

TRYPANOSOMIASIS IN THE EAST AFRICAN CAMPAIGN.

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IN the course of a military campaign in Tropical Africa, it was only to be anticipated that a considerable amount of sickness due to those diseases, which are of common occurrence in the tropics, would have to be contended with.

Amongst these malaria, dysentery (both amoebic and bacillary) and relapsing fever have furnished the vast bulk of the sickness met with among the troops and porters engaged in the East African Campaign.

Further, considering that operations were frequently taking place in areas known to be infected with trypanosomiasis, it was expected that instances of this disease were fairly certain to occur. However, although active operations have been in progress since 1914, it was not till March, 1918, that any instance of this disease was encountered. It is quite possible that unrecognized cases of this disease may have occurred before this date, but it is hardly likely that many can have occurred without suspicion as to their true nature being engendered.

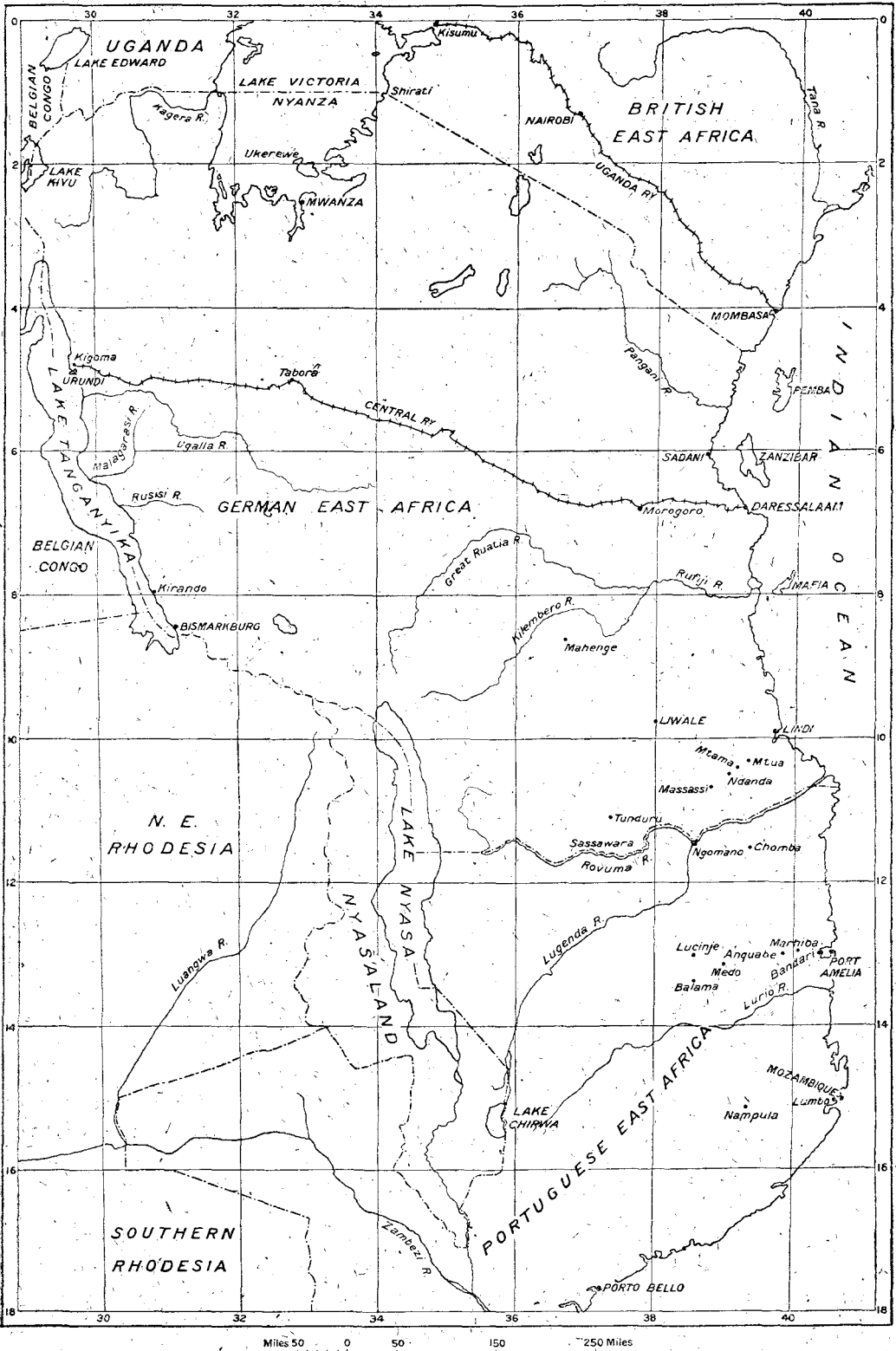
At the same time it should be pointed out that operations in the earlier part of the campaign were conducted in parts of the country not known to be infected, and it was not until the troops reached the southern part of German East Africa, in the later part of 1917, and were close to the Rovuma river, that they were fighting in an area definitely known to be infected.

