LIVERPOOL SCHOOL OF TROPICAL MEDICINE—MEMOIR XIII

REPORTS OF THE TRYPANOSOMIASIS EXPEDITION TO THE CONGO

1903-1904

OF THE

LIVERPOOL SCHOOL OF TROPICAL MEDICINE AND MEDICAL PARASITOLOGY

BY

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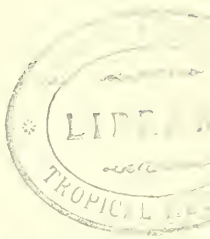

WITH A COMPARISON OF THE TRYPANOSOMES OF UGANDA AND THE CONGO FREE STATE

BY

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PREFACE

IN 1901 trypanosomes were discovered in the blood of a European by Dr. J. E. DUTTON, Walter Myers Fellow, while on an Expedition of the Liverpool School of Tropical Medicine to Gambia. In consequence of this observation an Expedition composed of Drs. DUTTON and TODD was sent in 1902 by the School to Senegambia to prosecute further researches in trypanosomiasis. The detailed report of the Expedition was published in 1903, and contained a study of the pathogenic trypanosomata of man and animals, several new species being described.

Prior to the return of this Expedition, the discovery of trypanosomes in the cerebro-spinal fluid of cases of Sleeping Sickness in Uganda by members of the Sleeping Sickness Commission of the Royal Society caused the subject of trypanosomiasis to assume great importance. At the same time it was brought to the notice of the Committee of the Liverpool School that in the Congo Free State the native population had from time to time suffered from very fatal epidemics of this disease. The Committee therefore decided to accept the invitation of His Majesty King LEOPOLD to send an Expedition to study Sleeping Sickness in that country. Drs. DUTTON and TODD were recalled from the Senegambia, and as soon as they had drawn up their reports they left for the Congo in September, 1903, and were soon after joined by Dr. CHRISTY, who had served previously on the Royal Society's Sleeping Sickness Commission in Uganda. On reaching the Congo the Expedition decided to make Leopoldville its headquarters. The authorities of the Free State at the same time attached Dr. INGE HEIBERG, an old pupil of the School, to the Expedition, and to him the members are greatly indebted for his aid in the work. A special hospital was erected by the State, in order that the observers might have the Sleeping Sickness cases under their care, and facilities were given for the study of a large number of patients. The results of these investigators are incorporated in the present volume, and illustrate the occurrence and distribution; describe the symptoms of trypanosomiasis in all its stages, both in Europeans and natives, and shew how Sleeping Sickness, so-called, is related to trypanosomiasis as a symptom of that disease.

At the same time the Committee resolved to continue the researches on trypanosomiasis in Liverpool, which had been started by Drs. Dutton and Todd in Senegambia ; Dr. Thomas was appointed to conduct the work, and aided by Dr. Linton experiments were immediately commenced, a preliminary note of their work being embodied in this report. The two groups of observers have throughout worked together, and in order that comparable data might be obtained, selected cases of Sleeping Sickness were, by permission of the Congo Free State authorities, sent to the observers in Liverpool. A later report will be published on these cases. As far as the very numerous and detailed observations of these workers go, they shew that the parasite identified with Sleeping Sickness in Uganda and the Congo does not differ from that described by Dutton in the Gambia. This view is also held by Laveran and Mesnil in France, and Bruce in this country. The question of a curative agent has for a considerable time engaged the attention of the members of the research, and experiments are now in progress to find a remedial agent which would have the same effect in trypanosomiasis that quinine has in malaria. A variety of drugs have been used with more or less success ; up-to-date, arsenic and trypan red, an aniline dye introduced by Ehrlich and Shiga, appear to be the most useful ; the parasite disappears for a time from the blood, and the life of the animal is prolonged, but with neither of the drugs is an absolute cure attained. A combination of the two appears to offer better results ; a large number of animals infected with different trypanosomes are under treatment. The present report also embodies an important note on the Tsetse-flies, by Mr. E. E. Austen, to whom the School is much indebted for describing and identifying the Diptera obtained during the Expedition.

Much important work remains to be done ; a further study of the disease from a clinical aspect, extended experiments on the transmission of trypanosomic diseases by biting flies, and researches on the lines of Schaudinn's work, together with therapeutical observations in patients and large animals naturally infected with trypanosomes, are urgently needed.

CONTENTS

HUMAN TRYPANOSOMIASIS ON THE CONGO

HUMAN TRYPANOSOMIASIS ON THE CONGO

(Being the First Progress Report of the Expedition of the Liverpool School of Tropical Medicine to the Congo, 1903) *

BY

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AT the request of His Majesty King Leopold II, King of the Belgians, this expedition was sent in September last to the Congo Free State to report upon the sanitation of the larger towns, and to continue the School's work on human trypanosomiasis. Through the kindness of the Governor-General of the Congo, and of Drs. Vourloud and Neilseng, government physicians at Boma, the hospital for natives at Boma was opened to us. It contained about sixty-five patients, and we saw there eighteen patients who had been admitted as cases of sleeping sickness.

Whether admitted to hospital by the medical officers, or pointed out to us by the missionaries as cases of sleeping sickness, with three or four exceptions, the cases which we have as yet seen have been, in our opinion, very unlike what has hitherto been described as sleeping sickness. Continued sleep or even abnormal sleep has been almost absent from many of the cases. It has been absent even in those who were believed to be in an advanced stage of the disease and who ultimately died. In only three or four of the cases observed by us has somnolence been a marked feature, and only in these few cases have the symptoms in any way coincided with those observed by one of us during the Uganda epidemic of sleeping sickness.[1]

From November 4 to November 29 two of us were occupied in travelling through the cataract region, in order to ascertain for ourselves the exact conditions existing there. Reports sent to the Congo Free State officials, and handed on to us, as well as correspondence received by ourselves, led us to believe that in this district we should find, not only an epidemic of sleeping sickness, but that the whole population was being 'decimated' by the disease. Although, in the course of our

* *Reprinted by permission of the publishers of the* British Medical Journal, *January* 23, 1904

journey, we visited many villages and made tiresome expeditions to visit those especially mentioned to us, not a single case of illness did we see in which sleep was a marked feature.

We stayed for some time at the Baptist Missionary Society's station at Wathen, in the Lutete district, where we received hospitality and assistance at the hands of those in charge of the mission. From here excursions were made into the surrounding district, and a large number of persons brought to us as cases of sleeping sickness were examined.

These consisted of a heterogeneous collection of men, women, and children, many of whom were found to be suffering from heart, lung, and other more or less common ailments. Amongst the boys it seemed that a diagnosis of 'worms' was sufficient in many cases to account for the symptoms. Cases of apparent starvation and neglect were also common, and it appeared from what we were told that, owing to there being a general belief in the contagiousness of 'manimba'—the native name for what is believed to be sleeping sickness—children and even adults were, as soon as the slightest symptoms developed, liable to be isolated and shunned by everyone, causing, eventually, a state of emaciation and filth which ended sooner or later in death. Apart from these, however, and eliminating the many common ailments, there still remains in the cataract region a class of cases which, undoubtedly, terminate fatally within a year or two. These cases, of which most villages visited by us contained one to three examples, have few very evident symptoms of illness beyond emaciation, and, in some cases, weakness, headache, enlargement of lymphatic glands, and dirty, dry, scurvy skin. In a proportion of these cases trypanosomes were found in the peripheral blood, and we think it probable that if a systematic examination were possible, the parasites would be found in a much larger number.

Trypanosomes have been found in the finger blood both of those cases in which the diagnosis of sleeping sickness was certain, and of those in which the case picture was atypical. In addition, trypanosomes have been frequently seen in the peripheral blood of apparently healthy individuals. The routine method adopted for the detection of the parasites in the peripheral blood of unsuspected cases was the simple examination of a rather thick, freshly-made, cover-slip preparation. All of the following persons were examined in this way :—

Place and Class of Native	Number Examined	Number Infected with Trypanosomes in Peripheral Blood	Number of Cases Admitted to Hospital or shown to us as Cases of Sleeping Sickness	
			With Trypanosomes	Without Trypanosomes
Boma				
1. Hospital at Boma. Native soldiers coming from all parts of the Congo	72	11	9 *	7
2. Prisoners in Boma gaol coming from all parts of the Congo	181	17	0	0
3. Children of native soldiers at Boma	19	0	0	0
4. Colony school. Native boys from all parts of the Congo	50	1	0	0
5. Native labourers and children from native quarter	34	0	0	0
Matadi				
1. Natives collected for examination by Dr. Sims	78	1	0	0
2. Children of native soldiers up to 8 years of age	10	0	0	0
3. Patients at the native hospital	22	3	2	2
4. Children at the Sansel, native quarters, up to 10 years of age	28	0	0	0
Cataract Region				
1. Carriers collected from Tumbar District ...	20	2	0	0
2. Carriers from Lutete District	23	0	0	0
3. Boys at Wathen Mission, ages up to 15 ...	35	1	0†	0
4. Natives examined indiscriminately at two small villages near Lutete	42	2	0	2
5. Natives coming to the mission for treatment, or sick natives seen in village within a twenty mile radius of Lutete	79	11	10	47
6. Cases collected for us as sleeping sickness cases by Chef de Post at Kusu from neighbouring villages	14	0	0	14

* Lumbar puncture was done in six of the sleeping sickness cases. In four trypanosomes were seen in the cerebro-spinal fluid. One of the four never showed trypanosomes in the peripheral blood.

† This boy had been suffering for twenty-four days previous to our arrival at the mission from a fever which was not amenable to quinine.

Case VI

Sleeping Sickness (September 30, 1903).—N'Bela, male, aged 24, agricultural labourer. A Mongo man. Patient never saw sleeping sickness before coming to Boma. Had lived in Boma for nearly three years. Illness commenced in July of this year. Admitted to hospital, August 1, 1903.

When the patient was seen (September 30) somnolence was already a marked feature of his condition. This symptom steadily became more marked as emaciation increased. When we left Boma (October 27) the patient was comatose, and death had been expected at any time during the preceding week.

General Condition.—Patient is thin and very weak. Questions are answered rationally, but only after a long interval. He displays a somnolent indifference to everything about him, and only by constant shaking can drowsiness be dispelled for a period long enough to permit him to speak.

Physical Examination.—Nervous system; co-ordination imperfect. Sense of weight good. Knee-jerks and superficial reflexes were obtainable, and showed no abnormality. Eyes reacted to light and to accommodation. Thoracic and abdominal examination showed nothing worthy of note. Lymphatic glands were all enlarged, hard, and freely movable. There was no haemorrhage into mucous membranes. Slight icterus of the conjunctiva was seen.

The accompanying chart indicates the course of the temperature in this case, and shows the results of the examinations for parasites. Trypanosomes were seen in both cerebro-spinal fluid and peripheral blood.

On October 9, 35 c.cm. of slightly clouded, colourless cerebro-spinal fluid were taken by lumbar puncture. No red cells were seen in the fairly profuse deposit formed by centrifuging. This precipitate contained a fair number of trypanosomes, many monuclear cells of large size, and numerous polymorphonuclear leucocytes.

Experimental Inoculation

The following animals have been inoculated from this case. The material inoculated was in each case demonstrated to contain living trypanosomes.

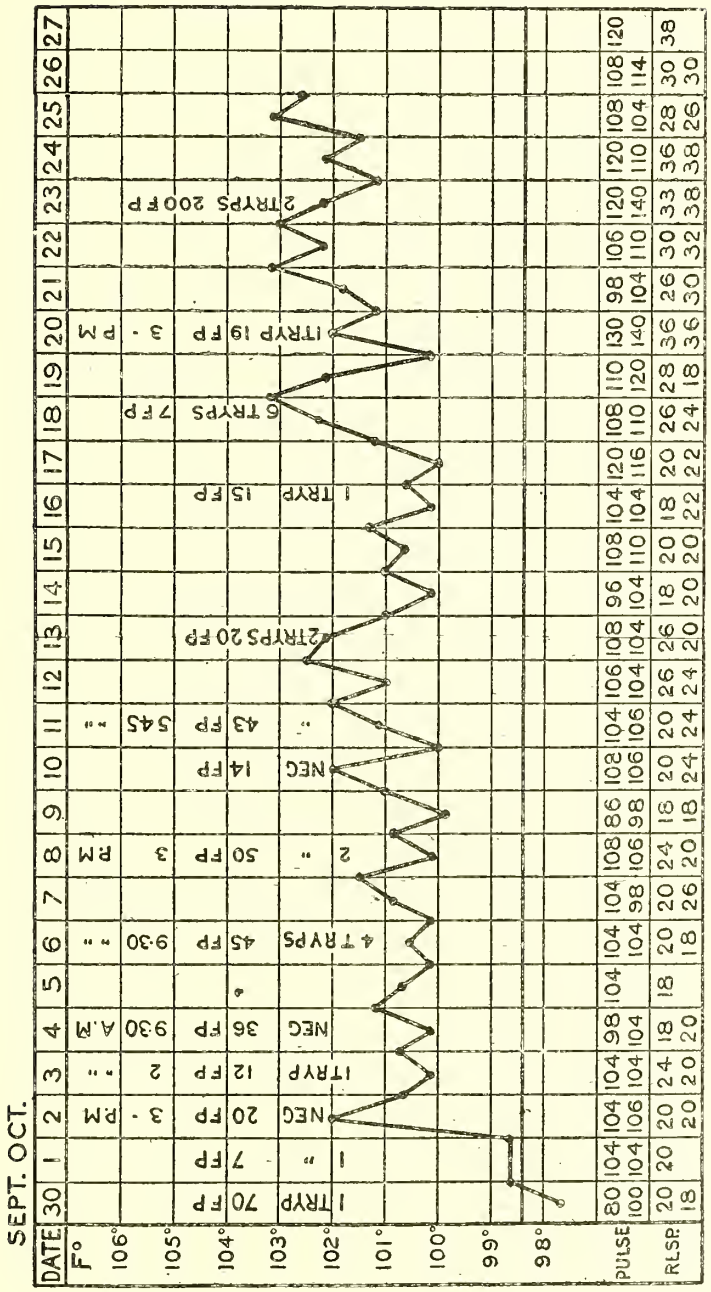
Animals Inoculated from Case 6

October 7. White mouse (Experiment 16) inoculated subcutaneously with 1 c.cm. cerebro-spinal fluid; infected October 25.

October 7. White mouse (Experiment 17) inoculated subcutaneously with 1 c.cm. cerebro-spinal fluid; never infected.

September 30. White rat (Experiment 5) inoculated subcutaneously with 5 c.cm. blood; never infected.

September 30. White rat (Experiment 6) inoculated intraperitoneally with 1 c.cm. blood; never infected.



TEMPERATURE CHART, CASE VI

Tryp. = Trypanosome. F. P. = Filaria perstans

October 9. White rat (Experiment 21) inoculated intraperitoneally with 4 c.cm. blood ; never infected.

October 9. White rat (Experiment 22) inoculated subcutaneously with 8 c.cm. blood ; infected October 23.

October 9. White rat (Experiment 23) inoculated intraperitoneally with 5 c.cm. blood ; infected October 23.

Case IV

Simple Trypanosomiasis (‘*Maladie de Dutton*’)[2], September 22.—J. P., male, aged twenty-eight, native of Sierra Leone, where sleeping sickness is not endemic. Came to Boma six years ago as a Free State soldier. Has always been in lower river districts. Entered hospital September 22 with gunshot wound. Had gonorrhoea in 1902, otherwise has not been ill during his stay in the Congo.

Patient is a strong, healthy man, well nourished, skin moist and clean ; slight oedema over both shins ; patient does not complain of feeling ill.

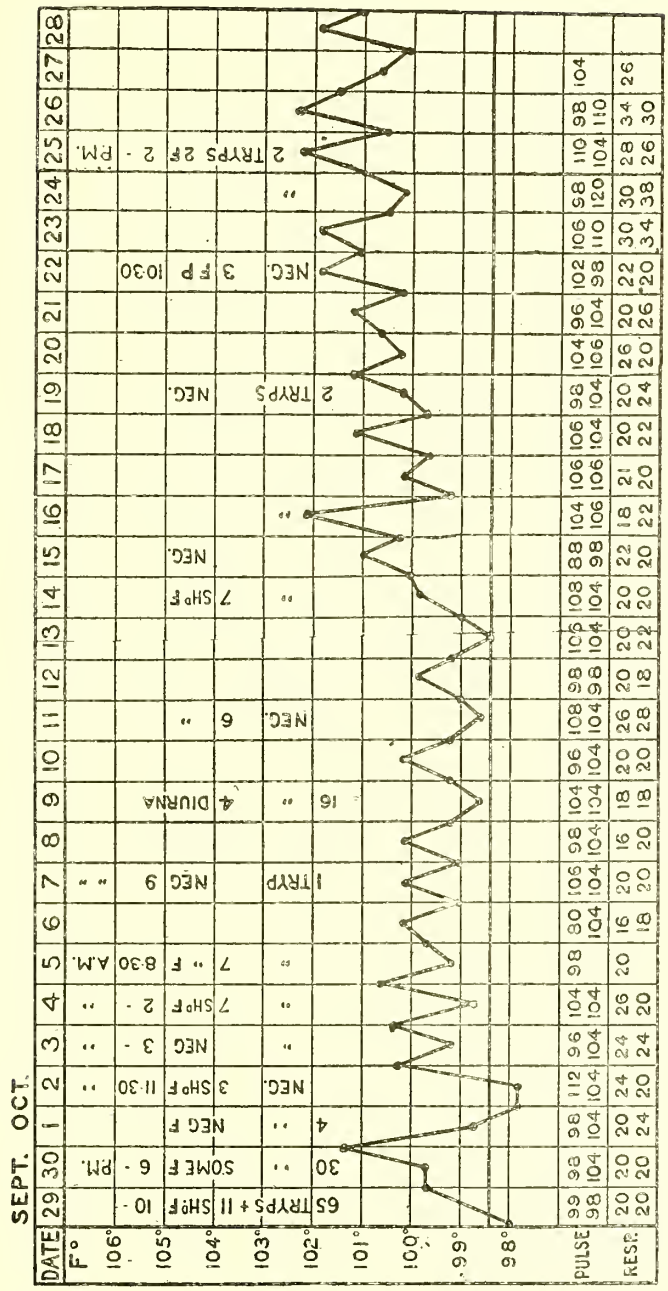
Glands are easily palpable, but are not markedly enlarged or hard. He is a bright, intelligent man, slightly deaf, but answers questions quickly and well. He is alert and interested in his surroundings. Mucous membranes are anaemic. There is a complete cataract of right eye. Heart and lungs are normal. Liver normal in size, but slightly tender. Spleen normal. Appetite good. Bowels constipated. Nervous system is normal.

Urine passed in twenty-four hours, 760 c.cm. ; specific gravity, 1,002, light straw colour, cloudy precipitate, acid ; small amount of albumen present, no sugar ; urea, 0·41 gram to 100 c.cm. of urine. Microscopically a few pus cells, probably due to a chronic gonorrhoea, were seen.

On October 26, 10 c.cm. of limpid cerebro-spinal fluid, as clear as distilled water, were withdrawn with some difficulty by lumbar puncture. The patient almost fainted before 8 c.cm. had been withdrawn. A very slight percipitate was obtained after long centrifuging. On examination it was found to contain a very few red cells, and still fewer small mononuclear white cells. No trypanosomes were seen during a long and careful search of the whole of the precipitate.

The cerebro-spinal fluid from this case presented a very different appearance, both macroscopically and microscopically, from that obtained from sleeping sickness cases. The fluid was clear, not clouded. None of the large mononuclear or smaller mononuclear and polymorphonuclear leucocytes seen in sleeping sickness cases were present.

The accompanying chart shows the course of the temperature in this case, and indicates the occasions on which trypanosomes were seen in the peripheral blood.



