected by this disease without the agency of the Tsetse Fly or by eating raw flesh, as, for example, by drinking contaminated water or eating soiled herbage. This point I am engaged in investigating.

And, lastly, up to the present we do not know of any drug which will prevent the disease or cure it in every instance when incurred.

I have received a letter from Mr. P. Ogilvie, Pretoria, kindly offering to assist in the elucidation of the "Fly" question, and asking for suggestions as to lines of work. For his information, and for the information of any others who may also wish to help in this matter, I would make the following suggestions:—

1. That all trustworthy information regarding seasonal prevalence be collected.

Dr. Lingard writes me that in India the rains generally commence on June 9th (bursting of the S.W. monsoon); Surra appears, according to the date of rain, in September, and continues during October, November, December, and sometimes into January. He wishes to know when the rains commence in South Africa, and when the disease is first recognised?

According to the Natal Almanac the dry season includes the months of April, May, June, July, and August; the wet season, September, October, November, December, January, February, and March. As far as I am aware Nagana is not confined to certain months of the year as Surra is, but I should like to have more definite information on this point.

2. Towards the end of the dry season or when the water pools are nearly dried up, can the Trypanosoma be discovered in this concentrated water by the microscope or by feeding or injection experiments?

3. The question of herbage.—Dr. Lingard states that in India after the rains have commenced the resting forms of the organism which have been clinging to the grass bordering upon the water-holes begin to soften, and when the animals feed on such vegetation, repeated doses of the contagium are taken into the stomach. It is probable that the small discs gradually gain an entrance into the blood stream and their development takes place in the liver, spleen and glands generally. All grass eating animals, therefore, may be the subjects of Surra, so that the opinions held with regard to the big game by the natives are possibly founded on fact.

In Africa we are, as yet, in dense ignorance respecting the resting stage of the Fly hæmatozoon; this would be a most important point to work up. In regard to the herbage, will such grass and water brought up from the Fly Country give rise to the disease in healthy animals kept in a healthy locality?

4. It would be important to find out as far as possible what species of wild animals, vultures, barbel, &c., harbour the parasites.

5. As many examinations of the blood and fluids in the body cavity of the Tsetse Flies should be made and the result noted. Injections of fluids in which several flies have been mashed up might also give results.

6. How long do the hæmatozoa retain their vitality in the body of a dead animal?

7. How long do the hæmatozoa retain their vitality in ordinary pond water?

8. The Fly Disease in the native sheep and goat has to be worked up. I have up to the present been unable to procure these animals from the natives here.

9. Further information is wanted regarding the breeding of the Tsetse Fly.

10. Are the young of animals affected by the "Fly" in any way immune to the disease?

11. Sir Walter Hely Hutchinson informs me that he has heard it stated that animals can be rendered immune to Tsetse by taking them into the Fly when young, at intervals. Is there any truth in this statement?

12. Will the passage of the parasite through a series of animals influence its morbific power?

13. Would large quantities of the serum of wild animals, say the wildebeeste, have any influence on the course of the disease if injected under the skin or into the peritoneal cavity of the animal affected by the disease?

14. Can anyone study the disease in tamed wild animals, for example the koodoo or any of the smaller buck? Or can anyone procure such animals and send them to Ubombo to enable me to try the effect of the disease on them?

15. Why should the wild animals be spared whilst tame animals suffer? What is it in domesticity that removes immunity? Can the domestic animal be supplied with what is present in the wild? Can this something be discovered?

These then are a few of the problems to be solved in the "Fly" question, and if anyone wishes to work at them and requires living Trypanosoma, I shall be happy to inject any living animal they may send to me for the purpose, and so supply their want.

APPENDIX

TO

Further Report

ON THE

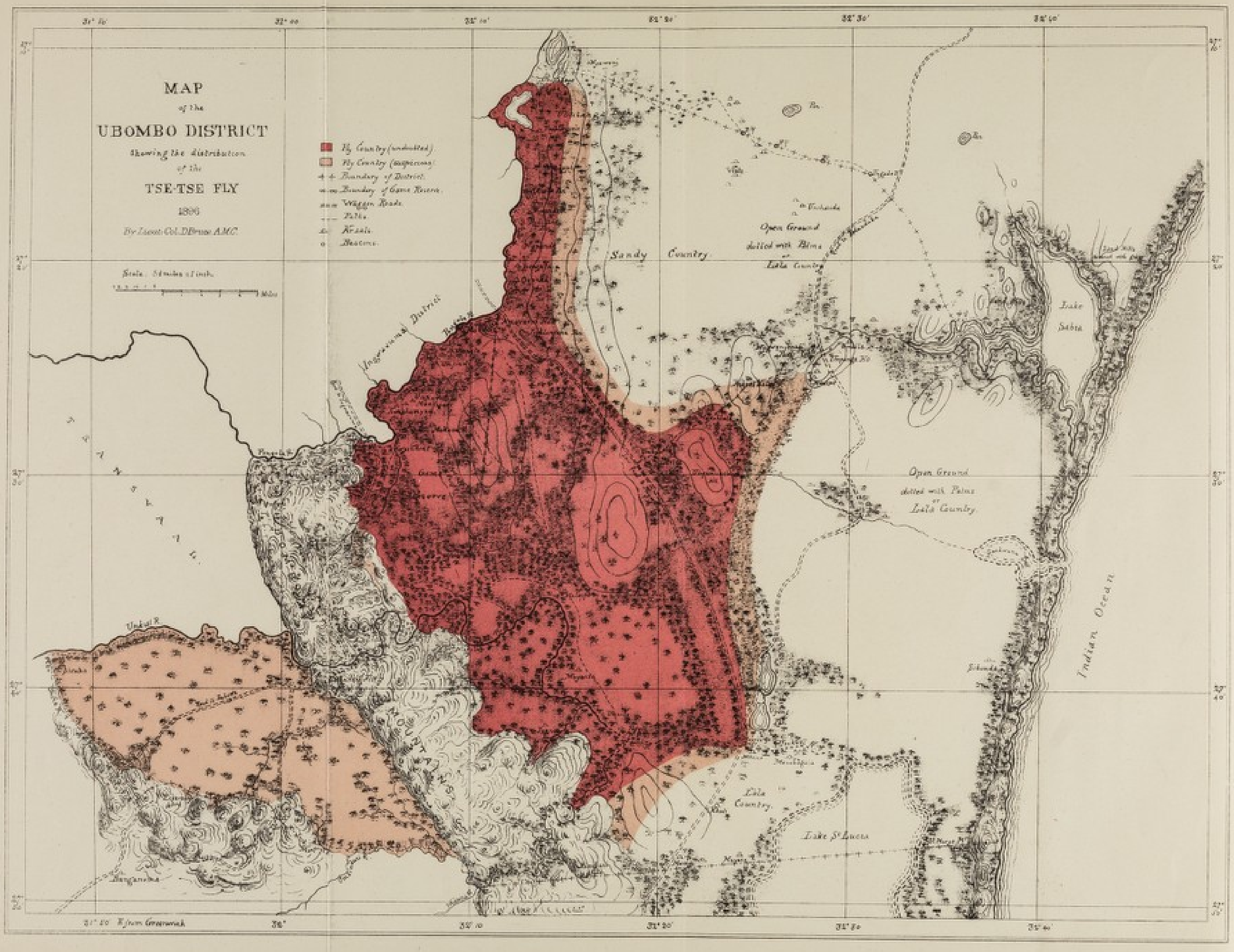
TSETSE FLY DISEASE OR NAGANA

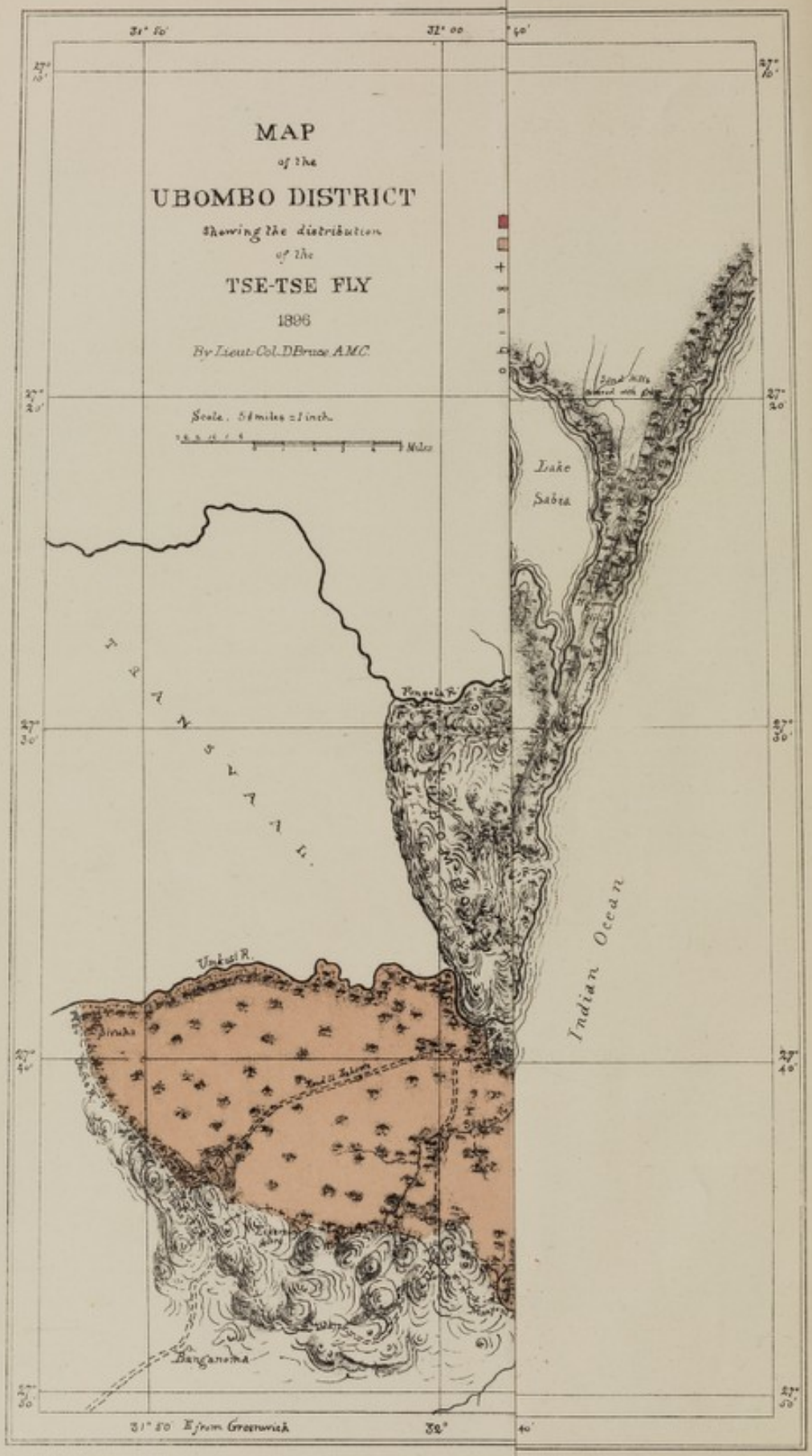
IN ZULULAND.