GEOGRAPHICAL DISTRIBUTION.

Very little accurate knowledge is available as to the exact distribution of the disease in German East Africa, nor has much detailed work been done on the types of *Glossina* to be met with, or their areas of distribution been properly delimited.

Before the war the Germans had investigated these points to a limited extent. Thus Dr. Marshall writes in 1908, that there were two foci of sleeping sickness on Lake Victoria, and one on Tanganyika. The disease had also been discovered on the islands Bumbire, and Iroba, and on the mainland of Ihangiro, opposite to them. The fly had not been found at the Kagero river, nor on the shore of the Lake near its mouth. *Glossina palpalis* was found widely distributed on the shore of the lake, and on the rivers in the Shirati district, and sleeping sickness was very prevalent there.

Cases of the disease had also been found on Ukerewe Island. As for the distribution of *G. palpalis* on the Lake there were only a few points at which the presence of this fly could be excluded.



The conditions on Tanganyika were worse even than those prevailing on the Shirati coast. The large rivers Malagarasi and Rusisi were fly haunted. At Urundi the percentage of infection was between sixty and ninety. Of 200 natives examined at Kirandö (eighty miles north of Bismarckburg) fifteen were infected.

In 1910 Wolf met with a case of trypanosomiasis, which came from the Rovuma river near its junction with the Sassawara river. In 1911 an extraordinary mortality of the natives in this area was reported, and on investigation this was found to be due to trypanosomiasis.

Following on this discovery search was made for tsetse, and Wolf reports "In spite of every search being made it was not possible to find in any part of upper Rovuma area the *G. palpalis*. After painstaking searches, it was established, that the *G. morsitans* is the transmitter of the Rovuma sleeping sickness in this area, and that the trypanosome shows characteristic peculiarities from the *Trypanosoma gambiense*. The trypanosoma of the Rovuma is identical with the *T. rhodesiense* discovered about 1909 by English physicians."

Professor Beck, as a result of his investigations, stated that eight to ten per cent of the *G. morsitans* in the Rovuma area were infected with trypanosomes.

Wolf in his report, further states "German doctors who have worked on the Rovuma consider it certain, that the principle source of infection is to be sought in the thickly populated districts of the Lugenda Valley, and especially in Kumembe (Ngomano). The Lugenda river flows through the Portuguese Colony in a north-easterly direction to enter the Rovuma near Ngomano. In 1917 five cases of sleeping sickness were identified in the hinterland of Kilwa on Lake Kiwlimira. Here there are no *G. palpalis*, so that *G. morsitans* must be accepted as the transmitting agent. It agrees likewise to the *T. rhodesiense*. I believe that the sleeping sickness from the Rovuma area or the Portuguese territory has been carried past Liwale."

From these few extracts of German writers it would appear that the known infected areas in German East Africa are the southern border of Lake Victoria Nyanza, the vicinity of Lake Tanganyika, and the neighbourhood of the Rovuma river (which forms the boundary between the German and Portuguese Colonies), from Sassawara on the west to Liwale on the east.

