

First report of the trypanosomiasis expedition to Senegambia (1902) of the Liverpool School of Tropical Medicine and Medical Parasitology / by J. Everett Dutton and John L. Todd ; with notes by H.E. Annett and an appendix by F. V. Theopold.

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TRYPANOSOMIASIS

BY
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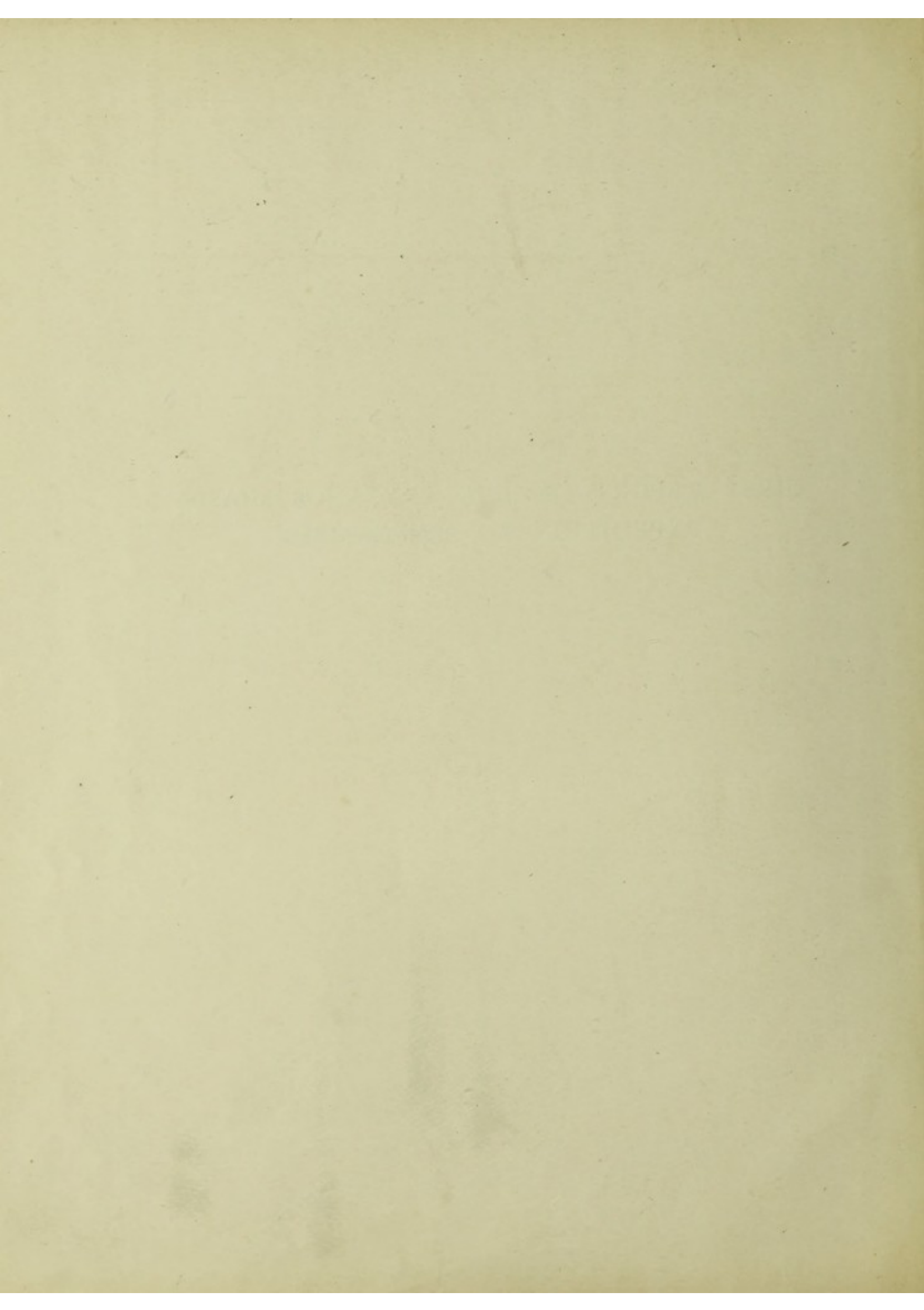


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FIRST REPORT OF THE TRYPANOSOMIASIS
EXPEDITION TO SENEGAMBIA



LIVERPOOL SCHOOL OF TROPICAL MEDICINE—MEMOIR XI

FIRST REPORT
OF THE
TRYPANOSOMIASIS EXPEDITION
TO SENEGAMBIA
(1902)

OF THE
LIVERPOOL SCHOOL OF TROPICAL MEDICINE
AND MEDICAL PARASITOLOGY

BY
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AND AN
APPENDIX BY F. V. THEOBALD, M.A.

