

that the recovery of ammonia is not likely to be profitable unless the nitrogen content exceeds  $1\frac{1}{2}$  per cent. calculated upon absolutely dry peat. Finally, he shows that under favourable conditions power can be generated from certain of these Canadian peat bogs at a cost equal to or below that at which it can be obtained by the utilisation of water-power. A little attention is also devoted to the question of the utilisation of the lignites of certain of the Western Provinces, where true bituminous coal is not obtainable locally, and it is shown that in certain circumstances it too can be employed profitably in the generation of gas.

The second report is intended to aid, not only the mining industry, but also the very large number of manufacturing industries that depend to a greater or lesser extent upon an adequate supply of raw materials in the shape of mineral products. The report deals with a very large number of miscellaneous minerals, of which asbestos, barytes, clay, lime, and sand are perhaps the most important, and it should be noted that such minerals as are used in Canadian manufactures but are not produced in Canada are referred to, as well as the minerals of domestic production. It is noteworthy that quite a considerable quantity of minerals is imported, although they could be produced in the Dominion, and one of the main objects of this report is to bring actual or possible producers and consumers into closer touch with one another. The object is an excellent one, and such reports as this should prove of the greatest value to both parties and should help towards that very desirable object, the industrial independence of this great Dominion.

The report of the Chief Inspector of Mines of the State of Mysore for the year 1913 has just been issued. Apart from the statistical portion, which shows that the value of the bullion produced during the year in question was 2,150,193*l.*, a decrease of 0.37 per cent. from the previous year, the chief general interest in this report is to be found in a careful investigation of a shaft accident at the Mysore Gold Mine. It appears that the steel pin, which secured a driving clutch, that connected the engine shaft and the winding reel suddenly broke, allowing the cage, in which forty-two miners were travelling, to fall to the bottom of the shaft. An investigation was held into the cause of the fracture of this pin and into the reason why the powerful brake attached to the winding engine did not hold the reel, and the report of the committee of inquiry is now given. It cannot be said that the cause of the fracture is satisfactorily explained, but the insufficiency of the braking arrangements is very clearly demonstrated. Having regard to the fact that this brake is of the construction that is in general use on winding engines in all parts of the world, this report deserves the careful attention of all who have to do with winding from deep shafts by means of the powerful winding engines that are in general use in modern mines; in particular it may be noted that the brake appears to have complied fully with the provisions of our Coal Mines Act, and yet was found inadequate to prevent the very serious accident in question.

Another interesting section in this report deals with accidents due to "air blasts," which caused no fewer than thirty-one deaths during the year in question. These air-blasts consist in the sudden flying-off of huge masses of rock from the walls of stoped-out portions of the deposit, the action being extremely violent and suggesting that the rock is under some condition of great strain that suddenly relieves itself. The phenomenon is as yet but little understood, and all measures taken for combating this danger are still of a more or less tentative character.

### TRYPANOSOMES CAUSING DISEASE IN MAN AND DOMESTIC ANIMALS IN CENTRAL AFRICA.<sup>1</sup>

THESE lectures are confined to a consideration of the trypanosomes causing disease in man and domestic animals in Central and Southern Africa. The conditions, however, which obtain on the east and west coasts of Africa between 20° N. and 30° S. latitude are much the same as those which are found in the central parts, and it is probable that the same trypanosome species are found throughout. So that in describing the species found in our own colonies it may be assumed that all the important pathological species found in Central Africa are being dealt with, although in other places they may be known by other names.

The central region—the tropical or equatorial—corresponds with the distribution of the tsetse-flies, and the trypanosomes causing disease in this region are carried from sick to healthy animals by various species of this genus of flies. In the north of Africa, outside the range of the tsetse-flies, two trypanosome diseases are found, one of the horse (dourine), and another of camels (surra), the former conveyed from sick to healthy horses by contagion, the latter almost certainly by large biting flies, the so-called horse-flies, or tabanidæ.

#### CLASSIFICATION OF THE AFRICAN TRYPANOSOMES.

The three characters mainly relied upon in this classification of trypanosomes are, in the first place, their morphology; secondly, their pathogenic action on animals; and, thirdly, their mode of development in the tsetse-flies. They may be divided into three groups, and these are set out in the following scheme:—

##### Group A. *Trypanosoma Brucei* Group.

1. *Trypanosoma brucei*.
2. *Trypanosoma gambiense*.
3. *Trypanosoma evansi*.
4. *Trypanosoma equiperdum*.