BY

LIEUT.-COL. DAVID BRUCE, F.R.S., R.A.M.C.

London:
HARRISON AND SONS, ST. MARTIN'S LANE,
PRINTERS IN ORDINARY TO HIS MAJESTY.
1903.


MAP
of the
UBOMBO DISTRICT
showing the distribution
of the
TSE-TSE FLY
1896
By Lieut.-Col. D. Bruce A.M.C.
Fly Country (undoubted)
Fly Country (suspicious)
Boundary of District
Boundary of Game Reserve
Waggon Roads
Paths
Kraals
Beacons
Sandy Country
Open Ground dotted with Palms or Lala Country
Lake Sabia
Indian Ocean
Lala Country
Lake St. Lucia
MOUNTAINS


MAP
of the
UBOMBO DISTRICT
Showing the distribution
of the
TSE-TSE FLY
1896
By Lieut-Col. D Bruce A.M.C.
Scale. 8 miles = 1 inch.
Miles
Lake
Sibia
Indian Ocean
Pongola R.
Umkusi R.
Sirudu
Bangweleni
31° 50' E from Greenwich
32°
40'
27° 10'
27° 20'
27° 30'
27° 40'
27° 50'

Tsetse Fly Disease or Nagana

IN ZULULAND.

INTRODUCTION.

On the 29th May, 1896, I sent to H.E. the Governor of Natal a "Further Report on the Tsetse Fly Disease or Nagana," which was printed by the Royal Society, at the request of the Natal Government, and issued on the 4th February, 1897.

I remained at Ubombo, in Zululand, until the 30th July, 1897, when I was driven away by the fact that the Nagana had overspread its usual boundaries and was destroying all the domestic animals, not only on the Ubombo, but for 50 miles further inland.

There was a widespread epidemic among the native cattle which was thought might be rinderpest. At the request of the Government I visited several of the native kraals, and everywhere found the disease to be Nagana. Many of the cattle belonging to the natives living near us on the Ubombo died. The Magistrate lost nearly all his trek donkeys, and several of the horses succumbed to the disease. My pointer, who had lived in good health for nearly two years on the top of the Ubombo, also took Nagana at this time and died.

It was impossible to find any reason for this sudden activity on the part of the Tsetse Fly, in so leaving its haunts in the low country and spreading far inland. It probably had something to do with the long-continued drought, which had driven herds of wild animals to seek for greener pasture in the mountain valleys. It was evident that Ubombo had lost its character as a healthy experimental station. It was now part of the Fly Country. I therefore abandoned the investigation, and returned to Pietermaritzburg and military duty.

Since then, my long-continued absence from England, on account of the Boer War, has prevented me from publishing the few further observations I made on Nagana in the beginning of 1897. As some of these notes are not without interest, I propose to bring them out as an Appendix to the last Report. Since 1897 many papers have been published on the Tsetse Fly Disease.

My offer made at the end of the "Further Report" to supply anyone with living Trypanosoma was taken advantage of. The Royal Society, in London, and Messrs. Bordet and Danyz, in Pretoria—the former by means of dogs, the latter by means of a horse—received the living disease.

At this point I would place on record my most hearty thanks to the Hon. Sir Walter Hely-Hutchinson, G.C.M.G., at that time Governor of Natal and Zululand. The initiation of the inquiry into the nature of Nagana is wholly due to him. In spite of much difficulty and obstruction he persevered in the furtherance of the investigation, and certainly without his active aid and encouragement the little that has been done could never have been attempted.

I also beg to thank Lieutenant-Colonel Sir Marshal Clarke, K.C.M.G., Acting Administrator, Zululand, and his successor, C. R. Saunders, C.M.G., for their aid and countenance. I ought also to express my indebtedness to the Tsetse Fly Committee of the Royal Society, and especially to Sir Michael Foster, for their encouragement and suggestions in the carrying out of this investigation.

My wife also has my best thanks. In her capacity of sole laboratory assistant she worked throughout the enquiry.

68, Victoria Street,
London, S.W.,
13*th January*, 1903.

1.—TOPOGRAPHY AND PHYSICAL FEATURES OF THE UBOMBO FLY DISTRICT.

In order to show the general features of this Fly District, I have prepared the accompanying Map, which was made partly by myself and partly from a map with which I was supplied shortly before leaving Ubombo.

At the same time it must be confessed that, on account of the difficulty of mapping a flat, thickly-wooded country, many of the features were put in by eye from the top of the Ubombo. The map, therefore, cannot claim to be very correct, but it is sufficient to show the general features of the country.

From the map it will be seen that the Lebombo chain of mountains runs nearly north and south. They rise sheer out of the plain on the western face and slope rapidly down on the eastern. At Ubombo the range is separated into two main ridges running north and south. These are nearly equal in height, and are separated by deep broken valleys.

The hut used as a laboratory was situated on the eastern ridge near the highest point of the hills, and (as nearly as I could make out) about 1,900 feet above the level of the sea.

The low country lying between the hills and the sea is divided fairly evenly into two parts by a line running through the centre from north to south. The western of these two halves is thickly covered with bush; the eastern consists of open country with a sandy soil covered with coarse grasses and dotted with palm trees, and known to the natives as the Lalla Country.

The Ubombo Fly District extends from the Pongola to some miles south of the Mkusi River, and to the eastward as far as the edge of the Lalla Country. North of the Pongola and between that river and the mountains, the country, as far north as the Usutu River, is curiously enough free from Fly Disease. As will be seen from the map, it is much more open than the Fly Country, but still there are numerous patches of bush scattered over it.

The Fly Country itself is very extensively covered with bush. The character of the vegetation varies in different positions. Between the ridges in the lower lying parts it may be described as dense thicket, the thorn trees composing it being only some 10 or 12 feet in height and matted and tangled together so as to be quite impassable, except along the game-paths made by the rhinoceros and other large animals. Here and there are beds of a kind of prickly pear or cactus, while in any open space which may be found the ground is covered with low aloes.

On the ridges the vegetation is, as a rule, not so dense. The mimosa trees, some 20 to 30 feet in height, are scattered irregularly about, with spaces of 30 or 40 yards between. There are also several large open spaces or plains, the principal one of which is known as the Makatini.

Along the rivers the vegetation is very dense. In many places it is impossible to approach the banks on account of the dense thicket of thorns. Huge fig trees, with thick green foliage, line the banks, affording shelter and food to numerous monkeys. Here, also, are seen the tall, graceful mimosas with pale greenish-yellow bark, known as Fever Trees, and which are never found far from water.

2.—THE TSETSE FLY.

In the 1897 Report I wrote that the Tsetse Fly is about 11 mm. or seven-sixteenths of an inch in length. To this may be added that the average length of 10 males was found to be 11 mm., that of the same number of females to be 12·5 mm.

The proportion of the sexes caught whilst feeding on animals seems pretty equal, since of 102 specimens caught at the same time, 51 were male and 51 female.

In experiments with the Tsetse Fly, as to how long it retains its infective power (Further Report, page 7), I showed that positive results could be got after 48 hours.

Exp. 339. Feeding Flies after 3 days.

August 1st.—Fed flies on affected animal.

August 4th.—Fed flies on Dog No. 339.

August 6th.—Fed flies on affected animal.

August 9th.—Fed flies on No. 339.

And so on till November 18th with negative results, when the experiment was stopped.

It would appear, then, that Tsetse Flies can give the disease to susceptible animals at least 48 hours after feeding on an affected animal, but that after 3 days the chances are much against their doing so.

3.—HOW LONG DOES THE BLOOD RETAIN ITS INFECTIVE POWER?

On page 11 of the 1897 Report I gave the results of experiments as to how long the blood of an affected animal remains capable of giving rise to the disease if kept in its naturally moist condition. In these experiments blood was found to retain its virulence for 4 days, but not for 7 days.

The following two experiments are added:—

Exp. 323. Dog, Native.

June 25th.—Inoculated with blood 6 days old.

July 3rd.—Hæmatozoa absent.

July 15th.—Hæmatozoa absent.

August 5th.—Hæmatozoa absent.

Dog healthy. Experiment stopped.

Exp. 325. Dog, Native.

June 25th.—Inoculated with blood 6 days old.

July 3rd.—Hæmatozoa absent.

July 15th.—Hæmatozoa absent.

August 5th.—Hæmatozoa absent.

Experiment stopped.

4. The question has often been asked how many flies does it take to give an animal the disease.

I cannot answer this question positively, but as bearing on it I may say that on several occasions I tried to set up the disease by allowing one fly caught in the low country to bite a healthy dog once, but always with negative results. I have no doubt one fly would give the disease if taken while feeding on an affected animal and placed straightway on a healthy one.