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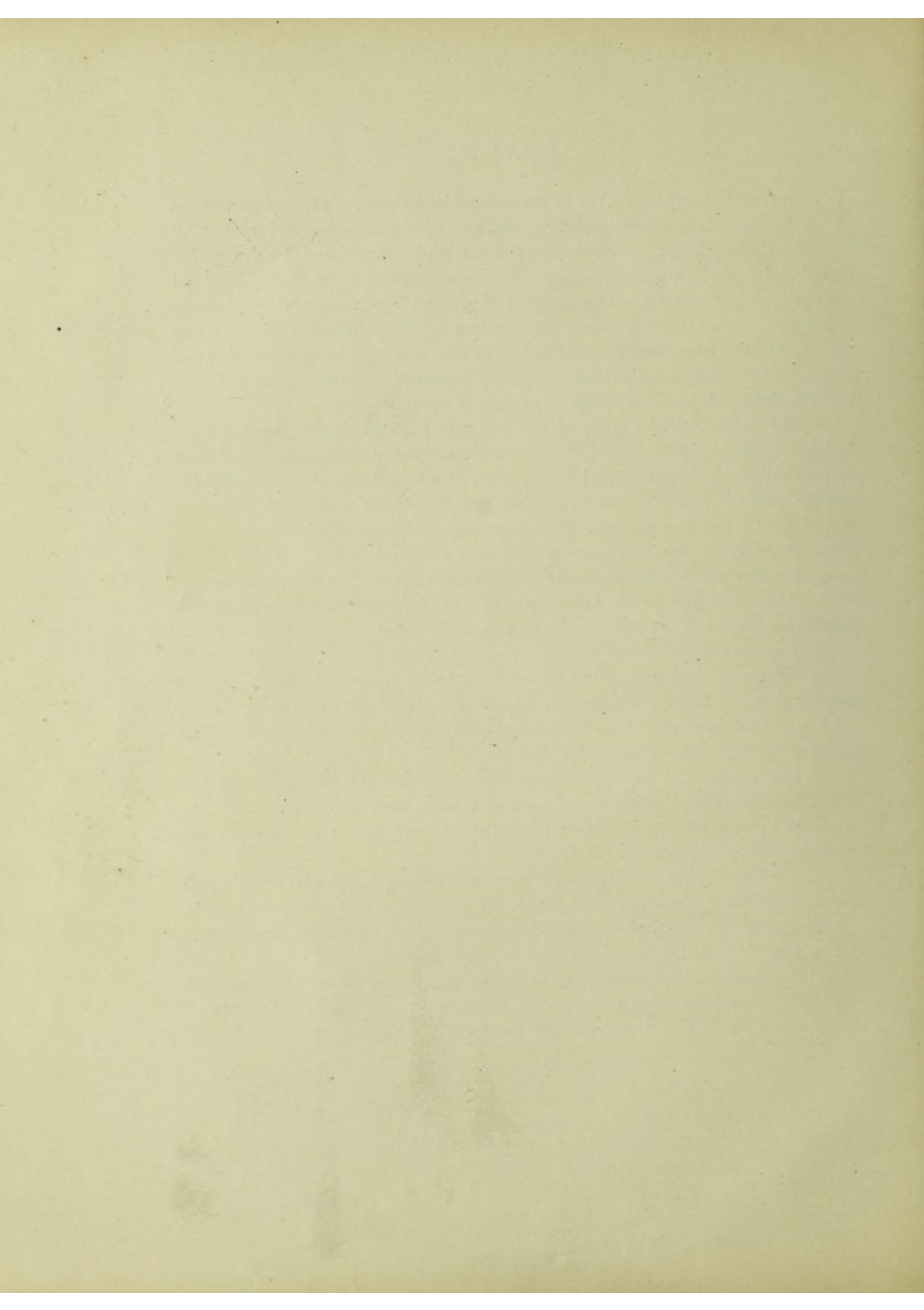
Monsieur MUTIN-BONDET, Veterinary Surgeon.