##### Group B. *Trypanosoma Pecorum* Group.

1. *Trypanosoma pecorum*.
2. *Trypanosoma simiæ*.

##### Group C. *Trypanosoma Vivax* Group.

1. *Trypanosoma vivax*.
2. *Trypanosoma capræ*.
3. *Trypanosoma uniforme*.

These names probably represent most of the principal pathogenic trypanosomes discovered up to the present time in Africa. The northern species, *Trypanosoma evansi* and *T. equiperdum*, are placed in the first group, as they seem by morphology and their action on animals to belong there. Each group is distinguishable or separable by well-defined characters.

*Group A. The Trypanosoma brucei* Group.—The species forming this group (Fig. 1) are all more or less polymorphic, varying in size and shape from short and stumpy forms without free flagella to long and slender forms with free flagella. The cytoplasm contains numerous dark-staining granules. The micronucleus or kinetonucleus is small, and is situated as a rule some distance from the posterior extremity. The undulating membrane is well developed and thrown into bold folds.

<sup>1</sup> Abridged from the Croonian Lectures delivered before the Royal College of Physicians of London on June 17, 22, 24, and 29, by Sir David Bruce, C.B., F.R.S.



In regard to their action on animals, the members of this group may be said generally to affect many different species of animals—as, for example, man, horses, cattle, dogs, and most of the smaller experimental animals. The two Central African members of the group, *T. brucei* and *T. gambiense*, develop in the tsetse-flies in the same way. At first the development takes place in the intestine; afterwards the parasites pass into the salivary glands, by way probably of the proboscis, and there complete their develop-

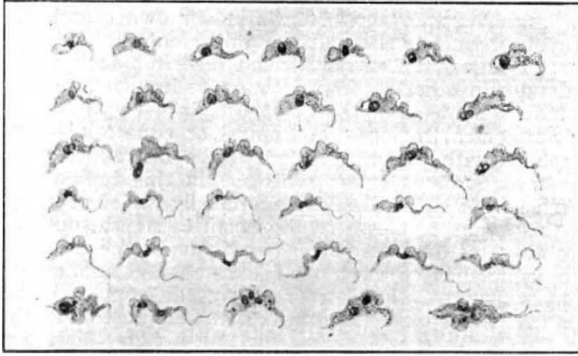


FIG. 1.—*Trypanosoma brucei* (Plimmer and Bradford) Zululand, 1913.  $\times$  about 700.

ment into infective forms. This is the only group in which the salivary glands are invaded. This group can be separated from the other groups by shape alone.

**Group B. The *Trypanosoma pecorum* Group.**—The trypanosomes are small and monomorphic. The cytoplasm is non-granular. The micronucleus is prominent, subterminal, and often seems to project beyond the margin. The undulating membrane is fairly well developed (Fig. 2).

The cycle of development in the tsetse-fly in Group B begins in the intestinal tract; afterwards the flagellates pass forward into the proboscis of the fly, and finally reach the salivary duct or hypopharynx, where they complete their development and become infective. The difference between Group A and Group B is that

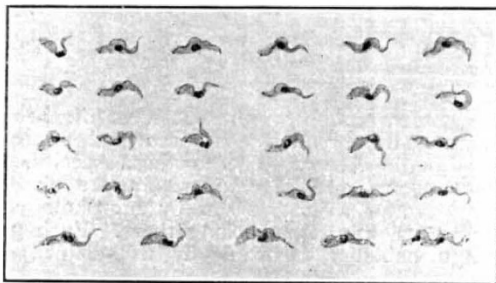


FIG. 2.—*Trypanosoma pecorum*.  $\times$  about 700.

in the latter the salivary glands are never invaded. There are only two species at present included in this group—*T. pecorum* and *T. simiae*. The former gives rise to the most important trypanosome disease of cattle in Africa, while the latter is remarkable for the rapidity with which it kills the domestic pig.

**Group C. The *Trypanosoma vivax* Group.**—The species making up this group (Fig. 3) are monomorphic, and remarkable for the extreme rapidity of their movements. The posterior extremity is enlarged. The cytoplasm is clear and hyaline. The

micronucleus is large and terminal, and the undulating membrane is little developed and simple. This species only affects horses, cattle, goats, and sheep. Monkeys, dogs, rabbits, guinea-pigs, and rats are refractory.

