It expands and contracts in an apparently normal manner. The vesicular murmur can be plainly heard in it, and percussion gives the same note as obtained from fresh lungs. The process will be seen to be a modification of the old glycerine-carbolic method, well known and often used to preserve anatomical specimens in an elastic condition. The modifications are important, however; and the results far better than any obtained in the old way. The idea of the method is, first, to fix the tissues by the chromic and acetic acid solution, then to introduce the glycerine-carbolic solution, and, finally, to get rid of this, and practically to make an 'oil-tanned' lung."

The composition of the three solutions and the mode of using them are then given. It does not appear that, at that time, he had prepared any specimen of both lungs, or that he subsequently exhibited, or published any description of, any such. That which he prepared (by his new method) to present to me at New York in the spring of 1892 may perhaps have been the first. These lungs were slightly pathological. They did not remain in efficient working much more than a year: partly, perhaps, owing to insufficient exercising. Meanwhile, in the early summer of 1892, I prepared, according to his latest directions, a perfectly normal and complete specimen of both lungs with trachea. This was a complete success. It was long used for demonstration at St. George's Hospital; and it still preserves its elasticity after the lapse of nearly twenty-three years. Unhappily, through a preventable accident, it has quite recently developed a bad "puncture" which now disables it, though it is still inflatable, for any complete and lasting inflation. That long record, and the fear lest, owing to his untimely death, the value of his latest method might otherwise be lost to science, make me anxious to publish those simple directions which enabled me to secure that success, without the help of any actual demonstration of his technique, or of any knowledge of the published details of his earlier method.

"Improved Method for Preparing Lungs in order to Preserve their Elasticity and Gross Anatomical Appearance.—The lungs must be removed intact, and the pulmonary blood vessels cut as close to the heart as possible. If it is intended to prepare both lungs the trachea should be cut as high as possible. If only one is prepared, cut the bronchus at its junction with the trachea. Insert and tie as large a cannula as possible in the bronchus or trachea, and in the pulmonary artery."

"1. Take of Müller's fluid 1,000 and of acetic acid 75. Fill the lungs through the air passages, and suspend them in solution for twenty-four or forty-eight hours; height of funnel not more than 3 ft. 2. Inject through the artery a mixture of glycerine 4, carbolic acid 1. 3. Dry as well as possible by suction applied to the air passages, then inject through the artery, under pressure of 5 or 6 ft. of water, cotton-seed oil 100, oil of origanum 10. Dry as well as possible, and keep in a crock."

II. Its Physiology.

The "Roosevelt elastic lung" is therefore remarkable for its durability as a whole; but much more so for that of its elastic fibre and of the elastic function in it. A striking contrast is displayed between the pathological history of the fibre in life and its artificial history under preservation in death. As shown by emphysema and by obesity, it is more prone than most living tissues to mechanical destruction by overuse or disuse. As shown by arterial degeneration, and by vascular syphilis, it is also exceedingly liable to destruction by microbes and by phagocytosis. This is doubtless correlated with an extremely low coefficient of nutritional activity and requirement; which is fully demonstrated by its endurance, when it is adequately protected against the scavengers of death.

We know little about it. But we do know that it is the last to die; and that it is also our *ultimum movens*, when, by a final vital recoil, the all-pervading "elastic skeleton of configuration" restores our normal lineaments after their muscular rigidity. That enigma, the last we have approached, might well hold our solution for another problem, not the least important of those yet to be solved—"the mechanics of our biomorphology." If, as it appears, the elastic fibre has in its mechanical keeping our individual polyorganic architecture, might it not conceivably be itself the builder; and, if so, the missing link between our physics and our vital energy? Adami's striking conception of "fibrogenesis" as a linear intraplasmatic

crystallization along each line of movement and of stress is suggestive of a great mechanical antithesis. Our "rigid" skeleton of support is mineralized through "rest"; our "pulsatile" elastic skeleton of organic architecture and nutrition would be the direct mechanical product from the individual. In vital "work," too, might possibly lie the true explanation for our chief clinical puzzle. The extreme diversity in our "normal" individual "sphygmopiesis" might be but an expression of the diversity in our "physical" or elastic coefficient, as the inviolable or irreducible individual factor in our vital "dynamic equation" between the nervo-muscular heart and the neuromyo-elastic artery.

The Croonian Lectures

ON

TRYPANOSOMES CAUSING DISEASE IN MAN AND DOMESTIC ANIMALS IN CENTRAL AFRICA.

DELIVERED BEFORE THE ROYAL COLLEGE OF PHYSICIANS OF LONDON.

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LECTURE IV.

In this concluding lecture I intend to sketch briefly the outstanding features of the trypanosomes belonging to Groups B and C. As far as we are aware, none of the trypanosomes attack man. They are, therefore, of less interest to us than the two species already described.

GROUP B.—THE TRYPANOSOMA PECORUM GROUP.

1. TRYPANOSOMA PECORUM.

The first of this small group, which only consists of two species, is *Trypanosoma pecorum*. It is probably the most important trypanosome disease of domestic animals in Central Africa.

Morphology.

Fig. 1 shows its general appearance. It is the smallest of all the African pathogenic trypanosomes, varying from 9 to 18 microns in length, with an average of 14 microns.

Fig. 1.—*Trypanosoma pecorum*.

Its breadth averages 1.96. This species is monomorphic, and short and stout in form. The contents of the cell are generally homogeneous. The nucleus is oval in shape, and situated about the middle of the body. The micronucleus is small and round, and situated near to, but not at the posterior extremity, and often appears to project beyond the edge of the trypanosome. The undulating membrane is simple, but fairly well developed, and there is no free flagellum.