TEMPERATURE CHART, CASE IV

Tryp. = Trypanosome. F. P. = Filaria perstans. Sh^d F. = Sheathed filaria.

Experimental Inoculations

The following animals were inoculated from this case :—

Animals Inoculated from Case 4

September 30. White rat (Experiment 5) inoculated subcutaneously with 5 c.cm. blood ; never infected.

September 30. White rat (Experiment 4) inoculated subcutaneously with 1 c.cm. blood ; infected October 8.

October 27. Guinea-pig (Experiment 27) inoculated subcutaneously with 4 c.cm. blood, infected November 7.

October 27. Guinea-pig (Experiment 28) inoculated subcutaneously with 5 c.cm. blood ; infected November 18.

October 27. Rabbit (Experiment 24) inoculated subcutaneously with 6 c.cm. blood ; never infected.

Necropsies were done at Boma on four cases which were admitted to the hospital for sleeping sickness. Two of these died before their blood or cerebro-spinal fluid could be examined. Repeated examinations of the blood (centrifuge used) of the third failed to demonstrate trypanosomes. Lumbar puncture was not done on this case. In spite of repeated and very careful examinations of the blood and cerebro-spinal fluid of the fourth case, trypanosomes were never seen.

A necropsy was also done on the body of a native admitted to the hospital as a case of encephalitis. This patient never showed the usual signs of sleeping sickness, and finally died of dysentry. Many trypanosomes were seen in his finger blood. Lumbar puncture was not done.

The *post-mortem* appearances in each of these cases were very similar to those described as occurring in sleeping sickness.[3] The usual increase of subarachnoid fluid, which had become cloudy, or occasionally almost purulent, was observed. The superficial and substantial vessels of the brain and spinal cord were turgid, and in two cases small sub-ependymal haemorrhages were noted.

In addition to these changes in each case, lymphatic glands were enlarged, several were congested or injected, not infrequently members of the various groups of glands were to the eye either partially or—especially the smallest glands—totally haemorrhagic. The naked-eye appearances of these glands were particularly interesting to us since we have observed very similar changes in animals infected by us with *Trypanosoma gambiense*.

Experimental Inoculations

The following experimental inoculations were made in white rats, white mice, rabbits, and guinea-pigs, with the results indicated :—

Twenty white rats were inoculated with cerebro-spinal fluid or blood taken

either during life or *post-mortem* from patients admitted to hospital as cases of sleeping sickness and from cases of trypanosomiasis. Living trypanosomes in eleven instances were seen in the material inoculated. In seven of these the material inoculated was from cases of 'sleeping sickness,' in four from cases of simple trypanosomiasis. Of the former, three became infected; of the latter, two. None of the rats inoculated with material taken *post-mortem*, in which no living trypanosomes were seen, have ever become infected.

Four white mice were inoculated with cerebro-spinal fluid taken from two cases of sleeping sickness. Trypanosomes had been found in both cerebro-spinal fluid and blood of the first case, in the second trypanosomes were never seen. Two mice were inoculated with fluid containing many trypanosomes from the first case. One has become infected. Neither of the mice inoculated from the second case has ever shown parasites.

Two rabbits were inoculated with blood containing trypanosomes. The blood for one experiment came from a case of sleeping sickness, for the other from a case of simple trypanosomiasis. Neither animal has become infected. Four guinea-pigs were inoculated with blood containing trypanosomes, two from a case of sleeping sickness, and two from a case of trypanosomiasis. Both of the latter have become infected.

The very slight susceptibility of laboratory animals to infection with the trypanosomes found in man in the Congo, the great chronicity of the infection produced when inoculation has been successful, and the periodicity with which the parasite has appeared in the peripheral blood of the experimental animals, are points which greatly resemble the animal reactions of *Trypanosoma gambiense*.[6] The number of experiments done is not yet sufficiently large to permit the mention of incubation periods. During the eight or nine weeks which the infected animals have been under observation none of them have ever shown any gross sign of disease.

Conclusions

The examination of trypanosomes seen in stained specimens of blood from cases of trypanosomiasis, from cases of sleeping sickness (typical or doubtful), in the specimens of cerebro-spinal fluid of the latter cases, and in the blood of experimental animals, infected with either of three above-mentioned fluids, has led us to the following conclusions:—

1. The trypanosomes seen in the blood of man, whether symptoms of sleeping sickness were present or not, have always been the same. The number of cases in which trypanosomes from the spinal fluid have been examined is at present too small to permit of a definite description of morphologic characteristics. We have seen forms in the cerebro-spinal fluid similiar to those described by Bruce[3] and Castellani,[4, 5] and also longer forms similar to those seen in the finger blood of the same cases.

2. None of the human trypanosomes seen, whatever their source, have presented any morphologic appearance incompatible with *Trypanosoma gambiense.*

3. The parasites seen in rats inoculated with the cerebro-spinal fluid of cases admitted to the hospital for sleeping sickness are the same as those seen in rats inoculated with the blood of cases of either sleeping sickness or simple trypanosomiasis.

4. The organisms seen in the blood of rats inoculated with trypanosomes from any of the three indicated sources have up to the present shown no differences from those observed in animals infected with *Trypanosoma gambiense.*

We have observed the extraordinarily long forms with prolonged flagella, and the stumpy forms with short flagella described as occuring in animals inoculated with the Gambian parasite.[6]

We have, therefore, no reason to suppose that the organisms seen by us in the Congo are other than *Trypanosoma gambiense.*

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1. Christy, T*he Epidemiology and Etiology of Sleeping Sickness in Equatorial East Africa*, with Clinical Observations, Royal Society Sleeping Sickness Commission, *Report III*, November, 1903.

2. Laveran and Mesnil, *Des Maladies à Trypanosomes, leur Réparition à la Surface du Globe*, Janus, August 15, 1903, pp. 395-402.

3. Bruce, Royal Society Sleeping Sickness Commission, *Report I*, August I, 1903.

4. Castellani, Royal Society Sleeping Sickness Commission, *Report I*, August, 1903.

5. Kruse, *Ueber das Trypanosoma Castellani den Erreger der Schlafkrankheit. Sitzungsberichten der Neiderrhein Gesellschatt f. Natur. u. Heilkunde zu Bonn*, May, 1903.

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HUMAN TRYPANOSOMIASIS AND ITS RELATION TO CONGO SLEEPING SICKNESS

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(Being the Second Progress Report of the Expedition of the Liverpool School of Tropical Medicine to the Congo, 1903) *

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THE Expedition arrived at Leopoldville on November 21, 1903. Two of its members who had made a short tour through the Cataract Region arrived a week later. During their journey through this district, in which Congo Sleeping Sickness was said to be extremely prevalent, many cases, with very anomalous symptoms, but called sleeping sickness, were seen. As already stated in our *First Progress Report*[1] trypanosomes were found in the peripheral blood of some of these cases. It therefore became necessary to commence a clinical study of human trypanosomiasis in order to see what relation it bore to Congo Sleeping Sickness.

Leopoldville offered particular advantages for such a study, and the members of the expedition decided to remain there for a few months. A large bungalow was placed at their disposal by the Congo government, and through the kindness of Dr. GRENADE, the State Medical Officer, and Dr. BRODEN, Director of the Leopoldville Bacteriological Institute, the patients at the native hospital, a good proportion of whom were infected with trypanosomes, were given to the expedition to study. In addition to this the presence of about two thousand state employees—labourers, soldiers, etc.—afforded an easily accessible, healthy, native population.

Later, the hospital for natives was found unsuited for a complete examination of the cases, and a new structure was built and supplied with the necessary appurtenances through the kindness of the local government. The members of the expedition have therefore for four months been in a position to keep patients under continued and careful observation.

* Received for publication, May 23, 1904. 1. *British Medical Journal*, January 23, 1904

The following table indicates the results of examinations of cover-slip preparations of finger blood taken from four hundred and sixty-five natives at Leopoldville. The results of similar examinations, made in the region lying between Stanley Pool and the sea, are appended in order to present a concise idea of the prevalence of human trypanosomiasis in the Congo :—

Class of Native	Number examined	Number with Trypanosomes in Finger Blood	Number of Cases with Trypanosomes previously diagnosed as sleeping sickness
Healthy labourers, prisoners, women, and children, all resident for varying periods in or near Leopoldville, but many of whom are natives of distant parts of the Free State	255	6	0
Outpatients visiting the clinic of the State Doctor at Leopoldville and chosen for examination because of their miserable appearance	53	3	2
Patients admitted by State Doctor to native hospital	157	45	34
Total for Leopoldville	465	54	36
Total of previous examinations at Boma, Matadi, and in the Cataract Region .	707	49	21
Totals	1,172	103	57

In the British Colony of the Gambia only six cases of human trypanosomiasis were found among 1,043 natives.[1] These cases presented no definite symptoms of illness and nothing abnormal was detected, with the possible exception of an occasional rise in temperature and increase of pulse frequency.

In the Congo we have also seen examples of the mild Gambian type, but the majority of our cases have shown marked symptoms of illness. From a close study of these cases it becomes evident that there is no well-defined line of demarcation between these two forms of the disease, though for descriptive purposes we propose to consider them under three main headings, A, B, and C :—

Type A. Cases with no definite symptoms of illness.

„ B. Cases with few symptoms.

„ C. Fatal cases showing well-marked symptoms, the most notable being fever, lassitude, weakness, and wasting.

1. Dutton, J. E., Todd, J. L., *First Report of the Trypanosomiasis Expedition to Senegambia* (1902), Liverpool School of Tropical Medicine, Memoir XI.

It is significant to note that a large percentage—thirty-six out of forty-four of those classed under types B and C—were believed by their friends to be suffering from 'sleeping sickness.'

We have seen cases coming under type C in which no sleep symptoms were ever present, as well as cases in which some of the diagnostic signs of classical sleeping sickness were noted.

We therefore subdivide type C into :—

1. Fatal cases showing no sleep symptoms.
2. Fatal cases showing sleep symptoms.

Here again no sharp division is possible between these two groups, still, we think it not inadvisable to separate them, since, from our experience here we believe that altogether too much prominence, as a diagnostic feature, has been given to what appears to us to be only a minor and inconstant feature of this disease, namely sleep. The prominence given to this symptom has tended to disguise the true nature of Congo Sickness which is, primarily at least, a trypanosome infection.

The following charts and abstracted clinical reports will illustrate the characteristics of the Congo disease.

TYPE 'A'

Cases with no Definite Symptoms of Illness

Case 79. Fariala. Male. Age thirty.

History.—Admitted to hospital for chronic ulcer of thigh, January 7. Is from Lukila, Kasai district ; has been for ten years in the neighbourhood of Leopoldville.

January 17. General condition. Is a well-nourished, sturdy man ; intelligence good, answers questions quickly and well, shows no unsteadiness of gait, and complains of nothing save the ulcer. Skin is dry and dirty ; there are several large scars and cicatrices on knees, ankles, and elbows, said to be due to injuries and burns. Face is pitted by smallpox. Slight oedema of right shin, probably due to old cicatrix.

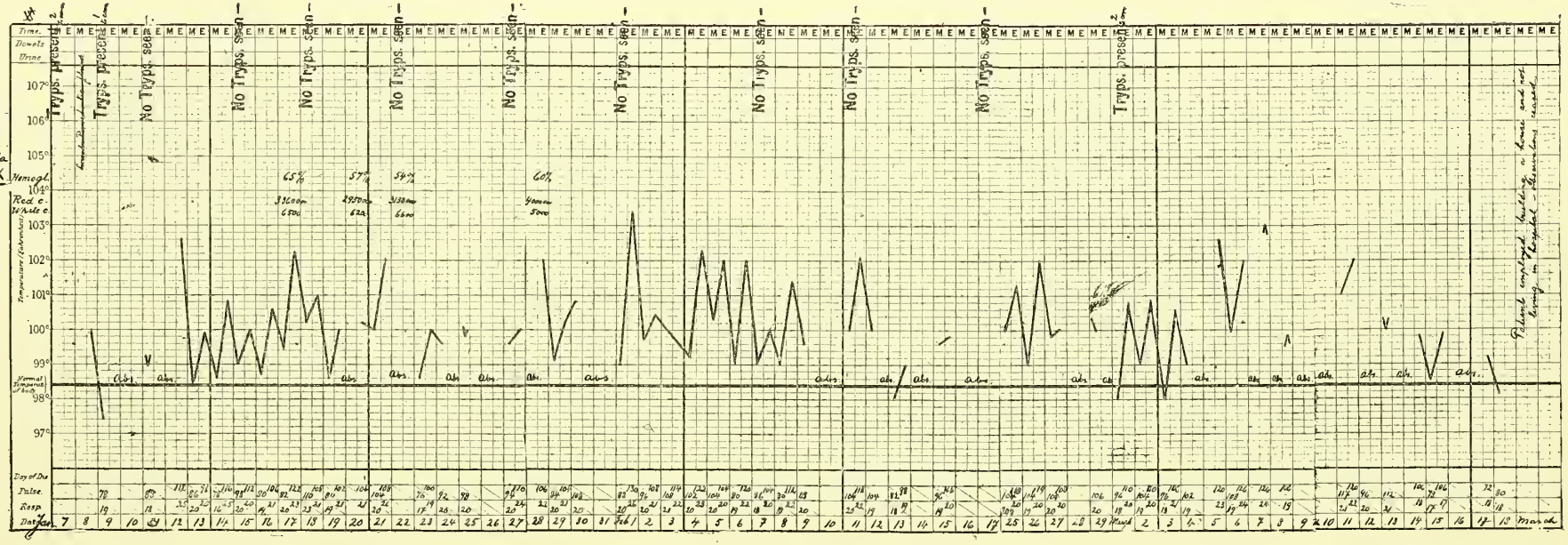
Lymphatic glands are all enlarged. Circulatory system : heart, position and dulness normal, loud mitral systolic bruit, aortic second sound accentuated. Respiratory system : normal. Alimentary system : tongue, teeth, and mouth normal ; bowels regular.

Liver and spleen not enlarged. Nervous system : co-ordination perfect, superficial and deep reflexes normal, pupils react to accommodation and light.

January 25. Complains of nothing, and, save for ulcer, seems quite well. Blood shows filaria diurna, filaria perstans, and trypanosomes.

March 31. Patient at work, and apparently in good health.

CASE LXXIX



Case 94. Kassongo. Male. Age twenty-three.

History.—A Batetela man. Has been for two years in Leopoldville as State labourer. Patient says that he never had fever.

February 23, 1904. General condition : Is a well-nourished, intelligent man, answers questions quickly and well. Has a stolid expression, but no dulness or vacancy whatever, actions not markedly slow. Oedema absent, no puffiness of face. Skin smooth and glossy. Lymphatic glands, all easily palpable, freely movable and hard ; parotids enlarged. Patient believes himself to be in perfect health. A careful physical examination reveals nothing abnormal in thorax or abdomen.

Nervous system. Co-ordination and sensation to touch and pain normal ; superficial and deep reflexes, normal ; pupils react to accommodation and light ; no tremors.

March 31. Physical examination repeated. With exception of a distinctly accentuated aortic second sound patient seems to be absolutely normal. He works willingly and well all day long.

TYPE 'B'

Cases, Intermediate Between Types 'A' and 'C,' showing very few signs of Illness, and not yet Definitely Believed by Their Friends to be Suffering From Sleeping Sickness.

Case 46. M'Pangila. Age seventeen. Male.

History.—Patient is a Lower Congo native. At age of thirteen he was admitted to the Baptist Mission Station at Wathen, Cataract Region. Here he was successively employed as garden boy, table boy, goat herd, and at the age of fifteen as cook. In December, 1902, he complained of fatigue while on the march, and was soon after dismissed because of untidy careless habits. His employers had at this time only a vague suspicion that these might be the prodromal symptoms of sleeping sickness. He had not since been seen until November 13, 1903, when, with another youth, he carried into the Mission a sick child from a village four-and-a-half hours distant. The only changes perceived in the lad were that much less care was taken of his personal appearance than formerly, and he was not so robust.

November 14, 1903. General condition. Patient is thin and has a certain, apathetic stolidity or dulness of expression. Oedema absent. Skin is soft and clean but dry. Lymphatic glands all considerably enlarged. Physical examination reveals nothing abnormal. Pulse, 96-110. Respiration, 20-24.

Temperature, with the exception of an evening rise to occasionally 99·5° F., is almost normal. Patient was sent to England, December 4, 1903. His condition since he has been under observation in Liverpool has not yet been ascertained.[1]

1. August, 1904, condition remains the same—parasites present in the blood.

Case 65. Mokoko. Male. Age twenty.

History.—Patient is a lower Congo native, and comes from a district in which sleeping sickness is present. He left his village a year ago, and has since been employed on a steamer plying on the Upper Congo. November 18, 1903, was admitted to hospital as a possible case of sleeping sickness. He says his illness commenced about the middle of September, with pain in his chest and knees, and a watery diarrhoea, no blood. These symptoms still continue.

December 12, 1903. General condition : Patient is very thin, muscles wasted. He is very weak and can only stand or walk with difficulty. He lies by the fire all day long without sleeping, and when spoken to insists that he has not 'sleeping sickness.' He complains of diarrhoea, and pain and tenderness in both hypochondria and epigastrium. There is no marked oedema. Skin is dry and dirty, slight 'craw-craw.' Lymphatic glands all slightly enlarged and hard. Respiratory system, slight bronchitis. Circulatory system, heart normal. Abdomen, distended and resonant. Liver, lower border not easily made out, apparently normal. Spleen, normal. Nervous system, co-ordination and sensation to touch and pain normal. Reflexes, superficial and deep, obtainable ; each muscle contracts to flicking ; pupils react to accommodation and light. Patient's condition seems to be wholly due to dysentery and bronchitis.

December 27. Functional murmur at pulmonary second sound.

January 5. Yesterday patient's blood showed over two hundred trypanosomes to the cover. His abdomen was distended and his feet oedematous, but there was little complaint. To-day the parasites are, perhaps, three times more numerous, the abdomen is more distended, and the feet oedematous. Patient complains of headache, pains in his joints, and of the abdominal distention. During these two days he has been more irritable, his breathing has been rapid and his skin moist. No murmurs.

January 6. No trypanosomes in blood, symptoms much alleviated. With the exception of the last two days patient has for some time been in much better condition than when he was admitted.