5. On page 16 of the Further Report, I state that there is no proof that the drinking of water or the eating of herbage plays any *rôle* in the infective process.

It was thought as well, however, to put this last statement to some kind of experimental proof. For this purpose there were brought up to the top of Ubombo specimens of water collected from pools in the country situated in the midst of bush notorious for fly. At the same time large bundles of grass were cut in the same localities.

A healthy horse was fed on the grass and water, and healthy dogs had small quantities of the water injected subcutaneously.

The following experiments show that the results of this procedure were negative :—

Exp. 319. Dog, Native.

June 15th, 1896.—Injected subcutaneously 5 c.cm. water from pool at Serpentine Camp.

August 5th, 1896.—Dog healthy.

Exp. 326. Dog, Native.

July 21st, 1896.—Injected 10 c.cm. water from pool in Fly Country.

September 2nd.—Dog healthy.

Exp. 341. Dog, Native.

August 27th, 1896. Injected 10 c.cm. water from pool in Fly Country.

October 16th.—Dog healthy.

Exp. 351.—Dog, Native.

September 14th, 1896.—Injected 10 c.cm. water from Fly Country.

September 18th.—Injected 5 c.cm. water from Fly Country.

September 25th.—Injected 10 c.cm. water from Fly Country.

December 7th.—Dog healthy.

Exp. 284.—Horse.

Between June 10th, 1896, and September 22nd, grass and water were brought up to the top of the Ubombo from the Fly Country on thirty-two occasions.

The water was sprinked over the grass, which was then given to the horse to eat. He remained perfectly healthy up to the 16th December. On that date, in order to prove that he was susceptible to Fly Disease, I took him for a few hours into the Fly Country.

On December 23rd, he had a swelling on the under surface of the abdomen and his blood contained numerous hæmatozoa.

6. In regard to the fate of the Trypanosoma when ingested by the Tsetse Fly (Further Report, page 18), I have to add a few notes on the examination of the proboscis, stomach, and intestines of the Tsetse Fly.

THE PROBOSCIS.

If the simple hypothesis that the Tsetse Fly merely acts a carrier of the adult living hæmatozoon from animal to animal is the true one, then it can only be by means of the proboscis that this transference takes place. If, on microscopical examination of the proboscis, numerous hæmatozoa were seen for several hours after feeding, then the acceptance of the hypothesis would be easy enough. But, unfortunately, nothing of the sort can be demonstrated. It is true that immediately after feeding the proboscis is found to be crammed full of red blood corpuscles, among which the hæmatozoon can be seen actively wriggling. So that if we picture a fly just beginning to feed on an affected animal, being whisked off by a flap of the tail, and then straightway passing to a fresh animal, there is no difficulty in understanding how the inoculation might take place, especially if we grant that the fly before drawing off any blood, probably injects a fluid to cause hyperæmia of the part, and so washes out the contents of the proboscis into the tissues of the animal attacked. But we have found by experiment that, even 48 hours after feeding, the flies are able to give rise to the disease. In this regard it must be confessed that it certainly took many efforts of many flies before the inoculation was effected. However it may be, the result of observations shows that even only 1 hour after feeding the proboscis contains few red blood corpuscles, and few if any hæmatozoa.

If, therefore, the Tsetse Fly does carry the disease from affected to healthy animals by simply transferring living hæmatozoa from one to the other, then we must assume that the dose of hæmatozoa required to set up the disease is a very small one, in fact we must almost assume that a single Hæmatozoon is sufficient.

The following table shows the result of the microscopical examination of the proboscis of Tsetse Flies at various times after feeding on an affected animal.

Hours.	Hæmatozoa.	Hours.	Hæmatozoa.
1	A few seen wriggling vigorously.	27	Nil.
2	Ditto.	28	One active hæmatozoon seen.
3	Ditto.	46	One active hæmatozoon seen.
4	One active hæmatozoon seen.	47	Nil.
5	Two active hæmatozoa seen.	48	Nil.
6	Nil.	49	Nil.
22	One active hæmatozoon seen.	50	Nil.
23	Nil.	72	Nil.
24	One active hæmatozoon seen.	97	Nil.
25	One active hæmatozoon seen.	118	Nil.
26	One active hæmatozoon seen.	144	Nil.

To get the above result, several flies were often examined at the same length of time after feeding before a positive result was obtained. In order, therefore, to make the list more complete, all that would be necessary would be the patience to examine a sufficiently large number of specimens. This, in my opinion, is not necessary, as the above list shows sufficiently clearly that, although isolated hæmatozoa may be found in the proboscis of the fly for many hours after feeding, still at no time are they numerous. It must, however, be stated that the proboscis is not a good object through which to look for such small objects as the hæmatozoa, so that others might have been present without being discovered. I must leave this subject in its present unsatisfactory condition, with the hope that some future observer may be able to throw more light on it. The most satisfactory solution would be the discovery of some metamorphosis of the Trypanosoma within the fly, such as occurs in the mosquito in malaria.

THE STOMACH.

When I came to examine the contents of the stomach, I was much surprised to find that the hæmatozoa retained their usual appearance and extraordinary activity as long as any vestige of blood remained in that viscus.

In some cases, at the end of 70 hours, the stomach would be found to contain no visible blood, all that remained being merely a small quantity of dark-coloured material consisting of acicular crystals and granular débris. When this condition obtained no living hæmatozoa would be seen.

In other cases, even up to 118 hours (5 days), a small quantity of reddish coloured material would still be found in the stomach. This, when examined microscopically, would be found to contain, unchanged, red blood corpuscles, among which the hæmatozoa could be seen to wriggle about as actively as ever. Up to 55 hours almost every preparation showed living hæmatozoa; after that time cases began to occur in which the blood, with its contained parasites, had disappeared from the stomach. At the end of another 24 hours these exceptions had become much more numerous, but up to almost the end of the 5th day rare individuals could be found in which the hæmatozoa were still numerous and vigorously alive. I always failed to find them on the 6th day.

It will be remembered that the flies were found to be capable of giving rise to the

disease after 48 hours, but failed to transmit it after 3 days. Judging from the above observation, that the Trypanosoma retains its vitality for 5 days in the fly's stomach, it may be supposed possible for the fly to transmit the disease up to that length of time.

It would be interesting to observe the behaviour of the Trypanosoma in the stomach of other blood-sucking flies, but it did not occur to me to experiment in this direction while in Zululand.

THE INTESTINE.

The contents of the intestine were examined by simply squeezing out of the living fly a small quantity and subjecting this to microscopical examination.

The material varied from yellow to almost black in colour, and consisted mostly of minute round or oval granules with larger crystals of an elliptic or rhomboidal shape. Among this crystalline matter I frequently observed motionless hæmatozoa up to 70 hours after the flies had been fed, but I have never seen at any time the least appearance of life in them. The parasites appeared unchanged in form, and I made some injection experiments to find out if these motionless hæmatozoa had any vitality left in them.

The droppings were collected by placing a piece of clean glass in the fly cage and washing off the deposit. During this time the flies were fed regularly on dogs suffering from Nagana, and whose blood contained numerous hæmatozoa.

The following experiments show the result :—

September 16th.—Injected droppings from 10 flies into a healthy dog. Dog remained healthy.

September 26th.—Injected droppings from 10 flies into a healthy dog. Dog remained healthy.

September 30th.—Injected droppings from 25 flies into a healthy dog. Dog remained healthy.

October 3rd.—Injected droppings from 25 flies into a healthy dog. Dog remained healthy.

October 8th.—Injected droppings from 23 flies into a healthy dog. Dog remained healthy.

October 22nd.—Injected droppings from 20 flies into a healthy dog. Dog remained healthy.

We may therefore look on it as highly improbable that the hæmatozoon is voided in the droppings of the Tsetse Fly in a form or condition capable of giving rise to Nagana.

7.—IS THE HÆMATOZOON CONFINED TO THE ALIMENTARY TRACT OF THE TSETSE? OR DOES IT MAKE ITS WAY THROUGH THE WALLS INTO OTHER TISSUES?

This is an important point. It seemed very probable that the hæmatozoa formed within the tissues of the fly is some resting or spore form, and that the connection between the Tsetse Fly and the disease was in some way or other bound up in this.

All I can say, in regard to this matter, is, that I have examined the bodies of some hundreds of flies at various times after they had fed on an animal affected by Nagana, and have never seen any appearance that would lead me to suspect that the hæmatozoa found their way out of the alimentary canal into the surrounding tissues. I have never seen the hæmatozoa change their shape while in the stomach of the fly, nor assume, as might be surmised, any globular or non-flagellate form. That the hæmatozoa do not pass into a resting stage capable of giving rise to the disease seems also negatived by the fact that the introduction under the skin of dogs of minced up flies whose stomachs are empty of blood has never given rise to the disease.

8.—THE INTRODUCTION OF MINCED UP TSETSE FLIES UNDER THE SKIN OF DOGS.

But at this point it must be stated that the introduction under the skin of susceptible animals of minced up flies whose stomachs do contain living hæmatozoa, also does not as a rule give rise to the disease.

For this curious fact I can give no explanation, as I left Ubombo while the experiments were in progress. The *modus operandi* of the experiment was to feed Tsetse Flies on an affected animal, and after a given time to mince them small, mix with a little water, and inject the suspension through a wide hypodermic needle under the skin of dogs.

The following experiments illustrate the point.

Exp. 449. Dog, Native.

July 5th.—Injected one minced up fly which had been fed on affected animal half-an-hour previously.

July 19.—Hæmatozoa numerous in blood of dog.

Exp. 451. Dog, Native.

July 16th.—Injected one fly fed 1½ hours previously.

August 2nd.—Dog healthy. No hæmatozoa in blood.

Exp. 452. Dog, Native.

July 16th.—Injected one fly fed 1½ hours previously.

August 2nd.—Dog healthy. No hæmatozoa in blood.

Exp. 429a. Dog, Native.

June 25th.—Injected one fly fed 24 hours previously.

July 21st.—Dog healthy. No hæmatozoa in blood.