There ought to be no difficulty in recognizing this species from morphology alone. If one or two hundred are drawn and measured, the average length would separate it from its neighbour *Trypanosoma simiae*. Another small trypanosome, *Trypanosoma uniforme*, differs in shape and in having a free flagellum.

Animals Susceptible to *Trypanosoma pecorum*.

Next in regard to the animals attacked by this trypanosome. This is essentially a disease of the herds. Horses, donkeys, oxen, goats, sheep, and pigs all fall victims.

One peculiarity about *Trypanosoma pecorum* is that it readily loses its virulence for certain animals by passage through certain other animals. For example, if this species—which is usually more or less infective to the monkey, dog, and rat—lives for some time in the blood of

the goat, it loses its power of infecting the former animals. This has given rise to the erroneous idea that a species, *Trypanosoma nanum*, exists. *Trypanosoma nanum* is in truth nothing but a strain of *Trypanosoma pecorum* which has lost its virulence for the other animals by its passage through the goat or allied species.

TABLE I.

Average Duration of Life, in Days, of Various Animals infected by *Trypanosoma pecorum*.

	Average Duration in Days.	No. of Animals Employed.
Donkey ...	87 (?)	1
Cattle ...	121 (?)	4
Goat ...	55	59
Pig ...	51	1
Monkey...	1-7	11
Dog ...	43	57
Guinea-pig	41	5
Rat ...	33	10

Percentage of Recoveries in Various Animals from *Trypanosoma pecorum* Infection.

	Percentages.	No. of Animals Employed.
Donkey ...	80	5
Cattle ...	35	17
Goat ...	12	70
Pig ...	0	1
Monkey...	0	11
Dog ...	1	63
Guinea-pig	0	5
Rat ...	0	10

This trypanosome does not seem to be very fatal to horses, mules, or donkeys. In Nyasaland there was no opportunity of testing it on horses, but out of five donkeys four recovered. Two-thirds of the cattle, and seven-eighths of the goats, succumbed.

The Carrier of *Trypanosoma pecorum*.

The chief carrier of *Trypanosoma pecorum* is *Glossina morsitans*. I am not aware of this trypanosome ever having been found in Nature in *Glossina palpalis*. In Uganda, where we made many feeding experiments with the wild *Glossina palpalis*, in not a single instance did a *Trypanosoma pecorum* infection take place. In Nyasaland, on the other hand, this parasite was the commonest of the trypanosomes with which *Glossina morsitans* was infected, as the following table will show.

TABLE II.—Proportion of Tsetse Flies (*Glossina morsitans*) Naturally Infective with *Trypanosoma pecorum* in Nyasaland.

1912.	No. of Flies Fed.	Mon- key.	Dog.	Goat.	1912.	No. of Flies Fed.	Mon- key.	Dog.	Goat.
Jan. 20	295	—	—	—	May 14	250	—	+	+
" 24	370	—	—	+	" 17	190	—	+	—
" 29	280	—	—	+	" 24	113	—	+	+
Feb. 2	295	—	—	+	" 29	120	—	+	+
" 9	220	—	—	+	" 29	230	—	+	+
" 13	200	+	—	—	" 29	320	—	+	+
" 16	195	—	—	—	" 29	240	+	+	+
" 21	170	—	—	—	" 29	100	—	+	+
" 26	170	—	—	—	" 31	175	—	+	+
Mar. 2	140	—	—	—	June 2	300	—	+	—
" 9	165	—	—	—	" 6	210	+	+	+
" 14	100	—	—	—	" 7	230	—	+	+
" 17	160	—	—	—	" 11	160	—	+	+
" 22	205	—	—	—	" 18	135	—	+	+
April 3	135	—	—	—	" 25	90	—	—	+
" 10	275	—	—	—	July 3	95	—	+	+
" 15	330	—	—	—	Sept. 25	70	+	—	—
" 18	200	—	—	—	" 27	25	—	—	—
" 18	180	—	—	—	Oct. 29	87	—	—	+
" 23	230	—	—	—	Nov. 5	145	—	—	—
" 23	140	+	+	+	" 11	150	—	+	+
" 26	100	—	—	—	" 18	157	—	+	—
" 27	260	—	—	—	" 21	95	—	—	—
May 3	155	+	+	+	" 25	180	—	+	+
" 3	95	—	—	—	Dec. 3	180	+	+	—
" 8	330	—	—	—	" 6	198	—	—	—
" 9	120	—	—	—	" 11	155	—	—	+
" 13	50	—	—	—	" 16	113	+	+	+

It will be seen from Table II that there were 56 experiments, and 10,081 tsetse flies (*Glossina morsitans*) were employed. In the 56 experiments *Trypanosoma pecorum* was found 46 times, more than twice as often as *Trypanosoma brucei*.

Nine monkeys, 34 dogs, and 35 goats were infected.

This gives a proportion of 4.6 per 1,000 flies infective with *Trypanosoma pecorum*. It is therefore abundantly evident that *Glossina morsitans* is a carrier of this trypanosome under natural conditions. But when once this disease has infected a herd there is some circumstantial evidence available to show that the infection may be spread by means of the ordinary cattle or buffalo flies, the Tabanidae.

The ordinary horse-fly of England belongs to this genus, and it is a matter of common experience that these flies give a severe bite or stab, as a rule drawing blood.

These Tabanidae are in their habits and distribution quite different from the tsetse flies. At a particular place for the greater part of the year they may be absent or rare, but at certain times they suddenly appear in enormous numbers, only to disappear again in a few weeks. When swarming in this way they are a veritable pest to cattle. The flies feed mostly at the hottest time of the day, and the cattle to protect themselves crowd together, so as to expose as small a surface of their bodies as possible. If there are any infected animals in the herd the conditions could not be more favourable for mechanical transmission. The infected animals are close beside the healthy, and the flies pass from one to another almost instantaneously.

