Fig. 16. Transverse section through the spermatheca, showing communication with the gut. *sp.amp.*, spermathecal ampulla; *comm.g.*, communication with gut; *d.v.*, dorsal vessel; *sep.gl.*, septal glands; *v.n.c.*, ventral nerve-cord.

17. Drawing of the ventral clitellar region. *lip*, lip overhanging the groove in which the male pore is situated.

18. Diagrammatic drawing of segments 10 and 11 of a young mature Enchytræid, showing the sperm-sacs. *d.v.*, dorsal vessel; *sp.s.*, sperm-sac; *v.v.*, ventral vessel; *ros.*, rosette of cells in the sperm-sac.

PLATE XLIX.

Fig. 19. Transverse section through two copulating Enchytræids. *ov.*, ovary; *cl.ep.*, clitellar epithelium; *sp.d.*, sperm-duct; *p.b.*, penial bulb; *p.*, penis; *op.*, male opening; *sp.gl.*, spermathecal glands; *sep.gl.*, septal glands; *amp.*, ampulla of spermatheca; *gl.*, glands in the ventral clitellar region; *m.*, muscles attached to these clitellar glands and the body-wall; *d.v.*, dorsal vessel.

20. Transverse section through segment 12 to show gland-like bodies in the ventral clitellar region. *d.v.*, dorsal vessel; *ov.*, ovary; *cl.ep.*, clitellar epithelium; *g.*, gland-like bodies continuous with the epidermis; *ep.*, epidermis; *v.n.c.*, ventral nerve-cord; *m.*, muscles connecting gland-like bodies obliquely with the body-wall; *v.v.*, ventral vessel; *c. in g.*, ciliates in gut.

21. Highly magnified section through the male opening. *sp.d.*, sperm-duct; *p.*, penis-like thickening; *op.*, male opening; *p.b.*, penial bulb; *m.*, muscles around the penial bulb; *g.*, ventral clitellar gland.

25. The Relationship of the Big Game of Africa to the spread of Sleeping Sickness. By Dr. W. YORKE, Liverpool School of Tropical Medicine *. With an Appendix containing Remarks by Sir JOHN BLAND-SUTTON, F.R.C.S., F.Z.S.; GUY A. K. MARSHALL, F.Z.S.; Prof. E. A. MINCHIN, M.A., F.R.S., V.P.Z.S.; The Hon. L. WALTER ROTHSCHILD, D.Sc., F.R.S., F.Z.S.; Sir HENRY SETON-KARR, K.C.M.G., F.Z.S.; and Sir ALFRED SHARPE, K.C.M.G., LL.D.; and Reply by Dr. YORKE.

[Received and Read March 18, 1913.]

Although Sleeping Sickness has been recognised as a disease on the West Coast for nearly two hundred years, human trypanosomiasis was unknown in Nyasaland and in the greater portion of Rhodesia until 1908. At the end of that year the first case of the disease was found in Nyasaland, and during 1909 and 1910 a considerable number of cases were discovered amongst the Europeans and Natives living in Nyasaland and Rhodesia. This state of affairs was not easy to understand, as the particular tsetse fly, *Glossina palpalis*, which is known to transmit Sleeping Sickness in other parts of Tropical Africa, has not been found in these countries.

In 1910, it was shown that the parasite causing the disease in Nyasaland and Rhodesia differed in certain respects from that

* Communicated by GUY CHETWYND, F.Z.S.

causing Sleeping Sickness in other portions of Tropical Africa. The name *Trypanosoma rhodesiense* was given to this new parasite.

Since these discoveries many cases of Sleeping Sickness have been found in Rhodesia and Nyasaland, and in 1911 the Chartered Company decided to have the matter thoroughly investigated; it was with this object that Dr. Kinghorn and I were sent to North Eastern Rhodesia.

The first problem that we had to solve was to ascertain the vector responsible for the spread of Sleeping Sickness in a country where *Glossina palpalis* does not exist. Now although *Glossina palpalis* has not been found either in Nyasaland or in the Luangwa Valley of Rhodesia, yet *Glossina morsitans*, the tsetse fly which is known to cause "fly" disease in domestic stock, is present in enormous numbers, and it was soon proved by Dr. Kinghorn and myself that it is this fly which is responsible for the spread of human trypanosomiasis. This discovery is one of great practical significance, for whereas the former fly, *Glossina palpalis*, is limited in its distribution to water-courses, and is never found far from the banks of certain rivers or the lake shores, the latter, *Glossina morsitans*, is ubiquitous, its distribution being quite independent of water. Hence, it is at once obvious that it is impossible to attempt to deal with Sleeping Sickness in Rhodesia and Nyasaland by any such simple method as removing the native population back from the water-courses and lake shores—a procedure which was attended with such remarkable results in Uganda. The problem of preventing the spread of Sleeping Sickness in these countries, which a few years ago were thought to be in no danger, is one of infinitely greater difficulty than was that which had to be faced in Uganda.