The cycle of development in Group C differs from that in Groups A and B in that it takes place at first only in the labial cavity of the proboscis, and later in the salivary duct or hypopharynx. No part of the cycle takes place in the intestinal tract or in the salivary glands.

These three groups are well marked, and it is fairly easy by microscopic examination alone to name what group a trypanosome belongs to, when seen in the blood of the vertebrate host or even in the tsetse-fly.

#### DESCRIPTION OF THE TSETSE-FLIES.

A description of *Glossina morsitans* and *Glossina palpalis* is given, with a figure illustrating the mouth parts of a tsetse-fly.

It is important to understand the structure of the proboscis, as this plays an important part in the development of Groups B and C. In the transverse section the parts are seen in position, the labrum and labium joined together form a tube through which the blood is drawn in the act of sucking, and known as the labial cavity; and the delicate terminal duct of the salivary glands or hypopharynx lying in the hollow of the labium, and opening near the tip of the proboscis. The salivary glands are long convoluted

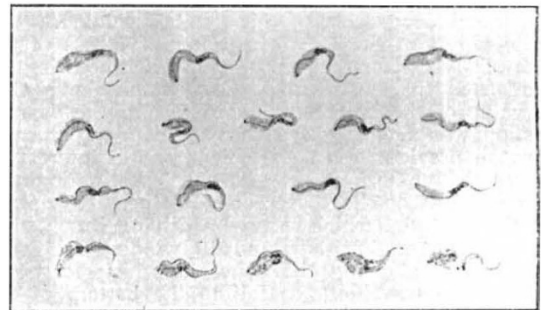


FIG. 3.—*Trypanosoma vivax* (Ziemann).  $\times$  about 700.

organs lying chiefly in the abdominal segment of the fly.

It was stated that probably all the tsetse-flies are capable of acting as carriers of all the pathogenic trypanosomes, at least in laboratory experiments. What makes one species of fly the especial carrier of a particular trypanosome is probably bound up in the natural history, the habits, and distribution of the fly.

It was shown that the two principal groups of tsetse-flies—the *G. morsitans* group and the *G. palpalis* group—differ from each other in well-marked characters, the former living in wild, unpopulated districts and trusting to the wild game for their food, the latter along rivers and lakes which are usually thickly populated, and trusting to man for a food supply, or in his absence living on the large reptiles, birds, and antelopes which frequent these places.

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

##### Group A. *Trypanosoma brucei* Group.

(1) *T. brucei*, the Nagana Parasite.—This was the first pathogenic trypanosome discovered in Central or South Africa. It was found in Zululand in 1894 in the blood of native cattle suffering from Nagana. The parasite was sent in the living condition to the



Royal Society in 1896, and at that time found its way into many laboratories, and much of the earlier work on trypanosomes was founded on it. The parasite causing the Rhodesian and Nyasaland form of sleeping sickness, and which had been named *T. rhodesiense*, is considered to be identical with *T. brucei*. Various strains, Zululand, 1894 and 1913, Nyasaland and Uganda, are compared, and the conclusion come to that they are identical in morphology.

SUSCEPTIBILITY OF ANIMALS TO *T. brucei*.

Many mammals, including man, horses, mules, donkeys, oxen, goats, sheep, monkeys, dogs, and many others, are attacked by this parasite. Birds and the cold-blooded vertebrates, such as crocodiles, lizards, and frogs, are quite unaffected by it. A single trypanosome seems to be just as efficacious in setting up infection as a million, and it does not seem to matter whether the kind of trypanosome injected is one of the long and slender forms or one of the short and stumpy.

Table I.—Giving (a) the Average Duration in Days of the Disease in Various Strains of *T. brucei*.

Strain	Man	Horse	Oxen	Goats and sheep	Monkeys	Dogs	Rabbits	Guinea-pigs	Rats
Human ... ..	90	—	134	42	25	34	28	67	30
Wild game ... ..	—	—	—	46	38	41	—	—	32
Wild <i>Glossina morsitans</i>	—	—	Recovered	54	38	29	47	81	26
Zululand, 1913 ... ..	—	33	310	77	29	18	33	44	27

(b) The Number of Animals Employed.									
Strain	Man	Horse	Oxen	Goats and sheep	Monkeys	Dogs	Rabbits	Guinea-pigs	Rats
Human ... ..	?	—	1	?	20	25	7	15	21
Wild game ... ..	—	—	—	5	9	13	—	—	6
Wild <i>Glossina morsitans</i>	—	—	2	16	14	25	—	10	19
Zululand, 1913 ... ..	—	3	1	7	8	17	—	10	23