In Uganda we had an opportunity of studying a sudden epidemic of this disease in a herd of milch cattle. In the course of two months 34 of the cows died out of a herd of 300. The evidence was all against this epidemic having been caused by tsetse flies.

For four months we had as many as 100 fly boys scouring the district for biting flies, but not a single tsetse fly was captured. We therefore came to the conclusion that one or more infected animals having got accidentally into the herd, the infection was spread by one of these swarms of Tabanidae. As soon as the affected animals were removed from the herd the epidemic ceased.

Another example of the same kind occurred among our experimental cattle at Mpumu in Uganda. The Tabanidae, which had been rare, suddenly appeared in swarms. They were first seen in the valley to the west of the hill in September, 1909, and a month later in the valley to the east. Soon after this the cattle, which had shown no signs of disease during the previous year, were found to be suffering from *Trypanosoma pecorum*. Those which grazed in the valley to the west were first affected, and afterwards those which grazed to the east of the hill.

Another writer, George E. Owen, has come to the same conclusion. He states that between 1908 and 1913 some 2,500 to 3,000 head of cattle died from trypanosome disease in the Barotse Reserve of Northern Rhodesia, though this district is free from *Glossina morsitans*. The mortality began each year in February and practically ceased after June. December and March are the months when flies are most numerous.

In 1912 Mr. Owen was sent to investigate, and was able to keep a herd of 800 without loss through 1813. The animals were carefully watched for six months during the season when the flies were scarce, and all the infected or suspected cattle removed before the flies again became numerous.

It may therefore, I think, be concluded with a fair degree of certainty that the trypanosome disease caused by *Trypanosoma pecorum* can be carried from sick to healthy animals by biting flies other than *Glossina*. What happens, probably, is this. One or more oxen previously exposed to the bites of tsetse flies are introduced into the herd living in a tsetse free district, and from these few infected animals the disease is spread by the Tabanidae.

I must apologize for discussing this point at such a length, but it is an important one, and shows how necessary it is that cattle owners living outside tsetse areas should keep strict watch on their herds in order to prevent the presence of trypanosome-infected cattle among them. They should include a microscope in their outfit.

The Cycle of Development of *Trypanosoma pecorum* in *Glossina morsitans*.

This trypanosome belongs to Group B, in which development takes place first in the gut and then passes forward into the labial cavity of the proboscis, and finally reaches the salivary duct or hypopharynx, where the trypanosomes revert to the original blood form and become infective. There is no infection of the salivary glands.

TABLE III.—Development of *T. pecorum* in *G. morsitans*.

Date.	Expt.	No. of Flies Used.	Experiment Positive or Negative.	No. of Infected Flies Found.	No. of Days before Flies became Infective.	Mean Temperature.
1912.						
May 16 ...	546	22	+	4	53	69° F. (20.5° C.).
July 2 ...	524	20	+	2	37	65° F. (18.3° C.).
1913.						
Jan. 3 ...	1732	60	—	0	—	84° F. (28.8° C.).
Jan. 7 ...	1737	40	+	3	19	84° F. (28.8° C.).
Feb. 10 ...	1853	25	+	5	24	84° F. (28.8° C.).
Feb. 24 ...	1950	33	+	6	21	84° F. (28.8° C.).
April 29 ...	2115	40	—	4	—	84° F. (28.8° C.).

Seven experiments were carried out with laboratory-bred flies. Five were positive and two negative.

Two hundred and forty flies were used, and twenty-four infected flies found (10 per cent.). The first two experiments were carried out at the ordinary temperature of the laboratory; in the others the flies were kept in the incubator.

It would appear from these five positive experiments that a period of from nineteen to fifty-three days may elapse before the cycle of development of *Trypanosoma pecorum* in *Glossina morsitans* is complete and the fly becomes infective.

Fig. 2.—*Trypanosoma pecorum* from proboscis.

Result of the Dissection of the Infected Flies.

Table IV gives the result of the dissection of the twenty infected flies found in the positive experiments. The second column gives the number of days between the first feed of the fly and its death and dissection.

TABLE IV.—Result of the Dissection of the Infected Flies. (Laboratory-bred Flies. Positive Experiments.)

Expt.	Time, Days.	Proboscis.		Pro-ventri-culus.	Crop.	Fore-gut.	Mid-gut.	Hind-gut.	Salivary Glands
546	30	—	—	—	—	++	+	+	—
546	64	—	—	—	—	++	++	++	—
546	84	—	—	—	—	++	++	++	—
546	84	++	—	++	++	++	++	++	—
524	27	—	—	—	—	++	++	++	—
524	55	+	—	+	—	++	++	++	—
		Labial cavity.	Hypo-pharynx						
1737	27	++	++	++	++	++	++	++	—
1737	28	+	—	+	—	++	++	+	—
1737	30	++	++	++	—	++	++	++	—
1853	17	++	++	++	—	++	++	++	—
1853	22	—	—	—	—	+	++	+	—
1853	23	+	—	+	—	+	++	++	—
1853	25	++	++	+	—	++	++	++	—
1853	26	++	++	+	—	++	++	++	—
1950	17	—	—	+	—	++	++	++	—
1950	19	++	++	—	—	++	++	++	—
1950	24	—	—	—	—	+	+	—	—
1950	26	++	++	+	—	++	++	++	—
1950	31	++	++	+	—	++	++	++	—
1950	31	++	++	++	—	++	++	++	—

TABLE V.—Showing the Result of the Dissection of the Infected Flies in the Negative Experiments. (Laboratory-bred Flies.)