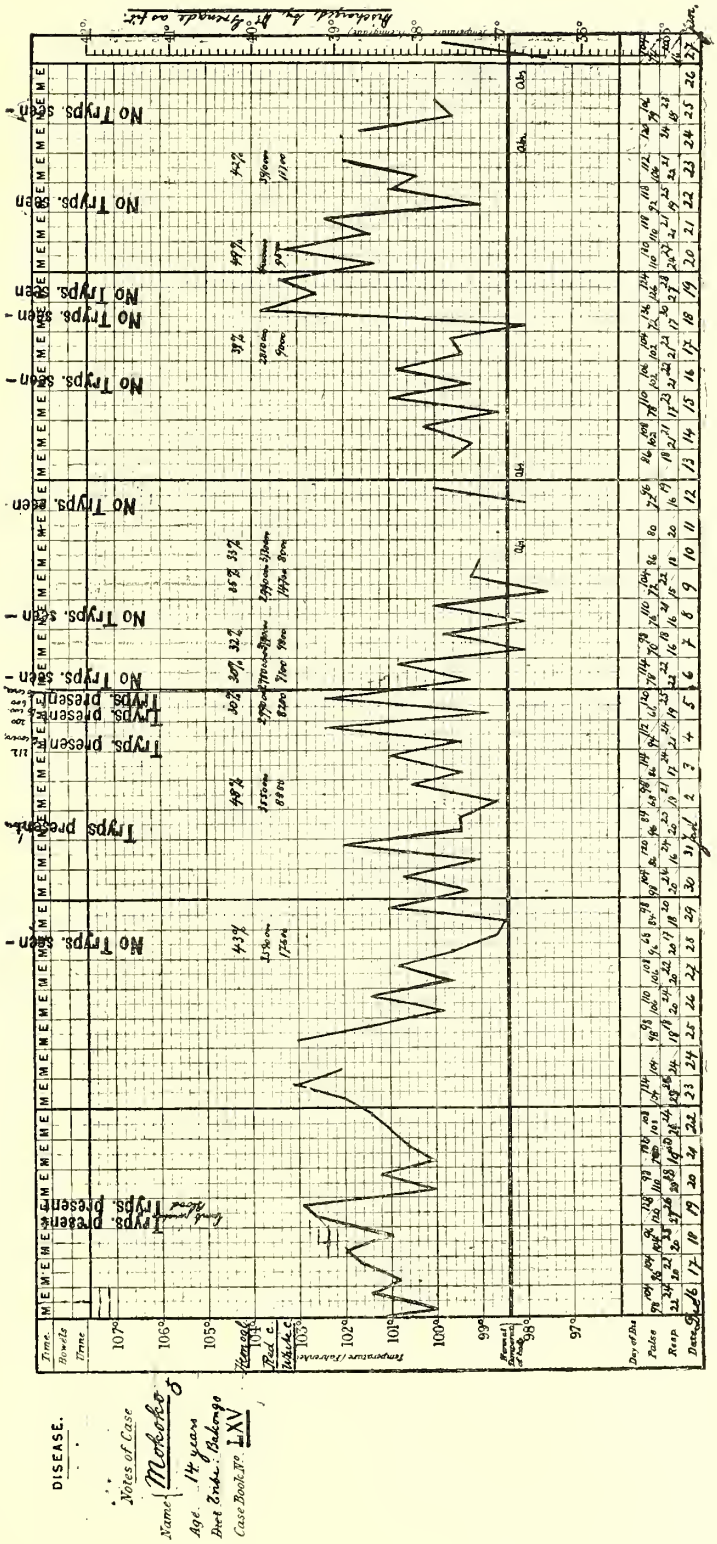
Faeces contain ova of *Anchylostoma duodenale*, *Ascaris*, and *Trichocephalus dispar*.

January 18. There is no oedema. Abdomen is distended and partially tympanitic. He complains of headache, but is otherwise comfortable. Is much stronger and strolls about.

January 28. Observations ceased, allowed to leave hospital.

February 13. Patient seen walking about with steady gait and with exception of abdominal distention and slight wasting, appeared to be well and expressed himself as such.

CASE LXV



TYPE 'C'

Cases Showing Well-Marked Symptoms.

The two following cases illustrate sub-group 'I' of this type. Fatal cases showing no sleep symptoms.

Case 82. Kimfuta. Female. Age eight.

History.—Patient is a Lower Congo native, and comes from a village four hours' march from Leopoldville. When brought to Leopoldville three months back she was an orphan and destitute, and a fortnight ago was sent to hospital, more through lack of friends to look after her than because of any suspicion of sleeping sickness.

December 29. General condition. Patient is miserably thin, and has the appearance of being half starved. She is very weak, her feet are full of chiggers, and she can only walk with difficulty. She has a dazed expression, and is constantly trembling. Tremors are increased on the slightest exertion.

Conjunctivae are congested and there is a slight purulent secretion. Intelligence is good, answers questions quickly and well. Skin, dry, imbricated, and filthy. Lymphatic glands, all easily palpable and hard, save femorals, which are much enlarged, soft, and matted together. The child complains of backache, and insists that she has sleeping sickness, but her condition is thought to be due mainly to starvation and ill-treatment. Circulatory system, normal. Respiratory system: slight bronchitis. Liver, normal. Spleen, just palpable. Alimentary system: tongue heavily coated, teeth tartrous, gums healthy, appetite excellent, abdomen flat and hard. Nervous system, reflexes all increased.

January 8. In spite of attention, cleanliness, and better food, patient is going down hill.

January 16. Greatly emaciated, tremors increased, patient can hardly stand and cannot walk, speech is thick but coherent. There is no drooping of lids, but eyes are fishy and vacant; intelligence is now very dull; she whines and complains on being disturbed; eats little; is seldom asleep, although she is always lying down, and passes faeces under her.

January 19. Emaciation extreme, is too weak to rise, lies with wide-open mouth and eyes, whines, moans, or attempts to speak if touched.

January 20. Spleen punctured, only a few parasites seen, although blood contained 100 to cover. Died during night.

January 21.

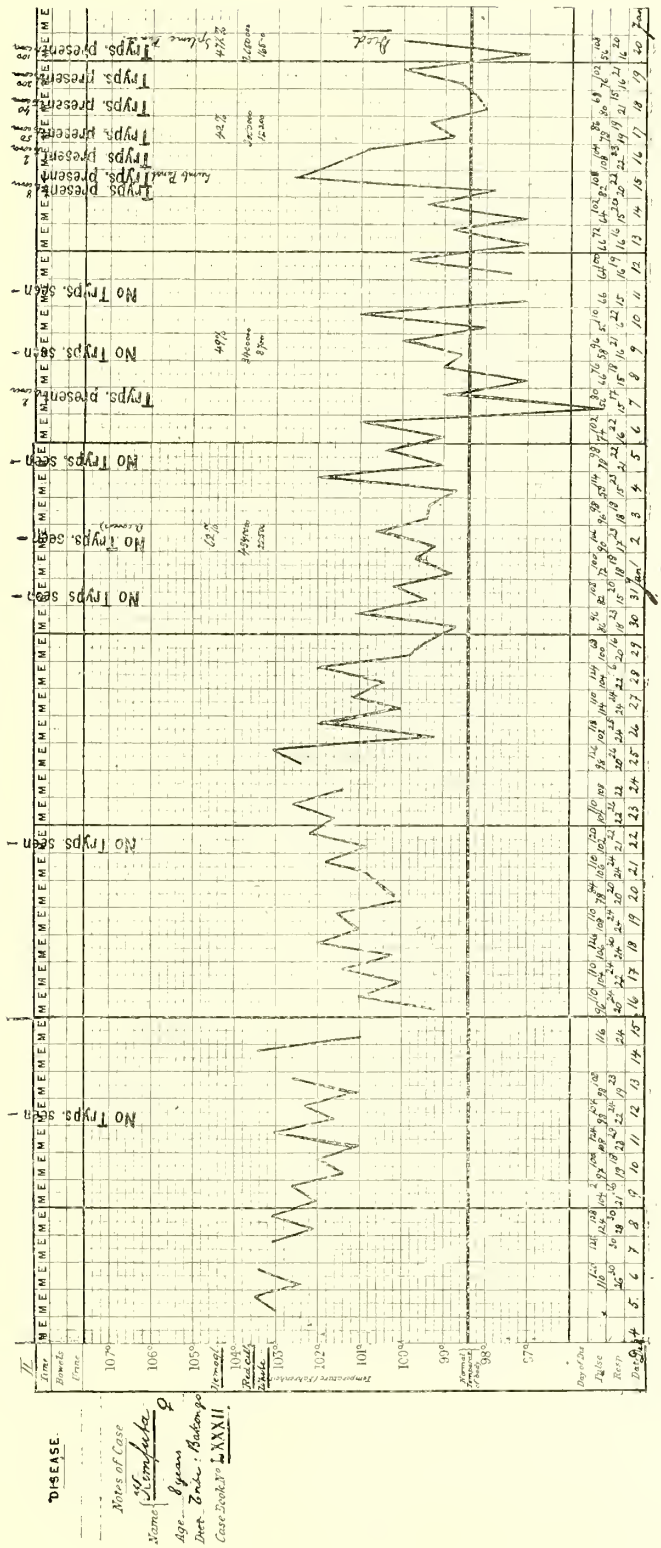
Necropsy commenced at 7 a.m., nine hours after death. Body much emaciated. No bed-sores, nor oedema. Ring-worm of scalp. Mouth foul, pyorrhoea alveolaris, teeth irregular, second dentition.

Thorax: Pleurae normal. Diaphragm: Right side, fourth space; left side, fifth rib.

Lungs: Left lung normal; right lung, old adhesions of upper and middle lobes.

Pericardium contained 5 c.cm. of turbid yellow fluid, in which no trypanosomes were found.

CASE LXXXII



Heart dilated, both sides contained large, firm, white agonal clots. Valves normal. In left ventricle was small area of sub-endocardial petechial haemorrhage. Several pin-head calcareous nodules along cardiac veins.

Lungs : Weight, right, 227 grammes ; left, 151 grammes. Both show profuse sub-acute bronchitis ; bronchi full of mucous.

Abdomen : Omentum drawn up and wrapped around spleen, small clot of blood between it and outer surface of spleen due to puncture. Peritoneum contained no fluid. Old adhesions between transverse colon and gall bladder.

Liver : Weight, 682 grammes. Yellow, slight interlobular congestion. Gall-bladder full of dark-green semi-fluid bile.

Spleen : Weight, 228 grammes ; enlarged, anterior border very much notched ; substance very diffluent and dark red in colour. One small spleniculus.

Kidneys : Weight (together), 151 grammes ; normal. Bladder normal, filled with urine.

Genitals normal, save for left tube bound down in Douglas' pouch by firm fibrous adhesions. Child has menstruated, remnants only of hymen.

Intestines normal, few anchylostomes in ileum.

Bone marrow (tibia), reddish orange in colour.

Brain : Dura not adherent to calvarium or pia. Superficial brain vessels much congested. vessels of basal ganglia very much so. There was fair amount of colourless, slightly turbid, sub-dural and sub-arachnoid fluid. Similar fluid occurred in ventricles and escaped from vertebral canal. No trypanosomes were seen in these fluids.

Lymphatic glands : Generally much enlarged, often oedematous, usually greatly congested and, in the abdominal and thoracic groups, haemorrhagic, sometimes excessively so. There were numerous patchy areas of dark sub-capsular pigmentation in the femoral and axillary groups.

Case 64. Dysiki. Female. Age twenty-six.

History.—Patient is from Lusambo, in the Kasai district, where sleeping sickness is said to be prevalent. She has been in Leopoldville for two-and-a-half years. Her illness is said to have commenced one-and-a-half months ago. Entered hospital because ' feet were sick and had difficulty in walking.'

December 8. General condition. Patient is very thin, expression somewhat dull and vacant, intelligence good, answers questions fairly quickly, muscles are wasted, and great weakness is apparent in her unsteady walk ; speech clear, but weak ; eyes very prominent ; lips puffy, dry, and cracked. Skin is dry, dirty, and scurfy. There is distinct pitting of shins and dorsa of feet but not of face or forehead. Lymphatic glands, all enlarged. Physical examination of abdomen and thorax showed no abnormality. Nervous system, co-ordination and muscular sense good, reflexes only just obtainable. Alimentary system : tongue steady, slightly furred, teeth and gums normal, appetite good, bowels regular.

December 15 to 19. Patient is very weak, sits most of the day outside the hut. Sleeps but very little, likes to sit in the sun.

December 21. Extremely weak, passes faeces in blanket without changing position. Prominence of eyes (ocular tension increased) and puffiness of eyelids and lips persists.

December 24. Patient in same state, helpless but conscious. Died during the morning.

Necropsy commenced one-and-a-quarter hours after death. *Rigor mortis* just commencing. Body thin, muscles wasted, eyes prominent, both pupils dilated (especially right). Oedema of shins, feet, and forehead. Marked cutaneous thickening of eyelids. Many chiggers in feet. Tongue bitten through, clenched between teeth (no history of a fit). Body very warm ; had been lying in sun, skin blistered. Panniculus scanty.

Thorax : Right pleura, few fibrous adhesions at base of lung. Left pleura, showed three small sub-pleural haemorrhages along vertebral column at level of fifth dorsal vertebra.

Pericardium contained 100 c.cm. clear yellow fluid.

Heart : Weight, 341 grammes ; fat, oedematous, valves normal, muscle pale, patchy thickening of endocardium of left ventricle, vessels normal.

Lungs : Slight bronchitis, oedematous. Weight : Right lung, 226 grammes ; left, 454 grammes.

Abdomen : Peritoneal cavity contained about 2 c.cm. clear fluid. Colon at level of umbilicus ; all abdominal blood-vessels turgid ; very extensive old fibrous pelvic adhesions ; several small broad ligament cysts full of clear fluid ; firm fibrous adhesions of liver and spleen to parietes.

Liver : Weight, 1,818 grammes : distinctly fatty, capsule over surface thickened. Gall bladder full of dark green fluid bile, ducts patent. Between liver and diaphragm was a layer, 5 c.cm. thick, of colourless gelatinous oedema.

Spleen : Enlarged and gorged with blood, substance soft and friable.

Kidneys, together, weighed 250 grammes, both showed cloudy change, capsules were adherent, and there was congestion of venae stellatae and around pyramids.

Pancreas and suprarenals normal.

Alimentary system : Mouth foul, gums soft, stomach normal, intestines normal, anchylostomes in jejunum.

Genitals : Vagina, slight mucous and cellular discharge, containing *Trichomonas vaginalis* and various bacteria ; no acute inflammation. Uterus nonparous, sub-acute metritis. Left ovary partially fibroid. Right ovary normal; no signs of recent inflammation in tubes.

Bone marrow (femur) : Very dark reddish orange.

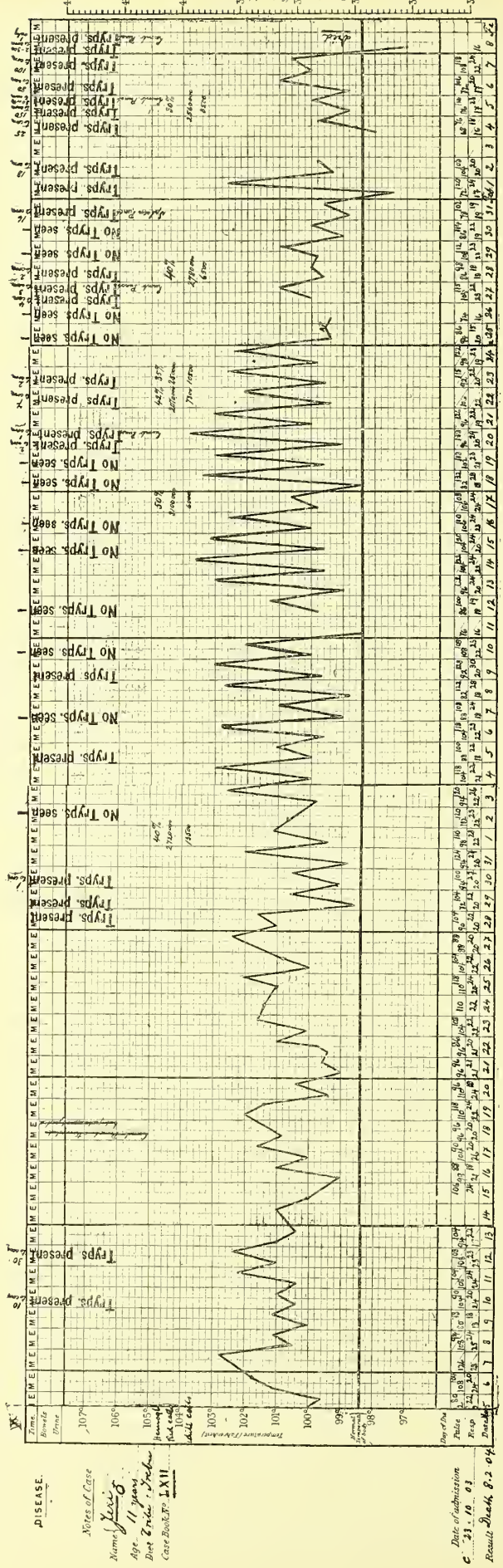
Brain : Dura not adherent ; superficial vessels congested, but not so much as in many of the cases ; sub-arachnoid fluid not greatly increased and only slightly turbid ; ventricles contained a few c.cm. of yellowish, slightly turbid fluid, ependymal vessels congested ; no haemorrhages seen ; spinal cord vessels turgid.

Lymphatic glands were nearly all enlarged, watery, and often congested. Some of pelvic and lumbar glands were chocolate colour and almost diffluent ; a small gland size of pea in much the same condition, but not diffluent, was found lying on head of pancreas. No actual haemorrhages into gland substance were seen. Fluids from pericardium, glands, receptaculum chyli, and oedematous tissues were examined, but no trypanosomes were found.

The following cases illustrate sub-group '2' of Type 'C.' Fatal cases showing sleep symptoms.

Case 62. Jeri. Male. Age eleven.

History.—Is a native of Irebu, a town near Lake Tumba, where sleeping sickness is said to be present. Left his village two years ago and has since lived in or near Leopoldville. He has been in hospital for one-and-a-quarter months. Was 'boy' to a white man who sent him to hospital as a suspected case of sleeping sickness. When



first seen on November 23, patient was a thin, sharp-witted lad, and denied that he had sleeping sickness, but was said by his companions to sleep a great deal.

December 11, 1903. General condition. He is a weak emaciated boy; intelligence good; answers questions quickly; no thickness of speech; no unsteadiness of gait; is a mouth breather, and his 'adenoid look' gives him a certain vacancy of expression. Skin is dry and scurfy and covered with early 'craw-craw.' Muscles wasted; mucous membranes anaemic; lymphatic glands all enlarged. Circulatory system, normal. Respiratory system, slight bronchitis. Alimentary system, tongue steady, slightly coated, teeth and gums covered with tartar. Liver, 1 cm. below ribs. Spleen, 2·5 c.cm. below ribs, not tender nor painful. Nervous system, reflexes normal; pupils react to accommodation and light; co-ordination and muscular sense, normal.

December 28. Patient still answers questions quickly and well, has no pain nor headache, complains of a constant desire to sleep, but is seldom seen sleeping. Heart: reduplication of first apical sound. No oedema. Ocular tension is very distinctly increased, and the eyes are prominent. Knee jerks exaggerated, tendon reflexes not obtainable. He is able to go some distance from the hospital to collect firewood to cook his food. His inordinate appetite has become the joke of the hospital.

January 19. Patient now sleeps most of the day, or sits dozing over his fire with his head drawn back and his mouth wide open.

January 21. Is too weak to walk about, but lies in the hut complaining and talking of his weakness.

January 25. Sleeps much more than previously and is becoming, if possible, more emaciated. Is easily roused when touched.

February 2. Is vivacious and talkative again. When lying down or dozing over fire, head is drawn back and chin protruded to utmost as noted on January 19.

February 5. Is much worse again, apparently dying, lies curled up on his side, and is roused with difficulty.

February 8. Dying, almost pulseless, Cheyne-Stokes respiration, lies on back in an extreme state of opisthotonos, with legs extended and head drawn back to such an extent that two fists, one above the other, can be passed beneath him. Died at 7 p.m.

Necropsy commenced one-and-a-quarter hours after death. Body much emaciated, still warm. Slight oedema of forehead, shins, and dorsa of feet.

Thorax: Pleural cavities normal. Pericardium contains 100 c.cm. of clear yellow fluid in which were seen divisional forms of trypanosomes.

Heart: Weight, 226 grammes, normal.

Lungs normal.

Abdomen: No fluid, old firm fibrous adhesions of right lobe of liver to parietes.

Liver: Weight, 610 grammes, slightly congested, otherwise normal. Gall bladder full of dark thick bile. Ducts patent.

Kidneys: Right showed marked peri-pyramidal congestion.

Spleen: Weight, 266 grammes. Slightly enlarged, substance firm, not slatey.

Pancreas and suprarenals normal.

Intestines contained very many ascaris, anchylostomes, and a few *Trichocephalus dispar*.

Bladder showed two submucous haemorrhagic areas near meatus.

Genitals normal.