Practically nothing was known before the war as to any infected areas in Portuguese East Africa. As has been shown above, the German doctors were confident that the Portuguese border of the Rovuma river was infected, but I can find no records of any investigations by the Portuguese authorities to ascertain the distribution of the disease in their territory.

In the course of military operations undertaken by us in Portuguese territory it became necessary to open up certain long lines of communication, and of these a line running from Port Amelia through Anquabe, Medo

Balama to Lucinje was the first. It was from this line that the first case of trypanosomiasis to be met with among the troops was derived. From a consideration of the history of this case, and of the other four cases of the disease in white men which subsequently occurred, it will be seen that infection in at least four of the cases must have been contracted between the coast and Anquabe, a distance of some forty-five miles up the line.

The Port Amelia line started from Bandari on the opposite side of the bay to Port Amelia itself, and ran first to Mahiba, a distance of fourteen miles, and thence to Anquabe, a further distance of some thirty miles. Thence it ran on to Medo, Balama, etc.

From Bandari to Anquabe the road runs the whole way through fairly thick bush. Several small streams run across this road, but in the dry weather dry up, though possibly pools may be left here and there where the streams run through the bush. On the occasion of my visit to this area the streams were dry, and I had no opportunity of following them up through the bush. Tsetse-flies were encountered in moderate numbers along the road, but were only noticed at Mahiba, and for a distance of some seven miles beyond this place in the direction of Anquabe. Captain McGregor, attached R.A.M.C., has investigated the fly of this area, and reports that he met with *G. morsitans* and *G. pallidipes*, but never saw a specimen of *G. palpalis*. *G. pallidipes* was by far the more common variety. He comments on the great variation in the numbers of flies encountered from day to day on this road. Usually the flies were in moderate numbers, but on one occasion he was attacked by veritable swarms. Other observers also have commented on the great variation in the numbers of flies encountered. One officer informed me that he had traversed this stretch of road without seeing a single fly, though he was on the look-out for them. In numerous journeys up and down this road McGregor first met flies at about seven miles from Bandari, and from that point to Anquabe they were met with here and there as far as Anquabe itself, the chief belt being for about six to seven miles extending from Mahiba in the Anquabe direction. Flies are known to occur beyond Anquabe, but no exact work on their distribution has been done. It is of interest to note that on this particular stretch of road between Bandari and Anquabe wild animals are extremely scarce, and, moreover, there are practically no natives living on this road, except a few at Mahiba and Anquabe. I am informed however, by officers who have penetrated the surrounding bush, that at a distance of about two miles from the road game is to be found in fair numbers, and it is presumably from this source that the flies derive their infection.

Another line of communication in Portuguese territory ran from Lumbo on the mainland opposite Mozambique in a direction practically east and west through Nampula towards Lake Nyassa.

McGregor informed me that he had never met with any fly between Lumbo and Nampula. The Army Veterinary Officers reported that beyond Nampula the fly belts are very extensive and reach to Lake Nyassa, and

that a great deal of trypanosomiasis exists in horses and cattle. Numerous specimens of flies sent to McGregor from this area were identified by him as *G. morsitans*, *G. pallidipes* and *G. fusca* in order of prevalence. One case of trypanosomiasis in a native was discovered at Lumbo, but it was impossible to ascertain where he had contracted the infection.

Recently McGregor has investigated the distribution of flies in the Lindi area. He reports, that as far as Lindi itself is concerned, tsetse are rare. He himself was not able to obtain any specimens here, but he had the opportunity of examining several specimens which had been obtained previously, and identified these as *G. pallidipes*. It was reported to him that both *G. palpalis* and *G. morsitans* had been captured some time before, but as the specimens had been lost their identification could not be confirmed. The general impression he gained was, that though tsetse might occasionally be found in Lindi itself they were undoubtedly scanty at the best of times.