Expt.	Time, Days.	Proboscis.		Pro-ventri-culus.	Crop.	Fore-gut.	Mid-gut.	Hind-gut.	Salivary Glands
		Labial Cavity.	Hypo-pharynx.						
2115	9	—	—	—	—	—	+	—	—
2115	9	—	—	—	—	—	++	—	—
2115	9	+	—	+	—	+	++	++	—
2115	11	—	—	+	—	+	++	+	—

In Experiments 546 and 524 there was no separate examination of the hypopharynx; it is included in the general term "proboscis." It was only when the importance of the hypopharynx became evident that an

Labrum: early infection.

Labrum. Hypopharynx.
Fig. 3.—*Trypanosoma pecorum*: Development in proboscis.

examination of the separate parts of the proboscis was made. In not a single fly was any invasion of the salivary glands noted, but it will be seen that in every positive experiment trypanosomes are found in the hypopharynx.

So much for the positive experiments; let us see what is the result of the dissection of the infected flies in the negative experiments. In none of the infected flies had development reached the hypopharynx, and consequently none of the flies had become infective.

From the consideration of these tables it will be seen that in *Trypanosoma pecorum* the development takes place at first in the intestine, then passes forward into the labial cavity, and finally invades the hypopharynx, and there is completed.

The Type of Trypanosomes found in the Infected Flies.

Fig. 2 represents the developmental forms of *Trypanosoma pecorum* found in the labial cavity of *Glossina morsitans*.

In regard to the forms found in the intestine, it may be said that these are indistinguishable from the developmental forms of other pathogenic trypanosomes.

The first seven figures represent early forms in the labial cavity. These were seen adhering singly by their flagella to the labrum.

The next group are the ordinary forms found clinging by their flagella ends to the labrum. It will be seen that they have assumed the crithidial stage—a stage which seems to be a *sine qua non* in the final stages of the cycle of development of all the pathogenic trypanosomes, and the interpretation of which is still obscure.

The small blood forms are from the hypopharynx of dead infective flies, and also from living infective flies induced to salivate on a cover-glass. They represent the final stage in the cycle of development, and are the only infective forms.

In Fig. 3 are seen drawings, at a magnification of 250 diameters, of the labrum and hypopharynx of a fly infected with this trypanosome. While the labial cavity

is seen to contain clusters of large ribbon-like trypanosomes, the hypopharynx is swarming with the small infective forms. From these drawings the ease and facility with which a tsetse fly can infect an animal will be readily understood.

Another way of showing how easy it is for a fly to infect an animal is to induce an infected fly to salivate on a cover-glass. If a hungry fly is placed on a large cover-glass, which covers a man's finger, the fly will attempt to bite through the glass, and in so doing will smear it with its saliva. If this is then stained and examined under a microscope, thousands of trypanosomes will be seen.

The Reservoir of *Trypanosoma pecorum*.

Table VI represents the number of times *Trypanosoma pecorum* was found among the 180 wild animals examined in Nyasaland and the species of game which harboured it.

TABLE VI.—Showing the Species of Animals Dealt with, the Total Number Examined, the Number found Infected, and the Species of *Trypanosoma* by which they were Infected.

Animal.	No. Examined.	No. Found Infected.	<i>Tryp. brucei</i> .	<i>Tryp. pecorum</i> .	<i>Tryp. simiae</i> .	<i>Tryp. caprae</i> .
Eland ...	10	6	0	6	0	1
Sable ...	5	0	0	0	0	0
Waterbuck ...	13	9	3	1	0	8
Koodoo ...	3	2	0	2	0	0
Bushbuck ...	10	7	0	7	0	1
Hartebeeste ...	35	6	5	1	0	0
Reedbuck ...	19	12	3	1	0	9
Orobi ...	26	4	1	1	0	1
Duiker ...	7	2	1	0	0	0
Buffalo ...	9	2	0	2	0	0
Lion ...	1	0	0	0	0	0
Hyaena ...	3	2	0	2	0	0
Warthog ...	33	7	1	3	3	0
Elephant ...	2	0	0	0	0	0
Wild cat ...	3	0	0	0	0	0
Porcupine ...	1	0	0	0	0	0
Total ...	180	59	14	26	3	20

It will be seen that 26 animals among the 180 harboured this parasite (14.4 per cent.), and the eland, the koodoo, the bushbuck, and the buffalo were the greatest sinners; 60 per cent. of the eland, 66 per cent. of the koodoo, 70 per cent. of the bushbuck, and 22 per cent. of the buffalo having *Trypanosoma pecorum* in their blood.

It is rather curious that *Trypanosoma pecorum* has picked out the animals in whose blood *Trypanosoma brucei* was absent.

2. TRYPANOSOMA SIMIAE.

This species of trypanosome is remarkable for the virulence it displays towards the monkey and the domestic pig, killing these animals in an incredibly short period of time, whereas it is harmless to oxen, antelope, dogs, and the smaller experimental animals. Curiously enough it affects goats and sheep, although oxen and antelope escape.

Trypanosoma simiae is similar to *Trypanosoma pecorum* in the rapidity with which its virulence becomes modified. If a cage containing wild tsetse flies (*Glossina morsitans*) infected with this parasite is placed on a monkey or a goat, both animals take the disease and the monkey in such an acute form that the average duration of disease is only a few days.

But if the attempt is made to pass this species of trypanosome from an infected goat to a healthy monkey, by the inoculation of the goat's blood, the experiment usually fails, showing that a short sojourn in the blood of the goat has almost nullified the virulence of the parasite for the monkey.