Lymphatic glands : Generally enlarged and watery, especially the abdominal groups, some of the latter were deeply congested ; one gland lying on the anterior surface of the pericardium had a distinctly haemorrhagic centre.

Brain : Slight congestion of superficial vessels and flattening of convolutions ; sub-arachnoid fluid in excess, ' ground-glass appearance ' of arachnoid. *Cord* : vessels congested, great increase in cerebro-spinal fluid.

None of cranial bone cells were abnormal.

Living trypanosomes were seen in preparations made *post-mortem* from finger blood and pericardial fluid, but not in heart blood.

Case 84. Moidi. Female. Age twenty-four.

History.—Is a Kasai woman. Has been living for past two months near Leopoldville. Was sent to hospital, January 12, as a case of sleeping sickness.

January 21. Patient is a big well-made woman, no emaciation, her expression is dull, pained, and stupid, almost vacant. She is continually making grimaces. She cannot walk and lurches forward in a half drowsy condition during examination. Speech is thick and slow. Oedema of shins and feet, face and lips puffy. Indeed, the whole body is more or less puffy and presents an appearance of plumpness. Skin dry and dirty. There is a very extensive cicatrix, from a burn, implicating the right arm, side, and thigh. Lymphatic glands not enlarged. Circulatory and respiratory systems normal. Alimentary system, tongue coated and moist ; teeth and gums foul. Tip of tongue only, after much persuasion, protruded. Liver normal. Spleen slightly enlarged.

January 25. Patient usually sits dozing over a small fire. She is occasionally found lying at full length, naked, on the floor, or asleep in an uncomfortable attitude on the board bed. She can be easily aroused by a touch, and can, by shouting, be persuaded to speak.

February 5. Patient is almost lethargic, and is aroused only with some difficulty. She now eats nothing and is becoming emaciated. Sensation is dulled ; hardly flinched during lumbar puncture.

February 6. Died early this morning.

Necropsy six hours after death. The body of a massive but wasted woman. *Rigor mortis* present in masseters and extremities. Panniculus in fair amount.

Abdomen normal. Diaphragm : Right side, upper border fourth rib ; left side, fifth rib.

Thorax : Pleurae normal.

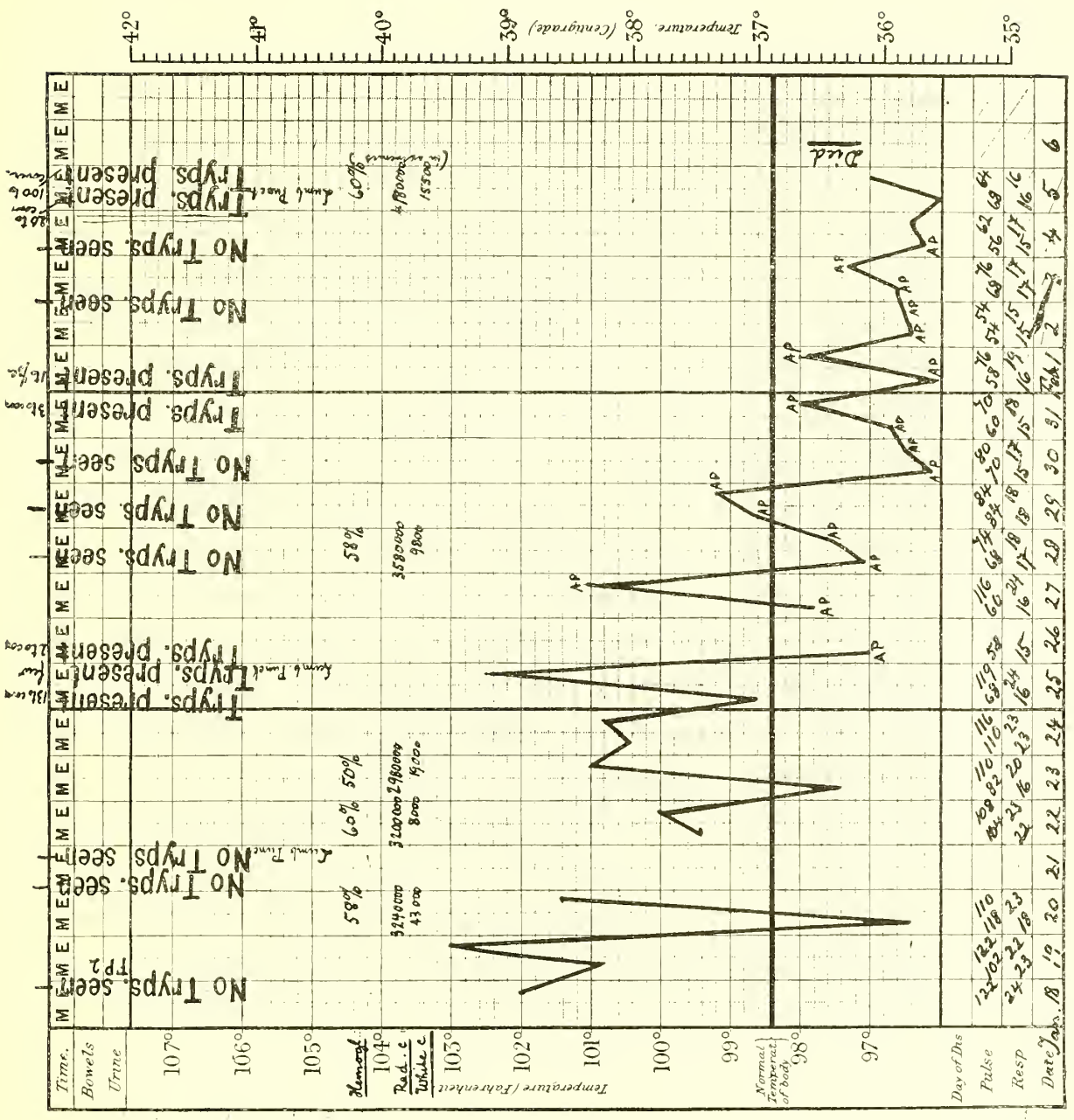
Pericardium contained a small amount of yellowish fluid.

Heart : Weight, 342 grammes ; contained firm white agonal clots.

Lungs : Weight, right, 454 grammes ; left, 335 grammes ; slight bronchitis and hypostatic congestion.

Liver : Weight, 1,591 grammes ; light in colour, central lobular congestion, soft and friable. Gall bladder full of dark, thick bile. Ducts patent.

CASE LXXXIV



DISEASE

Notes of Case

Name { Moridi
Age. 9
Diet

Case Book No LXXXIV

Date of admission Jan. 12 1904

Result Death Feb. 6

Spleen : Weight, 345 grammes ; measures 22 × 10 × 4·5 cm. ; substance firm, slightly fibroid.

Kidneys : Weight together, 338 grammes, normal.

Genitals, with exception of chronic vaginitis, normal. Mouth foul with sores. Intestines contained a few anchylostomes and *Trichocephalus dispar*. Alimentary canal otherwise normal.

Brain : Vessels all much congested, sub-arachnoid fluid increased and turbid. Microscopically cerebro-spinal fluid showed pus cells but no trypanosomes.

Lymphatic glands all much enlarged. Retro-sternal and mesenteric glands slightly injected. Decomposition had commenced in retro-peritoneal glands. Femoral and inguinal glands congested and in one or two of the latter pus formation had commenced.

The two following cases, not easily classable under the foregoing headings, are, we think, of interest, as they illustrate the extremely rapid course the disease occasionally takes after symptoms have once developed.

Case 69. Kabali. Female. Age twenty-six.

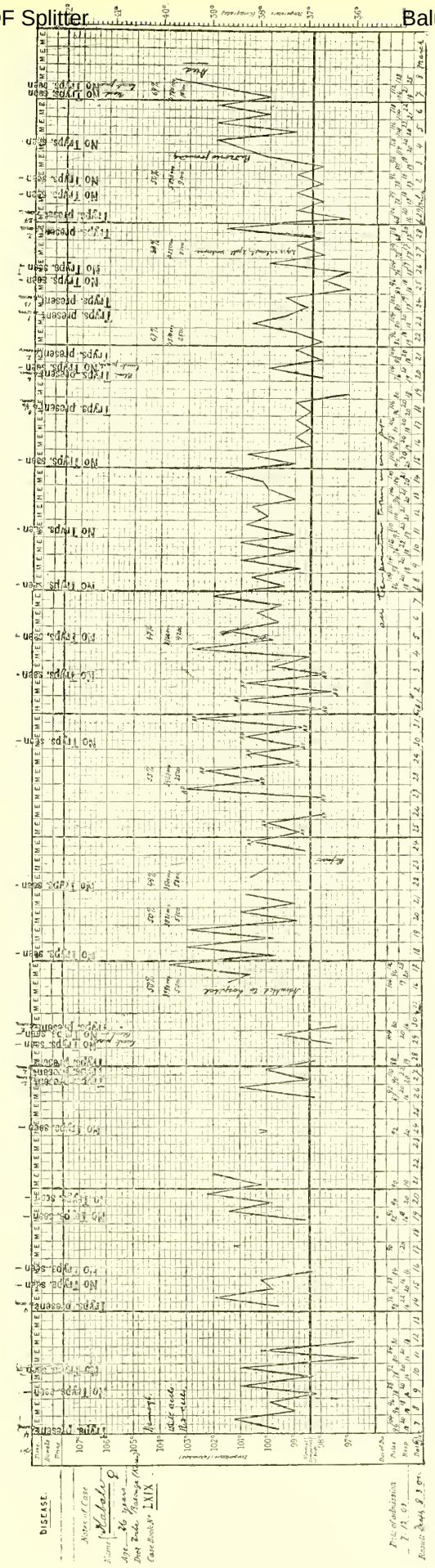
History.—Patient comes from the Kasai district, and left her native village, which is four days distant from Lusambo, ten years ago. Since then she has been in many places along the Upper Congo. Has lived in Leopoldville for the last two or three years.

When first seen on December 7, 1903, she stated that her illness had commenced four months previously, and began with rigor and fever ; the left side of her head swelled and was painful. There was no sore nor evacuation of pus. This condition lasted for six weeks. She says that she has since become thinner and weaker, particularly during the past two or three weeks. She has lost her appetite and has a tendency to diarrhoea.

December 25, 1903. General condition. Patient is a tall, well-developed woman, but thin, and her muscles are flabby. She is less active and vivacious than when first seen, and has a tired, listless gait. Her expression is rather dull, but she is intelligent and answers questions quickly and well. She speaks without hesitation or thickness. Complains of pain on pressure on shins. Skin is very abnormal. It is clean, though dry, and gives a rough feeling to the touch. This is most marked on shoulders, arms, and extensor surfaces of thighs and legs. In the parts most affected the skin has somewhat the appearance of an early exfoliative dermatitis, in which patches of glazed and thinned skin desquamate, slightly, at their edges along rectangular cracks and furrows. There is no itching nor discomfort, and the condition seems to be recent. At some spots are areas of crumpled tissue-paper-like wrinkles. No oedema. Lymphatic glands all enlarged. Tongue furred, moist, and steady. Circulatory system, pulmonary second sound is very much accentuated. Respiratory system normal. Liver normal. Spleen, considerably enlarged. Nervous system, co-ordination and muscular sense, normal ; reflexes, normal ; pupils react to accommodation and light, mental condition, alert.

January 18. Patient was admitted to hospital two days ago as a case of sleeping sickness. She is much thinner and weaker. Expression dull and sleepy. Skin

CASE LXIX



condition less marked ; tongue coated and dry but steady ; complains of sleepiness and dozed during examination.

January 25. Lies in bed all day, is drowsy but does not sleep much, is irritable and querulous.

February 2. Seldom asleep, and then at once aroused, is bright and intelligent ; complains of pains 'all over' and of insomnia at night ; staggers with weakness on attempting to walk.

February 12. Is much weaker, can hardly walk ; speech is thick, weak, and monosyllabic ; lips flabby and glued together ; sleeps a good deal but easily aroused.

February 16. Lies in a drowsy condition all day ; complains that she cannot walk. Food is brought to her, but unless assisted she does not eat.

February 24. Sleeps lightly all day ; is almost too weak to sit up alone ; calls for assistance to go to stool.

March 7. Pulse rapid and weak ; patient has not slept during night ; complains of pains all over ; cries out when touched, owing to bed-sores forming on sacrum and scapulae ; pupils equal and widely dilated ; axillary and femoral glands enlarged, but not nearly so large as when admitted to hospital ; cannot take her food, either milk or soup. Knee jerks increased ; arms persistently flexed at elbow ; hands tremulous. No oedema.

March 8. Died 4 p.m.

Necropsy commenced three-quarters of an hour after death. Body much emaciated ; bed-sores over sacrum and both scapulae ; skin clean, dry, and shows slightly on extremities the previously noted 'imbricated appearance.' No oedema ; pupils widely dilated, left slightly more.

Thorax : Left pleura contained 30 c.cm., very turbid fluid with floating flakes of lymph, right pleura contained 10 c.cm. of similar fluid. Pericardium contained 40 c.cm. slightly turbid yellow fluid.

Heart : Weight, 223 grammes ; normal ; full of firm clot.

Lungs, each weighed 231 grammes. Marked hypostatic congestion, with sub-acute bronchitis of both lungs. At posterior border of left lung, level of seventh dorsal vertebra, there was a recent deposit of fibrous lymph on pleura, with intense subjacent congestion.

Abdomen : Peritoneum dry, mesenteric vessels congested ; spleen and liver adherent to parietes and adjacent organs by fairly extensive, old, fibrous adhesions.

Liver : Weight, 1,585 grammes, large, nutmeg, with commencing fatty change.

Gall bladder filled with thick grumous bile.

Spleen : Weight, 335 grammes, $19 \times 11 \times 4.5$ c.cm. Anterior border deeply notched, substance dark, firm, but friable.

Kidneys weighed together 225 grammes, normal.

Pancreas and suprarenals : Normal.

Alimentary canal : Mouth, stomach, large intestine, and jejunum normal. A Peyer's patch about 60 cm. above ileocaecal valve was greatly congested. On either side of patch for some 10 cm. were numerous disseminated congested areas, varying in diameter from 1 to 4 mm. Ascaris and anchylostomes present.

Genitals : Uterus small, nonparous small myoma. All genital organs firmly bound together by old adhesions ; vagina normal.

Brain: dura easily detached, sub-arachnoid space filled anteriorly with thick-formed lymph, posteriorly sub-arachnoid fluid very cloudy and semi-purulent; all vessels moderately congested. About 50 c.cm. turbid yellowish cerebro-spinal fluid escaped on opening tentorium. Cranial bone sinuses normal.

Bone marrow (femur): Very dark and diffluent, like clotted blood.

Lymphatic glands: All retro-peritoneal and pelvic glands very deeply congested; on section, soft, with a good deal of bloody fluid. One or two omental glands in a similar condition.

Glands from the other parts of the body enlarged but otherwise normal. Trypanosomes were not found by coverslip preparations in any of the body fluids or blood.

Case 86. Yaiyai. Female. Age twenty.

History.—Patient was sent to us on January 7 by native dispenser at a settlement near Leopoldville. She was said to have recently come from a district in which sleeping sickness was present. She was supposed to be in the 'initial stage' of that disease. Patient was well dressed, clean, and neat. She seemed in good health. She had, however, a peculiar vacancy of expression, and answered questions in an excitable and voluble manner, making grimaces. She stated that she had sleeping sickness, but, as yet, not badly. Patient was kept under observation for two days, but nothing abnormal was noted, save her expression and some display of irritability.

On *January* 24, she was carried into hospital in a listless, dazed, and sleepy condition, unable to walk, and apparently unable to speak.

February 4. General condition: Is a well-nourished woman; expression dull and vacant; manner apathetic and listless. Is able to walk with slow shuffling steps. Intelligence much dulled. Tongue coated and moist, slight tremors. Skin normal. Glands all enlarged save posterior cervical. Considerable oedema of shins and insteps; lips and eyelids puffy. Appetite good. Patient munches her food in a slow way, peculiar to many advanced cases. She frowns continuously. Circulatory, respiratory, and alimentary systems normal.

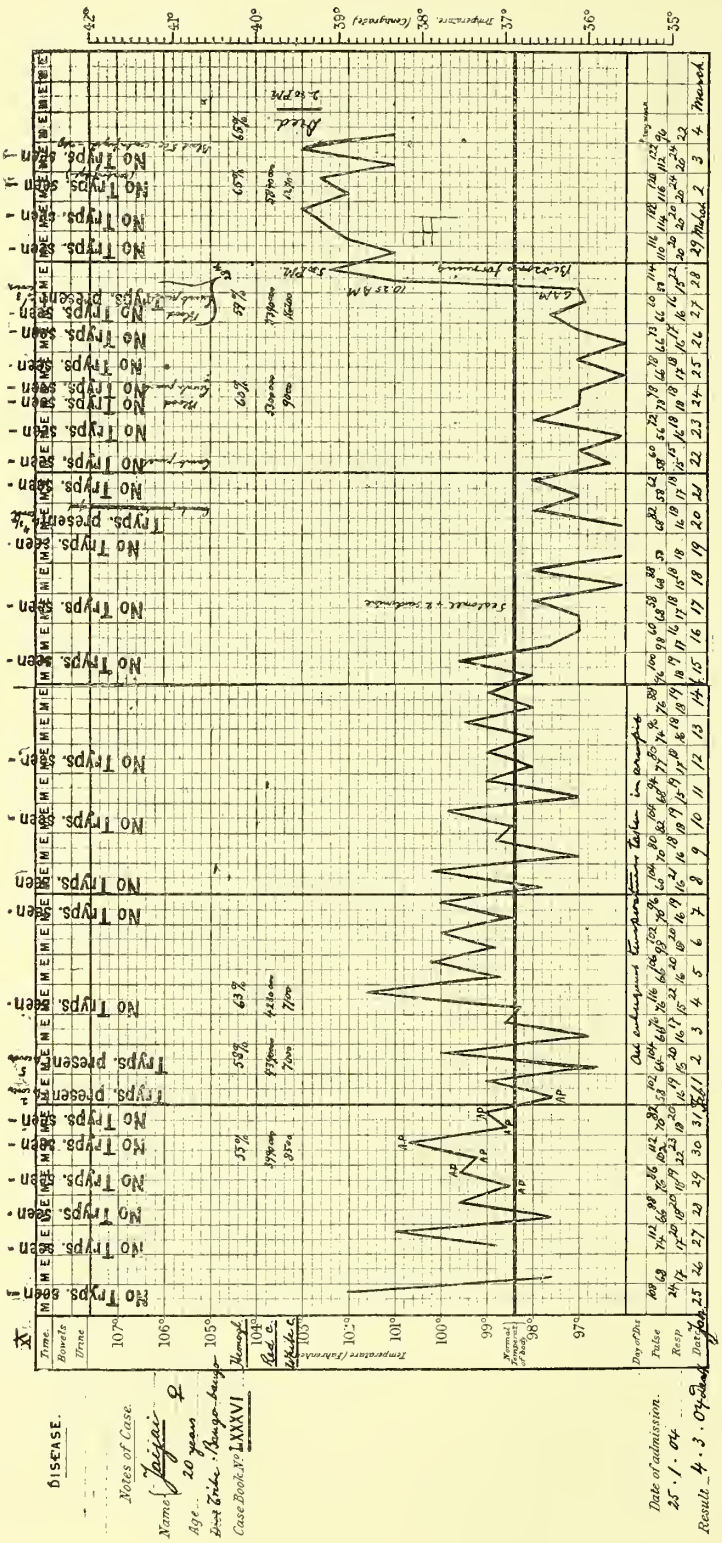
February 10. Sleeps a good deal but is much better than she was, is not so apathetic, and can talk and laugh while eating; appetite good.

February 16. Vacancy of face is again marked; patient is very weak and trembles all over; she speaks in weak voice only after much persuasion.