Table I. gives the average duration in days of the disease caused by various strains of this trypanosome, also the number of animals employed. From this it will be seen that this disease runs a fairly rapid course in man, killing him as a rule in three or four months. This, as we shall see, is in marked contrast to the much more chronic course of the Congo sleeping sickness caused by *T. gambiense*. In horses, donkeys, and mules nagana runs its course on an average of thirty-eight days. No opportunity of studying the disease in horses occurred in Uganda or Nyasaland, as horses are very seldom seen in these countries. In the ox the disease is chronic and a certain proportion recover. In the other animals it may be said broadly that the disease runs a fairly similar course, and that little or no difference in the virulence is seen between the different strains.

Nagana is, as a rule, a fatal disease. With the exception of the oxen, almost all the other animals die. Out of 318 employed in these experiments only three recovered.

From its action on animals, then, just as from its morphology, it is apparent that *T. brucei* as it occurs in Zululand differs in no way from the Nyasaland strain called by Stephens and Fantham *T. rhodesiense*.

THE INFECTIVITY OF WILD TSETSE-FLIES (*G. morsitans*).

The tsetse-flies in Nyasaland were examined in order to find out how many of them were naturally infected. There were fifty-six experiments, and 10,081 tsetse-flies (*G. morsitans*) were employed. In the fifty-six experiments *T. brucei* was found twenty times (35.7 per cent.). Nine monkeys, fourteen dogs, and eleven goats were infected. This gives a proportion of 1 in 500, or 2 flies per 1000 caught in the sleeping

sickness area, Nyasaland, infective with nagana. This is only allowing one infective fly to each series of flies fed on the experimental animals, and is therefore the irreducible minimum.

TRYPANOSOMES FOUND IN THE BLOOD OF WILD ANIMALS LIVING IN THE SLEEPING-SICKNESS AREA, NYASALAND.

When an animal was shot a small quantity of its blood was taken in a sterilised bottle containing citrate of potash to prevent coagulation. Smear preparations were made at the same time. As the animals were often shot thirty or forty miles away from the camp, a motor-cycle was used to get the blood up the hill as quickly as possible. When the blood arrived at the laboratory it was at once injected into a goat, a monkey, and a dog. In this way 180 specimens of blood of wild game living in the fly area were examined, and fifty-seven were found to harbour pathogenic trypanosomes (32 per cent.).

This is, however, probably much below the actual percentage. A wild animal is only examined once, and that often under unfavourable conditions. If it were possible to examine the same animal several times it is probable that many more would be found infected. The parasites come and go in the blood; one day they may be present, the next day absent. The big game live in the "Fly Country" among swarms of infected flies, and are constantly liable to infection and re-infection.

The following table (Table II.) represents the number of times *T. brucei* was found among the 180 wild animals examined, and the species of game which

Table II.—This Represents the Number of Times *T. brucei* was Found Among the 180 Wild Animals Examined and the Species of Game which Harboured it.

Species of animal	Number examined	Number infected with <i>T. brucei</i>	Species of animal	Number examined	Number infected with <i>T. brucei</i>
Eland ...	10	0	Duiker..	7	1
Sable ...	5	0	Buffalo..	9	0
Waterbuck	13	3	Lion ...	1	0
Koodoo ...	3	0	Hyæna..	3	0
Bushbuck...	10	0	Elephant	2	0
Hartebeeste	35	5	Warthog	33	1
Reedbuck...	19	3	Wild cat	3	0
Oribi ...	26	1	Porcupine	1	0

harboured it. From this it will be seen that fourteen animals among the 180 harboured the nagana parasite (7.8 per cent.), and that the waterbuck, hartebeeste, reedbuck, and duiker seem to be the most dangerous neighbours to man. Twenty-three per cent. of the waterbuck, 14 per cent. of the hartebeestes, 16 per cent. of the reedbuck, and 14 per cent. of the duiker had *T. brucei* in their blood. If, then, the contention that this parasite found in the wild game is the cause of Nyasaland sleeping-sickness be proved to be true, then it is abundantly obvious how dangerous these wild animals are to man; and it must be borne in mind that in this Nyasaland fly area *T. brucei* is only one of the pathogenic species of trypanosome found in the wild game. Other three species pathogenic to the domestic animals are also found, *T. pecorum*, *T. simiae*, and *T. caprae*; *T. pecorum* in 14.4 per cent., *T. simiae* 1.7 per cent., and *T. caprae* in 11.1 per cent. of the wild game examined. It is self-evident that these wild animals should not be allowed to live in "Fly Country," where they constitute a standing danger to the native inhabitants and the domestic animals. It would be as reasonable to allow mad dogs to live and be protected by law in our English towns and villages. Not only should all game laws