Exp. 427a. Dog, Native.

June 21st.—Injected three flies, fed an affected animal yesterday.

July 21st.—Dog healthy. No hæmatozoa in blood.

Exp. 438. Dog, Native.

June 20th.—Injected three flies fed three days previously.

July 5th.—Dog healthy. No hæmatozoa in blood.

Exp. 427. Dog, Native.

May 26th.—Injected one fly fed 5 days previously.

June 21st.—Dog healthy. No hæmatozoa in blood.

Exp. 429. Dog, Native.

May 27th.—Injected one fly fed 6 days previously.

June 21st.—Dog healthy. No hæmatozoa in blood.

Other experiments on the same lines were made, but always with the same result, Half an hour after feeding, the minced up flies gave rise to the disease, if injected at a later period the result was negative. It is a matter for regret that this experiment was stopped at 6 days and not tried at longer intervals.

I also tried on several occasions to set up the disease by injecting minced up flies brought direct from the low country, but without result.

It then occurred to me that perhaps the injection of these flies might in some way or other confer immunity on the experimental animal. Curiously enough there is a popular idea to this effect among the Boers who come in contact with the disease.

I therefore tried many experiments in different ways to find out if there was any truth in this. From one to eight flies were injected at the same time, and at varying times afterwards the dogs were inoculated with 1/10 c.cm. of blood containing the hæmatozoa. Or several flies were injected into one part of the body of the dog and the Nagana blood inoculated at the same time into another part.

In other cases the minced up flies were injected on several occasions both before and after inoculation. In no case was the incubation period of the disease perceptibly shortened, or the death of the animal delayed.

This point must therefore remain for the present as I have stated it. A fly, a few hours after feeding on an affected animal, crammed with blood showing active hæmatozoa under the microscope, if minced up and injected under the skin of a susceptible animal, fails to give rise to the disease.

9.—IS THE VIRULENCE OF THE HÆMATOZOA AFFECTED BY ITS PASSAGE THROUGH ANY SPECIES OF ANIMAL?

I have tried this in the case of the horse, donkey, ox, and goat, and found no diminution or change in virulence. Dogs inoculated with blood from any of these animals took the disease as readily and died as quickly as those inoculated from affected dogs.

10.—IS THE TSETSE FLY ARTIFICIALLY REARED CAPABLE OF GIVING RISE TO THE DISEASE IN SUSCEPTIBLE ANIMALS?

Freshly extruded larvæ broken up and placed under the skin of dogs always gave negative results.

11.—DOES THE HÆMATOZOON PASS FROM THE MATERNAL INTO THE FŒTAL CIRCULATION?

This question, as far as my experiments go, must be answered in the negative. Blood taken from newly born pups whose mothers were affected by Nagana, and injected into healthy dogs, always gave negative results.

These pups were always prematurely born and only lived a few hours after birth. I had therefore no opportunity of trying if they possessed any immunity against the disease.

12.—RELATION OF THE BIG GAME TO THE FLY DISEASE.

I have to add the following additional experiments in which the blood of wild animals was injected into healthy dogs to ascertain if such blood contained the Trypanosoma.

Exp. 327. Dog, Native.

August 18th, 1896.—Injected 5 c.cm. blood from Burchell's Zebra.
September 29th.—No hæmatozoa in blood. Dog healthy.

Exp. 334. Dog, Native.

August 20th, 1896.—Injected 5 c.cm. Leopard's blood.
September 30th.—No hæmatozoa in blood. Dog healthy.

Exp. 335. Dog, Native.

July 10th, 1896.—Injected 5 c.cm. Buiker's blood.
September 2nd.—No hæmatozoa in blood. Dog healthy.

Exp. 337. Dog, Native.

July 8th, 1896.—Injected 5 c.cm. Koodoo blood.
September 2nd.—No hæmatozoa in blood. Dog healthy.

Exp. 340. Dog, Native.

August 23rd, 1896.—Injected 6 c.cm. Leopard's blood.
September 30th.—No hæmatozoa in blood. Dog healthy.

Exp. 335. Dog, Native.

September 14th.—Injected 5 c.cm. Roi Rei Buck's blood.
„ 30th.—Hæmatozoa absent. Dog healthy.

Exp. 357.—Dog, Native.

September 20th, 1896.—Injected 2·5 c.cm. Blood from "Bopeva," a small weasel-like animal. Skin sent.
November 5th.—Hæmatozoa absent, in blood.

Exp. 405. Dog; Native.

March 26th.—Injected 5 c.cm. Reed Buck's blood.
May 1st.—Hæmatozoa present in blood.

Exp. 408. Dog, Native.

March 28th.—Injected 10 c.cm. Jackal's blood.
May 30th.—Dog healthy.

Exp. 412. Dog, Native.

April 25th.—Injected 5 c.cm. Koodoo's blood.

April 30th.—Hæmatozoa present in blood.

Exp. 414. Dog, Native.

April 15th.—Injected 5 c.cm. Roi Rei Buck's blood.

May.—Hæmatozoa absent.

Exp. 434. Dog, Native.

June 12th.—Injected 5 c.cm. Koodoo's blood.

July 14th.—Dog healthy.

Exp. 437. Dog, Native.

June 18th.—Injected 10 c.cm. Koodoo's blood.

July 19th.—Dog healthy.

13.—THE FINDING OF THE TRYPANOSOMA BY DIRECT MICROSCOPICAL EXAMINATION OF THE BLOOD OF WILD ANIMALS.

Before these experiments of the injection of the blood of wild animals into dogs were begun, I had tried on several occasions to discover the parasite in the blood of various kinds of game by direct microscopical examination, but always without result. It was possible that the hæmatozoon existed in the blood of wild animals in some other form than that seen in the domestic and more susceptible animals. It therefore seemed of importance to again subject the blood of wild animals to a careful microscopical examination, in order either to discover the parasite in its known form, or to find out if any extraneous bodies could be seen in such blood.

The search in this case resulted in the discovery of the adult form in the Koodoo, Reedbuck, and Steinbuck. The hæmatozoa seen in the blood of these wild animals were indistinguishable from those found in the domestic animals. The parasites were few and far between, and it was only by long and patient searching that they could be found at all.

14.—DESCRIPTION OF THE FLY DISEASE OR NAGANA AS IT OCCURS IN DOMESTIC ANIMALS.

In the "Further Report" I described the disease as it appears in the Horse, Donkey, Ox, and Dog.

To the account of Fly Disease in the Dog I add one experiment.

Exp. 362. Dog, Native. Brought from Matoppi's kraal in Fly Country. To ascertain if dogs bred in Fly Country are immune to inoculated Nagana.

October 4th, 1896.—Inoculated 5 c.cm. blood from dog suffering from Nagana.

October 16th.—Hæmatozoa present in blood. Very numerous.

October 17th.—Dog died.

This dog's mother, according to native statement, was brought to Matoppi's from a healthy district. She had pups, and afterwards died of Nagana. Her two pups thrived. It is difficult to get natives living in the Fly Country to part with their dogs, as few are found there. My opinion is that these dogs require frequent renewal from healthy districts.

15.—NAGANA OR TSETSE FLY DISEASE IN NATIVE GOATS.

From the fact that native goats are often found at kraals where no cattle are kept, and where cattle cannot be kept, I was led to believe that the goat was immune to Nagana. Popular opinion seemed to strengthen this belief.

It seemed reasonable to think that the blood of the goat might contain some principle antagonistic to the Nagana hæmatozoon, and that this might be discovered and the knowledge made use of for immunising purposes.

It was with some curiosity then that I began the inoculation of goats with Nagana blood. At first all seemed to go well, the animals appeared to be not in the least affected by the disease, but unfortunately after the lapse of some months all of them died, except one, which was accidentally killed.

This fact, in itself, makes the immunity of goats against Nagana appear to be of very doubtful value. That all died of Nagana I cannot assert, but in my opinion the fatality among them was bound up in some way or other with this disease. But at the same time it must be admitted that although the native goat cannot be said to be absolutely immune, still he is to a very great extent refractory. On examination of the five cases given, it will be seen that only one goat died with undoubted symptoms of Nagana, and that the disease only showed itself after three months of apparent health. In this case profound destruction of red blood corpuscles took place, in the others no such destruction occurred. The temperature curve is very little affected in any of the cases, and it is only very exceptionally that the hæmatozoa are detected in the blood. In spite of this, blood taken from any of the goats and injected into healthy dogs gave rise to the disease.

Exp. 318. Goat, Native.

June 14th, 1896.—Procured this goat from a neighbouring kraal. The animal appears to be perfectly healthy, and is accompanied by a small kid.

June 19th.—Injected in the region of the right groin 5 c.cm. blood from a dog suffering from Nagana.

August 26th.—This goat has been in perfect health up to the present, that is to say, for more than two months, but to-day a small abscess was discovered in the right groin close to the site of the injection of the dog's blood. I opened the abscess, which was about the size of a walnut, and removed a quantity of cheesy-looking pus.

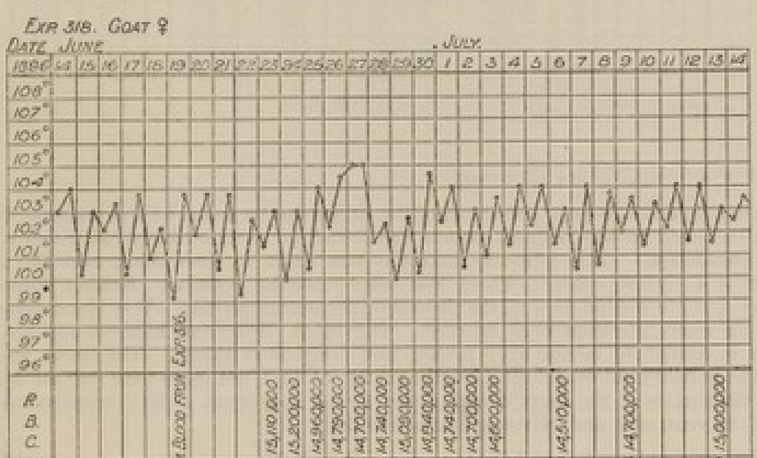
In order to see if there is any difference in the action of the hæmatozoa after living in the goat for more than two months, I to-day injected 2 c.cm. of this goat's blood into a healthy dog. Ten days afterwards the hæmatozoa were found to be present in this dog's blood, and he died 23 days later; 33 days after inoculation.