February 28. Since last note patient's condition has not altered. She has been unable to get off her bed; passes motions into bed; has only been able to take liquid food, and that only with assistance, is usually wide awake and conscious of all her surroundings, and, until now, has shown no very marked signs of wasting. To-day temperature, 102·4 F., profuse perspiration; crepitation and dullness at base of right lung.

March 3. Is now helpless, with sunken eyes and haggard face; body and limbs show marked wasting; oedema of shins and insteps and puffiness of face have disappeared; tremors, which were very marked, have ceased; she cannot take even soup; large bed-sores have formed on either side of sacrum.

CASE LXXXVI



March 4. Patient dying, but perfectly conscious, even acutely observant of all that goes on around her. Died at 2.30 p.m.

Necropsy commenced one-and-a-half hours after death.

Body warm, wasted though not distinctly thin. *Rigor mortis* just commencing, pupils evenly and widely dilated. Tissues dry, panniculus fairly abundant. Muscles normal.

Thorax : Pleurae normal.

Pericardium contained a few drops of fluid.

Heart : weight, 148 grammes, small, muscles pale and firm. Mitral valve showed distinct, reddish, gelatinoid thickening.

Lungs : Weight, right, 300 grammes ; left, 223 grammes. There was slight bronchitis of both lungs and marked hypostatic congestion, the latter condition marked in the right ; vessels contain firm, stratified clots.

Abdomen : Pelvic peritoneum congested and genitals firmly bound down by extensive fibrous adhesions.

Liver and spleen adherent slightly by old fibrous adhesions to adjoining organs and parietes.

Liver : Weight, 1, 582 grammes, slightly nutmeg. Gall bladder moderately filled with golden bile.

Spleen : Weight, 221 grammes, $17 \times 9 \times 5$ cm., upper lobe almost entirely divided by deep indentation from lower, substance dark, firm, friable.

Kidneys : Weight together, 218 grammes, normal.

Pancreas and suprarenals : Normal.

Alimentary Canal : Normal.

Bladder : Normal.

Genitals : Uterus small, non-parous, metritis with acute intense cervicitis ; fallopian tubes and ovaries bound down to uterus by old adhesions, acute vaginitis.

Bone marrow (femur) : Reddish brown.

Brain : Superficial vessels congested, particularly over posterior half of hemispheres (patient lying on back for several days before death). Relatively small amount, though increased, of sub-arachnoid fluid ; no ependymal haemorrhages ; about 30 c.cm. cerebro-spinal fluid escaped on cutting tentorium. Cranial bone sinuses normal.

Lymphatic glands : Retro-peritoneal and pelvic glands were enlarged ; the latter group being congested; the remaining groups of glands were normal.

In this attempt to illustrate the course of sleeping sickness, as found in the Congo, we have tried, as far as possible, to exclude from the illustrative cases those in which obvious secondary lesions were demonstrated *post-mortem*. It will be seen that deep sleep, continued sleep and lethargy, symptoms described as characteristic of sleeping sickness, are not features of the Congo disease as observed by us up to the present.

Trypanosoma Gambiense as a Probable Cause of Congo (Sleeping) Sickness

(*A*) As already indicated, in nearly every case in which sleeping sickness was diagnosed, or suspected, trypanosomes have been found in either blood, cerebro-spinal fluid, or both.

(*B*) We have shown that there is a very evident clinical connexion between those cases which have only very slight symptoms ('Trypanosoma fever') and the advanced cases of 'sleeping sickness' seen in hospital.

(*C*) In Gambia there is an undoubted trypanosome disease in horses which is characterized, in its first stage, by the absence of obvious symptoms ; and later, by fever, emaciation, and weakness. The course of this disease may be chronic and death delayed for a long period.

We have a horse, in good condition, under observation in Liverpool, which was first found to be naturally infected on October 30, 1902. A letter, dated January 18, 1904, from an officer in Gambia, says that his horse, found naturally infected in April, 1903, 'could not be better, and is in excellent condition.'

This Gambian horse disease seems to present a striking analogy to the human trypanosome disease seen in the Congo. The dull, listless expression, the weakness, the wasting and lack of energy, the irregular elevation of temperature, the frequent disappearance of the parasite from the peripheral blood (to ordinary examination), and the chronic course of the disease, are features characteristic of both infections. The similarity seems to us to be a point in favour of the casual relation of trypanosomes to Congo sleeping sickness.

(*D*) The sleeping sickness of the Congo is known to have a long latent period. Symptoms are said to have developed in some cases two to five years after leaving an infected locality.[1]

It is interesting to note that natives may be infected with trypanosomes for many months without showing signs of illness.

We have just heard that a Gambian native in whose blood trypanosomes were seen over a year ago is still in the very best of health.

Dr. Zerbini, State Physician at Boma, has just sent us a report on cases observed by us in Boma during October last. His report is based on either his own observations or on the statements of heads of departments in those instances in which the cases had passed from his care. The report on five cases is indistinct or vague. Five had been in perfect health during the whole six months. Three have had surgical troubles, but are now well. One has had persistent diarrhoea, but has quite recovered and has been sent to the Upper Congo. Two cases have died of pneumonia, and the remaining four, only one of whom was in perfect health when we saw them, show slight oedema, lack of energy, or some other ailment. In none is there any suspicion of sleeping sickness.

(*E*) Europeans[2, 3, 4] infected with *Trypanosoma gambiense* show symptoms which are quite comparable with those observed in uncomplicated trypanosome infections of Congo natives.

(*F*) In Gambia, where cases of sleeping sickness are rarely seen (Dr. M. Forde, Principal Medical Officer at Bathurst, stated that perhaps one case a year came under his care), only six out of one thousand odd natives were found to be infected,[5] by the examination of fresh cover-slip preparations. In an almost exactly equal number of a similar class of natives examined in the same way in the Congo, forty-six have been infected In Uganda, where the disease occurred in epidemic form, the percentage of infection among the general population was still higher.[6]

1. Guérin, *Archives de Médecine*, VIe séries, vol. 14, Paris, 1869.
2. Manson, *Brit. Med. Jour.*, March 28, May 30, 1903.
3. Dutton, Todd, Christy, *Brit. Med. Jour.*, January 23, 1904.
4. Broden, Les injections à Trypanosomes au Congo chez l'homme et les animaux. (*Extrait du Bulletin de la Soc. d'études Coloniales*, Févr., 1904).
5. Dutton, Todd, *Ibid.*
6. Reports of the Sleeping Sickness Commission, Royal Society.

We have not yet met with an epidemic of sleeping sickness in the Congo, although at least one has been recorded.[1] From the answers to inquiries, which we have received from state doctors and officials in different parts of the country, we do not think that the disease exists in any part of the Congo in a much more severe form than at Leopoldville, and certainly there is no epidemic comparable to that in Uganda.

It will be noted that the Congo type of case, as we have described it, bears a close resemblance to some of the cases described by BRUCE and NABARRO on the Victoria Nyanza, at a time many months after the epidemic wave had passed eastward, and at a place Entebbe, situated at the extreme tail of the epidemic area. Moreover, one of us is able to recognize a similarity between many of the cases seen here, particularly those in which emaciation, lengthened period of illness, and absence of sleep, are the main features, and many of the cases seen by him on Buvuma Island, also situated in Victoria Nyanza, and towards the tail of the epidemic area.[2]

THE COURSE OF THE DISEASE

Duration. It has been impossible to decide for how long a period a native may be infected with trypanosomes and still show no definite signs of disease. One man already mentioned, is known to have been infected for over a year and is still perfectly well. It would appear from one or two early cases which have been observed that the transition from this 'latent' stage to one in which symptoms are noticed may be very gradual. We have only observed eight fatal cases of Congo sickness in which the necropsies showed no obvious secondary infection. The duration of the disease, dating from the recognition, either by the friends of the patient or by ourselves, of obvious signs of ill-health, has been from two to four months.

Recovery. In our experience no native who has shown definite and constant signs of ill-health has recovered.

Since we have been in Leopoldville we have frequently seen a case of trypanosomiasis in a European, reported by Dr. BRODEN[3] and Sir PATRICK MANSON.[4]

Mrs. M., a missionary stationed at Leopoldville, states that her illness commenced on October 1, 1900, with a fever which lasted for three months and was not amenable to quinine. Since that time she had not been free from constantly recurring fever, except for two periods, in 1901 and 1902, each of three months duration, up to March 19, 1903. Since the last date she has been absolutely free from fever or other signs of trypanosoma infection up to the present (April, 1904). She is apparently in perfect health and has increased in weight. The trypanosomes were found by Dr. BRODEN on February 7, 1903, during an attack of fever which lasted for a few days. During the last year Dr. BRODEN has seen no parasites in the blood of this case.

Death. We do not think that we have sufficient evidence to state that death is produced by the trypanosomes alone.

1. Bergh St. Marie in the Portuguese Commission's Report.
2. Christy, Reports of the Sleeping Sickness Commission, Royal Society, No. III.
3. Broden, *Ibid.*
4. Manson, *Ibid.*

Secondary Infections and Complications

Secondary bacterial infections seem to determine the fatal issue of many cases of Congo sleeping sickness. Thirteen out of twenty-two necropsies performed at Leopoldville showed complications or obvious secondary infections.

They were as follows :—

Purulent meningitis	4
Purulent pleurisy and pneumonia	1
Pneumonia and localized tubercle of lung	1
Localized gangrene of lung	1
Enlarged caseating and breaking down glands in thorax and abdomen. No tuberculous lesions in organs (tubercle bacillus in glands seen in one case)	2
Dysenteric ulceration of large bowel (perforation in one case) ...	2
Universally adherent pericardium (recent)	1
Infiltration of pus in femoral, inguinal, and internal iliac glands (gonorrhoea)	1

According to the *Post-mortem* Reports of Colonel D. Bruce and Dr. Nebarro very similar lesions were observed in Uganda, where ten out of twenty autopsies on sleeping sickness patients showed obvious secondary infections and in three instances purulent meningitis.

It will be noted that purulent meningitis has been the most frequent complication both in Uganda and in the Congo. The brain in these cases (see case 69) presents a very abnormal appearance. The convolutions, especially on the upper surface, are covered and glued together by a thick layer of tenacious lymph, and the vessels of the pia arachnoid are intensely congested. It is evident that such morbid changes are explicable on bacteriological grounds, and we have found, microscopically, in all such cases an almost pure culture of a diplococcus occurring in small chains. These observations make us doubt whether the acute congestion of cerebral vessels accompanied by an increase of pia arachnoid fluid containing pus cells, seen by us here, and described by others as typical of sleeping sickness, can be attributed to the trypanosome alone.

The Portuguese Sleeping Sickness Commission,[1] Dr. Broden in Leopoldville,[2] and Dr. Castellani in Uganda,[3] have all described bacterial infections in a very large percentage of sleeping sickness cases examined before as well as after death.

It must be noted that such purulent changes have not been described in animals dying from other trypanosome infections.

1. Bettencourt, Koppe, de Rezende and Mendes, *La Maladie du Somneil*, 1903.
2. Broden, *Ibid.*
3. Castellani, Report of the Sleeping Sickness Commission, Royal Society.

OBSERVATIONS ON THE PARASITE

Periodicity and Frequency of Occurrence in the Peripheral Blood

Frequent examinations of the finger blood of trypanosome cases have shown that the parasites may, to the ordinary methods of examination, be absent from the peripheral blood for varying periods. This periodicity is illustrated by the two following cases :—

Case 101. Oporanga. Regular observations were made about every third day, commencing on January 3, 1904, when no parasites were seen. The periods of presence or absence of the parasite from that date until death were as follows :—

22	days	absent	...	11	observations.
3	,,	present	...	3	,,
7	,,	absent	...	4	,,
3	,,	present	...	3	,,
33	,,	absent	...	14	,,
4	,,	present	...	4	,,
6	,,	absent	...	3	,, Died.

This case shows comparatively long periods of absence.

Unless especially stated the routine method of examination employed for the detection of the trypanosomes was the examination of a freshly made three-quarter inch square, cover-slip preparation of finger blood ringed with vaseline.

Case 90. Bongwendie. Routine examinations commenced on January 19, when trypanosomes were found to be present. They had not been seen for four days previously.

3	days	present	...	3	observations.
9	,,	absent	...	4	,,
6	,,	present	...	4	,,
6	,,	absent	...	4	,,
1	,,	present	...	1	,,
2	,,	absent	...	1	,,
1	,,	present	...	1	,,
5	,,	absent	..	4	,,
2	,,	present	...	1	,,
3	,,	absent	...	1	,,
6	,,	present	...	5	,,
10	,,	absent	...	7	,, Died.

This case shows shorter periods of absence.

In some cases parasites have been almost constantly present in the blood (Case 85); in others, as is shown by the two following cases, they have been rarely found.

Case 77. Ejoli. Under observation for twenty-one days before death. Parasites seen only twice on two consecutive days; sixteen observations.

Case 103. Belamo. Still under observation. Parasites not seen for thirty-eight days; seventeen observations. They have been seen in the blood of this case only by centrifugalizing.

The frequency with which the parasite is obviously present in the peripheral blood bears no relation to the severity of the symptoms.

Number of Parasites Appearing in the Blood

The number of parasites seen in ordinary fresh cover-slip preparations is generally small, but in some patients, very large numbers have been recorded. In two cases a large increase has occurred during the few days immediately preceding death.

Case 87. Patesa. Admitted to hospital for acute dysentery; under observation for four days preceding death. Parasites increased from two to a cover on the first day to twenty to a field on the day of death. There was no accompanying rise of temperature.

Necropsy showed very extensive dysenteric ulceration of the large intestine.

Case 88. Boyo. Parasites increased during the four days preceding death from two to one hundred to a cover. They had not been previously seen in such large numbers. At the necropsy on this case very general enlargement and caseation of abdominal and thoracic glands was noted. There were no tubercular lesions in the lungs or other organs.

A similar increase of parasites occurred just before death in Cases 62 and 82 (see charts); but in the two following cases they were absent for some days before death :—

Case 89. Kapinga. Parasites not seen for ten days preceding death; six observations.

Case 77. Ejoli. Parasites not seen for nineteen days before death; fourteen observations.

Sudden Disappearance of the Parasite from the Finger Blood

The following are examples of cases in which the parasites have gradually increased from small to fairly large numbers (twenty to cover-slip or more) and then suddenly disappeared on the day after their acme was reached.

Case 65. Mokoko. See chart.

Case 99. Mozao. At the end of the first examination on March 5 seven trypanosomes were seen to a field. On March 6, 7, and 8 none were seen.

Case 102. Kondolo. On February 24, seven parasites were seen to a cover; on February 25, seventy to cover; on February 26 and eight following days, none were seen, although observations were made every other day.

A second similar acme and sudden disappearance was recorded in this case. On March 10, six parasites were seen to cover; on March 11, two hundred and fifty; but on March 12, none were found.

Case 90. Bugwendi. On February 26, 27, 29, and March 1, parasites were seen in the following numbers respectively, eight, five, five, and one to a cover. On March 2, twenty-four were seen; but from March 3 to date of death (March 21) they were absent.

Case 101. Oporanga. On March 1, 2, 3, parasites were seen in numbers six, forty-eight, thirty-one, respectively, to the cover-slip. On March 4, one hundred and twelve were noted; but on March 6, 7, and 8, not one was to be found. (No examination on March 5).

Case 66. Tenda. On February 23, 24, and 25, the number of parasites recorded per cover was four, five, and seven, respectively,. On the 26th, seventy to a cover; but on the 27th, and for fifteen subsequent days, none were found.

Case 85. Banja (see below). Illustrates a somewhat opposite condition. The parasites increased in this case to an acme which lasted for two or three days, and then a marked diminution in number was observed. This patient, who is still under observation, illustrates the constancy with which parasites are seen in the peripheral blood of some cases. It will be noted that his general health has latterly improved.

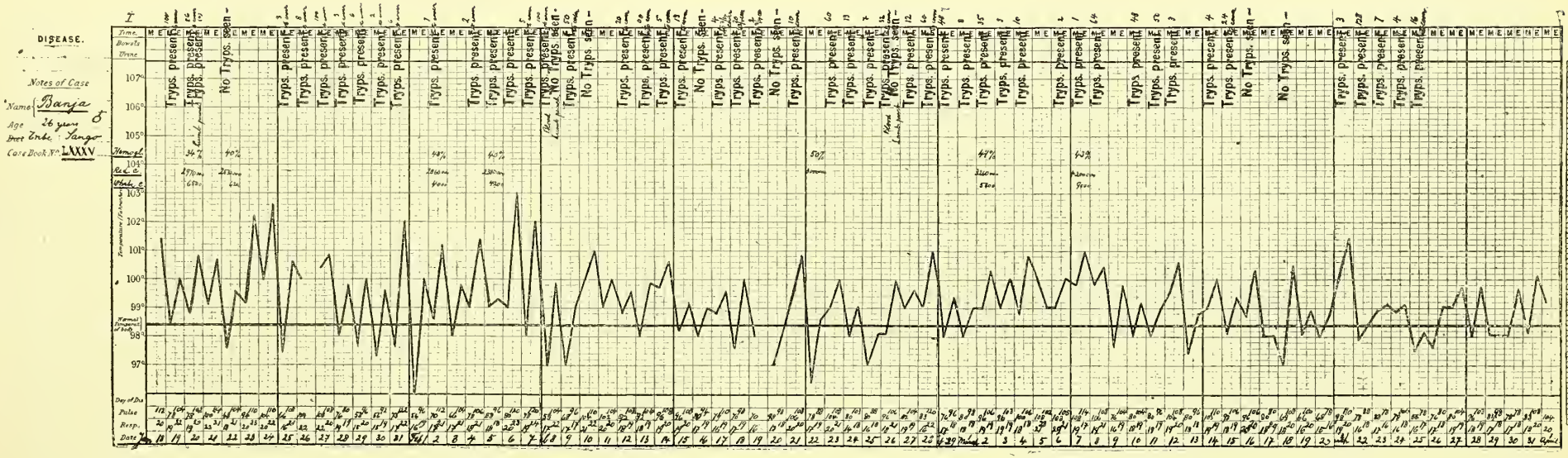
Case 85. Banja. Male. Age twenty-six.

History.—Patient comes from the Sango district of the Bangala country, where Congo Sickness, so far as we know, is not present. He has been employed for some years on steamers plying on the upper river. A month before being sent to hospital was imprisoned on a charge of cannibalism. No history of illness before going to prison.

January 20, 1904. General condition: Somewhat emaciated; expression pained and anxious; intelligence fair, answers questions slowly but correctly; breathing laboured—is apparently very ill. His skin is dry and filthy, no 'craw-craw.' Slight oedema of forehead, considerable of shins and feet; lymphatic glands all enlarged, especially femoral and inguinal; tongue coated, moist, and steady.

Circulatory system, heart sounds booming at apex; pulmonary second sound accentuated and dicrotic. Respiratory system, dullness at base of right lung, loss of breath sounds, pleuritic rub and moist rales. Liver enlarged and painful. Spleen not enlarged. Nervous system, patellar and cremasteric reflexes not obtainable, epigastric increased; pupils equal and react to light.

CASE LXXXV



February 2. Is in better condition, and is brighter than when admitted; abdomen distended; oedema of loins marked.

February 16. Shows no signs of sleep, walks now without any unsteadiness. Appetite large.

February 24. Seems quite well; slight oedema of shins, but none elsewhere; no puffiness of face; can walk about with ease.

March 18. Gait normal, expression contented, no dulness, is quite intelligent, answers questions quickly and well; glands all enlarged, but femorals and inguinals apparently smaller than when admitted to hospital; slight oedema of shins; liver still enlarged, but not tender; heart and lungs, normal; knee jerks, normal; epigastric and cremasteric reflexes obtainable; appetite good; no drowsiness; patient works willingly around the hospital.