restricting their destruction in "Fly Country" be removed, but active measures should be taken for their early and complete blotting out. It must be strictly borne in mind that this only refers to wild animals living in "fly" areas. No pathogenic trypanosomes have up to the present been found by the Commission in the blood of animals living in fly-free areas.

(2) *T. gambiense*, the Parasite of Congo Sleeping-Sickness.—*T. gambiense* (Fig. 4) is very similar in size and shape to *T. brucei*, but it would appear to be possible to distinguish them by the presence of the blunt-ended, posterior-nucleated forms which are so common in the blood of animals infected by the nagana parasite and quite absent in animals infected by the other. But as these posterior-nucleated forms are absent or scarce in the blood of man, this method of diagnosis requires the inoculation of experimental animals and the study of many preparations of their blood. It would appear to be impossible at present to distinguish between the two species by the microscopical examination of preparations made from the blood of man alone.

SUSCEPTIBILITY OF ANIMALS TO *T. gambiense*.

A marked difference exists between *T. gambiense* and *T. brucei* in regard to their virulence towards animals.

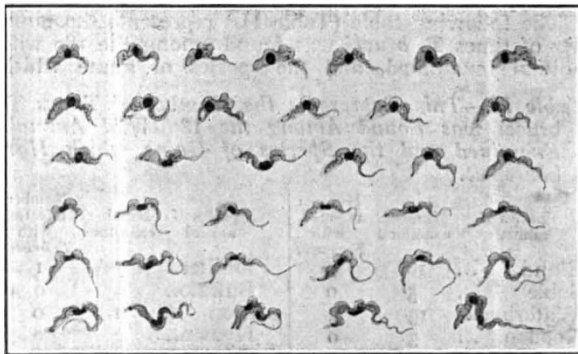


FIG. 4.—*Trypanosoma gambiense* (Dutton). Tanganyika, 1913.   
 × about 700.

It is almost impossible at first to give this disease to goats, monkeys, dogs, and guinea-pigs. The rat is the animal which is least refractory.

TABLE III.—Showing the Average Duration in Days of the Disease caused by *T. gambiense*, Tanganyika, compared with that caused by *T. brucei*, Zululand.

	Monkey	Dog	Guinea-pig	White rat
<i>T. gambiense</i> ...	159	96	264	137
<i>T. brucei</i> ...	26	34	67	30

The disease in animals caused by *T. gambiense* is thus much more chronic than that caused by *T. brucei*, and this character, combined with the morphology already described, affords the surest and safest means of separating these species.

*G. palpalis* THE CARRIER OF *T. gambiense*.  
INFECTIVITY OF WILD *G. palpalis*.

In 1903 at Entebbe, the Government cantonment, the tsetse-flies had plenty of opportunity of becoming infected, since they were caught in the vicinity of the hut-tax labourers' camp. These men came in thousands to Entebbe to work for Government for one month in lieu of paying hut-tax. They lived in

rudely-built grass huts near the lake shore, and on examination of their blood some 30 per cent. of them were found to harbour the parasite. In 1903, while these highly-infected labourers were living on the lake shore, the proportion of infective flies was found to be as high as 11.2 per 1000. The Government removed the hut-tax labourers from the vicinity of the lake, which became deserted, and a year afterwards the proportion of infected flies fell to 1.2 per 1000. When the Commission returned to Uganda in 1908 and took up camp at Mpumu at the north end of Lake Victoria we found the lake-shore flies in the vicinity still infective, although the population had been removed early that year. The examination of 7200 flies gave a proportion of 1.8 per 1000. But we had given the Government to understand that as soon as the natives were removed the flies would become harmless. It was therefore important to find out how long the lake-shore flies remained infective, and why they remained infective. For this purpose they were examined every year until 1912.

TABLE IV.—Showing the Results of Yearly Examinations of wild *G. palpalis* from 1903 to 1912 inclusive.