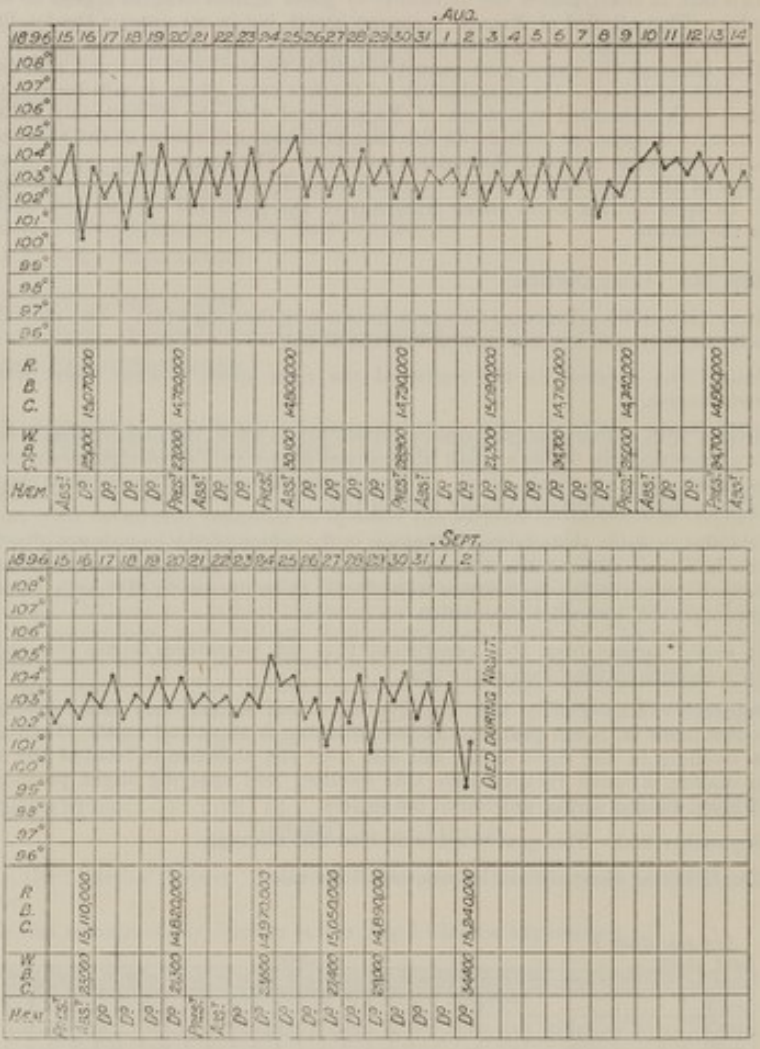
It would appear from this experiment that the hæmatozoa do not lose anything in virulence by a sojourn in the blood of the goat.

September 1st.—During the last five days this animal has fallen off rapidly in condition. She looks ill, is not grazing, and her breathing is short and rapid. There is a large œdematous swelling in the right axilla, and also over the abdomen in front of the mammary gland. On making a small incision into this swelling and applying slight pressure a large quantity of blood-stained serum was forced out in jets.

September 2nd.—This goat was found dead this morning.

The following chart shows the progress of the case.





Post-mortem, 7 hours after death.

On skinning this goat, the whole of the subcutaneous tissue on the under surface of the body from the neck to the perineal region was found to be extensively infiltrated with a thin gelatinous exudation. This jelly-like material was quite an inch thick over the abdominal region, and of a pinkish colour. There was some muscular wasting. Neither cornea showed any opacity. There was no fluid in the pleural or peritoneal cavities. The blood-vessels on the surface of the small intestines were injected. The lungs, liver, and kidneys appeared healthy. The spleen measured 3¾ inches by 3 inches, and weighed 6 ounces. The tissue was soft and friable. The pericardium contained a small quantity of fluid, not more than half an ounce. The vessels on the surface of the heart were injected, giving the organ a dark crimson colour. The interior of the ventricles and valves appeared healthy.

Remarks.—It is doubtful if this animal died of Nagana. On examining the temperature chart it is seen that the hæmatozoa are only rarely present in the blood, and that in small numbers, the temperature is seldom above the normal, and the red blood corpuscles show no signs of diminishing in numbers.

On the other hand, an abscess in the groin was opened by a free incision on the 26th August, and afterwards the œdematous swelling occurred. The formation of the abscess, though tardy, was probably connected with the injection of the dog's blood, and the condition leading to death seems to have been brought about through the opening of this abscess.

Exp. 330. Goat, Native.

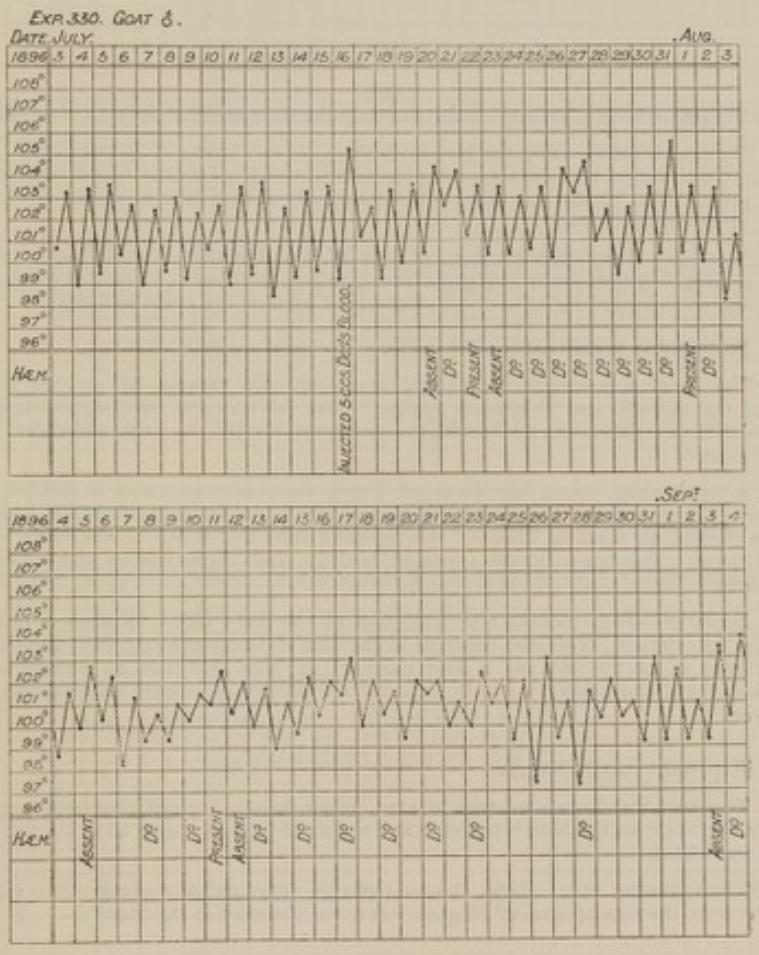
July 16th.—Injected under the skin of the abdominal region 5 c.cm. blood from a dog suffering from Nagana.

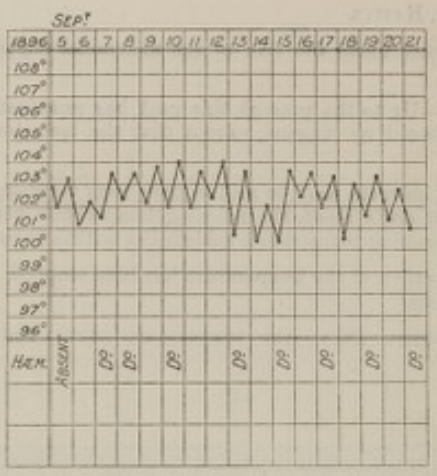
September 30th.—Up to the present this goat has remained in excellent health. He has never been visibly affected by Nagana, except that the hæmatozoa have been seen in his blood on three occasions.

His temperature, however, may be said to have been persistently a little above normal. Before the injection of the dog's blood, the morning temperature usually lies between 99° and 100°, but afterwards the morning temperature seldom lies within these limits.

To-day the observation of the temperature has been stopped, as it is thought the animal will show no more symptoms.

The following chart shows the temperature curve and presence or absence of hæmatozoa in the blood:—





November 17th.—To-day a hard swelling was discovered at the site of inoculation.

November 24th.—Since the last entry this goat has fallen off in condition. To-day he is suffering from vertigo, and since morning has been turning constantly round from left to right as on a pivot. The pupils are equal. He frequently falls forwards on his head, and finds difficulty in regaining his feet.

November 25th.—The symptom of vertigo is absent this morning, but he can scarcely walk, only staggering forward a few paces at a time, as he tries to nibble the grass. As there seemed to be no chance of his recovery, he was killed by bleeding about mid-day.

Post-mortem, immediately after death.

The blood appeared to be normal in colour. On making an incision through the parietes of the thoracic and abdominal regions, some fat was seen in the subcutaneous tissue over the abdomen. There was no fluid in the peritoneal cavity, and the abdominal organs all appeared healthy.