April 1. Has certainly put on flesh lately and increased in general robustness.

Symptoms Associated with the Presence of the Parasites in the Blood

The only patient in whom a large increase of parasites was associated with a rise in temperature and the presence of symptoms which might be attributed to the parasite was Mokoko (Case 65), whose case is given above.

From our observations we cannot make out any definite relation between the temperature and pulse and the appearance of the parasites in the peripheral circulation. A rise of temperature is not necessarily associated with an increase of parasites in the blood.

It appears, therefore, that the number or constant presence of the parasites in the peripheral blood bears no relation to the severity of the disease.

Occurrence of Parasites in Serous Fluids

On two occasions 10 c.cm. of fluid was drawn from a flabby hydrocele (in Case 104) and centrifugalized. Parasites were seen each time, although they were absent from the blood to ordinary examination, and probably had been absent for some days previous to the second tapping. The cerebro-spinal fluid of this case was also examined on the same date as the first tapping, but no parasites were seen, although the deposit of the centrifugalized hydrocele fluid showed fourteen to a cover. In another case (number 92), in which the penis and scrotum were very oedematous and there was a small right hydrocele, no parasites were seen in either oedema or hydrocele fluids, although the blood showed thirty to a cover. We have not found parasites in the urine (centrifugalized) in the few cases examined in which the blood showed many parasites.

The Occurrence of the Parasites in Tissues and Fluids after Death

Our examination of film preparations of tissues and fluids taken at autopsies are not completed, but it is interesting to note here the frequency with which the parasites have been seen in the pericardial, pleural, and peritoneal fluids when examined fresh. In three cases (62, see chart, 64, 76) parasites were seen, without centrifugalizing, in the pericardial or peritoneal fluid; one-and-a-half, two, two-and-a-half hours, respectively, after death. In two of these cases numerous longitudinal divisional forms were seen in the pericardial fluid. In three other cases actively motile trypanosomes were seen in the pericardial fluid after centrifugalizing, fourteen, fifteen-and-a-half, twenty-and-a-half hours after death. In all these cases parasites were seen either the day before or a few days previous to death. In only one case has a trypanosome been seen in fluid from a lymphatic gland (omental); but in this case parasites were easily detected in the blood, *post-mortem*, and were very numerous in the pericardial and peritoneal fluids.

Occurrence of Parasites in the Cerebro-Spinal Fluid

During the stay of the expedition at Leopoldville, lumbar puncture was performed on forty-nine natives coming from many parts of the Upper and Lower Congo. Of these thirty-eight were proved to be suffering from trypanosomiasis, the parasites being found in the blood. In the remaining eleven cases no trypanosomes were seen in either the blood or cerebro-spinal fluid, and in some of them a diagnosis of some other disease, *e.g.*, tubercle, dysentery, etc., was established either during life or *post-mortem*.

In twenty-five of the thirty-eight trypanosomiasis cases the parasites were found in the cerebro-spinal fluid, but in thirteen no parasites were seen, although in one case (No. 101) the fluid was examined on five occasions.

If, however, those punctures in which the cerebro-spinal fluid was mixed with the blood in greater proportion than from three to four red cells to a field (Zeiss 1/6 objective, No. 4 eye-piece, diaphragm removed), and in which the parasites were found by coverslip examination to be present in the peripheral blood on the same day as the puncture, be excluded, then we find that the above result is very different, namely, thirty-two cases, in sixteen of which the parasites were found in the cerebro-spinal fluid, and in sixteen they were not. The amount of cerebro-spinal fluid drawn off and centrifugalized at each operation varied from 10 to 30 or even 40 c.cm. This, if necessary, was not only centrifugalized a second or third time, but from one to six coverslip preparations of the resulting deposit were examined before a negative result was recorded.

If the cerebro-spinal fluid is mixed with blood it has a slight yellowish tinge, and is opalescent or clouded according to the amount of blood it contains. If

normal cellular elements are much increased the fluid has also an opalescent or cloudy appearance, but there is no yellowish tinge. It is doubtful whether the presence of the parasite in the cerebro-spinal fluid has any influence upon the increase of its cellular elements. It would seem, from a study of our cases, that the fluid as a rule, whether the parasites be present or not, is perfectly clear and limpid as in health. Only in a few instances, in both positive and negative cases, have the cellular elements shown an apparent increase, consisting mainly of small mononuclear cells, together with some mononuclear large cells. In Case 93, the only one in which a large number of parasites—fifty to a cover—were found in the cerebro-spinal fluid, a great increase of small mononuclear cells was noted at the same time. A month later the cerebro-spinal fluid was again examined, but only one parasite could be found, and the cellular elements were scanty. This case, up to the present date, nearly two months after the discovery of large numbers of trypanosomes in the cerebro-spinal fluid, has shown no tendency to sleep during the daytime, no wasting, nor any of the more noticeable symptoms associated with the later stages of many of our cases of trypanosomiasis.

As a rule, we have found that the parasites occur in extremely scanty numbers in the cerebro-spinal fluid, seldom more than one to three or four, very occasionally from ten to twenty to the cover-glass preparation of the sediment left after centrifugalizing.

As already stated, neither at Leopoldville, nor anywhere on the Lower Congo, up to the present time, have we met with a case of Congo sickness in which sleep has had a prominent place among the clinical symptoms, and in those few cases in which it has been noted a few days before death, or at irregular intervals during the course of the disease, it has not been describable as deep or continuous sleep, but merely a drowsy or somnolent condition from which the patient was at once aroused by being touched or, perhaps, by being spoken to. On comparing the cases in which parasites were found in cerebro-spinal fluid with those in which they were not found, we see that most of the few cases in which drowsiness was a feature, together with cases in which head symptoms, *e.g.*, mild mania, epileptic attacks, flexure contractions, and convulsive seizures, were conspicuous are upon the positive side. On the negative side similar cases are also found, but it is noticeable that there are many of those cases which showed hardly any symptoms, and which, if no fatal complications intervened, lived on month after month in an advanced state of emaciation, retaining their faculties, speech, appetite, etc., almost to the moment of their death.

It is, therefore, not improbable that the presence of the parasites in the cerebro-spinal fluid at a late stage in the disease may tend to increase the gravity of the case by predisposing to one or other of many complications, or, in other ways, hasten a fatal termination ; but that this is not invariably so is proved by at least two of our cases, one of which (Case 93) is now one of the most sturdy patients under observation.

Animal Experiments

We have infected rats, mice, guinea-pigs, rabbits, and monkeys with human trypanosomes taken from cases of Congo Sleeping Sickness, both in its early and its latest phases. As was stated in our first Progress Report the course of the infection in these animals has given us no reason to suppose that we are dealing with more than one species of trypanosomes, or that the parasite is other than *Trypanosoma gambiense*.

All our inoculations have been made with small or medium doses (1 to 5 c.cm.) of infective material, either cerebro-spinal fluid or blood—diluted or no—in which, in almost every case, living trypanosomes, sometimes in enormous numbers, were demonstrated. About 50 per cent. of such inoculations have failed to infect. These failures seem to bear no relation either to the source of the material inoculated, to the site of inoculation—subcutaneous or intraperitoneal—or to the approximate number of parasites injected.

Once more, no animals inoculated with material in which trypanosomes were seen, taken *post-mortem* from cases of trypanosomiasis, have ever become infected. Parasites have been seen in the peripheral blood of infected animals only at more or less irregular intervals. Guinea-pigs have, perhaps, shown themselves the most satisfactory of ordinary laboratory animals, since, as a rule, when once infected, parasites are constantly present in large numbers.

Death, in the small number of our infected animals which have died, can in no instance be certainly said to have been due to the trypanosome alone. These are all points in which the Congo trypanosome resembles *Trypanosoma gambiense*. In addition, the 'incubation period,' that is, the period intervening between the inoculation of small or medium doses of infective material and the detection of the parasite in the peripheral blood of the experimental animal, is much the same for both parasites.

	Congo trypanosome	Trypanosoma gambiense
Rats	5—20	7—20
Mice	5—16	6—7
Rabbits	17—27	21
Guinea-pigs ...	11—25	12—21

Monkeys of two different varieties, both belonging to the sub-family *Cercopithecus*, have been infected. Only one has shown slight symptoms of ill-health. On two

occasions a slight rise of temperature and loss of vivacity has accompanied one of the irregularly appearing intervals during which parasites have been seen in the peripheral blood. This monkey has been infected for nearly three months.

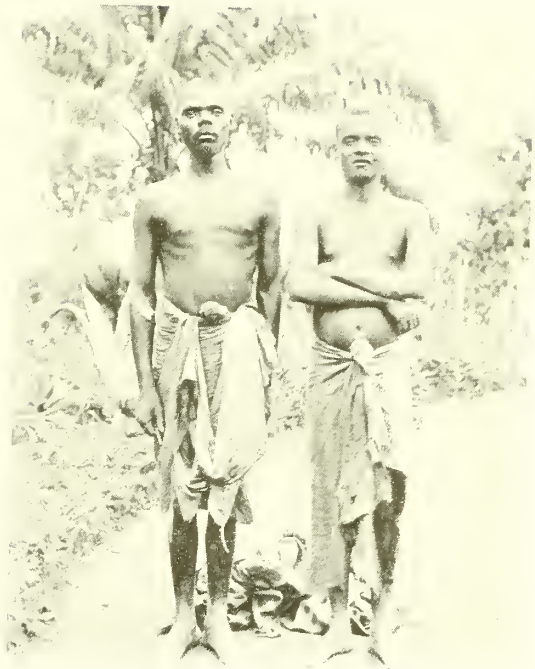
We have again failed, even with large doses of blood containing huge numbers of trypanosomes, to infect two dog-face monkeys (*Cynocephalus*).

Transmission Experiments

We have attempted to repeat the transmission experiments made by Bruce, in Uganda, with the tse-tse fly. These flies are rather numerous in the bush along the river banks on either side of Leopoldville, and a fair number are daily brought to the laboratory by a gang of boys supplied for the purpose by the local authorities. Practically all the flies brought in by them have been *Glossina palpalis* (native name, 'mavekwa'). Unfortunately, we have had the greatest difficulty in obtaining monkeys and have only been able to use two for these experiments. Both in the Congo and in the Gambia, experiments have shown that the guinea-pig is, perhaps, the laboratory animal most susceptible to *Trypanosoma gambiense*. We, therefore, determined to employ it, lacking monkeys, in our transmission experiments. At the time of writing both experiments with monkeys remain unsuccessful, and only one, a direct transmission experiment with guinea-pigs, in which the flies were made to feed alternately on an infected and an uninfected animal, has given a positive result.

In conclusion, we wish to thank Dr. Ingle Heiberg, who has been attached to the expedition by the Government of the Congo Free State, for his untiring kindness and the help he has given us in our work.

Fariala (Case LXXIX). Illustrating an early stage with general absence of symptoms except rise of temperature.



Two cases from Cataract Region of Congo, showing practically no symptoms. Carried heavy loads for many days without complaint.

Trypanosomiasis cases in the hospital for blacks at Leopoldville, Upper Congo, waiting for their daily ration of 'Kwanga' (Casava bread) to be served out to them.

To follow page 45

Kondolo (Case CII). Main symptoms severe and continued headache. No sleep.

Boyo Mitchel (Case XCI). Showing puffy face and lips, and drowsy dull expression. Taken six weeks before death.

Oparanga (Case CI). Showing extreme emaciation. Taken three days before death.

To follow page 43

THE CONGO FLOOR MAGGOT

THE CONGO FLOOR MAGGOT

A BLOOD-SUCKING DIPTEROUS LARVA FOUND IN THE CONGO FREE STATE

(*The First Interim Report of the Expedition of the Liverpool School of Tropical Medicine to the Congo*, 1903. *Received January*, 1904)

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IN correspondence, during our stay at Boma, with the Rev. Holman Bentley and Mr. Sutton Smith, both of the Baptist Missionary Society Corporation, we learned of the existence in the Lower Congo of what were called 'floor maggots,' which they described as 'keen blood-suckers.'

It was not, however, until camped at a place called Nkanga, on our way from Tumba to Lutete, in the cataract region of the Congo, that we had an opportunity of seeing specimens of these maggots. Here, the head man of a neighbouring village, after being questioned on the subject of native pests, collected for us during the night, a number of what appeared to us—at first sight—to be ordinary blow-fly maggots. On a closer inspection many of them were seen to contain bright red blood.

A day or two afterwards, when visiting a native village, we had the opportunity of seeing the natives collect these blood-suckers by digging with the point of a knife or scraping with a sharpened stick in the dust-filled cracks and crevices of the mud floors of their huts. We were soon able to find them ourselves as easily as the natives, and unearthed many larvae which contained bright red blood. In collecting them the natives selected those huts in which the occupants slept on floor mats, saying that where people slept on beds or raised platforms the maggots were not so numerous. They informed us, however, that those who slept in beds which were not raised more than eighteen inches from the ground were also bitten, and credited the maggot with the power of jumping to that height. In some of the huts we collected, in a short space of time, as many as twenty from only a small proportion of the floor crevices. Many were turned up from a depth of three inches. In some of the cracks, and in moist, soft earth, they were found at greater depths. There is

no doubt that these maggots feed only at night. Mr. BENTLEY told us that as many as fifty could sometimes be found beneath a single mattress, and that he had known boys to be so pestered by them that they had preferred to sit all night outside a house to sleeping within it.

In one village, Nzungu, we visited a hut, measuring eight feet by ten feet, in which seven boys were sleeping on a small mat, and in the dust beneath a bed-platform on which slept a man and a woman. In the corner of the hut was the usual small fire and a sleeping pye-dog. Although we did not see the maggots actually feeding, we collected from beneath the mats and from amongst the boys' legs some half-dozen which were filled with recently sucked blood. The natives said that the maggots dropped off at once if the limb on which they were feeding was moved. There were specimens of all sizes, ranging in length from 2 to 15 mm. amongst those brought to us, and so far we have obtained them in every village we have visited. When ready to pupate, the larva lies dormant upon the surface, changes in colour to a pinky-brown, and later becomes a dark-reddish or brownish-black, chitinous, segmented, and oblong puparium.

We have never been able to substantiate the assertion, made to us on several occasions, that the maggot is able to jump to a height of eighteen inches. We think it more probable that they reach the raised beds by crawling up either the supports or the grass wall against which the bed is usually placed. We have, however, satisfied ourselves that it feeds mainly or entirely at night, and that it probably feeds nightly since blood in varying stages of digestion, and ranging in colour from bright red to black, can often be seen in its alimentary canal.

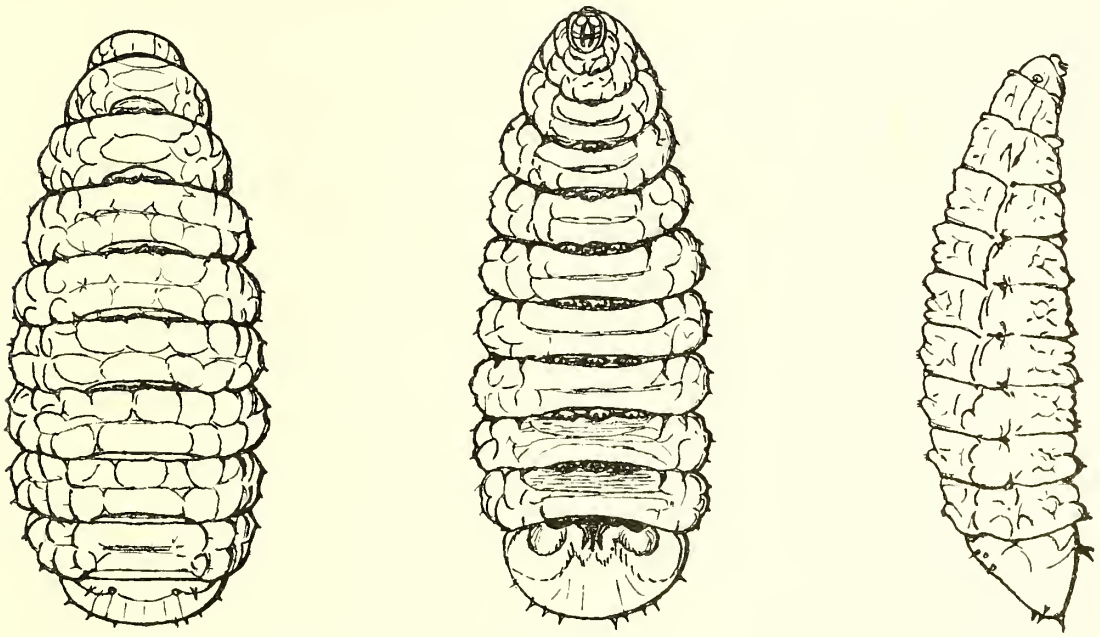
The distribution of this larva seems to be very extensive. We have collected it all over the Lutete and surrounding districts, and at Leopoldville. We have heard of it as being common at San Salvador, in Portuguese territory, on the Congo at Matadi in the cataract region, and at Tchumbiri one hundred and fifty miles above Stanley Pool.

Some of the native names for the maggot are as follows :—

At Tchumbiri, north of Leopoldville, the Bateke, according to Mrs. E. BILLINGTON of the Royal British Nurses' Association, call it 'Mabinzu.' At Leopoldville the Bateke call it 'Nchichi.' At Wathen (Lutete), in the cataract region, Mr. BENTLEY states that all maggots are called 'Ntunga,' and that there was no other special name for this one. At Matadi the native name is probably 'Mvidi.' In the Bangala district it is called 'Kiso.'

This larva maggot is semitranslucent, of a dirty white colour, acephalous, and amphipneustic. It resembles, when adult, the larvae of the bot-flies, and consists of eleven very distinct segments. The first or anterior one is divisible, by a slight constriction, into two portions, the foremost of which is small, bears the mouth parts, and is capable of protrusion and retraction to a considerable extent.

The larva is broadest at the ninth and tenth segments, is roughly ovoid in transverse section, and has, distinctly, dorsal and ventral surfaces. At the junction of the two surfaces is a row of irregular protuberances, two or more being placed on each segment. On each protuberance is a small posteriorly directed spine and a small pit. The central part of the ventral surface is flattened, and at the posterior margin of each segment is a set of three foot-pads, transversely arranged, each covered with small spines directed backwards. These aid the larva in its movements, which are fairly rapid and peculiar in that the mouth parts are protruded to the utmost and the tentacula fixed, as a purchase, first on one side then on the other, while a wave of contraction runs along the body as each segment is contracted and brought forward.



Dorsal Surface Ventral Surface Lateral Surface

CONGO FLOOR MAGGOT (× 4·5)

The last segment is larger than any of the others. Its upper surface is flattened, and looks backwards and upwards at an angle of about forty-five degrees with the longitudinal axis of the larva. This surface is roughly hexagonal and bears anteriorly, one on either side, the posterior spiracles which are seen with a pocket magnifying glass as three transverse, parallel, brown lines. Around this flattened surface, towards its border, are placed groups of rather prominent spines. The ventral surface of this segment is also flattened, and is thrown into folds by muscular contractions. The anus is situated in the anterior portion of this segment in the middle line, and is seen as a longitudinal slit, surrounded by a low ridge. Posterior to it, and on either side, is a large conspicuous spine. The anterior segment is roughly conical, and bears the mouth parts in front. Posteriorly, on the dorsal

surface, almost covered by the second segment, two spiracles, on either side, are seen with a low power as small brown spots. Two black hooks or tentacula protrude from the apex of this segment. They are curved towards the ventral surface of the maggot. The apex of each hook is blunt, and its base surrounded by a fleshy ring. Between them is the oral orifice. The tentacular processes are continued for some distance into the body of the maggot as black chitinous structures with expanded bases. There is probably, as in *Oestrus ovis* (Linn.), an articulation between the external and internal chitinous structures, since the arrangement of the mouth parts seems to be the same as in the maggot of that fly. Paired groups of minute spicular teeth are placed around the two tentacula so as to form a sort of cupping instrument. The arrangement of these teeth is as follows :—A rather large tubercle is situated on either side of and above the tentacula ; each is mounted by two or more groups of very small chitinous teeth. Just above each tentaculum is another small group of teeth. On either side of these black tentacula two irregular rows of small teeth are placed one above the other. The two latter groups are not placed upon tubercles. The integument of the larva is thick and difficult to tear. The larva is able to withstand a good deal of pressure without injury.