Year.	Locality	Number of flies examined.	Number of flies infective	Proportion of infective flies per 1000	Remarks.
1903	Entebbe.	?	?	11.2	—
1904	"	?	?	1.2	—
1908	Mpumu.	7,200	11	1.8	1 in 654
1909	"	18,691	7	0.4	1 in 2670
1910	"	27,179	4	0.14	1 in 6795
1911	"	23,899	1	0.04	1 in 23899
1912	"	28,279	4	0.14	1 in 7070

From this it will be seen that although there had been a steady decrease in the proportion of infective flies, a few remained, and these showed no sign of disappearing. The mistake made by the Commission was first in believing that the transmission of the *T. gambiense* was mechanical, and that a fly lost its power of infection within three days after feeding on an infected animal; and, secondly, in believing that man was the sole reservoir of the virus. It was found that a fly may remain infective for several months, and that man is by no means the only source of the virus.

THE CYCLE OF DEVELOPMENT OF *T. gambiense* IN *G. palpalis*.

This prolonged infectivity which some flies possess is due to the fact that in these the trypanosomes do not die off, but proceed to further multiplication. It was shown that a very small proportion of flies which feed on an infected animal show this cycle of development. In one series of experiments, forty-two in number, only one fly in 212 (0.5 per cent.) became infective. An average of thirty-six days is required to complete the cycle. The long account given may be summarised as follows.

Trypanosomes taken into the alimentary canal of tsetse-flies retain their shape and infectivity for some eighteen hours. They then degenerate and lose their power of infection, and as a rule have disappeared altogether from the majority of the flies by the fifth or sixth day. In a small percentage of flies, male as well as female, the trypanosomes maintain their position, they continue to multiply, and in a short time swarm in the alimentary canal of the fly. These multiplication forms bear little or no resemblance to the original trypanosomes. After some twenty or thirty days the developing flagellates find their way into the salivary glands, resume their original blood form, and regain their infectivity.



THE RESERVOIR OF *T. gambiense* (CONGO SLEEPING-SICKNESS).

Besides man, who is probably the most important reservoir of the virus, native cattle and the antelope living on the lake-shore in Uganda were found to harbour the parasites in their blood.

The prophecy that the fly would become harmless shortly after the natives were removed from the lake shore has unfortunately proved wrong, and before the islands are repopulated some other measure will have to be taken to get rid of the fly danger.

GROUP B.—THE *T. pecorum* GROUP.

I.—*T. pecorum*.

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

Morphology.

Fig. 2 shows the general appearance of the trypanosome. It is the smallest of all the African pathogenic trypanosomes, varying from 9 to 18 microns in length, with an average of 14 microns.

Animals Susceptible to *T. pecorum*.

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.

TABLE V.—The Average Duration of Life, in Days, of Various Animals Infected by *T. pecorum*.

	Donkey	Cattle	Goat	Pig	Monkey	Dog	Guinea-pig	White rat
Average duration in days ...	87?	121?	55	21	129	48	41	33
Number of animals employed ...	1	4	59	1	11	57	5	10

The Percentages of Recoveries in Various Animals from *T. pecorum* Infection.

Percentages ...	80	35	12	0	0	1	0	0
Number of animals employed ...	5	17	70	1	11	63	5	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 *T. pecorum*.

The chief carrier of *T. pecorum* is *G. morsitans*. In Nyasaland, this parasite was the commonest of the trypanosomes with which *G. morsitans* was infected. There were fifty-six experiments, and 10,081 tsetse-flies (*G. morsitans*) were employed. In the fifty-six experiments *T. pecorum* was found forty-six times, more than twice as often as *T. brucei*. Nine monkeys, thirty-four dogs, and thirty-five goats were infected. This gives a proportion of 4.6 per 1000 flies infected with *T. pecorum*.

THE CYCLE OF DEVELOPMENT OF *T. pecorum* IN *G. 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.

THE TYPE OF TRYPANOSOMES FOUND IN THE INFECTED FLIES.