Another interesting point in regard to this species is, that as far as is known, the warthog is the only animal among the wild game which harbours it. It is probable

that it will also be found in the blood of the bush-pig, but that has not been done yet.

Morphology.

If Fig. 4 be compared with Fig. 1, it will be seen that *Trypanosoma simiae* is longer than *Trypanosoma pecorum*. It is monomorphic, varying from 14 to 24 microns in length—average 18. The trypanosomes are fairly uniform in shape. The body is elongated, undulating, and frequently extends in a straight line. The contents of the cell are clear and free from granules. The nucleus is oval,

Fig. 4.—*Trypanosoma simiae*.

and situated about the middle of the body. The micro-nucleus is small and round, situated almost invariably about $1\frac{1}{2}$ microns from the posterior extremity. The undulating membrane is well developed and thrown into bold undulations.

It is difficult to say whether this species has a free flagellum or not. By careful staining and good illumination it would seem in most cases as if the undulating membrane extended to the tip of the flagellum. In some preparations, however, the last two or three microns of the flagella appear to be free.

Susceptibility of Animals.

Table VII shows the average duration of life in various animals infected by *Trypanosoma simiae*, also the percentages of recoveries from the disease.

TABLE VII.

Average Duration of Life in Various Animals infected by *T. simiae*, Nyasaland. Mixed Infections are not included. (The letter R stands for refractory.)

	Average Duration in Days.	No. of Animals Employed.
Ox ...	R	4
Antelope ...	R	5
Goat and Sheep ...	46.6	5
Pig ...	5.3	9
Baboon ...	R	3
Monkey ...	10.8	24
Dog ...	R	21
Rabbit ...	R	10
Guinea-pig ...	R	5
Rat ...	R	5

Percentages of Recoveries in Various Animals from *T. simiae* Infection. Mixed infections are included.

	Percentages.	No. of Animals Employed.
Ox ...	R	4
Antelope ...	R	5
Goat and Sheep ...	37.5	32
Pig ...	0.0	13
Baboon ...	R	3
Monkey ...	14.3	35
Dog ...	R	21
Rabbit ...	R	10
Guinea-pig ...	R	5
Rat ...	R	5

It will be seen that the ox, antelope, baboon, dog, rabbit, guinea-pig, and rat are refractory.

In the whole range of the trypanosome diseases of animals there is surely nothing so striking as the rapidly fatal action of *Trypanosoma simiae* on the domestic pig. In nine experiments the average duration was only 5.3 days. This, not from the time of the appearance of the trypanosome in the blood, but from the date of the infection. Further, this rapid action is not the result of an exaltation of virulence by numerous passages through the pig, but natural to the trypanosome.

The Carrier of *Trypanosoma simiae*.

The carrier of *Trypanosoma simiae* in Nyasaland is *Glossina morsitans*. Table VIII gives the proportion of flies naturally infected with this parasite.

TABLE VIII.—Proportion of Tsetse Flies (*Glossina morsitans*) Naturally Infective with *Trypanosoma simiae* in Nyasaland.

1912.	No. of Flies Fed.	Mon-key.	Dog.	Goat.	1912.	No. of Flies Fed.	Mon-key.	Dog.	Goat.
Jan. 20	296	+	—	—	May 14	250	+	—	+
" 24	370	++	—	—	" 17	190	—	—	+
" 29	280	++	+	—	" 24	113	—	—	+
Feb. 2	295	++	+	—	" 29	120	—	—	—
" 9	220	++	+	—	" 29	230	—	—	—
" 13	200	+	—	—	" 29	320	—	—	—
" 16	195	—	—	—	" 29	240	—	—	—
" 21	170	—	—	—	" 29	100	—	—	+
" 26	170	—	+	—	" 31	175	+	—	+
Mar. 2	140	—	—	—	June 2	300	—	—	+
" 9	165	—	—	—	" 6	210	—	—	—
" 14	100	—	—	—	" 7	230	—	—	—
" 17	160	—	—	—	" 11	160	+	—	—
" 22	205	—	+	—	" 18	135	—	—	—
April 3	135	—	—	—	" 25	90	—	—	—
" 10	275	+	—	—	July 3	95	—	—	—
" 15	330	+	—	+	Sept. 25	70	—	—	—
" 18	200	++	—	—	" 27	25	—	—	—
" 18	180	++	+	—	Oct. 29	87	—	—	—
" 23	230	++	—	—	Nov. 5	145	—	—	—
" 23	140	—	—	—	" 11	150	+	—	—
" 26	100	+	—	+	" 18	157	+	—	+
" 27	260	++	—	—	" 21	95	—	—	—
May 3	155	+	—	—	" 25	180	+	—	—
" 3	96	—	—	—	Dec. 3	180	+	—	—
" 8	330	+	—	—	" 6	198	+	—	—
" 9	120	+	—	—	" 11	156	+	—	—
" 13	50	—	—	+	" 16	113	+	—	—

As will be seen from this table, there were 56 experiments, and 10,081 tsetse flies were employed. In the 56 experiments *Trypanosoma simiae* was found 34 times (60.7 per cent.). Twenty-six monkeys and seventeen goats were infected. This gives a proportion of 3.4 per 1,000 flies infective with *Trypanosoma simiae*. It must be noted that warthogs were numerous in the sleeping sickness area, Nyasaland, which accounts for the fairly high proportion of infected flies.

The Cycle of Development of *Trypanosoma simiae* in *Glossina morsitans*.

This species belongs to Group B, and the cycle of development is similar to that which has already been described as occurring in *Trypanosoma pecorum*.

The Reservoir of *Trypanosoma simiae*.

In Table VI is shown the number of times *Trypanosoma simiae* was found among the 180 wild animals examined in Nyasaland, and the species of game which harboured it.