The following gross internal anatomical structures have at present been made out—intestinal canal, salivary gland, nervous system, and fat body. The intestinal canal commences as a short oesophagus, which ends in a proventriculus. A remarkable dorsal diverticulum, corresponding to the food reservoir of the muscid larvae, opens into the oesophagus near its anterior end. After the maggot has fed, the diverticulum is a very conspicuous object, since it is seen through the semi-transparent body wall as a bright red area when full of blood, extending from the head to about the fifth segment. The caeca, behind the proventriculus, have not as yet been well made out. The mid-intestine or chyle stomach is short, when compared with the hind intestine, and extends from the proventriculus to the junction of the urinary tubules with the gut. It lies in one or two coils. The hind intestine is very much coiled, and occupies the greatest part of the body cavity.

The urinary tubules are four in number, two on either side of the intestine. Each lateral pair combines to form a broad plate, from which is given off a single process for attachment to the gut.

Each salivary gland of the larvae consists of one very long acinus made up of large granular cells. The gland ends in a chitinous ringed duct, which joins its fellow of the opposite side to form a common duct, opening near the base of the free portion of the tentacula. The body of the maggot is lined by a white, loosely reticulated fat body. The minute anatomy of the mouth parts and sucking apparatus has not been studied.

The time required for the maturation of the larva is not yet known. The puparium is a dark-brown or black, cylindrical, segmented body, measuring 9-10·5 mm.

in length and 4-5 mm. in width. The anterior end is roughly conical, the posterior is rounded. All the external structures seen in the larva can be made out on the external surface of the puparium. The cuticle shows annular ridges.

The duration of the puparial stage is from a fortnight to three weeks.

During our stay at Wathen, Mr. BENTLEY showed us, among his collection of insects, a large light-brown fly which he believed to be developed from the floor maggots. Specimens caught in the boys' dormitory at the Wathen Mission were soon after brought in by one of our collectors, and later, while searching a native hut infested with 'floor maggots,' we saw one of these flies resting on the grass wall. Many others were subsequently found in the same building, sitting motionless amongst the beams and cob-webs of walls and roof. Because of their colour, which corresponded exactly with the smoke-stained straw and rafters of the huts, they were difficult to see, and in the dark interiors more difficult to catch.

This fly, seldom one of any other species, has since been found in many huts infested with maggots. We were told that the fly deposited its eggs on the ground of a hut, particularly in spots where urine had been voided. As a rule the fly is silent, but on one occasion we observed it buzzing loudly, fly in at the door and go directly beneath some bed mats which were raised by a low platform, some eight centimetres from the floor.

Both Mr. BENTLEY and the natives state that this fly never bites men.

The native name at Wathen for all flies is 'Nwanzi' or 'Mbwanzi.' The name for the fly which we describe is 'Nkulu Mwanzi.' The fly is thick set, and is of about the size and build of a 'blue bottle.' It is about 10-12 mm. in length, and once seen can be easily recognized. Its general colouring is tawny, but the small black hairs covering its body give it a smoky appearance. The head is large, as broad as the thorax, and protrudes in front of the eyes, which are when fresh a reddish brown in colour. The eyes are separated from each other, below, by a considerable interval and appear small in comparison with the size of the head. The proboscis is folded beneath the head in a deep groove, and is inconspicuous while in this position.

The palpi are club-shaped and covered, more particularly at their apices, with conspicuous black bristles. The third joint of the antenna is long, yellow, flattened from side to side, and rounded at its apex. It bears an arista which, thickened at its base (probably jointed), tapers to a fine point. The arista bears fine black hairs along its upper and lower borders; long at its base, the hairs become short and slanting at its apex. The dorsum of the thorax is flattened, and marked by longitudinal black and brown stripes, the transverse suture is well marked. The thorax is covered with fine black hairs and studded with rows of black bristles, which are particularly long on the sides. The squamae are very large, yellow in colour, and completely cover the yellowish-white halteres. The abdomen apparently consists of five segments. It is covered with long black hairs, and bears a few long bristles

on the last two segments. The second segment is the longest, and here the width of the abdomen is greatest, the upper surface of this segment is characteristically marked. A dark-brown or black line runs down the centre of the segment to meet at right angles a similar line which borders its posterior edge. There are no other marks on this segment, it is transparent, and its general colour is that of the rest of the body. The third segment is, except for a narrow yellow streak along its anterior border, very dark brown in colour, more marked laterally. The fourth segment is of the same dark colour, and is bordered posteriorly by a narrow lighter brown band. The fifth segment is small, and contains the genital apparatus. The wings are of a smoky brown colour, and show a well-marked venation. The legs are of the same buff as the rest of the body, and are covered with black hair and bristles. A noticeable feature of the legs is the jet-black fifth tarsal joint, which stands out prominently against the large cream-white pulvilus.

We have been able to allow a number of these maggots to feed on rats and guinea-pigs. We purpose carrying out a series of experiments with the object of determining whether they are able to play a part in the transmission of the human trypanosome. In none of the entomological works, which we can at present command, are we able to find any reference to habits or morphology by which we can identify this fly.

Specimens of the flies and larvae were submitted for identification to Mr. E. E. Austen, the dipterologist to the British Museum, together with a copy of the above report, and he has been kind enough to write as follows :—'I have read the report on the "Blood-Sucking Floor Maggot" with the greatest possible interest. Whether this maggot prove to be in any way concerned in the dissemination of trypanosomiasis or no, Messrs. Dutton, Todd, and Christy have come across an entirely novel and most interesting fact in the biology of diptera, and they are heartily to be congratulated on being the first to bring it to notice.

The flies are specimens of *Auchmeromyia luteola*, Fabr. (a species of the family Muscidae), which is widely distributed in tropical and subtropical Africa. Before reading the report, I was under the impression that the larvae of this fly were well known subcutaneous parasites of human beings and dogs, but I have now no doubt that *A. luteola* has been confused with another fly, very similar to it in appearance, but belonging to a different genus, the larvae of which are unquestionably subcutaneous parasites in man, dogs, and monkeys. Nevertheless,' says Mr. Austen, 'I have evidence, apparently reliable, which seems to show that the larvae of *A. luteola* may, perhaps, under exceptional circumstances, live subcutaneously in man. The fly itself, so far as I am aware, is otherwise harmless, and is incapable of sucking blood. Specimens in the national collection show that it ranges from Northern Nigeria to Natal ; it is therefore somewhat surprising that the habits of the larva have not been reported before.'

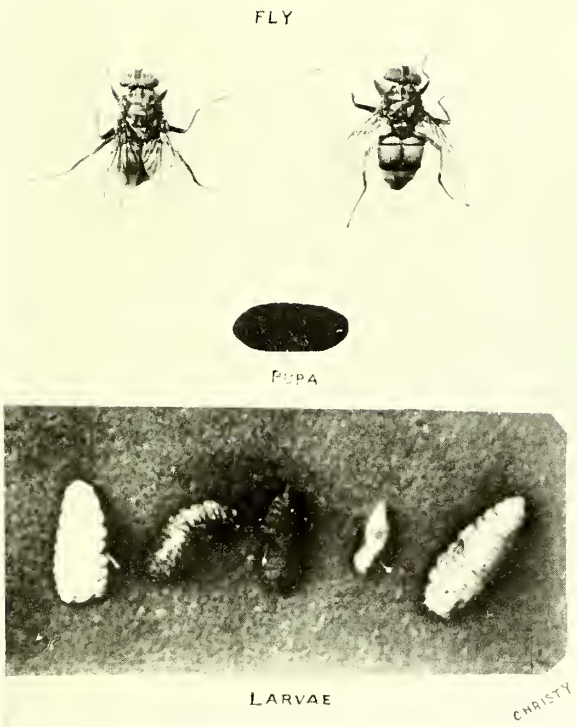


Fig. 1

Fig. 2

Fig. 3

CONGO FLOOR MAGGOT AND FLY

Fig. 1. Flies, pupa and larvae (nat. size). Fig. 2. Markings in the sand, produced by larvae.
Fig 3. Larvae immediately after, and some hours after, feeding. (× twice).

To face page 54

THE CEREBRO-SPINAL FLUID IN SLEEPING SICKNESS (TRYPANOSOMIASIS)

THE CEREBRO-SPINAL FLUID IN SLEEPING SICKNESS (TRYPANOSOMIASIS)

104 LUMBAR PUNCTURES

Second Interim Report of the Expedition of the Liverpool School of Tropical Medicine to the Congo, 1903

BY

CUTHBERT CHRISTY, M.B., C.M., Edin.

THE majority of the punctures recorded in the accompanying tables were performed whilst working with Drs. J. E. Dutton and J. L. Todd, in the Congo Free State. All, with the exception of three, were performed by myself either at Boma, Leopoldville, posts further up the Congo, or since returning to England. Some conclusions drawn from a number of them are published in our last conjoint report.[1]

Out of a total of sixty-four natives operated upon, the fifty-four in Table I were proved to be cases of sleeping sickness by the discovery of trypanosomes in the blood or cerebro-spinal fluid, or in hydrocele fluid ; while in the remaining ten in Table II, parasites were never found, although the majority of them were more or less suspicious cases, and all were in hospital at Leopoldville.

In thirty-four of the fifty-four sleeping sickness cases the parasites were found sooner or later in the cerebro-spinal fluid, whereas in twenty of them no parasites could be found, although in one (Case 17) the fluid was examined on five occasions.

If, however, those punctures in which the cerebro-spinal fluid was mixed with blood, and in which the parasites were found by coverslip examination to be present in the peripheral circulation on the same day as the puncture, be excluded, then we find that the result is very different, namely, forty-nine cases only, in twenty-five of which trypanosomes were found in the cerebro-spinal fluid, and twenty-four in which they were not found.

A reference to Cases 19, 20, 21, will show how important it is to exclude from all statistics those punctures in which the fluid contains trypanosomes with blood cells when the parasites are known to be in the blood stream. Cases 20 and 21 show clearly that when the fluid is mixed with blood the number of parasites appearing in it is closely in proportion, not only to the number in the blood, but to the amount of blood admitted by unskilful puncture. I therefore in this analysis will exclude from consideration all punctures in which the cerebro-spinal fluid and the blood both show trypanosomes when blood is admitted into the fluid. In the Tables these are marked with an asterisk in the name column.

1. *Second Progress Report.*

TABLE I.—SLEEPING SICKNESS CASES

\+ Indicates trypanosomes present. − Indicates trypanosomes absent. C.Cms. stands for cubic centimetres.
Those marked with an asterisk are excluded from statistics owing to blood appearing in the C.S.F. when trypanosomes are known to be present in the circulation. Centrif. stands for centrifugalisation.

No.	Name	Date	Sex	Age	Trypanosomes in Finger Blood	Cerebro-Spinal Fluid						Remarks on Duration of Case, Prominence of Sleep, or other Symptoms, etc., and Mode of Death
						C.Cms drawn off	Days before death	Trypanosomes	Clear or Cloudy	White Cells	Red Cells	Date of Death	
1	Elombo	17-12-03	♂	40	− 1 cover examined.	18	11	− 4 covers examined.	Clear.	..	None.	28-12-03	Duration about five months. Extreme emaciation. No symptoms of sleep. Comatose from sun exposure when punctured. Complete revival after withdrawal of fluid.
2	Lisasi .. .. *	16-12-03	♂	20	+ 5 to cover.	10	0	+ 7 to cover.	Cloudy.	..	Many.	16-12-03	Always slightly drowsy, increased before death.
3	Salamo.. .. *1	18-12-03	♀	24	+ 3 to cover.	15	2	+ 4 to cover.	Slightly cloudy.	Increased.	A few.	20-12-03	Duration about three months. Seldom seen dozing till a few days before death. Many division forms of trypanosomes in C.S.F., pericardial fluid, and bone marrow one-and-a-half hours *P.-M.*
	2	20-12-03			+ 13 to cover.	40	*P.-M.*	+ many.	Cloudy.	Increased.	None.	..	
4	Mokoko .. *	19-12-03	♂	21	+	17	..	+ 3 to cover.	Cloudy.	Increased.	Many.	..	Few symptoms. After being two months in hospital was discharged by medical officer as fit for duty. On January 5 his blood showed 1000 trypanosomes to the cover.
5	Dysiki	19-12-03	♀	26	− 2 covers examined.	18	2	+ 1 in 4 covers.	Clear.	Increased.	A few.	21-12-03	Emaciation but no sleep symptoms till day before death.
6	Bamliki	19-12-03	♂	16	− 2 covers.	6	6	− 1st centrif. 5 covers.	..	..	..	..	Extreme emaciation; slept a good deal, and was semi-comatose for three days before death. Bed-sores.
								+ 2nd centrif. 3 to cover.	Cloudy.	Scanty.	Many.	25-12-03	
7	Polulu .. *1	21-12-03	♂	21	+ 1 in 2 covers.	20	21	+ 1 in 2 covers.	Cloudy.	Scanty.	Many.	..	December 19 had severe left hemiplegic seizure. No sleep symptoms at any time. *P.-M.*—Extensive purulent meningitis. Cysticercus in triangularis.
	2	31-12-03			+ 3 to cover.		11	− 1st centrif. 2 covers.	Slightly cloudy.	Scanty.	None.	11-1-04	
								+ 2nd centrif.					
8	Tenda 1	23-12-03	♂	30	− 2 covers.	30	103	− 4 covers. 2 centrif.	Clear and limpid.	Very scanty.	None.	..	Duration about five months. Progressive emaciation. Frequently found dozing during January, but subsequently marked absence of nearly all symptoms. Retained faculties and was able to walk till hour of death. Pericardial and peritoneal fluids both showed active division forms twenty-and-a-half hours after death.
	*2	16-1-04			+ 6 to cover.	3	79	+ 1 in 2 covers.	Cloudy.	Increased.	A few.	..	
	3	27-2-04			− 2 covers.	25	37	− 4 covers. 2 centrif.	Clear and limpid.	Very scanty.	None.	..	
	4	31-3-04			− 2 covers.	16	4	+ 1 in 2 covers.	Clear.	Scanty.	A few.	4-4-04	
	5	5-4-04			− 2 covers before death.	47	*P.-M.*	− 5 covers. 13 hours *P.-M.*	Slightly cloudy.	Increased.	None.	..	

TABLE I.—*Continued*

No.	Name		Date	Sex	Age	Trypanosomes in Finger Blood	Cerebro-Spinal Fluid							Remarks on Duration of Case, Prominence of Sleep, or other Symptoms, etc., and Mode of Death
							C.Cms drawn off	Days before death	Trypanosomes	Clear or Cloudy	White Cells	Red Cells	Date of Death	
9	Manteka ..	1	24-12-03	♂	19	−	30	21	− 4 covers.	Clear.	Scanty.	A few.	..	Duration about three months. Rapid emaciation. Irritable and troublesome. Helpless, dazed, semi-comatose condition for two days before death.
		2	5-1-04			+	16	9	+	Clear and limpid.	Scanty.	None.	14-1-04	
10	Manga.. ..	*	26-12-03	♂	22	+	30	9	+	Slightly cloudy.	Increased.	A few.	4-1-04	Duration about five months. Marked absence of symptoms till ten days before death when sudden fit, followed by semi-comatose condition, great salivation, abolished reflexes, and erection of penis. Improved somewhat before death.
11	Kalenga ..	*1	26-12-03	♂	24	+ 1 in 2 covers.	18	45	+ 2 to cover.	Cloudy.	Scanty.	Many.	9-2-04	Marked sleep symptoms, no emaciation. Death due to sun exposure; temperature 107. Blood showed 100 trypanosomes to cover five hours before death.
		2	9-2-04			+ 2 covers 1½ hours *P.-M.*	24	*P.-M.* 1½ hrs.	− 4 covers. 2 centrif.	Cloudy.	Much increased.	None.	..	
12	Maiadina ..	*1	29-12-03	♂	18	+ 2 to cover.	14	28	+ 36 to cover.	Cloudy.	Scanty.	Many.	..	Duration about four months. Had epileptic attacks, few sleep symptoms, simple-minded condition. Six days before death, tremors of limbs, opisthotonos and semi-coma. *P.-M.* Pneumonia and purulent meningitis.
		2	22-1-04			− 2 covers.	18	4	− 3 covers. 2 centrif.	Clear and limpid.	Scanty.	None.	26-1-04	
13	Kabali	1	29-12-03	♀	27	− 2 covers.	5	70	− 8 covers. 2 centrif.	Cloudy.	Increased.	A few.	..	Duration about five months. Progressive weakness, drowsiness, and emaciation from the first. No marked nervous symptoms. Bed-sores.
		2	20-2-04			+ 6 to cover.	32	17	− 5 covers. 2 centrif.	Clear.	Scanty.	A few.	..	
		3	7-3-04			− 2 covers.	15	1	− 5 covers. 2 centrif.	Clear.	Scanty.	None.	8-3-04	
14	Molumba ..	1	7-1-04	♂	32	− 2 covers.	14	..	− 5 covers. 2 centrif.	Cloudy.	Increased.	Many.	..	No marked symptoms, no drowsiness or dulness. Recurring attacks of dysentery.
		2	5-2-04			− 1 in 2 covers.	22	..	− 3 covers. 2 centrif.	Clear and limpid.	Scanty.	None.	..	
15	Kondolo ..	1	8-1-04	♂	32	− 2 covers.	20	80	− 5 covers. 3 centrif.	Clear and limpid.	Very scanty.	None.	..	Three months in hospital. Marked absence of symptoms except headache. Ten days before death headache severe. Suicide by drowning.
		2	14-3-04			− 2 covers.	22	14	− 2 covers. 2 centrif.	Clear and limpid.	Very scanty.	None.	28-3-04	
16	Ejoli		7-1-04	♂	28	− 2 covers.	8	19	− 3 covers. 3 centrif.	Clear.	Increased.	None.	..	Drowsiness. Great emaciation. Few symptoms. Conscious till hour of death.