A large number of wild *Glossina morsitans* was examined in the Luangwa Valley in order to ascertain the proportion capable of infecting man with trypanosomiasis. This information is important, as it affords an approximate idea of the potential danger of the district. We found that 1 in 500 wild *Glossina morsitans* was infective in nature. This is an astonishingly large proportion, and it is at once evident that some host other than man must be infected with the human trypanosome in order to account for the large number of naturally infective *Glossina morsitans*. With the object of ascertaining what was the chief vertebrate reservoir of the virus, we examined a large number of the wild fauna of Africa. In all, we examined 250 wild animals (including elephant, rhinoceros, hippopotamus, lion, buffalo, 14 different kinds of antelope, caracal, galago, squirrel, genet, hunting dog, giant rat, and wild rabbit), 256 monkeys, 35 domestic stock, 142 wild rats, and 15 wild mice, making a total of 698. The results were striking, a large proportion of the antelope being found to be infected with the parasites which cause Sleeping Sickness in man and trypanosomiasis in domestic stock. As a conservative estimate, the percentage of big game infected with

the trypanosomes of man or domestic stock might at Nawalia in the Luangwa Valley be placed at 50, and at Ngoa on the Congo Zambesi watershed at 35.

TABLE I.—Percentage of various species of game found infected with trypanosomes pathogenic to man or domestic stock at Nawalia, Luangwa Valley.

Animal.	Number examined.	Percentage harbouring trypanosomes.
Bushbuck	9	66·6
Waterbuck	28	60·7
Kudu	7	57·1
Hartebeest	6	16·6
Roan	8	12·5
Warthog	9	11·1
Puku	10	10·0
Mpala	29	6·9

TABLE II.—Percentage of various species of game found infected with trypanosomes pathogenic to man or domestic stock at Ngoa, Congo Zambesi watershed.

Animal.	Number examined.	Percentage harbouring trypanosomes.
Sitatunga	2	50·0
Waterbuck	27	44·4
Eland	15	26·6
Duiker	9	22·2
Roan	5	20·0
Puku	8	12·5

This investigation, therefore, made it perfectly clear that the main reservoir of the trypanosomes of man and domestic stock is the big game.

Having ascertained these two essential facts, namely that the tsetse fly, *Glossina morsitans*, is the vector by which the disease is spread, and that the big game is the inexhaustible reservoir of the virus which causes the disease, we are faced with the problem

of what, in the light of this knowledge, can be done to stamp out Sleeping Sickness or to limit its spread in Nyasaland and Rhodesia. Obviously the most satisfactory means of prevention would be the extermination of *Glossina morsitans*, which conveys the parasite from one vertebrate host to another. Unfortunately, however, this is out of the question at the present stage of our knowledge. The only known method of getting rid of the fly from a district is by clearing away the bush. In the immediate vicinity of villages such a procedure is doubtless feasible and would be attended by valuable results, and natives should be encouraged to do everything possible in this direction. The labour involved in clearing large tracts of country would, however, be so great that this can be at once set aside as impracticable. Moreover, it must be remembered that not only would the country have to be cleared, but it would require to be kept cleared. Everyone who has had experience of Tropical Africa is familiar with the dense shrub growth which springs up in the site of old garden clearings, two or three years after the natives have ceased cultivating the land. This shrub growth is exceedingly favourable to *Glossina morsitans*, so that unless the country be constantly kept cleared the last state of the district is worse than the first.

At present but little is known of the bionomics of *Glossina morsitans*. The results of investigations carried out up to the present indicate that this tsetse fly has no particular breeding places, but that its pupæ are deposited in a more or less haphazard manner in hollow trees and excavations where they are not likely to be disturbed by game-birds. Regarding the liability of the fly to disease and of its natural enemies we know nothing. Much more information is required on this subject, but it seems only too obvious that the investigations will be beset by great difficulties and that the information will only be forthcoming as the result of much slow and tedious work. In fact, to those familiar with *morsitans* country the extermination of the fly must seem an almost impossible procedure.

In Uganda, where the disease is spread by *Glossina palpalis*, the removal of the population a short distance away from the lake shores and water-courses was followed by most excellent results. Such a measure, however, is impossible in Nyasaland and Rhodesia, where the vector *Glossina morsitans* is practically ubiquitous in its distribution and not limited to water-courses as is *Glossina palpalis*.

In view of the impossibility of exterminating the fly and of the equal impossibility of removing the population from the fly belts, we must consider the only way that remains of combating the disease, that is the advisability of attempting to destroy the reservoir of the virus. It is obvious that the mere isolation of infected human beings is futile in view of the fact that the main reservoir of the virus is the blood of the big game.