It will be seen that only 3 animals among the 180 harboured this parasite (1.7 per cent.). This was, of course, due to the fact that it is only the warthog among the wild game which acts as a reservoir; 30 warthogs were examined and 3 found infected (9 per cent.).

GROUP C.—THE TRYPANOSOMA VIVAX GROUP.

The three species forming this group have a strong family resemblance, and but for size might almost be included in one species.

1. TRYPANOSOMA VIVAX.

This is the cause of one of the most important diseases of cattle in Uganda. We did not meet with it in Nyasaland, where its place seems to be taken by *Trypanosoma caprae*. It is, however, widely distributed in Central Africa. It has been reported from Senegal and the Sudan

Fig. 5.—*Trypanosoma vivax*, Ziemann.

in the north to Rhodesia in the south. It is easily recognized on account of its extreme activity during life, its characteristic morphology in stained specimens, and the fact that it only affects horses, cattle, goats, and sheep, while monkeys, dogs, rabbits, guinea-pigs, rats, and mice are refractory.

In Uganda the tsetse flies on the lake shore were found to be infected with it, and it was also found in the blood

of a bushbuck shot at the same place at which the flies were collected.

2. TRYPANOSOMA UNIFORME.

This trypanosome resembles *Trypanosoma vivax* very closely except that it is smaller. Up to the present it has

Fig. 6.—*Trypanosoma uniforme*.

only been found in Uganda. Its carrier there is *Glossina palpalis*, and its reservoir the wild game on the lake shore.

3. TRYPANOSOMA CAPRAE.

This species has only been reported up to the present from Lake Tanganyika and Nyasaland. It, like the other two species belonging to this group, only affects cattle, sheep, and goats. Monkeys, dogs, and the smaller experimental animals are immune.

Morphology.

Fig. 7 shows its general appearance. During life this trypanosome, like the other members of the group, is characterized by its extreme mobility.

Fig. 7.—*Trypanosoma caprae*, Kleine.

It is a monomorphic species, varying from 18 to 32 microns in length, the greatest number of individuals being 25 microns long. Measured across the broadest part, *Trypanosoma caprae* averages 3 microns in breadth (maximum 4.25, minimum 1.75). It differs from *Trypanosoma vivax* in that it is heavier built and altogether has a larger and clumsier appearance. The posterior half is swollen, and its end is bluntly angular or rounded. The anterior extremity is narrower and pointed. The contents of the cell are clear, with a delicate alveolar structure and free from vacuoles and granules. The nucleus is oval, compact, and lying about the middle of the body. The micronucleus is large and round, situated as a rule close to the posterior extremity; the undulating membrane is more developed than in *Trypanosoma vivax* and thrown into bolder folds and undulations. There is a well-marked free flagellum which averages 6.5 microns in length (maximum 9.4, minimum 4).

Susceptibility of Animals to *Trypanosoma caprae*.

Table IX gives the average duration of the disease, and the percentages of recoveries in various experimental animals.

TABLE IX.

Average Duration of Life in Various Animals infected by *T. caprae*, Nyasaland. (The letter R stands for refractory.)

	Average Duration in Days.	No. of Animals Employed.
Ox ...	...	...
Goat ...	56.5	19
Sheep ...	115.3	3
Monkey ...	R	12
Dog ...	R	12
Guinea-pig ...	R	2
Rat ...	R	2

Percentage of Recoveries in Various Animals from *T. caprae* infection.

	Percentages.	No. of Animals Employed.
Ox ...	100	2
Goat ...	32.2	28
Sheep ...	25.0	4
Monkey ...	R	12
Dog ...	R	12
Guinea-pig ...	R	2
Rat ...	R	2

The Carrier of Trypanosoma caprae.

The carrier of *Trypanosoma caprae* in Nyasaland is *Glossina morsitans*.

Table X shows the proportion of tsetse flies naturally infected with this parasite in Nyasaland. In 56 experiments, in which 10,081 tsetse flies were used, *Trypanosoma caprae* was found thirty-five times (62.5 per cent.) Since

TABLE X.—Proportion of Tsetse Flies (*Glossina morsitans*) Naturally Infective with *Trypanosoma caprae* in Nyasaland.

1912.	No. of Flies Fed.	Mon-key.	Dog.	Goat.	1912.	No. of Flies Fed.	Mon-key.	Dog.	Goat.
Jan. 20	296	—	—	—	May 14	250	—	—	+
" 24	370	—	—	+	" 17	190	—	—	+
" 29	280	—	—	—	" 24	113	—	—	+
Feb. 2	255	—	—	+	" 29	120	—	—	—
" 9	220	—	—	+	" 29	230	—	—	—
" 13	200	—	—	+	" 29	320	—	—	+
" 16	195	—	—	—	" 29	240	—	—	+
" 21	170	—	—	—	" 29	100	—	—	+
" 26	170	—	—	—	" 31	175	—	—	—
Mar. 2	140	—	—	+	June 2	300	—	—	+
" 9	165	—	—	—	" 6	210	—	—	+
" 14	100	—	—	+	" 7	230	—	—	—
" 17	160	—	—	—	" 11	160	—	—	+
" 22	205	—	—	+	" 18	135	—	—	—
Apr. 3	135	—	—	—	" 25	90	—	—	—
" 10	275	—	—	+	July 3	95	—	—	—
" 15	330	—	—	—	Sept. 25	70	—	—	—
" 18	200	—	—	+	" 27	25	—	—	—
" 18	180	—	—	—	Oct. 29	87	—	—	+
" 23	230	—	—	+	Nov. 5	145	—	—	+
" 23	140	—	—	—	" 11	150	—	—	+
" 26	100	—	—	—	" 18	157	—	—	+
" 27	260	—	—	+	" 21	95	—	—	—
May 3	155	—	—	—	" 25	180	—	—	—
" 3	96	—	—	—	Dec. 3	180	—	—	+
" 8	330	—	—	+	" 6	198	—	—	+
" 9	120	—	—	—	" 11	156	—	—	—
" 13	50	—	—	+	" 16	113	—	—	—

this species is not pathogenic to the monkey or dog, of course it was found only in the experimental goats. This gives a proportion of 3.5 per 1,000 flies infected with *Trypanosoma caprae*.