TABLE I.—*Continued*

No.	Name	Date	Sex	Age	Trypanosomes in Finger Blood	Cerebro-Spinal Fluid							Remarks on Duration of Case, Prominence of Sleep or other Symptoms, etc., and Mode of Death
						C.Cms drawn off	Days before death	Trypanosomes	Clear or Cloudy	White Cells	Red Cells	Date of Death	
17	Oparanga .. 1	7-1-04	♂	18	– 2 covers.	16	62	– 3 covers. 2 centrif.	Clear and limp.	Scanty.	None.	..	Duration five or six months. Progressive weakness and emaciation from the beginning. Extreme emaciation at death. No dulness or marked symptom at any time, except drowsiness during January, but less subsequently. Could walk, and retained faculties till hour of death. Trypanosomes in blood scanty and seldom seen.
	2	27-1-04			+ 3 to cover.	20	42	– 7 covers. 3 centrif.	Clear and limp.	Scanty.	A few.	..	
	3	11-2-04			– 2 covers.	20	27	– 2 covers. 2 centrif.	Clear and limp.	Scanty.	None.	..	
	4	23-2-04			– 2 covers.	14	15	– 3 covers. 2 centrif.	Clear and limp.	Scanty.	None.	..	
	5	2-3-04			+ 48 to cover.	10	7	– 6 covers. 2 centrif.	Clear and limp.	Scanty.	None.	9-3-04	
18	Kimfuta	15-1-04	♀	9	+ 8 to cover.	16	5	+ many.	Clear and limp.	Very scanty.	None.	20-1-04	Duration about two months only. Rapid emaciation, weakness, tremors, and vacancy. Sleep symptoms only present towards the end.
19	Banja *1	20-1-04	♂	26	+ 80 to cover.	8	..	+ many.	..	..	Much blood.	..	Was admitted January 5 from prison, chained to two other prisoners, and charged with cannibalism. Emaciation due to starvation. By end of February quite well and putting on flesh. At end of April started for England, arriving apparently in robust health. Now fat and well, and is employed in Johnston Laboratory, Liverpool.
	2	8-2-04			+ 100 to cover.	50	..	– 4 covers. 2 centrif.	Clear and limp.	Scanty.	None.	..	
	3	26-2-04			+ 32 to cover.	20	..	– 4 covers. 2 centrif.	Clear and limp.	Scanty.	None.	..	
20	Ieri 1	20-1-04	♂	11	+ 2 to cover.	20	19	+ 1 in 2 covers.	Clear and limp.	Very scanty.	None.	..	Duration about four months. Progressive weakening and wasting interrupted by periods of partial recovery. Somnolence marked towards the close. Appetite voracious to within three days of death. Marked retraction of head and opisthotonos for several days before death. *P.-M.*—No signs of purulent meningitis.
	2	27-1-04			+ 4 to cover.	10	12	+ 4 to cover.	Clear and limp.	Very scanty.	None.	..	
	*3	5-2-04			+ 150 to cover.	10	3	+ 40 to cover.	Cloudy.	Scanty.	Many.	..	
	*4	8-2-04			+ 250 to cover.	15	0	+ 1 in 2 covers.	Clear.	Scanty.	A few.	8-2-04	
21	Moidi 1	21-1-04	♀	24	– 2 covers.	6	16	– 3 covers. 2 centrif.	Clear.	Scanty.	A few.	..	Duration about three months. Drowsiness a marked symptom latterly. Very little wasting. Sensations much dulled. Lumbar puncture without cocaine.
	*2	25-1-04			+ 13 to cover.	35	12	+ 3 in 4 covers.	Clear.	Scanty.	A few.	..	
	*3	5-2-04			+ 100 to cover.	15	1	+ 20 to cover.	Slightly cloudy.	Scanty.	Many.	6-2-04	
22	Kapinga	23-1-04	♀	24	+ 1 in 2 covers.	20	12	+ 1 in 3 covers.	Slightly cloudy.	Much increased.	None.	4-2-04	No drowsiness at any time. Progressive weakness, vacancy, and emaciation.

TABLE I.—*Continuea*

No.	Name		Date	Sex	Age	Trypanosomes in Finger Blood	Cerebro-Spinal Fluid							Remarks on Duration of Cases, Prominence of Sleep and other Symptoms, etc., and Mode of Death
							C.Cms drawn off	Days before Death	Trypanosomes	Clear or Cloudy	White Cells	Red Cells	Date of Death	
23	Patesa		3-2-04	♀	12	+ thousands, 15 to field.	10	0	– 6 covers. 2 centrif.	Clear.	Scanty.	A few.	3-2-04	Only four days under observation. Admitted for dysentery. Emaciation extreme. No drowsiness or vacancy. L.P. four hours before death. Spleen punctured at same time showed 80 trypanosomes to field.
24	Boyo		9-2-04	♂	19	– 2 covers.	32	0	– 3 covers. 2 centrif.	Slightly cloudy.	Increased.	None.	9-2-04	Duration three to four months. L.P. soon after convulsive seizure, with high temperature and unconsciousness, due to sun exposure. Six hours before puncture, finger blood showed 100 trypanosomes to the cover.
25	Benjamin		9-2-04	♂	28	+ 2 to cover.	13	1	+ 9 to cover.	Slightly cloudy.	Greatly increased. Early pus.	None.	10-2-04	Duration about three months. Progressive weakness and emaciation. Seldom drowsy. Tremors almost amounting to rigors before death, which was hastened by sun exposure. *P.M.*—Intense cerebral congestion.
26	Boyo Mitchel	1	11-2-04	♂	16	– 1 cover.	18	103	+ 3 to cover.	Cloudy.	Increased.	Many.	..	Duration about five months. Drowsiness very marked. Extreme emaciation during last month on voyage to England. Died on the way to Liverpool.
		2	27-2-04			– 2 covers.	16	99	– 4 covers. 2 centrif.	Clear.	Increased.	None.	..	
		3	31-3-04			– 2 covers.	25	54	+ 1 in 2nd centrif.	Slightly cloudy.	Increased.	Many.	.	
		4	23-5-04			+ 5 to cover.	35	1	+ 1 in 5 covers.	Clear and limp.	Scanty.	None.	24-5-04	
		5	24-5-04			– 1 cover.	30	*P.-M.*	–	Cloudy.	Increased.	None.	..	
27	Kabongo		13-2-04	♂	35	– 4 to cover.	4	..	– 4 covers. 2 centrif.	Clear and limp.	Scanty.	None.	..	Very few symptoms. No drowsiness. Still under observation. On March 21, hydrocele fluid negative, three covers examined; blood positive, 12 to cover.
28	Kikoffi	1	20-2-04	♂	19	+ 10 to cover.	30	..	+ 60 to cover.	Slightly cloudy.	Much increased.	None.	..	Still under observation. Nervous tremors and exaggerated reflexes on admission, but improved subsequently.
		2	25-3-04			– 2 covers.	26	..	+ 1 in 1 cover.	Clear and limp.	Scanty.	None.	..	
29	Yaiyai	1	24-2-04	♀	20	– 2 covers.	42	9	– 3 covers. 2 centrif.	Slightly cloudy.	Increased.	A few.	..	Duration two months only. Rapid progressive weakness and wasting. Symptoms of drowsiness not marked. Extensive bed-sores. Conscious till hour of death.
		2	28-2-04			– 4 covers.	10	7	+ 1 in 3 covers.	Clear.	Increased.	None.	..	
		3	4-3-04			– 2 covers.	15	0	– 4 covers.	Clear and limp.	Scanty.	None.	4-3-04	

TABLE I.—*Continued*

No.	Name		Date	Sex	Age	Trypanosomes in Finger Blood	Cerebro-Spinal Fluid						Remarks on Duration of Cases, Prominence of Sleep and other Symptoms, etc., and Mode of Death
							C.Cms drawn off	Days before Death	Trypanosomes	Clear or Cloudy	White Cells	Red Cells	Date of Death	
30	Plekai		26-2-04	♂	27	– 1 cover.	22	9	– 5 covers.	Clear and limp.	Scanty.	None.	4-3-04	Admitted February 2 for dysentery. No drowsiness. Great emaciation.
31	Bugwendi		2-3-04	♂	16	+ 48 to cover.	25	19	– 5 covers. 2 centrif.	Clear and limp.	Scanty.	None.	..	Duration about three months. Irritable, hysterical, and troublesome. March 16, wasting and incoordination of movements. March 20, suddenly drank his urine from pot. Then developed incoherent jabber, spasmodic contraction of arms, with athetosic movements of fingers, and picking at blanket. *P.-M.*—Lymph round cord, ventricles of brain distended, etc.
			21-3-04			– 2 covers.	15	0	– 3 covers. 2 centrif.	Slightly cloudy. Straw colour.	Scanty.	Many.	21-3-04	
32	Belambo ..	1	27-2-04	♂	26	– 4 covers.	24	46	+ 1 to 4 covers.	Cloudy.	Greatly increased.	Many.	..	Duration about three months. Drowsiness at no time very marked, but nervous symptoms, tremors, and increased reflexes from day of admission. On March 19 developed spasmodic flexion of arms with convulsive movements of face and many muscles of body. No wasting. Appetite good, and conscious till hour of death. Reflexes nearly absent at death.
		2	9-3-04			..	18	35	+ 1 in 4 covers	Clear.	Increased.	None.	..	
		3	30-3-04			– 2 covers.	20	14	+ 80 to cover.	Cloudy.	Increased.	Many.	..	
		4	13-4-04			– 2 covers.	40	0	+ 1 in 6 covers.	Clear and limp.	Scanty.	None.	13-4-04	
33	Litali		29-2-04	♂	26	– 5 covers.	18	2	+ 3 to cover.	Slightly cloudy.	Greatly increased.	None.	2-3-04	Admitted to hospital as a jabbering lunatic. Not suspected of sleeping sickness. Under observation for two months. Trypanosomes never found in blood. Slept a good deal latterly.
34	Mozao	1	11-3-04	♂	16	+ 3 to cover.	18	7	– 5 covers. 2 centrif.	Clear and limp.	Scanty.	None.	..	No very marked symptoms, but extreme emaciation.
		2	18-3-04			– 2 covers.	4	0	– 3 covers.	Clear.	Increased.	None.	18-3-04	
35	Batanga		14-3-04	♂	27	– 2 covers.	24	24	+ 2 to cover.	Clear.	Slightly increased.	None.	7-4-04	Duration about three months. Marked sleep symptoms. Epileptic attack March 17. At L.P. pressure of fluid considerable. Improvement in symptoms after puncture.
36	Bandela ..	1	16-3-04	♂	10	– 2 covers.	22	30	– 4 covers. 2 centrif.	Clear.	Increased.	None.	16-4-04	Duration five months. March 12, had appearance of bloated cretin, with marked sleep symptoms, which disappeared for several days after L.P. Salivation, no wasting. *P.-M.*—Large quantity ventricular and subarachnoid fluid.
		2	16-4-04			– 1 cover from heart.	50	*P.-M.*	– 3 covers. 8 hours' *P.-M.*	Cloudy.	Much increased.	None.	..	

TABLE I.—*Continued*

No.	Name	Date	Sex	Age	Trypanosomes in Finger Blood	Cerebro-Spinal Fluid						Remarks on Duration of Cases, Prominence of Sleep and other Symptoms, etc., and Mode of Death
						C.Cms drawn off	Days before Death	Trypanosomes	Clear or Cloudy	White Cells	Red Cells	Date of Death	
37	Mengo	10-3-04	♂	24	– 2 covers.	26	..	– 6 covers. 2 centrif.	Clear.	Scanty.	A few.	..	Admitted March 2 with very marked nervous symptoms; childish and irritable. One flick with finger on patella caused uncontrollable clonic movements of legs, and a light tap caused violent extension of whole body and legs, so powerful as to throw him off a chair yet able to walk about and do light work. Observations up to April 29, 1904. * Refers to hydrocele fluid only.
	Hydrocele fluid	10-3-04			– 2 covers.	..	..	+ 14 to cover.	Cloudy.	Large quantity.	None.	*	
	" "	25-3-04			– 2 covers.	..	..	+ 2 in half deposit.	Cloudy.	Large quantity.	None.	*	
38	Tumba .. 1	26-3-04	♂	21	– 2 covers.	35	4	+ 20 to cover.	Slightly cloudy.	Scanty.	Many.	..	Duration about three months. Marked dulness and sleep symptoms. March 22, developed tremors and flexions of arms, which increased after L.P. on 26th. Conscious up to time of death.
	2	30-3-04			– 2 covers.	34	0	– 4 covers. 2 centrif.	Clear and limp.	Scanty.	None.	30-3-04	
39	Molar	26-3-04	♂	14	– 2 covers.	15	..	+ 2 covers.	Clear.	Scanty.	A few.	..	Mission boy, only under observation for a few days.
40	Mokindi	2-4-04	♂	18	+ 4 to cover.	20	..	– 5 covers. 2 centrif.	Clear and limp.	Very scanty.	None.	..	An early case. Did not admit that he was sick. Marked dulness, but no sleep. Distension of belly.
41	Nakunyi	4-4-04	♂	17	+ 16 to cover.	18	4	– 4 covers. 2 centrif.	Clear and limp.	Very scanty	None.	8-4-04	Admitted a month before death for dysentery. Great emaciation and weakness, otherwise few symptoms, and none of sleep.
42	Pania	6-4-04	♂	18	+ 4 to cover.	18	..	– 5 covers. 2 centrif.	Clear and limp.	Scanty.	None.	..	An early case. Few marked symptoms and none of sleep.
43	Kitambala	7-4-04	♂	13	– 4 covers.	18	..	– 4 covers.	Clear and limp.	Scanty.	None.	..	An early case. Marked dulness, but no sleep.
44	Katambue	14-4-04	♂	24	– 2 covers.	26	..	+ 300 to cover.	Slightly cloudy.	Increased.	A few.	..	An advanced case. Only under observation for one day. Was doing duty up to February 27, 1904.
45	Kabela	14-4-04	♂	26	– 2 covers.	20	..	+ 16 to cover.	Slightly cloudy.	Scanty.	A few.	..	An advanced case. Only under observation for one day. Was doing duty up to February 17, 1904.
46	Ekongo	18-4-04	♂	21	– 2 covers.	22	..	+ 6 to cover.	Slightly cloudy.	Increased.	A few.	..	Advanced case seen on the march, and punctured under difficulties in the grass.
47	Mbeka	18-4-04	♂	19	+ 2 to cover.	20	..	– 4 covers. 2 centrif.	Clear and limp.	Scanty.	None.	..	An early case, with no symptoms.

TABLE I.—*Continued*

No.	Name	Date	Sex	Age	Trypanosomes in Finger Blood	Cerebro-Spinal Fluid							Remarks on Duration of Cases, Prominence of Sleep, or other Symptoms, etc., and Mode of Death
						C.Cms. drawn off	Days before death	Trypanosomes	Clear or Cloudy	White Cells	Red Cells	Date of Death	
48	Kitambue	23-5-04	♂	24	+ 4 to cover.	18	17	+ 1 in 4 covers.	Slightly cloudy.	Much increased.	None.	9-6-04	Admitted to hospital beginning of March. Started for England April 29. Weakness increased during voyage and sleep a marked symptom. Admitted Royal Infirmary, Liverpool, May 24. Slept almost continuously before death.
49	Tomi	23-5-04	♂	17	+ 5 to cover.	4	25	− 3 covers. 2 centrif.	Clear and limp.	Slightly increased.	None.	..	Boy from Kinshassa Mission, Leopoldville. Duration of case about five months. No sleep symptoms. Progressive wasting and weakness.
		15-6-04			+	30	1	+ many. 5 to field.	Clear.	Increased.	None.	17-6-04	
		17-6-04			+	45	*P.-M.*	− 2 covers.	Slightly cloudy.	Much increased.	None.	..	
50	Nbela	9-10-03	♂	24	..	35	23	+ many.	Slightly cloudy.	Much increased.	None.	1-11-04	An advanced case seen at Boma. Marked somnolence. Admitted August 1.
51	John Paka	26-10-03	♂	28	+ 2 to cover.	10	..	− 6 covers.	Clear and limpid.	Very scanty.	A few.	..	An early case. A Sierre Leone boy seen at Boma. No symptoms and no complaint.
52	Louis .. .. *	16-10-03	♂	16	+ 11 to cover.	15	..	+	Slightly cloudy.	Increased.	A few.	Died.	An advanced case in hospital at Boma.
53	Somi	13-10-03	♀	22	+ 6 to cover.	20	..	+ many.	Slightly cloudy.	Increased.	None.	Died.	An advanced case seen in hospital at Boma.
54	Bulumbu	29-10-03	♀	16	− 1 cover.	10	..	+	..	..	..	Died.	An advanced case seen at Boma.

TABLE II.—DOUBTFUL CASES NOT PROVED TO BE SLEEPING SICKNESS

No.	Name	Date	Sex	Age	Trypanosomes in Finger Blood	Cerebro-Spinal Fluid							Remarks on Duration of Case, Prominence of Sleep, or other Symptoms, etc., and Mode of Death
						C.Cms. drawn off	Days before death	Trypanosomes	Clear or Cloudy	White Cells	Red Cells	Date of Death	
1	Mwandu	22-12-03	♂	21	– 2 covers.	20	8	– 6 covers.	Clear.	Scanty.	A few.	31-12-03	Apparently a case of miliary tubercle. No symptoms of sleeping sickness.
2	Lembi	24-12-03	♂	23	–	5	9	– 5 covers.	Clear.	Scanty.	A few.	2-1-04	Probably a sleeping sickness case. Extreme emaciation.
3	Salabantu	30-12-03	♂	19	–	15	..	– 4 covers.	Clear and limpid.	Scanty.	None.	..	Discharged as fit. No symptoms of sleeping sickness.
4	Saoka	7-1-04	♂	17	–	10	..	– 3 covers. 2 centrif.	Clear and limpid.	Very scanty.	None.	..	A doubtful case, with symptoms of dulness and childishness, admitted December 26 and discharged as fit March 23, 1904.
5	Luisi	10-1-04	♀	24	–	14	0	– 5 covers.	Clear and limpid.	Scanty.	None.	10-1-04	A doubtful case, with marked emaciation and some sleep symptoms.
6	Kanyinki	22-1-04	♂	24	–	18	33	– 3 covers. 3 centrif.	Clear and limpid.	Scanty.	None.	25-2-04	A doubtful case, admitted for severe dysentery. Extreme emaciation but no dulness or drowsiness.
7	Sungula	5-2-04	♂	30	–	40	0	– 4 covers. 2 centrif.	Milky.	Large deposit of pus.	None.	5-2-04	Doubtful history of sleeping sickness; no emaciation. Retraction of head and convulsive movements of arms. Died a few hours after admission. *P.-M.*—Purulent meningitis and ethmoidal sinuses full of pus.
8	Fatiala	8-2-04	♀	20	– 2 covers.	22	4	– 3 covers. 2 centrif.	Clear.	Increased.	A few.	12-2-04	A septic case, with large collection of pus in arm when admitted.
9	Pongor	10-2-04	♂	24	– 3 covers.	15	0	– 1 cover.	Greenish-yellow cloudy fluid.	Pus.	None.	10-2-04	Semi-comatose when admitted. Somewhat similar case to No. 8. Excessive continuous rigors, and pus oozing from nostrils. Violent spasmodic contraction of limbs.
10	Kabinda	27-2-04	♂	18	– 1 cover.	25	..	– 2 covers. 2 centrif.	Clear and limpid.	Very scanty, almost none.	None.	..	Only in hospital half-an-hour asking for treatment for craw-craw.

In studying the column in Table I, in which is enumerated the number of days intervening between the puncture and the date of death, it will be seen that the probability of trypanosomes being found in the cerebro-spinal fluid tends to increase as one nears the fatal termination. This is shown by the following two Tables compiled from the forty-nine cases under analysis.

TABLE III

Thirty-five cases known to have proved fatal at the date of compilation of Table I

Days before Death	Number of Punctures	Trypanosomes present	Trypanosomes absent
Within ten days	27	14	13
Between ten and thirty	19	8	11
Between thirty and one hundred and ten ...	11	4	7
Totals	57	26	31

TABLE IV

Fourteen cases not known to be fatal at the time of compilation of Table I

Days before Death	No. of Punctures	Trypanosomes present	Trypanosomes absent
The majority are comparatively early cases, and presumably many days from death	17	6	11

A larger series of lumbar punctures in the earlier stages would be of the greatest interest.

In the Congo disease, as we have seen it, mainly at Leopoldville, there is an endless variety of types. In most cases sleep is absent, in some dulness and apathy are prominent, in others nervous symptoms and mania are conspicuous, while a proportion of cases have only progressive emaciation and fever. In classifying these symptoms it does not seem possible to definitely connect them with the appearance or non-appearance of trypanosomes in the cerebro-spinal fluid.

Cases 8 and 17, each of them punctured several times with negative results, except in one instance, were conspicuous for general absence of all symptoms except fever and emaciation. They each were able to walk and retain their faculties up to the time of death. Case 13 is in many respects similar to the foregoing two, but developed increased drowsiness some time before death, although showing no parasites in the cerebro-spinal fluid.