Fig. 5 represents the developmental forms of *T. pecorum* found in labial cavity of *G. morsitans*. 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 contains the ordinary forms found clinging by their flagellar ends to the labrum. It will be seen that they have assumed the crithidial stage, a stage which seems to be a *sine quâ 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. They represent the final stage in the cycle of development and are the only infective forms. On the same figure are seen drawings 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

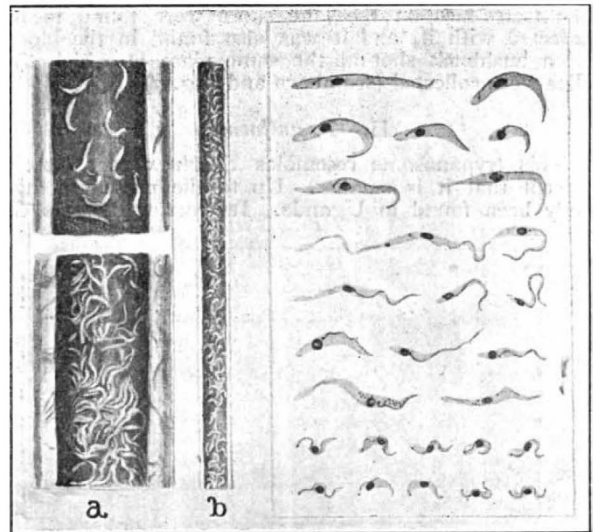


FIG. 5.—Developmental forms of *Trypanosoma pecorum* found in the labial cavity and hypopharynx of infected flies. a, Labrum. b, Hypopharynx.

small infective forms. From these drawings the ease and facility with which a tsetse-fly can infect an animal will be readily understood.

II.—*T. 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.

In the whole range of the trypanosome diseases of animals there is surely nothing so striking as the rapidly fatal action of *T. 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.

Another interesting point in regard to this species is that, so 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.

#### GROUP C.—THE *T. vivax* GROUP.

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

##### I.—*T. vivax*.

This is the cause of one of the most important cattle diseases in Uganda. We did not meet with it in Nyasaland, where its place seems to be taken by *T. caprae*. It is, however, widely distributed in Central Africa. It has been reported from Senegal and the Sudan in the north to Rhodesia in the south. It is easily recognised 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 (see above and Fig. 3).

##### II.—*T. uniforme*.

This trypanosome resembles *T. vivax* very closely except that it is smaller. Up to the present it has only been found in Uganda. Its carrier there is *G.*

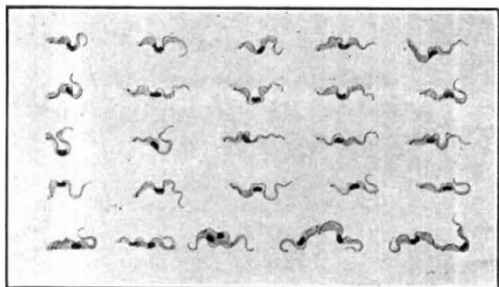


FIG. 6.—*Trypanosoma simiae*.  $\times$  about 700.

*palpalis*, and its reservoir the wild game on the lake shore.

##### III.—*T. 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 smaller experimental animals are immune.

#### CONCLUSION.

This concludes the Croonian Lectures on the trypanosomes causing disease in man and domestic 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.

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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 which 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 *T. brucei* and *T. 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 doubt that if the wild game were driven out of the fly country trypanosome diseases such as those caused by *T. brucei* and *T. 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 habitable, then clearing and cultivation must be resorted to. By these means, in all probability, *G. palpalis* will be driven away, and with it the disease.

#### UNIVERSITY AND EDUCATIONAL INTELLIGENCE.

DR. H. G. EARLE has been appointed to the chair of physiology in the University of Hong-kong.

DR. J. A. MENZIES has been appointed professor of physiology in the University of Durham College of Medicine, Newcastle-upon-Tyne.

IN the prospectus of the University College of North Wales a reference to "Aeroplane and Other Researches" occurs in the schemes of study of the department of applied mathematics. In view of the important part played by aeroplanes in the present war, we hope that Prof. Bryan will make every effort to enlist the services of his pupils in the solution of the many unsolved problems which he has enumerated, and that he will encourage them to take up this work in preference to studies of a more examinational character. We understand from Prof. Bryan that he would be glad to secure the assistance of students from other universities possessing the necessary training in applied mathematics who are able and willing to enter the college at Bangor for a post-graduate course of research in the subjects in question.

THE prospectus of the University courses in the Municipal School of Technology, Manchester, for the session 1915-16, which is now available, serves admirably to give the inquiring student an excellent idea of the resources and equipment of this great technical college. It will be remembered that a faculty of technology in the University of Manchester was established in 1905, with the principal of the School of Technology as dean of the faculty and with the heads of the mechanical and electrical engineering, applied chemistry, and architecture departments of the School of Technology as professors of the University.