And now we come to a subject to which I particularly wish to

draw your attention—a subject which has excited considerable controversy already, and one which will, I expect, excite still more in the near future. I refer to the connection between big game on the one hand and trypanosomiasis of man and domestic stock on the other.

Since the beginning of last year, when Dr. Kinghorn and I published our paper announcing the fact that a large proportion of the wild fauna of Africa harboured the trypanosomes of man and domestic stock, a considerable polemic has arisen over the question of the advisability of attempting to exterminate the big game in the vicinity of human habitations.

In discussing this subject, it appears to me that I could not do better than attempt to answer some of the objections which have been raised against any prophylactic measure being adopted which involves interference with the African fauna.

It has been suggested that if the big game be destroyed in any district, the fly, being deprived of its natural source of food, might turn its attention solely to man and his flocks and herds. It appears to me that but little importance should be attached to this hypothesis. In the first place, cattle do not as a rule live in the presence of *Glossina morsitans*. It was suggested that cattle and other domestic stock might harbour the human trypanosome for considerable periods without detriment to health. This, however, is not true in the case of the human trypanosome of Nyasaland and Rhodesia, which we proved rapidly killed horses, cattle, donkeys, goats, and dogs. Moreover, even if the human parasite did not kill domestic stock, these would still die from the ordinary cattle trypanosomes such as *T. pecorum*, *T. nanum*, and *T. vivax*, with which we found the wild *Glossina morsitans* to be heavily infected ; so that it is quite obvious that domestic stock cannot have the same significance as a reservoir of the virus as the antelope, which are tolerant of the trypanosomes pathogenic for man and domestic stock. Secondly, the tsetse fly does not invade the clearings in and around villages to any great extent, and therefore man is only attacked when for any reason he goes forth into the bush, and it is hard to believe that he would suffer to any much greater extent in the absence of game. Thirdly, and this is the most important point: if the game were removed the reservoir of the virus is destroyed, and therefore in a short time the fly would tend to become non-infective. The bite of a non-infective *Glossina morsitans* hurts nobody. Finally, there is absolutely no evidence indicating that if the big game in any particular district were slaughtered, the tsetse fly, unable to obtain blood from these animals, would attack man and the domestic animals to a greater extent than at present. It might equally well be argued that if the food-supply of the fly be removed the fly would disappear. There is, moreover, a considerable amount of evidence that the tsetse fly spreads with the game. For example, since the rinderpest swept through Central and South Africa sixteen or seventeen years ago the big game have increased

enormously in numbers, and with this increase in game there has been a corresponding increase in the number of tsetse fly. At Nawalia, in the Luangwa Valley, where we were stationed, *Glossina morsitans* was present in enormous numbers, and natives sent out to collect the flies had no difficulty in capturing large numbers within a short distance of the laboratory. Nawalia is the site of an old Government station which was closed a few years ago on account of Sleeping Sickness. The magistrate who was stationed there in 1905 told me that he only occasionally saw tsetse flies in this district at that time.

Again, it has been suggested that the big game might be only one of the reservoirs of the disease, and that the infection might equally well be conveyed by the small vermin. It must be remembered, however, that the small vermin are to a considerable extent nocturnal in their habits, and although *Glossina morsitans* does occasionally bite at night, especially when the moon is full, yet nobody who has lived in "fly" areas can have any doubt but that this is exceptional, and that for practical purposes the fly feeds in the daytime only. Dr. Kinghorn and I examined a large number of small vermin—rats, mice, wild rabbits, etc.—without finding a single instance of natural infection. Furthermore, it might be remarked that there is no evidence to show that the small vermin are tolerant of the human trypanosome as are the big game. In those which we infected experimentally the disease ran an acute course and the animals died. If this be the case with the majority of the small vermin, they cannot have the same significance as reservoirs of the virus as have the big game, which can probably harbour the parasite for long periods of time without exhibiting signs of disease.

We return, therefore, to the original position. The big game is the natural reservoir of the infection, and the rôle of the tsetse fly, *Glossina morsitans*, is to transfer the virus from the big game to man and his flocks and herds. At the present state of our knowledge we are unable to attack successfully the tsetse fly, nor, unfortunately, is there any prospect of our being able to do so in the near future. Whether anything would be gained in this direction by slaughtering the big game is still a moot point: therefore I will not consider this side of the question, but advocate the advisability of attempting to drive back the game from inhabited regions solely because the game are the reservoir of the infection.

It has been asserted that the power to slaughter all game animals in an infected district is unsound in principle, because the game, when harried, would betake itself to places difficult of access to man, or scatter in small herds, or in pairs, or singly, over wide areas, and that should this occur, it is highly probable that it might be followed by tsetse, thus spreading the danger of infection to wide areas now free from game and fly.

To such criticism as this it is not difficult to reply. If the game when harassed betook itself to places difficult of access to

man, surely this is exactly what is desired. In such places it would no longer be a menace to civilisation.