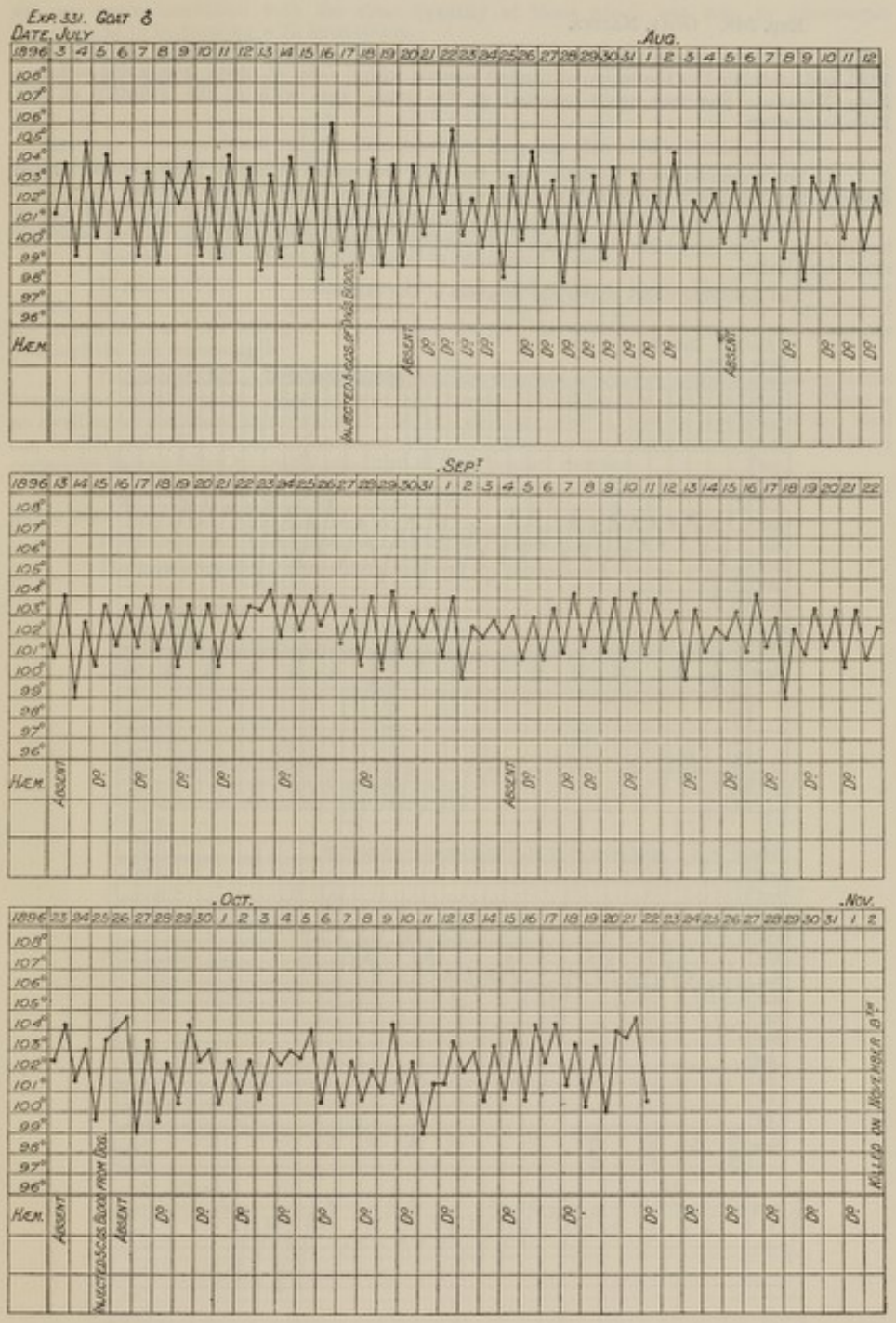
A few bladder worms like those of *Tænia marginata* were seen in the neighbourhood of the liver. There was no fluid in either pleural cavity nor in the pericardium, and the lungs and heart showed no signs of disease. On opening into the cavity of the brain an excess of fluid was found. The brain tissues appeared healthy, and there was no sign of cisticerci or other tumour.

Remarks.—Four months after injection of the dog's blood an abscess formed at the site of the inoculation. This also occurred in the case of the goat (Exp. 349) mentioned above, after the lapse of nearly two and a half months. In Exp. 348 also, an abscess formed after 2 months at the site of injection, and in Exp. 349 a collection of caseous material was discovered in the liver.

Exp. 331. Goat, Native.

This goat was inoculated with 5 c.cm. blood from a dog suffering from Nagana on the 16th July, and again on the 25th September, and remained in good health until the 8th November when he was killed.

The following chart shows the temperature curve and the observations on the blood. As will be seen, the hæmatozoon of Nagana was never observed in the blood :—



Remarks.—This goat was inoculated with blood from the same dog and at the same time as the goat, Exp. 349.

It is to be regretted that he was killed at so early a date, as it is quite possible he might have developed symptoms later on. Exp. 349 did not show any signs of being affected by Nagana until the beginning of December.

Exp. 348. Goat, Native.

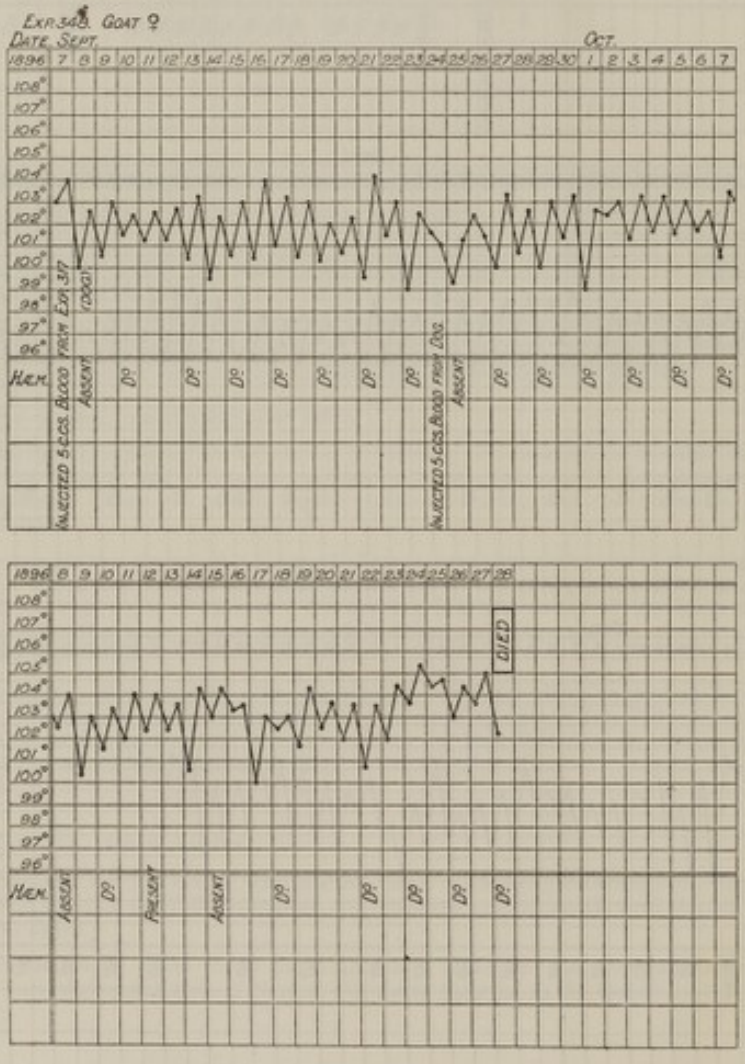
This goat, accompanied by her kid, was procured about the beginning of September, 1896, and inoculated in the region of the left scapula with 5 c.cm. of blood from a dog suffering from Nagana. She was again injected at the same spot with a like quantity on the 25th September.

The hæmatozoa were only observed on one occasion in her blood.

She remained quite healthy until the end of October, when a huge abscess formed starting from the site of the inoculation and extending to the right shoulder and leg.

October 26th.—To-day I injected a small quantity of blood from this goat into a healthy dog. Ten days after the dog's blood contained hæmatozoa, and he died on the 22nd day after inoculation.

The following chart shows the progress of the case :—



Remarks.—It is curious that nearly two months elapsed before any signs of inflammation showed at the site of the inoculation. I cannot but connect the injection of the Nagana blood with the formation of this abscess.

Exp. 349. Goat, Native.

September 1st, 1896.—This goat accompanied by a kid 8 days old was procured to-day, and at once inoculated with 5 c.cm. of blood from a dog suffering from Nagana. On the 25th September,

she was again inoculated with the same quantity of blood which was seen by microscopic examination to be teeming with hæmatozoa.

November 6th.—This goat appears to have remained up to the present in perfect health. The hæmatozoa have only been seen in her blood on three occasions. As I imagined the goat might have recovered from the disease and all the hæmatozoa introduced into her blood be killed off, I injected to-day a small quantity of her blood, obtained from an ear vein, into a healthy dog. Contrary to my expectation, however, this dog (Exp. 361) showed the hæmatozoa in numbers in his blood twelve days afterwards, thereby proving that the parasite still existed in a living condition in the blood of the goat. This is very similar to what obtains in the wild game. The blood of an animal apparently healthy and in which no hæmatozoa can be detected by the microscope can nevertheless give rise to the disease.

November 18th.—Goat again noted to be in good health.