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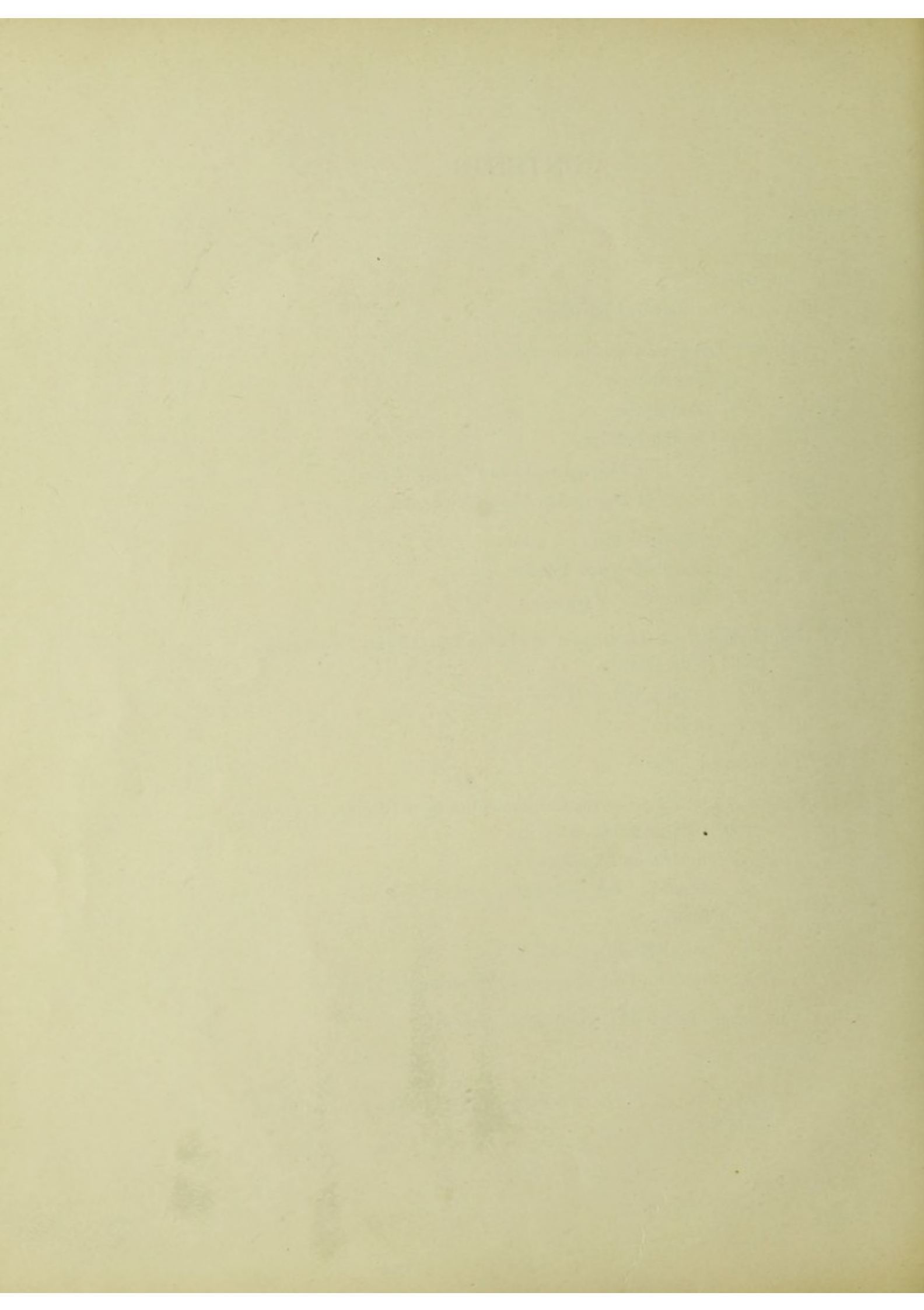
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FIRST REPORT OF THE TRYPANOSOMIASIS EXPEDITION TO SENEGAMBIA (1902)

I. INTRODUCTION

WE arrived in Bathurst on the 2nd of September, 1902, and remained there for three weeks. Cape St. Mary, seven miles from Bathurst, then became our headquarters, and we stayed there until the first week in January, when we went to McCarthy Island, which we made our base until we left for Senegal on the 7th of May. Subsequently, we remained in French territory until we left for England about the middle of June, 1903. While we were in the Gambia we arranged our movements so as to allow the study of the distribution and of the properties of the Gambian trypanosome to proceed simultaneously. To this end we established our laboratory at a convenient spot, as a base at which experimental work could be carried on, and from which short journeys could be made with the object of determining the frequency of trypanosomiasis among the natives.