The country immediately surrounding Lindi was described by him as being in many places typical "tsetse" country. Although at the time of his investigation he was only able to catch one living specimen of glossina, which proved to be *G. pallidipes*, in this area he saw several others, and from questioning natives and others he ascertained that tsetse were reasonably common in this district at certain times of the year, usually just after the rains. Captain McGregor next proceeded to investigate the distribution of fly on the road from Lindi to Ngomano on the Rovuma at its juncture with the Lugenda river, and also on the road branching off the Lindi—Ngomano road at mile 108 and proceeding to Tunduru.

Summarizing his investigations he states:—

(1) From Lindi to Ngomano and Tunduru, four species of glossina have been found, viz., *G. morsitans*, *G. pallidipes*, *G. fusca* and *G. brevipalpis*.

(2) *G. morsitans* is far and away the most common of the species, except in the Lindi area, where *G. pallidipes* is the common species, at any rate at this time of the year (November).

(3) Species of *G. fusca* were taken near Mtua, and a male and female *G. brevipalpis* were seen near Luanda on the Tunduru road.

(4) No very sharply defined fly-belts occur anywhere on the line, but there are places where the flies are especially numerous, and other places where the fly does not seem to exist—to be qualified again by saying at this time of the year (November).

(5) The points at which the fly has been found to be especially bad are:—

(a) Between Chirimaka and Mtua; (b) between miles 127-130 on the Ngomano road; (c) between miles 134-139 on the same road; (d) seven miles from Ngomano on the Chomba road, in Portuguese territory; (e) between miles 25-30 on the same road; (f) between miles 125-127 on the Tunduru road; (g) between miles 193-196 on the same road.

Flies can however be found in varying numbers between most of these points.

(6) The parts of the line apparently free from tsetse are:—

(a) From Mtama to Ndanda; (b) from Ndanda to Massassi; (c) from Massassi (mile 93) to mile 108; (d) from a point 6 miles east of Tunduru to Tunduru itself.

(7) The percentage of glossina infected with a species of trypanosome is high, averaging about 11 per cent. At one place however, on the Chomba road, seven out of seventeen flies carried trypanosomes.

(8) No cases of human trypanosomiasis were seen anywhere on the line. Among the post-commandants at the different posts, the political officers, and others, there seems a general belief that sleeping sickness exists. I have met no one, nevertheless, who can give a reason for holding this belief, beyond saying that they "have heard it said," and that "the Germans reported it."

(9) None of the natives or native Jumbes (chiefs) whom I met knew of, or were able to recognize from my description, a disease among them which would point to trypanosomiasis.

(10) I conclude, therefore, that if it exists at all in the areas I have visited, cases must be very rare indeed, and that if there is an endemic centre along the Rovuma, it must be higher up than I was able to get under present conditions.

ETIOLOGY.

It is obviously very difficult indeed to arrive at any estimate of the duration of the period of incubation. It has been suggested by Manson that the actual infective bite in many cases is accompanied by more than usual pain and irritation, which may persist for several days.

Certainly I have seen several cases of the disease from other parts of Africa in which a short time before symptoms of the disease appeared the patient was bitten by tsetse, and remembered one particular bite which caused an excessive amount of irritation, or even severe pain, more intense than had ever been experienced before.

I have made careful inquiries on this point from the five white men who contracted the disease here. Of these only S—n and M—n can remember any particular bite. S—n stated that he had very frequently been bitten by tsetse, and knew the sensation well. On February 25, 1918, he was bitten on the shoulder, and this caused very severe pain lasting for some hours, and was so marked that he asked a comrade to put some iodine on it. On March 2 he was taken ill with what proved to be trypanosomiasis. If we accept this bite as being the infective one, then the period of incubation in this case is of five days only.

In the case of M—n he remembered having been bitten on the lip about a fortnight before he was taken ill. This caused great pain and swelling.

Further, from experiments in monkeys, carried out by Captain Hughes, I.M.S., it was shown that infection in those animals was very readily

obtained by injection of the patient's blood containing trypanosomes. The parasites could be detected in the monkeys in six to seven days, and death in two cases took place on the twelfth day, and in a third case on the seventh day after inoculation.

From these data I am inclined to think that the incubation period is a short one, somewhere between five and fourteen days.