The Cycle of Development of Trypanosoma caprae in Glossina morsitans.

Trypanosoma caprae belongs to the *Vivax* group, in which the development of the trypanosome is restricted to the proboscis.

TABLE XI.—Development of *T. caprae* in *G. morsitans*.

Date.	Expt.	No. of Flies Used.	Experiment Positive or Negative.	No. of Infected Flies Found.	No. of Days before Flies became Infective.	Mean Temperature.
1912.						
April 16...	444	12	+	1	16	71° F. (22.1° C.).
June 3 ..	617	33	—	0	—	65° F. (18.3° C.).
June 3 ...	1215	22	+	1	21	65° F. (18.3° C.).
1913.						
Jan. 18 ...	1777	35	+	11	19	84° F. (28.8° C.).
Jan. 22 ...	1784	35	+	20	19	84° F. (28.8° C.).
April 1	2046	33	+	13	20	84° F. (28.8° C.).

Six experiments were made with laboratory-bred flies; 5 were positive and 1 negative.

One hundred and seventy laboratory bred flies were used and 46 infected flies were found (27.1 per cent). The first three experiments were carried out at the ordinary temperature of the laboratory; in the last three the cages containing the flies were kept in an incubator. It is difficult to understand the difference in the number of infected flies found. In Experiments 444 and 1215 only 8 and 5 per cent. respectively of the flies became infected, whereas in the last three experiments an average of more than 40 per cent. was found. The flies in the second group were kept, it is true, at a temperature similar to that which they would find in summer in their native haunts in the low country, while the first three experiments were done in winter, and at the ordinary temperature of the laboratory. This would no doubt explain the difference to some extent. Again, goats and sheep infected with *Trypanosoma caprae* are unsatisfactory animals to feed flies on. One day the

trypanosomes are present in small numbers in the blood, the next day it may be impossible to find any; very seldom are they in large numbers. It is possible, then, that flies may feed on an infected goat or sheep without taking in a single trypanosome.

It would appear from the five positive experiments that an average period of nineteen days elapses before the cycle of development of this parasite is complete in *Glossina morsitans*, and the fly becomes infective.

Now 46 infected flies were found among 170 used, and the next table gives the result of the dissection of these flies.

TABLE XII.—Result of the Dissection of the Infected Flies.

Expt.	Time, Days.	Proboscis.	Pro-ventri-culus.	Crop.	Fore-gut.	Mid-gut.	Hind-gut.	Salivary Glands.
444	25	+	—	—	—	—	—	—
1215	32	+	—	—	—	—	—	—
1777	21	+	—	—	—	—	—	—
1777	26	+	—	—	—	—	—	—
1777	30	+	—	—	—	—	—	—
1777	30	+	—	—	—	—	—	—
1777	30	+	—	—	—	—	—	—
1777	30	+	—	—	—	—	—	—
1777	30	+	—	—	—	—	—	—
1777	30	+	—	—	—	—	—	—
1777	30	+	—	—	—	—	—	—
1777	30	+	—	—	—	—	—	—
1777	30	+	—	—	—	—	—	—
1777	30	+	—	—	—	—	—	—
1784	19	+	—	—	—	—	—	—
1784	21	+	—	—	—	—	—	—
1784	23	+	—	—	—	—	—	—
1784	21	+	—	—	—	—	—	—
1784	29	+	—	—	—	—	—	—
1784	29	+	—	—	—	—	—	—
1784	29	+	—	—	—	—	—	—
1784	29	+	—	—	—	—	—	—
1784	29	+	—	—	—	—	—	—
1784	29	+	—	—	—	—	—	—
1784	29	+	—	—	—	—	—	—
1784	30	+	—	—	—	—	—	—
1784	30	+	—	—	—	—	—	—
1784	30	+	—	—	—	—	—	—
1784	30	+	—	—	—	—	—	—
1784	30	+	—	—	—	—	—	—
1784	30	+	—	—	—	—	—	—
1784	30	+	—	—	—	—	—	—
1784	30	+	—	—	—	—	—	—
1784	30	+	—	—	—	—	—	—
2046	23	+	+	—	—	—	—	—
2046	23	+	—	—	—	—	—	—
2046	24	+	+	—	—	—	—	—
2046	26	++	++	—	—	—	—	—
2046	28	+	—	—	—	—	—	—
2046	28	+	—	—	—	—	—	—
2046	28	++	++	—	—	—	—	—
2046	29	+	++	—	—	—	—	—
2046	29	+	++	—	—	—	—	—
2046	29	+	++	—	—	—	—	—
2046	29	++	++	—	—	—	—	—
2046	29	++	+	—	—	—	—	—
2046	30	+	++	—	—	—	—	—

It will be seen from Table XII that it was not until the last experiment that the labial cavity and hypopharynx were examined separately. In the previous experiments the presence or absence of trypanosomes in the proboscis as a whole was noted.

In the first two experiments only a single infected fly was found in each. In Experiment 1777 eleven, and in 1784 as many as twenty were found.