Our stay in Senegal was almost totally occupied in studying the sanitary conditions of the principal towns, and in drawing up a scheme, at the request of M. ROUME, the Governor-General of French West Africa, on which to base an anti-mosquito campaign.

A description of the work done in Senegal has been embodied in another report. This, 'The First Report' of our expedition, is intended to describe only a very small portion of the work done in the Gambia on trypanosomiasis.

Although trypanosomes were found in men, horses, rats, mice, birds, snakes, tortoises, and frogs, we were able to study only those parasites found in men and horses. The distribution, clinical symptoms, and the morphology of these latter parasites, together with a synopsis of their reactions in experimental animals, will be first described; there will then follow a brief description of the organisms seen in the remaining hosts.

We shall only briefly mention our unsuccessful attempts to transmit the disease from infected to uninfected animals by means of biting flies.

A second report will contain a more extended study of the results of animal inoculations of the various trypanosomata found in the Gambia. It will also contain our, at present incomplete, observations on their agglutinative powers and on the

possibility of altering their virulencies. We reserve for this report descriptions of the microscopical *post-mortem* appearances in infected animals, and a detailed comparative examination of all known pathogenic trypanosomes.

LABORATORY METHODS

Blood was examined, both fresh and stained. When it was possible the centrifuge was used in preparing the fresh specimens, which were examined as soon as prepared. We found that if trypanosome-infected blood were mixed with equal parts of the following solution and centrifuged, the great majority of the parasites were collected with the white cells in a thin white layer, which could be easily removed with a fine pipette:—

Sodium citrate	1 gramme.
Normal saline solution	100 c.c.

For centrifuging the blood of animals from which it was possible to obtain several cubic centimetres, urine centrifuging tubes were used. These were far too large, however, for examining the blood of natives, small birds, and animals, of which we could obtain only a few drops at a time. For examining such small quantities, varying in amount from 0.1 c.c. to 0.25 c.c., we adopted the following method. Rather thin-walled, easily fusible glass tubing, having an internal diameter of five mm., was drawn out into bulb-shaped capsules having an arm at either end, and capable of being placed in the haematokrit arm of a centrifuge.* Blood was allowed to enter the bulb by capillarity, until it was half or three-quarters filled. The sodium citrate solution used as a diluting fluid was then allowed to fill up the remaining space, and the bulb, its longer arm having being carefully sealed in the flame of a spirit lamp, was placed in the centrifuge.

In fifteen minutes a well-marked white ring is present. The serum and diluting fluid can very quickly be removed by gently scratching and then breaking the glass about four or five mm. above the ring, and smartly tapping the bulb containing the undesired clear fluid. The white ring can then be easily removed by a capillary pipette and examined under a coverslip as a moist preparation.

We nearly always used a one-sixth (Zeiss D) objective with a No. 4 ocular (the diaphragm of which had been removed) in searching fresh blood for trypanosomes. By doing this we secured a large field, and were able to examine slides very quickly. Since a living parasite is always 'spotted' by its movements rather than its form, we lost but little efficiency in so doing. Even with the aid of this manoeuvre, ten to fifteen minutes were required to examine the amount of blood covered by a three-quarter inch square coverslip, the size which we used throughout our work.

Blood films on slides were taken from every case for permanent preparations and further study.

* Kanthack, Durham, and Blandford, *Royal Society Proceedings*, vol. lxiv, No. 404, 1898; Durham, *Thompson Yates Laboratories Report*, Vol. IV, pt. ii, 1902.

Films were fixed either by absolute alcohol or by the vapour of the following mixture, in which preparations were allowed to remain for from five to fifteen seconds, as recommended by PLIMMER and BRADFORD.*

Two per cent. solution Osmic Acid	}	Equal parts.
* Glacial Acetic Acid - - -		

We found the latter to be the better fixative. Its results were more constant, and the minute structure of the parasites, particularly the chromatic granules, were better preserved.

All blood films, except where it is otherwise stated, were stained by the following modification of ROMANOWSKY's stain, suggested by Dr. MACCONKEY:—

<i>Solution A.</i> Medicinal methylene blue (Hoechst)	-	-	0.5	gramme
Sat. solution of chemically pure borax in dis-				
tilled water	-	-	-	50.0 c.c.
Incubate for four days at 37°C., then add absolute				
alcohol	-	-	-	50.0 c.c.

and allow to stand for one day before using.

<i>Solution B.</i> Eosin, extra B.A. crystal (Hoechst)	-	-	0.25	gramme
Distilled water	-	-	-	50.00 c.c.
Absolute alcohol	-	-	-	50.00 c.c.