The parasite, as seen in human blood, is a polymorphic trypanosome indistinguishable morphologically from *T. gambiense*. In all cases it occurred in fairly large numbers, and was readily found in the peripheral blood.

Examination of the patient's blood in the living state showed auto-agglutination, especially marked in the case of L—n.

Experiments on rats showed that infection of these animals was readily induced by direct inoculation from man to animal, the period of incubation averaging six days, the rats surviving on the average about twelve days.

Subinoculations from rat to rat followed much the same course, but in none of these experiments on rats were there any true posterior nuclear forms encountered.

On two occasions in inoculated monkeys forms were seen in very scanty numbers approaching the so-called posterior nuclear forms described as occurring in subinoculations of *T. rhodesiense*. These forms were only observed on one day in each case, and although there was considerable dislocation of the nucleus of the parasites, it was never so far displaced as to get behind the micro-nucleus.

I am inclined to consider, however, that the parasite is probably identical with *T. rhodesiense*, or, at any rate, very closely related.

It is true that in our hands the parasite never yielded post-nuclear forms in rats, and only on two occasions in monkeys, but from the rapid way in which animals could be directly infected from man, its marked virulence in them, its non-amenability in man to atoxyl treatment, and the fact that no *G. palpalis* occur in the areas where the disease was contracted, render it highly probable that we have to deal with *T. rhodesiense* rather than with *T. gambiense*.

From the consideration of the following table it will be seen that altogether we have had eighteen cases of the disease, of whom five of the patients were white men, whilst the remainder were natives. This list does not quite exhaust the number of cases which have been recognized, as some five other cases in natives have occurred in various parts of the country, but as they did not come through our hands, and no details as to their condition or the results of treatment are available, we have not included them in the above table. The place of infection in the case of the white men has been easy to determine, as full particulars as to the area in which they were employed are available. Thus S—n, who was a motor driver, worked on the Port Amelia line between Bandari and Anquabe, and was never in any other fly area.

TABLE I.

	Name	Place of infection	Result
Europeans	S—n	Port Amelia Line	Evacuated to South Africa. Died.
	M—n	" "	" "
	L—n	" "	Reported rapidly sinking. "
	M—k	" "	Evacuated to England.
	C—k	? Lugenda Valley	Under treatment.
Natives	Boki (1)	Port Amelia Line	Under treatment. Doing well.
	Mikambo (2)	" "	" "
	Dorakani (3)	" "	Died from acute tick fever with pulmonary symptoms.
	Bikali (4)	" ?	Under treatment. Doing well.
	Somiba (5)	" ?	Was doing very well till he got pneumonia following influenza and died.
	Munguma (6)	Port Amelia Line	Under treatment. Doing well.
	Inwalo (7)	" "	" "
	Haminsi (8)	" "	Was doing well till he contracted pneumonia following influenza and died.
	Nigeria (9)	" "	Under treatment. Doing well.
	Mehago (10)	" ?	Was doing well till he contracted pneumonia following influenza and died.
	Opengo (11)	Port Amelia Line	Died of dysentery shortly after admission to hospital.
	Singali (12)	" "	Died shortly after admission.
	Oroni (13)	" "	" " "

M—n was employed in the Road Corps, and worked on the same stretch of road as S—n.

L—n also was in the Road Corps, and was employed with M—n on the same piece of road.

Both these men had never worked in any other fly area. M—k, who was a dispatch rider, also worked between Bandari and Anquabe. It is true that for a time he had also been farther up the line where tsetse occurred, but a consideration of his case seems to show that the place where he was infected was undoubtedly the same as where the other three derived their infection.

C—k does not appear to have been infected on the Port Amelia line. Up to October 3, 1918, beyond a few attacks of malaria he had kept well. His last attack of malaria was at the beginning of August. On October 3 he started to march from Lucinje to Ngomano, crossing the Lugenda river on October 9, and arriving at Ngomano on October 15. On the night of his arrival at the latter place he had fever. This ran a more or less irregular course, and he was transferred to Lindi where, on his blood being examined, trypanosomes were found.