Regarding the second alternative, that the hunted game might scatter into small herds or in pairs and be followed by the tsetse fly, thus spreading the infection over large districts, the obvious rejoinder is that should this occur the game must be destroyed in the new areas, provided these happen to be inhabited regions. Such a contingency is, however, very unlikely, as it is very questionable if small herds of hunted game scattering over wide areas would cause the tsetse fly to migrate with them.

The Colonial Secretary pointed out that "To talk of the extermination of the wild fauna of a subcontinent was to talk wild nonsense." This is perfectly true, but it is no reason why the game in the vicinity of human habitations should not be destroyed. It was further stated by the Colonial Secretary that an attempt made in Nyasaland to get the game in a certain area killed off was, after twelve months, unsuccessful, though the natives were encouraged to shoot. This, again, is no argument against the policy suggested, but merely a confession that the experiment was not efficiently performed. In this connection it is of interest to note the remarks of Dr. J. B. Davey at a recent meeting of the Society of Tropical Medicine. He said:—

"I do not think there would be much difficulty in doing this (destroying the game) if the natives were employed. It is work they would take up with some avidity. Having lately been a member of the Commission in Nyasaland, I remember that considerable anxiety was expressed lest we should not be able to get sufficient material, because during the previous year fifty rifles had been served out in that area, and the natives had been encouraged to shoot game. If in that short time fifty rifles could clear a large area like that, after a year or two they could make a great impression. As a matter of fact, that permission was only in force for about three months, and they made a considerable impression on the amount of game."

Such experiments as these are unsatisfactory, and do not yield any definite results. For any reliable information to be obtained, it is necessary that the work should be done in a scientific manner, and on a sufficiently large scale in some particular district. A locality which is fairly well populated and which contains plenty of tsetse fly and game should be chosen. An exact census of the population should be made, and the proportion suffering from Sleeping Sickness determined. The same must be done in the case of the domestic animals, if such exist. An index of the percentage of infective tsetse fly must be ascertained. This is most important, as it gives one a definite idea of the potential danger of the district. Finally, the game must be completely eradicated, and at the same time the percentage infected with the human and cattle trypanosome determined, and when once the game has been driven out, it must be kept back by vigorous action and not allowed to return. After an interval

of a couple of years or so the population, domestic stock, and tsetse fly must again be carefully examined. Then we should be in a position to decide definitely whether or not driving the fauna back from the site of human habitations would pay. Such an experiment as this would take some years to accomplish. That the big game is the reservoir of the human infection there can no longer be any doubt, as the work of Kinghorn and myself has already been confirmed. I submit, therefore, that the time for temporising is past. Sleeping Sickness has already crossed the Zambesi, and cases have been recorded in Southern Rhodesia. In my opinion the natives living in fly areas should be allowed to kill game in their own way, and they might also to an extent be armed with rifles of some uncommon bore, so that a control could be kept over the ammunition. Europeans ought to be allowed to shoot what they like. Protecting the reservoir of the trypanosomes causing fatal disease in man and his flocks and herds by heavy licences, appears to be rather an anomaly. Finally, I consider that some such decisive experiment as I have outlined is urgently required, as even under the most favourable conditions several years must elapse before we should be in a position to recommend definitely that vigorous steps be taken to drive back the big game from the neighbourhood of human settlements on a large scale throughout Tropical Africa.

It may seem an act of vandalism to slaughter the wonderful fauna of Africa; but surely when it is definitely proved that this fauna is antagonistic to civilization, then that which stands in the path of progress must be removed.

APPENDIX.

Sir John BLAND-SUTTON, F.R.C.S., F.Z.S., did not feel qualified to express an opinion of value in regard to the drastic scheme for extirpating the living reservoirs of Sleeping Sickness proposed by Dr. Yorke. It is not always necessary to destroy the reservoirs of a disease in order to protect human beings from infection. For example, when bacteriologists discovered that the goats of Malta, from which the island derives its milk supply, were the reservoirs of the *Micrococcus melitensis*, the cause of Malta (or Mediterranean) fever, the Governor was strongly urged to order the wholesale destruction of the goats. He explained that such an order would bring about a revolution in the island, for the Maltese are devoted to their goats. The micrococcus is conveyed in the milk; it was a simpler plan to banish goat's milk from the military and naval dietary. Sailors and soldiers at Malta are not now allowed to drink goat's milk; as a consequence, Malta fever has disappeared from the Navy and the Army.

It is probable that the band of bacteriologists and entomologists (of which Dr. Yorke is a brilliant member) engaged in

studying Sleeping Sickness will find some prophylactic or remedial measure less obnoxious than the wholesale destruction of the great game animals of Africa.