These are the stock solutions. For laboratory use dilute each with distilled water, one part of stain to nineteen of water. Support the slide to be stained face downwards. Mix equal parts of the diluted stains in a small flask and pour immediately into the staining dish. The length of time necessary for staining varies from three to six minutes.

When staining is complete, wash the specimen quickly but thoroughly in water and allow it to dry in the open air.

The quantitative estimations of blood cells were made with the THOMAZEISS 'blood counting apparatus.' A Zappert ruled cell was used, and both erythrocytes and leucocytes were estimated from the same preparation. The haemoglobin was estimated with a FLEISCHEL's haemoglobinometer.

The qualitative leucocyte counts were made almost wholly from slides stained by the above-mentioned modification of ROMANOWSKY's method. When it was considered necessary to corroborate the results obtained by these methods, slides fixed by heat and coloured with EHRLICH's tri-acid mixture were also examined.

In order that our tables of differential leucocyte counts may convey a real meaning we have thought it well to define as exactly as possible what are the characteristics which have determined the headings under which we have classified the leucocytes. Cells having one of the three recognized types of granulation, as described

* Plimmer and Bradford, *Quart. jour. mic. science*, Vol. XLV, part iii.

by EHRLICH* present no difficulty. It is in the estimation of the non-granular, mononuclear elements that there is a lack of uniformity. The criteria by which various observers decide exactly to what class a doubtful cell belongs are not always the same.

Although there are two, or perhaps three, well-marked 'types' of mononuclear leucocytes, forms intermediate between them may occur. There is no definite characteristic by which such intermediate forms may be classified; and in differential counts, as often presented, the personal equation is a regrettable factor. It is in an effort to avoid this personal error, and to present our work in a form which will have a real clinical value, that we have grouped our mononuclear elements in the manner here described.

The deeply basophile lymphocyte of about the same size as a red cell, with deeply staining cytoplasm and central, relatively large, nucleus is a constant, easily recognizable type. It must be remembered that, though forms are often seen whose protoplasm is not more basophile than their nuclei, yet these cells possess every other characteristic of small lymphocytes, and as such they must be classified. A study of a sufficient number of films will lead from these small forms, by insensible gradations, through a long series of mononuclear cells to a type of leucocyte possessing all the characteristics of a 'large mononuclear.'

The typical large mononuclear leucocyte, as described by EHRLICH (*loc. cit.*), occurs in normal blood in very small numbers (one per cent.) and is also a constant easily recognizable form. It is a huge cell, two to three times the size of a red cell, and possessing a relatively large amount of usually palely staining cytoplasm and an oval eccentrically placed nucleus. The cytoplasm is finely, the nucleus coarsely, reticulated.†

Between these two extremes there is an intermediate group of cells possessing characteristics which do not conform to those of either division.

Many observers, however, do not strictly hold to EHRLICH's definition of a 'large mononuclear leucocyte,' but classify under this head almost any cell which is slightly larger than a lymphocyte, possessing at the same time the staining reactions, structure, and often bean-shaped nucleus of a 'large mononuclear.' By so doing they avoid a complex classification. It is the difficulty of determining exactly what combination of properties is sufficient to place members of this large unclassified group into either of the two main divisions which has produced a third class 'large lymphocyte.' The nuclei of any of these mononuclear cells may be 'incurved,' 'horseshoe shaped,' or even sub-divided.

Cells of the small lymphocyte type with irregular nuclei (rarely 0.5 per cent. in normal blood) are classified as lymphocytes. Cells of the large mononuclear type

* Ehrlich und Lazarus. *Die Anaemie*, Wien, 1898.

† Ewing, *Clinical Pathology of the blood*, 1901.

possessing 'horseshoe nuclei,' whether possessing sparse neutrophilic granulation or not, are often called 'transitional.' Whatever the name employed, the clinical fact remains that in malaria, the type of disease in which a 'large mononuclear increase' occurs, the cells which become more numerous are those mononuclear large cells possessing the following characteristics:—They have slightly basophilic, finely reticular cytoplasm, often possessing nodal thickenings which simulate granulation. Their nuclei, though usually taking a deeper stain than the protoplasm, are still not 'deeply basophilic,' and are coarsely reticulated and often irregular in shape. The size of such cells varies from one-half to even four times that of an erythrocyte, and the proportion between their cytoplasm and nucleus varies greatly.