I regard it as highly probable that infection in this case took place on the march from Lucinje to Ngomano, i.e., in the valley of the Lugenda.

It is noteworthy, that on this march C—k was accompanied by a force of some 2,000 to 3,000 men, and yet he was the only one to be infected. The blood of every man in the force was examined microscopically on arrival at Lindi, and none were found who showed the infection, nor has any case of the disease since arisen amongst these men.

In endeavouring to ascertain where the native cases became infected, one is at once confronted with great difficulties. Most natives have very little idea where they have been. Their ideas of time, distance and loca-

tion are hazy in the extreme. When asked what places they have visited only a very few of them can remember the names of such, and frequently they have no idea of the name of the port at which they disembarked, or from which port they have come, when they re-embarked for the base. It is almost an impossibility to trace their movements from official records. A few of them are reasonably intelligent and can give sound information, but with the majority of them very little information of any value can be obtained. All these native cases were evacuated through Port Amelia, and would therefore have been working on that line, with the exception of the German porters. Of these native cases Nos. 1, 2, 7, 9 give a fairly clear history of having been employed between Bandari and Anquabe and nowhere else on this line, and the inference is very strong, therefore, that it was somewhere between those two places that they derived their infection. Of the cases No. 3, 11, 12, 13, who died shortly after admission to the Carrier Dépôt Hospital at Dar-es-salaam, beyond the fact that they were employed on the Port Amelia line, no information as to their movements is available.

Cases 4, 5 and 10 were German porters, who were taken prisoners from Von Lettow about 200 miles up the Port Amelia line. They had marched with that redoubtable commander over a very large part of both German East Africa and Portuguese East Africa, and it is therefore impossible to say where they were infected. They were under treatment by the Germans for trypanosomiasis when captured.

Cases 6 and 8 can give no information of any value as to their movements, and beyond the fact that they were employed on the Port Amelia line no further information is available.

The date of onset of the disease could only be ascertained in the cases of the white men. With the onset of the initial fever they all thought they had malaria, and it was only later, when their blood was examined, that the true nature of the illness was detected.

It is utterly impossible to say in the cases of the natives when their illness started. The cases with the exception of the German porters were only diagnosed by routine blood examination, when they were admitted to hospital, and it is impossible to say how long the disease had existed in them before they reported sick and came into hospital.

In considering the results of the cases it will be seen that, as far as the white men are concerned, they are bad.

S——n did badly from the first. He never stood the treatment adopted at all well, it was never found possible to increase the doses of antimony up to a point sufficient to control the fever and the parasites, he lost heart, and when evacuated to South Africa where his home was, was undoubtedly a doomed man. His death was reported as occurring in South Africa in August, 1918. The total duration of his illness was a little over five months, but no details of the manner of his death have been received.

M—n also did badly. He was unable to stand the antimony in sufficient doses and died five and a half months after the commencement of his illness, but no details as to the manner of his end are available.

L—n at first did fairly well. The fever appeared to respond favourably to the antimony administered, but he too was unable to stand a sufficient dosage to completely control the fever and parasites. He has slowly got weaker, and latest reports show that a fatal result is to be anticipated.

M—k, on the other hand, did well from the beginning. One was able to push the antimony up to the maximum dosage, and although when he left this country the fever was not completely controlled, he was much better and there is every hope that with a continuance of the treatment in England he should have a good chance of complete recovery.¹

C—k has only been under treatment for a very short time. He is not standing the antimony injections well, and is extremely anæmic, so that one is not able to give a very favourable prognosis.²

The native cases on the other hand appear to respond well to treatment. They were all treated at the Carrier Dépôt Hospital at Dar-es-salaam.

Of the cases that died, in none of them could the cause of death be ascribed to trypanosomiasis alone. No. 3 died a few days after coming in of very acute relapsing fever.