Mr. Guy A. K. MARSHALL, F.Z.S., urged that the essential question for consideration was whether or no the game constituted a reservoir of *Trypanosoma rhodesiense*, as opposed to *T. brucei*; and from this point of view it was somewhat unfortunate that in giving his percentages of infected game Dr. Yorke had lumped these two forms together, as this might possibly lead to misconceptions. He felt very strongly that the unqualified statement that human trypanosomiasis had "spread" to south of the Zambesi was not justified in the present state of our knowledge, and might well cause unnecessary alarm. He agreed with Mr. Austen that there was no evidence to show that the disease was really spreading in the countries north of the river, though its existence there had now been known for nearly five years. A few years ago, when sporadic cases of Sleeping Sickness were first discovered in Nigeria, the fear was expressed that an epidemic similar to that in Uganda would take place. Yet no epidemic had occurred, nor was such an event probable; for it was now generally recognised that the disease is there endemic, the bulk of the population being therefore immune, and these sporadic cases merely indicated that more or less susceptible individuals are still being born. The incidence of human trypanosomiasis in Nyasaland and Rhodesia presents a very striking similarity with that which we find in West Africa, and, in conjunction with other considerations, strongly suggests that we are dealing, not with a new disease, but with one which has already been endemic for some time and has merely escaped detection, as in West Africa. If this be a sound conclusion, we must recognise the probability that the immune natives may themselves be reservoirs of the trypanosome, in which case Dr. Yorke's assumption that the removal of the game must necessarily eradicate the disease may prove entirely fallacious. Finally, Mr. Marshall pointed out that Dr. Yorke had recorded that 1 in 500 of the wild flies was infected with *T. rhodesiense*, and had referred to this as an unusually high percentage. But the latter statement seemed hardly in accord with the results obtained by other workers, and notably the data for *T. cazalboni* and *T. gambiense* given by Roubaud in a recent paper. Indeed, considering the high percentage of infected game in the Luangwa Valley, as estimated by Dr. Yorke, his record of infected flies seemed quite remarkably low and did not suggest any probability of an epidemic.

In considering any measures for coping with human trypanosomiasis in Rhodesia and Nyasaland, it is a matter of the very highest importance, especially from an administrative point of view, that we should ascertain whether the disease is really a new one in those countries, or whether it is merely endemic. The

matter was so inadequately discussed when Dr. Yorke read his paper to the Society that it seems desirable to consider it here in somewhat greater detail. In his published papers Dr. Yorke has throughout assumed the disease to be a new one, and gives no sign of having even contemplated the other alternative. When, at the meeting referred to, I suggested that the disease is probably endemic, and that in consequence immune natives may prove to be a reservoir of the trypanosome, he rejected the suggestion on the grounds (1) that the disease had only recently been discovered, and it was incredible to suppose that so distinctive an organism as a trypanosome had been previously overlooked by medical men; and (2) that the virulence of the disease was so great in all the cases of infection investigated (there being no recoveries) that he found it impossible to believe that human beings could harbour this organism with impunity.

As one who resided in Southern Rhodesia for nearly thirteen years, I cannot seriously accept the first of these arguments. The methods of blood-examination which are now matters of every-day routine for any young doctor trained in our modern Schools of Tropical Medicine, were certainly not practised in Rhodesia until quite recently, and the chances of the disease being correctly diagnosed, at least up to 1906, would have been extremely remote. With regard to the second contention, the mere fact that a disease is highly virulent in susceptible persons is no valid evidence as to the non-existence of immune individuals. It is now generally admitted that the natives of West Africa are very largely immune to *Trypanosoma gambiense*, and there seems to be no good reason for assuming that a similar power of resistance to *T. rhodesiense* cannot exist in East Africa. Indeed, the more virulent the disease, the more rapid will be the development of a general immunity; and further, the more deadly the parasite, the less likely are there to be intergrades between complete immunity and fatal susceptibility in the host.

It is true that a new disease will usually exhibit what is known as primary virulence; but this virulence is essentially in relation to population, and not merely in relation to the individual. In other words, we may reasonably assume a disease to be newly introduced if there is a very high percentage of cases and a low percentage of recovery; but if the percentage of cases is small (as it certainly is in Rhodesian trypanosomiasis), the individual severity is no proof that the disease is new. To take an example. The endemicity of yellow fever in West Africa is now hardly disputed, yet in the epidemics in Accra and Sekondi in 1910 to 1912, out of 23 Europeans attacked no fewer than 22 died. Another aspect is perhaps worth consideration. There can be no question that Sleeping Sickness was a new disease in Uganda, and there was a marked primary virulence in its true sense; yet in spite of this, the individual course of the malady, though eventually fatal, was less severe and much less rapid than in Rhodesian trypanosomiasis.