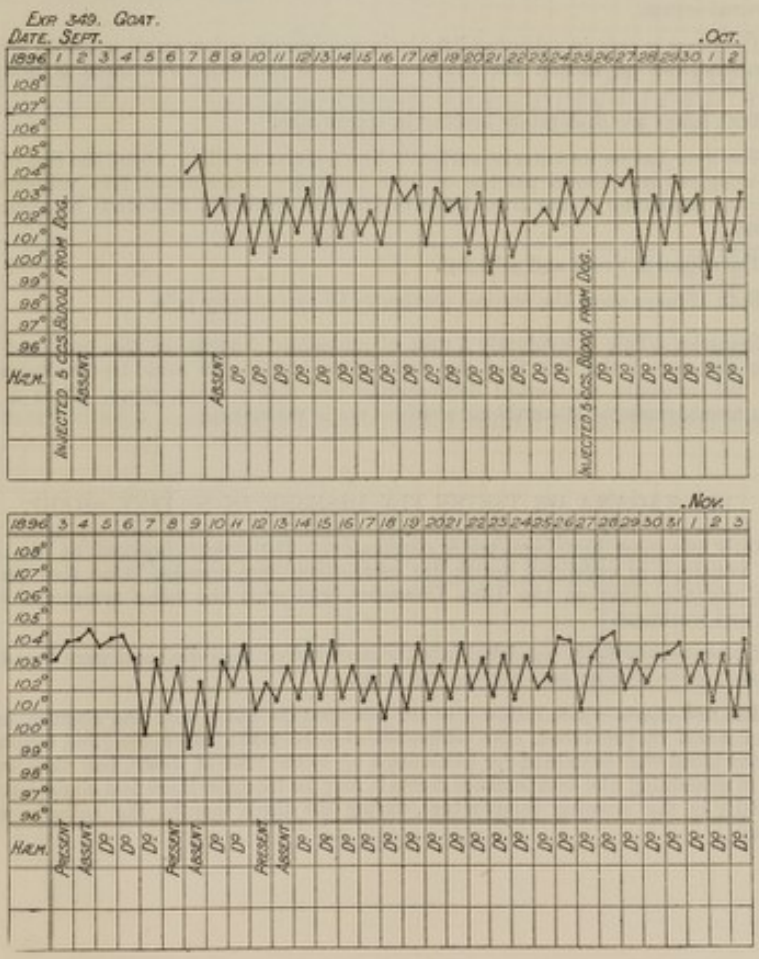
December 9th.—The hæmatozoa have not been detected in the blood for almost two months, and yet this goat is now beginning to shows signs of Nagana. Since the beginning of the month she has been falling off in condition, the mucous membrane of the lips and gums are pale and the left cornea milky.

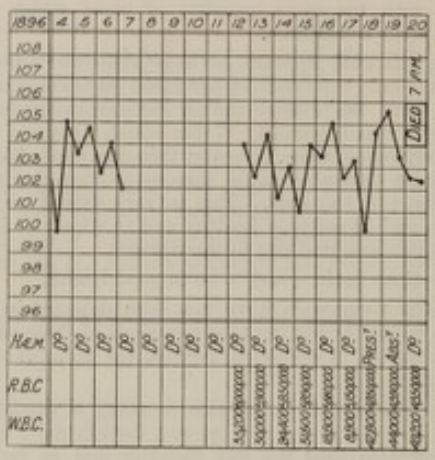
December 12th.—This animal shows this morning a marked muscular wasting in the lumbar and sacral regions. Both cornea are opaque, the left more so than the right. There is no swelling at the site of injection over the left shoulder, and in fact no swelling of any kind can be detected. The mucous membrane over the gums is exceedingly bloodless, the red blood corpuscles having fallen to nearly one-third of their normal number.

December 18th.—To-day the opacity of the right cornea has disappeared, and the left is much less opaque than six days ago.

December 20th.—The goat died to-day at 7 p.m.

The following chart shows the course of the disease.





December 21st.—*Post-mortem* examination 15 hours after death.

The body is much emaciated and presents no external swelling. The left cornea is milky, the right moderately clear. The peritoneal cavity contains 3 oz. of fluid and the intestinal coils appear atrophied and pale.

Both pleural cavities and the pericardium contain a small quantity, 4 oz. in all, of clear straw-coloured serum.

The heart is pale in colour and flabby in consistence, a few small petechiæ are scattered over the surface, and on section the muscular tissue is pale, otherwise there is nothing noteworthy.

Both lungs are moderately healthy in appearance.

The liver has on its anterior surface a firm nodular swelling, about the size of a walnut, which on being cut into shows patches of liver substance degenerated into yellowish caseous material. The remainder of the liver tissue is pale and evidently in a state of advanced fatty degeneration.

The spleen is small and softened.

Both kidneys show the cortical portion to be exceedingly pale.

Remarks.—This is evidently a case of undoubted Nagana in the goat, and is chiefly curious on account of the fact that the hæmatozoa hardly ever could be demonstrated in the blood, and yet extensive anatomical degeneration had slowly taken place. It is a matter of regret that hæmacytometer observations were not made throughout.

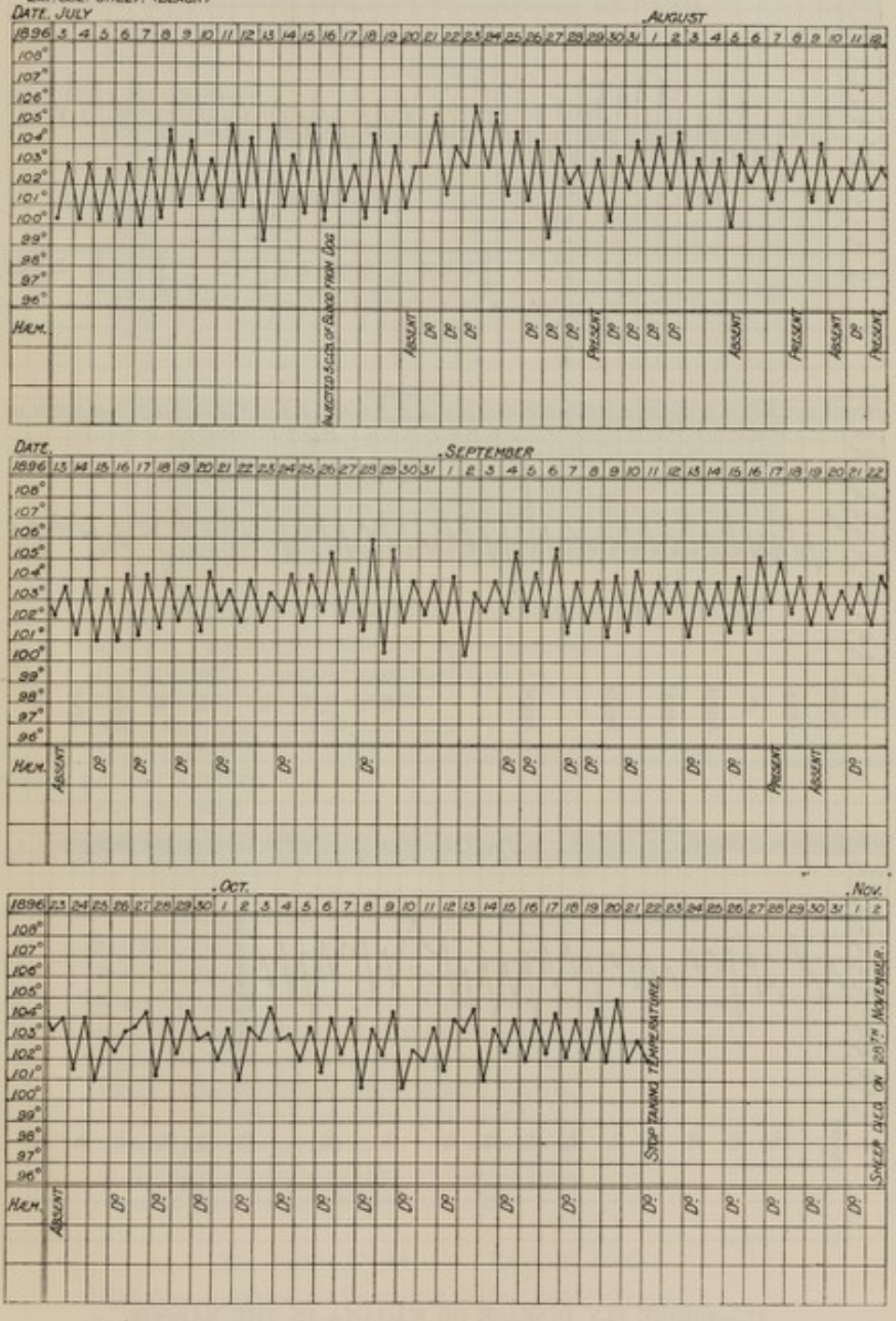
16.—NAGANA OR TSETSE FLY DISEASE IN NATIVE SHEEP.

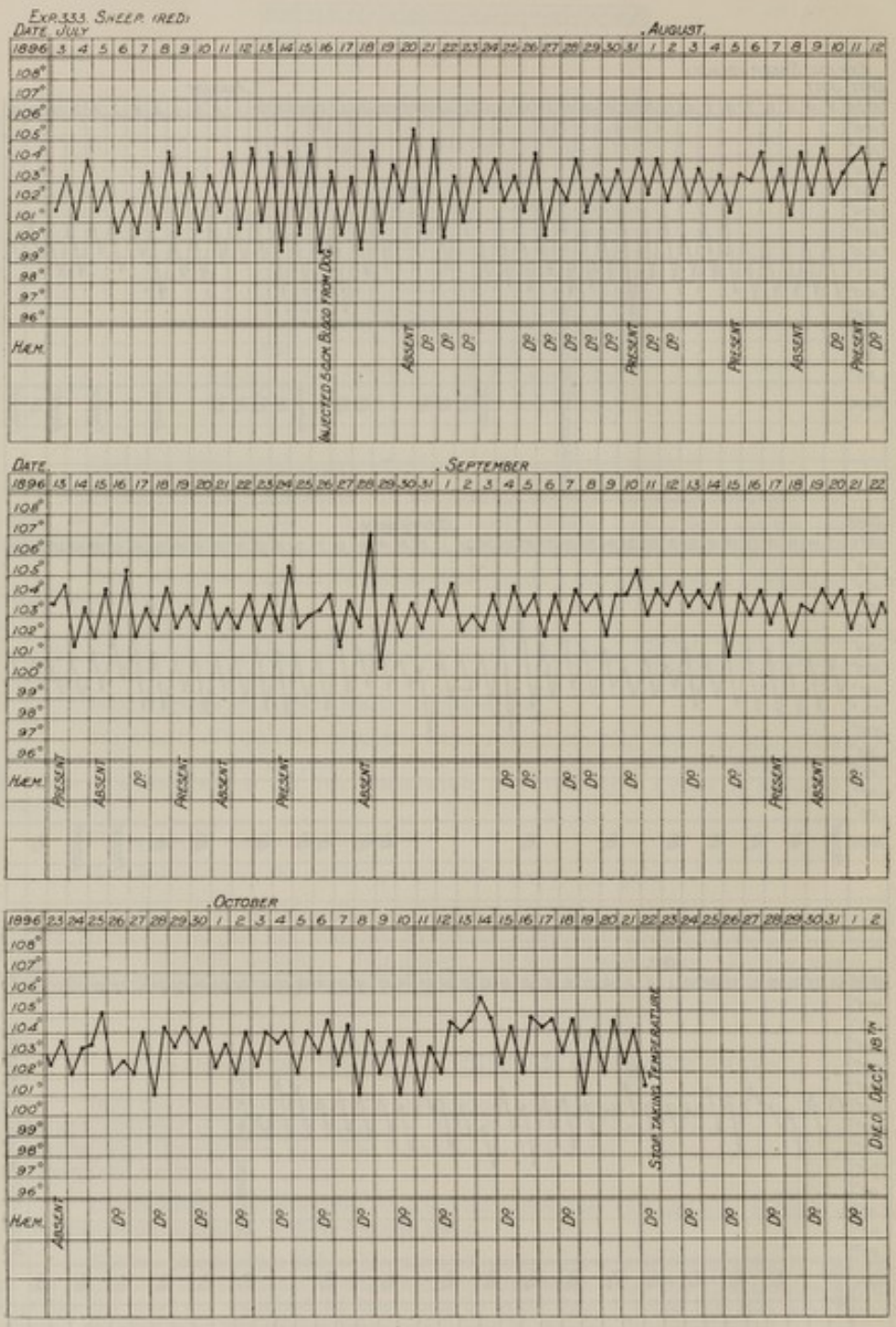
Only two cases have been under observation. Both were injected on the 16th July, 1896, and remained in excellent health for more than four months. One died on the 28th November, and the other on the 18th December, and with the same symptoms of vertigo, staggering and weakness.