This case is of particular interest in that the patient had marked ascites due to extensive cirrhosis of the liver. His blood showed numerous spirochætes and trypanosomes. Examination of the ascitic fluid withdrawn during life showed large numbers of trypanosomes therein, but no spirochætes. No. 5 died of pneumonia, following influenza. He had been free from trypanosomes in his blood for over three months, and had showed no signs or symptoms of the disease during that time. No. 11, 12, 13, died within a day or two of being admitted to hospital at Dar-es-salaam. Two of these suffered from pneumonia, whilst the other was the subject of profound anæmia, due to ankylostomiasis and dysentery, and was practically moribund when admitted.

No. 10 was extremely lethargic when admitted, and it was thought that the trypanosomiasis had progressed to the sleeping sickness stage. Spinal puncture was performed, but no parasites were found in the fluid withdrawn. He was found, however, to be the subject of marked ankylostomiasis, which undoubtedly was the cause of his lethargy, as under treatment with thymol the lethargic condition rapidly disappeared. He, however, succumbed later to pneumonia, following influenza.

¹ I have since ascertained that this man died of influenza about a month after his arrival in England.

² From a letter received from South Africa dated June, 1919, I have learned that this man has much improved. He has put on weight, stands his antimony treatment well, and parasites are only found in his blood in small numbers and at lengthening intervals.

No. 8 was doing well till he also contracted pneumonia, following influenza, and died.

All other cases at the time of writing are doing well.

SYMPTOMS.

There was a singular absence in nearly all the cases of many of the symptoms usually associated with trypanosome infection. The common symptom was irregular fever associated with a moderate degree of headache; and some lassitude. The white men generally considered that they had got an attack of malaria, whilst in the natives the fever was at first usually ascribed to malaria or relapsing fever. It was only when the cases came under routine blood examination that the true nature of the infecting agent was manifest. The temperature charts in all cases were of an irregular intermittent nature, and showing from time to time exacerbations of the fever associated with increases in the number of parasites in the peripheral blood.

Enlarged Glands.—It has been pointed out that enlargement of the superficial lymphatic glands in the cases of *T. rhodesiense* infection is the exception rather than the rule, and certainly in the cases I have met with here this was well marked.

In only two cases, namely, those of M—n and L—n, was any enlargement of the glands to be detected. In M—n's case only very moderate enlargement of the glands in the posterior triangle of the neck was detected in the first three weeks of his illness. Enlargement of the cervical glands in the case of L—n came on in the later stages of his illness. There was an entire absence of glands of the large "juicy" type so commonly associated with infections with *T. gambiense*. Enlarged glands were never seen in the native cases.

Œdema.—As is well known, local œdema of the eyelids or part of the face is by no means an uncommon symptom in trypanosome infections, but such was only met with in the case of C—k, who stated that this had been noted on one or two occasions previous to his coming into my hands. It has not so far been seen by me.

Splenic Enlargement.—No marked enlargement of the spleen was to be noted in any of the cases. Usually it was just palpable, but in one or two cases the organ extended about one finger's breadth below the costal margin.

Rash.—This was only seen in two cases, viz., L—n and C—k. In L—n it appeared on the left side of the chest in the form of two small erythematous rings in about the sixth week of his illness. It was very poorly marked and disappeared in three days' time. It was noted again in his case some three months later.

The characteristic rash was also seen in C—k on his admission to hospital in Dar-es-salaam. It was confined to the trunk, and disappeared in two days' time.

A very careful outlook for the appearance of the rash in the native cases was maintained, but in none of them was it ever detected.

Kerandel's symptom, or deep hyperæsthesia, was met with in M—k's case only. It was present early in the disease, and was particularly well marked over the shins, but gradually got less as the temperature came more under control as a result of treatment.

Heart.—Easily excited rapidity of the heart's action was observed in all cases in the early stages of the disease. In the native cases, the only ones I was able to watch over a considerable length of time, this disappeared as the fever became thoroughly controlled by treatment.

OTHER SYMPTOMS.

More rarely occurring symptoms, such as periostitis and irido-cyclitis, which have been described as occasionally met with in cases of trypanosomiasis were never encountered. In the case of L—n marked swelling of one testicle was noted late in the disease.