The position, then, is as follows. We have in Rhodesia and Nyasaland a very virulent form of trypanosomiasis, carried by a *Glossina* which has a wide local distribution and which is a much more persistent biter than *G. palpalis*, and, further, a considerable percentage of the game is estimated to be infected by *T. rhodesiense*. If this be really a new disease, it would seem that the native population is faced with an epidemic beside which that in Uganda would be a mere circumstance, and the most drastic remedies that youthful enthusiasm can devise would appear to be justified.

On the other hand, we have to consider that the disease has been known to exist in this area for nearly five years and has probably been there a good deal longer; for we can hardly be asked to believe that by a remarkable coincidence *Trypanosoma rhodesiense* chanced to be discovered at the precise moment of its origin. Yet, in spite of the presence of these factors making for the rapid development of a severe epidemic during all these years, nothing of the kind has happened; nor have we any real reason to suppose that an epidemic is imminent. The evidence, such as it is, seems to indicate that we are dealing with an endemic disease, which is not likely to become worse if the present conditions are maintained. And while further experimental investigations should certainly be carried on, there is no justification for anything in the way of panic legislation.

Professor E. A. MINCHIN, M.A., F.R.S., F.L.S., V.P.Z.S., said that in Dr. Yorke's paper he had heard nothing to criticise or controvert, so far as statements of fact were concerned. The two African trypanosomes deadly to man, namely *Trypanosoma gambiense* and *T. rhodesiense*, were members of a large group of trypanosomes which was typified by the well-known *T. brucei*, and might therefore be called the *brucei*-group. There could be hardly any doubt, in the present state of knowledge, but that the *brucei*-group of trypanosomes was one which was primitively parasitic upon wild ungulates, and that many species had been carried secondarily by the agency of biting flies to other vertebrate hosts, in which they had been able to maintain themselves. The fact that the trypanosomes of this group were harmless to the wild ungulates but deadly to other animals showed that the former were their natural hosts. Thus, *T. gambiense* and *T. rhodesiense* were harmless to antelopes but deadly to man; *T. brucei* was harmless to wild ungulates but very deadly to domesticated horses, cattle, or dogs. This conclusion did not apply, however, to the human trypanosome of Brazil, *T. cruzi*, nor to cattle trypanosomes of the *theileri*-type, forms which were quite distinct from the *brucei* group.

While agreeing with Dr. Yorke on matters of fact, Prof. Minchin thought that in the present state of our knowledge the utmost caution should be exercised in putting into practice administrative measures based upon the data so far established. The inter-

relationship of different organisms in nature was very complex, and a sudden change brought about in the conditions might have results altogether different to what was anticipated originally. Dr. Yorke had argued, it seemed to him, as if the distribution and occurrence of tsetse-flies were fixed and immutable, and had denied that there was any danger of their migrating towards human habitations if the big game, their natural source of food in the bush, were destroyed. Prof. Minchin found it difficult to believe, however, that if the tsetses in the bush were deprived of their food, they would sit down and die of starvation; he thought it far more probable that the flies would migrate in search of food, which they would find in human beings and the cattle surrounding their habitations. [Dr. Yorke maintained that the shelter of the bush was necessary for the flies; but such shelter might be found on cultivated land, especially in the banana-plantations which often surrounded native huts or villages.]

Considering the question, therefore, purely from a utilitarian point of view, Prof. Minchin thought it within the bounds of possibility, to say the least, that the wholesale destruction of the big game might lead to a condition of things more dangerous and disastrous than that existing at present. He urged that such measures should be undertaken, at first experimentally, on a small scale and in a restricted area, in order that accurate knowledge might be obtained of the effects produced by the elimination of the wild ungulates before destroying them wholesale.

Letter from The Hon. L. WALTER ROTHSCHILD, D.Sc., F.R.S., F.Z.S.

Dear Dr. Chalmers Mitchell,

I am extremely sorry that, as I leave for Monaco on the 18th, I cannot attend the meeting. But I trust you will read this letter at the meeting. Dr. Yorke suggests the extermination or partial extermination of the ruminants and large game animals. I wish to protest against this most emphatically on zoological and ethical grounds. However, in order to prove to the utilitarians the absolute uselessness of this proceeding, I should like to point out that the extermination of the game animals in any large area would be a task of several years' duration, and the following would take place. As year by year the large animals grew scarcer, the tsetse-flies *Glossina palpalis* and *morsitans*, which are the means of spreading Sleeping Sickness in men and N'gana in animals, would be driven to bite monkeys, carnivora, rats, mice, and the numerous small animals of those regions; these would be infected and the trypanosomes of the disease would gaily survive. This would not only mean the continuance of the disease in its present degree, but would cause a sharp increase of both diseases. The reason for this increase, to my mind, would be very evident, for at present, owing to the abundance of large game animals, the

flies do not on sight attack EVERY *human being* or *domestic animal* they perceive; but once let them be reduced to small animals for their normal supply of blood, it is certain that on the first appearance of any *domestic animal* or *human being* they would instantly precipitate themselves on it. This would mean that many more would be bitten than at present, and many more cases of the disease would occur. I am sure if the game is exterminated, the fly will not be; and, on the contrary, the disease will be increased and not diminished.