In regard to the number of trypanosomes in the labial cavity this may vary greatly. Sometimes the lumen of the tube will be seen to be densely crowded; at other times a single colony will be seen. For example, in Experiment 1777 the first infected fly, dissected on the twenty-first day, is noted to have had the lumen of the proboscis swarming with clusters of torpedo-shaped flagellates attached to the labrum by their flagellar ends, a few swimming free. In the seventh infected fly, dissected on the thirtieth day, only three colonies are noted. In the same way, at one time the hypopharynx may contain few, at another time it is seen to be densely packed with swarms of actively moving trypanosomes. In unstained specimens the difference in size and shape between the trypanosomes in the labial cavity and those in the hypopharynx is quite manifest.

From this table the broad facts stand out boldly—that in this group of trypanosomes the development is confined to the labial cavity and hypopharynx, and does not take place in any other part of the fly.

The Type of Trypanosomes found in the Infected Flies.

No attempt was made by the Commission to study the development of *Trypanosoma caprae* in *Glossina morsitans* in the earliest stages. This can only be done if a large number of laboratory-bred flies are available, and this was not the case in Nyasaland.

In Figs. 8 and 9 are represented some of the developmental forms found in the labial cavity and hypopharynx of infected flies. It will be seen that most of the flagellates found attached to the labrum are crithidial in type, with well defined nuclei, micro-nuclei, and free flagella. Some are ribbon shaped, others have elongated posterior extremities, and again others are torpedo-shaped.

The last three rows are from the hypopharynx, and have been obtained, as a rule, by causing the fly to salivate on the cover-glass. They represent the final stage in the cycle of development, the reversion to the infective or blood form. They are rather smaller than those found in the blood of the vertebrate host, but resemble them closely in every other way.

Fig. 8.—*Trypanosoma caprae* from proboscis.

The Reservoir of Trypanosoma caprae.

Among the 180 wild animals examined in Nyasaland 20 harboured *Trypanosoma caprae* (11.1 per cent.); 61 per cent. of the waterbuck and 47 per cent. of the reedbuck acted as reservoirs.

CONCLUSION.

This concludes the Croonian Lectures on the trypanosomes causing disease in man and animals in Central Africa. These lectures deal with but a small part of the subject, which has in the course of the last twenty years grown to huge proportions. Nothing has been said about medicinal treatment, and even measures of prevention have been left a good deal to the imagination. Taking a look back over the whole field, the outstanding features

may be said to be, first, that some order is beginning to reign in what was lately chaos in regard to the classification of the pathogenic trypanosomes. They may all now be referred to three groups, and nine species.

In regard to the transference of the virus from sick to healthy animals by the fly, this has been made clearer and easier of comprehension by the discovery of the part the salivary glands and hypopharynx play in the various modes of development which the trypanosomes undergo in the fly. It results that it would almost appear impossible for an infective fly to pierce even momentarily

the skin of a healthy susceptible animal without causing infection.

Another important feature is the proof brought forward that *Trypanosoma brucei* and *Trypanosoma rhodesiense* are the same.

Finally, in regard to the prevention of these trypanosome diseases of man and domestic animals. We have seen that the wild game in the fly country is heavily infected. It is impossible to doubt that they are the reservoir and source of many of these diseases. There can be little

Fig. 9.—*Trypanosoma caprae*: Development in proboscis.

doubt that if the wild game were driven out of the fly country, trypanosome diseases such as those caused by *Trypanosoma brucei* and *Trypanosoma pecorum* would disappear.

In regard to the measures of prevention against the most important of all the trypanosome diseases—Congo sleeping sickness—it has been shown by experience that the removal of the natives from the fly area is a simple and efficacious way of stopping an epidemic.

In these sparsely inhabited countries where spare land and food are easily obtained, there is as a rule no difficulty in effecting this migration.

If it is desired to go a step further and render the sleeping sickness area inhabitable, then clearing and cultivation must be resorted to. By these means in all probability *Glossina palpalis* will be driven away, and with it the disease.

Memoranda:

MEDICAL, SURGICAL, OBSTETRICAL.

TREATMENT OF RIGID OS UTERI.

I HAVE read with interest Dr. John A. Irvine's communication on this subject in the JOURNAL of June 12th, p. 1002. The administration of tartar emetic successfully rectified the uterine complication. The effects of antimony, however, are far from pleasant to the patient, and liable to cause dangerous depression. There is, in my opinion, a better way by which the relaxation of the rigid os uteri may be effected *cito, tuto, et jucunde*.

As I have pointed out in my book *Clinical Memoranda* (Baillière and Co.), the application of a tampon of lint or absorbent cotton-wool, soaked in a solution of cocaine, or β eucaine (10 grains to the ounce of boric acid solution), to, or into, the os and cervix, acts like a charm.

The irritated nerves and muscular fibres are anaesthetized, and the spasm relaxes directly—even within five minutes—the os dilating widely. On many occasions I

have had cause to bless this precious drug which has never failed me.

Not only does the cocaine relieve the spasm of the os, but, as the solution spreads all over the vagina, it also anaesthetizes the mucous membrane, permitting the head to pass through the outlet with a minimum of discomfort.

Driffield, E. Yorks.

A. T. BRAND, M.D.

RECURRENT VESICULAR RASH.

I HAVE at present a young married woman under my care who has a blister which appeared spontaneously over the lower end of the ulna. About nine months ago a somewhat smaller blister arose over the big toe. I then thought a shoe must have wrung her foot. She had a patch of zoster over the glutei in the winter, and when the ulna blister appeared there was a fresh patch of zoster. All these manifestations were on the left side. She is quite healthy otherwise except for slight grating in some joints and slight occasional asthma. I regard the whole as a manifestation of fibrositis.

Bournemouth.

GEORGE MAZARD.