Unfortunately both wandered into the bush before they died and were found in a state of decomposition, so that no *post-mortem* was made.

The two charts which follow give the temperature curve and the absence or presence of the hæmatozoa in the blood. No temperatures were taken after the 22nd October. After that date the blood was examined for hæmatozoa up to the time of death, but they were always absent. On examining the temperature curve it may be thought there is a slight but persistent rise, especially in the morning temperature, during the three months the animals were under observation.

EXP 332. SHEEP (BLACK)





17.—TSETSE FLY DISEASE OR NAGANA IN THE BABOON.

I only had the opportunity of trying the effect of the injection of blood containing the Nagana hæmatozoa into one baboon, and with the negative results, as the following experiment will show:—

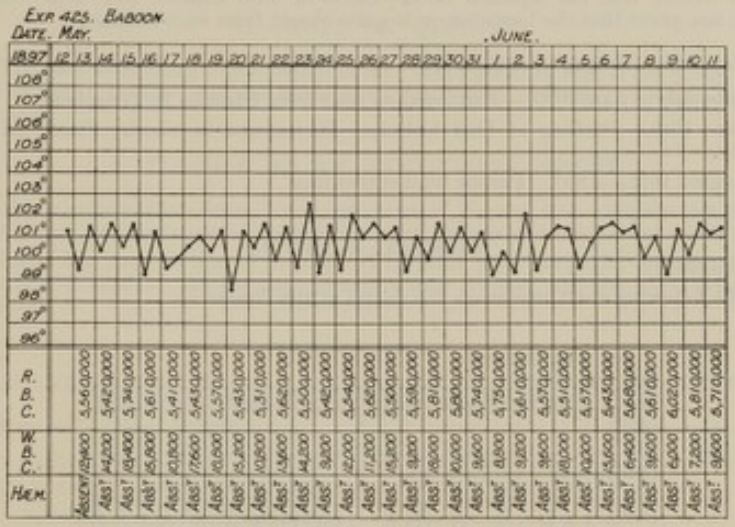
Exp. 425. Baboon, Young.

May 12th, 1897.—Injected 0·2 c.cm. blood from dog suffering from Nagana.

June 4th.—Injected 2 c.cm. blood from dog suffering from Nagana.

August 4th.—Baboon healthy.

The following chart gives the temperature, &c.:—



18.—TSETSE FLY DISEASE OR NAGANA IN THE MONKEY.

Exp. 423. Monkey, 2 months old. Species unknown.

June 4th, 1897. Injected 1 c.cm. blood from dog.

June 25th.—Monkey in good health.

June 30th.—Apparently in good health.

July 4th.—Refuses to eat. Blood swarming with hæmatozoa. Much more numerous than in the blood of the dog.

July 6th.—Died.

Remarks.—This is getting perilously near man. The hæmatozoa were more numerous in the blood of this monkey than in the blood of any animal I have examined.

19.—INJECTION OF NAGANA BLOOD INTO FOWLS.

Exp. 350.

September 7th.—Injected 5 c.cm. Nagana blood into two fowls.

September 25th.—Injected 5 c.cm. Nagana blood into same two fowls.

September 30th.—Hæmatozoa absent in blood of fowls.

October 4th.—Hæmatozoa absent in blood of fowls.

October 10th.—Hæmatozoa absent in blood of fowls.

October 16th.—Hæmatozoa absent in blood of fowls.

October 18th.—Hæmatozoa absent in blood of fowls.

October 22nd.—Hæmatozoa absent in blood of fowls.

December 31st.—Both fowls alive and healthy.

20.—ARSENIC AS A CURATIVE AGENT.

I add three experiments to those given in the "Further Report"—

Exp. 345. Horse, "Sweep."

August 6th.—R.B.C., 3,910,000. W.B.C., 11,600. Hæmatozoa, 800. Placed on 12 grains arsenic daily.

November 26th.—Died of Nagana.

Remarks.—On October 22nd the hæmatozoa having been absent on microscopical examination from "Sweeps" blood for some time, a small quantity of his blood (1 c.cm.) was injected into a healthy dog to ascertain if his blood was still infective. This dog remained healthy up to December 7th, thus showing that an animal suffering from Nagana, and afterwards dying of the disease, may have his blood at times injected into a healthy dog without setting up the disease.

It follows that the injection of the blood of wild animals into dogs with negative results does not prove that the hæmatozoon is quite absent from such animals.

Exp. 346. Horse, "Beffusa."

June 5th to June 12th.—Employed on road from Ubombo to Tongaland.

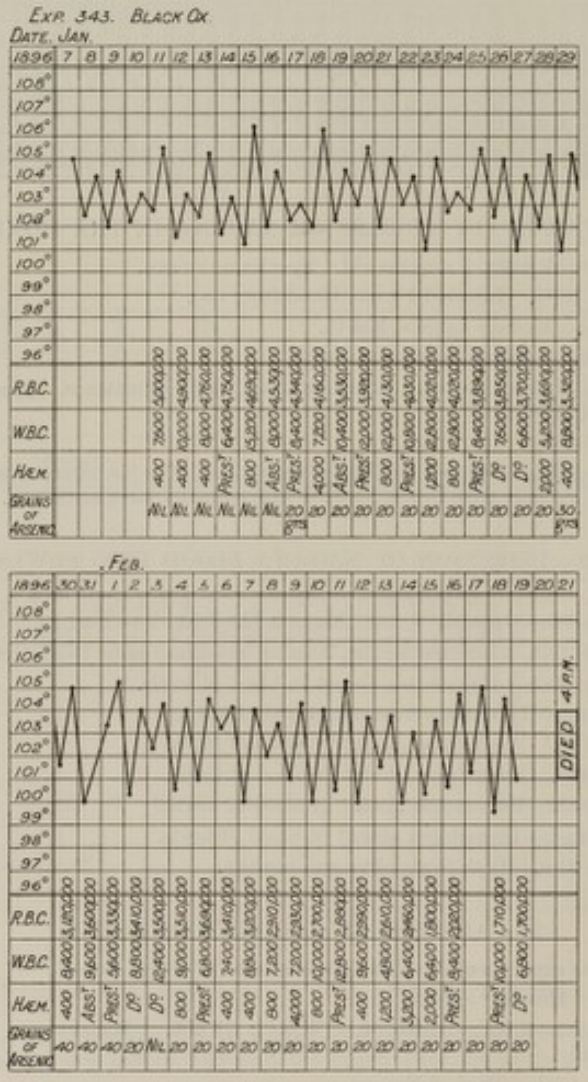
June 16th.—Swelling of hind fetlocks. Hæmatozoa in blood. Placed on 12 grains arsenic daily.

August 15th.—Died. Paresis hind extremities.

Treatment in Cattle.

Exp. 343.

The following chart represents the course of the disease:—



Remarks.—From this case it is evident that arsenic has little influence on the course of the disease in cattle.

On page 62 of the "Further Report" is found Exp. 261, showing the effect of arsenic on a donkey, which was still alive when that paper was written. I have to add that this donkey died in January, 1897, of Nagana, having been kept alive for nearly a year by the administration of arsenic.

21.—NOTE ON QUININE.

The only other drug I tried was quinine. This, given to dogs to the extent of 30 grains daily, had no apparent effect on the hæmatozoon, and did not delay the death of the experimental animal.

22.—BILE AS AN IMMUNISING AGENT.

The injection of bile from a dog just dead of Nagana into a healthy dog does not give rise to the disease, but, as far as my experience went, had no protective effect whatever.

I also injected the bile of koodoo, wildebeeste, and other wild animals into dogs, but also with negative results. All the dogs promptly took Nagana, and died after inoculation with virulent blood.

23.—SERUM OF WILD ANIMALS AS AN IMMUNISING AGENT.

Exp. 447. Dog, Native.

July 2nd.—Injected 40 c.cm. defibrinated blood from reed-buck, and at the same time inoculated into a small incision in the dog's ear a drop of Nagana blood.

July 10th.—Hæmatozoa swarming.

July 17th.—Dog died of Nagana.