Yours sincerely,

(Signed) WALTER ROTHSCHILD.

Zoological Museum,
Tring,
Herts, England.
March 15th, 1913.

Letter from SIR HENRY SETON-KARR, K.C.M.G., F.Z.S.

Dear Dr. CHALMERS MITCHELL,

I am exceedingly sorry I cannot attend the meeting to-night to hear Dr. Yorke's paper on the relation of Big Game in Africa in spreading Sleeping Sickness, and the discussion thereon.

The subject is one of deep interest and importance, to which I gave considerable attention during my travels in South and East Africa in 1911. So far as my own information and observation go, I am entirely opposed to any proposed destruction of Big Game, or any relaxation of the restrictions on the shooting of game until the case against them as spreaders of Sleeping Sickness has been fully and clearly established, which is far from being the case at present.

The subject was fully discussed in the South-African press a few years ago by many well-known authorities, and I would suggest that the letters on this subject be, if possible, obtained, printed, and circulated.

The main difficulties of the problem, as I understand them, are:—

1. That there would appear to be other agencies, apart from Big Game, that spread this dreadful scourge, and that some of these agencies have yet to be ascertained.
2. That inasmuch as there are Sleeping Sickness areas where Big Game are not plentiful, and Big Game areas where there is no Sleeping Sickness, the direct connection between the two has yet to be fully established.
3. That other causes exist for the spread of the disease, such as, for example, the greater freedom of intercourse among native tribes under white protection.
4. That the destruction of Big Game in any given areas is a task of immense difficulty, which, if attempted, might not have the desired effect. Some game would merely be driven elsewhere, and the smaller mammals and game be left, which are also a possible spreading agency of the disease.

I feel sure that Dr. Yorke will initiate a most interesting and valuable discussion, and no doubt throw new light on this important and mysterious subject. But I earnestly deprecate any too hasty or premature a conclusion on the matter, and before all authorities have been fully heard and consulted. If it is conclusively shown that Big Game undoubtedly spread Sleeping Sickness, then Big Game must go. On the other hand, it would be an irreparable calamity of the worst kind to permanently injure or destroy the indigenous wild life of Africa *to no purpose*, and this might be the result of premature action.

Yours truly,

47 Chester Square,
London, S.W.
March 18th, 1913.

(Signed) HENRY SETON-KARR.

Sir ALFRED SHARPE, K.C.M.G., LL.D., remarked: "The question appears to resolve itself into the following heads:—

"1. What varieties of tsetse are proved to be carriers of the trypanosomes of any form of Sleeping Sickness, and (which is even more important) what *other* carriers are there?

"2. Does tsetse really depend entirely on game for its existence?

"3. Assuming even that tsetse are the only distributing agents of all forms of Sleeping Sickness—and that fly depends solely on game for its existence—is it possible to destroy every form of life upon which tsetse can exist?

"There is a desire on the part of a certain section of the European population of our African possessions to do away with all restrictions on the killing of game. This is fomented in some cases by the local press; and every argument is made use of which may further their object. In most cases those who carry on this local agitation are ignorant of the actual conditions governing the existence of tsetse, and its relation to game and to Sleeping Sickness.

"1. With regard to my first heading. We know that *Glossina palpalis* is considered to be the only distributing agent of Sleeping Sickness in Uganda. *G. morsitans* is now credited, however, with being a distributer in Nyasaland and N.E. Rhodesia. What we require as regards *morsitans* is (*a*) more definite proofs that it distributes under natural conditions, (*b*) more knowledge as to whether there are not other distributing agents in Nyasaland and Rhodesia. It is remarkable that in these two districts, in spite of the fact that *G. morsitans* exists not only in the low country but up to a height (in Rhodesia) of 4000 feet above sea-level, Sleeping Sickness has only been found under the same conditions as in Uganda, *i. e.* in close proximity to the banks of the large rivers and lakes.

"2. As to the second heading, my experience is opposed to the

supposition that tsetse depends solely on game for its existence. There are striking instances in Nyasaland and Rhodesia of game-less districts full of fly, and flyless districts full of game.

"3. Referring to the third heading—how would it be *possible* to annihilate game? It must be remembered that it is not only what is known as 'big game' that fly feeds on, but *all* game, big and small, and almost every form of life in the bush. It would be necessary to annihilate practically every form of life and to remove also every native. This would obviously be impossible.

"In any case it would be a terrible mistake, which could never afterwards be remedied, to countenance anything in the shape of an attempt to annihilate game in any part of Africa, unless we are *absolutely certain beforehand* that such a course would not only ensure the extinction of all varieties of tsetse fly, but make sure that all forms of Sleeping Sickness would disappear. At present the grounds for such an assumption are totally inadequate."