TREATMENT.

Atoxyl seemed quite inadequate to control the disease as met with here. In several cases it was given at first in doses of three grains three times a week, but failed completely to check either the fever or the parasites. It is true that it seemed to reduce somewhat the number of trypanosomes in the blood, but it entirely failed to banish them, even temporarily, from the peripheral blood. Chief reliance was therefore placed on injections of antimony (tartar emetic). Some cases were given atoxyl as well, whilst others were treated on antimony alone. It did not appear that the ones in which both drugs were given showed any better results than those in which antimony alone was administered.

The antimony was given by intravenous injection twice weekly, starting with a dose of one grain of the tartar emetic, and working the dose up until $2\frac{1}{2}$ grains were taken twice weekly. The measure of success seemed to be dependent on the ability of the patient to stand this latter dose. Thus in the case of S—n, M—n and L—n, neither of them was ever able to stand a larger dose than $1\frac{1}{2}$ grains, and this proved inadequate to control either the fever or the parasites. If an attempt was made to give a larger dose marked reaction occurred. Usually a severe rigor took place, accompanied by headache, nausea, or in the case of M—n frequent vomiting. Great depression ensued and marked anorexia.

M—k, on the other hand, tolerated the drug well, and he was having doses of $2\frac{1}{2}$ grains twice weekly and standing it well, before being evacuated to England. The native cases tolerated the antimony exceedingly well. Beyond a little coughing at the end of the injection, which passed off in about five minutes, no untoward symptoms were observed and none complained of headache or any other symptoms. In the case of Boki it took practically three months of treatment on these lines before the trypanosome condition was thoroughly under control; no trypanosomes were found in the blood after this time in spite of daily examinations for

a further period of over three months, when a relapse occurred, and the parasites again appeared in the blood for two days, and slight fever occurred. The dose of antimony was then increased to three grains twice weekly, which caused no inconvenience.

Other native cases followed much the same course. They mostly took about two months treatment before the disease could be said to be under control, and three of the cases showed a subsequent relapse at intervals of one to two and a half months. Whenever a relapse occurred after a considerable interval of freedom from parasites, the dose of the drug was at once increased to three grains twice weekly, and one and all stood this very well. The cases were all controlled by daily blood examinations of thick films.

An interesting point of these native cases is that in practically every one of them relapses of benign tertian malaria occurred from time to time. Frequently when the trypanosomes were well under control, when none had been found in the blood for several weeks, and the patient was receiving the full doses of antimony, a sudden rise of temperature would take place suggesting a relapse due to trypanosomes, which, on blood examination, would prove to be due to a benign tertian infection.

This phenomenon was seen in ten of the native cases. Further, Cases 1 and 3 showed infection also with *Sp. duttoni*. As has been stated, Case 3 died of acute tick fever with the blood crowded with spirochætes, and with severe pulmonary involvement.

Case 1—Boki—showed numerous relapses due in many instances to spirochætes, and on other occasions to benign tertian parasites. These cases are of marked interest as showing how ineffectual tartar emetic is either against *Sp. duttoni* or benign tertian parasites. In spite of prolonged and maximum dosage with tartar emetic, these parasites continued to thrive, and from time to time manifest their presence, causing a rise of temperature.

PROPHYLAXIS.

As will be seen from the foregoing, undoubtedly the chief focus of the disease we had to deal with was on the Port Amelia line, between Bandari and Anquabe. It is remarkable that, with the continuous movement of troops and porters which was taking place along this line, more people were not infected.

As soon as the first case of the disease was discovered, measures were taken as far as was possible to protect the men. Clearing of the bush on both sides of the road was quite impracticable.

The area was much too large, and would have entailed an enormous amount of labour over a long time to make any impression, and such labour was not obtainable. Steps were therefore taken to protect as far as possible the regular users of this road, such as motor drivers, dispatch riders, road corps men, etc. Veils were served out to protect the head and neck, the use of shorts was forbidden, and bamber oil was issued for the frequent anointing of the hands and bare parts of the arms.