Reply by Dr. W. YORKE.

As it is impossible in the limited time at my disposal to reply in full to the volume of criticism which my paper has evoked, I must content myself with dealing with a few of the more important and useful points which have been raised.

Mr. Marshall points out that in giving the percentages of infected game I grouped together all the trypanosomes pathogenic to man and domestic stock, and that this might lead to misconception. In our published papers Dr. Kinghorn and I have separated the parasites one from the other, and full particulars are given as to number of each antelope infected with each of the various pathogenic trypanosomes. I might state here that at Nawalia in the Luangwa Valley 16 per cent., and at Ngoa on the Congo-Zambesi watershed 3·3 per cent., of the wild animals were infected with *Trypanosoma rhodesiense*. The statement that in my published papers I have throughout assumed the disease to be a new one in these countries, and not even contemplated the other alternative, is incorrect. Whether the disease be new or old appears to me to be only of secondary importance. What is of the first importance, however, is whether or not the disease is spreading—whether more cases are occurring now than, say, eight or ten years ago. I admit that from the statistics available the question is difficult to answer; but what evidence there is suggests strongly that during the past few years sleeping sickness has been on the increase. Obviously the most reliable information is to be obtained from an examination of the incidence of the disease in Europeans. I cannot think it likely that even anterior to 1909 many white people could have succumbed from trypanosomiasis without the disease having been recognised; yet in spite of the fact that the main roads to the Congo-Zambesi watershed crossed the Luangwa Valley, and that these roads were traversed by

large numbers of European officials and others, it was not until towards the end of 1909 that the first white case was discovered. Since then, although the Luangwa Valley has been closed, quite a number of Europeans have contracted the disease in North-Eastern Rhodesia. Moreover, we must remember that some time before 1909 scientific experts had travelled through the Luangwa Valley without discovering a single case either native or European.

The hypothesis that human beings can harbour the parasite for long periods of time, as do the wild fauna, without exhibiting signs of disease, is one which I cannot support. Amongst the cases discovered by us, several presented practically no symptoms, the only indication of the disease being the presence of trypanosomes in the blood; yet without exception they were all dead within six months.

Professor Minchin suggests that if the game in a "fly" area were destroyed, the "fly" would enter the villages and attack human beings and the cattle surrounding their habitations. To this objection I have already referred in my paper. Cattle are not as a rule found in villages situated in "fly" districts, and the "fly" does not invade villages around which there is a clearing, even though at the present time they could by so doing obtain food still more readily than they do in the bush.

Regarding the letter of the Hon. Walter Rothschild, I have already in my paper discussed the suggestion that after the extermination of the game the fly would be driven to attack monkeys, rats, and mice. Even if this occurred, these animals quickly succumb from trypanosomiasis, and therefore cannot have the same significance as reservoirs of the disease as the big game which are tolerant of the parasites.

With regard to the first objection raised by Sir Henry Seton-Karr, I must ask what are the other reservoirs of the virus; and if there be any, is that a reason why the main reservoir should not be destroyed? Passing to the second point, I submit that the connection between big game and sleeping sickness has been fully and amply established. As to the third point, the greater freedom of intercourse among native tribes under white protection has undoubtedly played a part in the spread of the disease. But if civilisation and progress is to continue, this freedom of intercourse must also continue, and the population will of necessity run more risk of infection than previously. Recognition of this fact makes it all the more essential that we should endeavour to render the "fly" as non-infective as possible by destroying the reservoir of the virus. As the last point is fully discussed in my paper, I need not refer to it again.

In reply to Sir Alfred Sharpe, I must point out that we have established beyond all doubt that *Glossina morsitans* does transmit sleeping sickness under natural conditions. I cannot see how further knowledge as to whether there are other distributing agents affects the question, beyond the fact that if this be proved

to be the case it merely emphasizes the almost hopelessness of attempting to get rid of the transmitting agents, and demonstrates clearly that if anything is to be done it must be in the direction of destroying the reservoir from which *Glossina morsitans* and the other (hypothetical) vectors derive their infection.

As I pointed out in my paper, whether the tsetse fly depends entirely on game for its existence is beside the question. This subject has already been most fully discussed on more or less theoretical grounds, and we now require facts, and not further discussion. Definite information on this, as on many other points raised in this discussion, can only be obtained by means of the limited experiment which I advocate.

In conclusion, may I state my position once more. I have not, as some of the speakers appear to think, made the wild statement that the whole of the game in Tropical Africa should be destroyed, but merely that the restrictions should be removed in "fly" areas, and that natives and Europeans should be encouraged to kill game in these areas, especially in the vicinity of human habitations. Finally, I am strongly of the opinion that some such limited experiment as I have outlined should be undertaken, as it is only by this means that the data necessary for our guidance in the future can be accumulated.