Practically speaking, then, in malaria all the mononuclear elements, save the small and deeply basophilic lymphocytes, are increased, and it is solely for the purpose of presenting a simple definite clinical picture of the somewhat analagous changes in our cases of trypanosomiasis that we have adopted the following classification.

- Neutrophiles.
- Eosinophiles.
- Mast cells.
- Mononuclear small cells {
 - Lymphocytes.
 - Lymphocytes with irregular nuclei.
- Mononuclear* large cells {
 - Large lymphocytes.
 - Large mononuclears.
 - Transitionals.

The headings neutrophiles, eosinophiles, mast cells, and lymphocytes need no explanation. The term 'lymphocytes with irregular nuclei' explains itself. We have classed as 'large mononuclears' only those cells which conform to EHRLICH'S description, but have admitted as 'transitionals,' despite his definition, any large cell with a horseshoe shaped or doubled nucleus whether it contained sparse granules or not.

INOCULATIONS

Infected material was, as a rule, diluted with approximately equal parts of the usual sodium citrate solution before inoculation.

Both intraperitoneal and subcutaneous inoculations were practised.

* The simple division of all mononuclears into two general classes, large and small, was suggested by the classification employed by Türk.† We have endeavoured, while taking advantage of its simplicity, to retain that greater precision of description ensured by a more complex classification. It will be at once seen that the division styled 'mononuclear large cells' includes both Ehrlich's 'large mononuclears' and other cells for which that name cannot be employed.

† Türk, *Klinische Untersuchungen ueber das Verhalten des Blutes bei acuten Infektionskrankheiten*, 1898.

II. HUMAN TRYPANOSOMIASIS

We have decided to retain the term Human Trypanosomiasis¹⁵ as a convenient heading under which to discuss all cases in which the human subject is infected with trypanosomes, quite irrespective of the clinical symptoms characterizing the case under consideration. At the same time we recognize that a more extended subsidiary nomenclature may become necessary to indicate special morbid types of infection produced by these flagellata. For example, the type of illness now several times observed in Europeans infected with them has been called 'trypanosome fever.'

DISTRIBUTION AND PREVALENCE

Our first endeavour on reaching the Gambia was, naturally, to find a second case of trypanosomiasis.

A native child was known to have been infected, and we, therefore, determined to examine the blood of as many persons as possible, both black and white. Since parasites very similar to those already seen in man were known to occur in mammals, and since it was not impossible that they might be infected with the human trypanosome, it was necessary to include them in our examinations, all of which were made in the same way.

Our routine method for the detection of parasites in the peripheral circulation was, we are aware, imperfect. The exigencies of travelling, together with the length of time necessary to make preparations, prevented the constant employment of the centrifuge and animal inoculations as diagnostic methods. A coverslip preparation, for immediate examination, and a film for staining subsequently, were taken from each individual examined. Names were also recorded, and, in the event of infection, the case was recalled for further examination.

DISTRIBUTION

The accompanying map of the Gambia, though not minutely exact, will give a very excellent idea of the localities in which we worked, and will, with the following table, indicate very precisely the numbers of natives examined in each district. Those towns in which infected individuals were found are in italics.

TRYPANOSOMIASIS EXPEDITION TO SENEGAMBIA

7

Date	Place	Number examined	Number infected
Sept. 11-26 ...	Bathurst	41	0
Oct. 15-23 ...	Cape St. Mary (patients)* ...	12	0
Nov. 1-Dec. 9 ...	" " (") ...	6	0
Oct. 24	Bakau	34	0
" 25	Wasaloonka	23	0
Nov. 5	Sakuta	16	0
" 7	<i>Lammin</i>	35	3
" 10	Busumballa	46	0
" 11	Brefet	38	0
" 15	<i>Gunjur</i>	32	1
" 15	Birkama	36	0
Dec. 6	Tunjina	14	0
" 7	Farrabubunta	32	0
" 9	Kalfuta	43	0
" 11	Bajana	19	0
" 12	Brefet	38	0
" 14	Somita	27	0
" 17-20	Vintang	84	0
Jan. 10	McCarthy Island	28	0
Mar. 29	" " (Labourers) ...	30	0
Jan. 19-21	<i>Kuntur</i>	90	1
" 24	Kununko	15	0
" 26	Edah	10	0
Feb. 10	Manna	31	0
Mar. 10	Maka (French territory) ...	62	0
" 12	Boulenbou (" ") ...	26	0
" 13	Walia (" ") ...	59	0
" 28	Sharnum	2	0
" 29	Yarbutenda	13	0
" 31	Sunkunda	16	0
April 7	Berif	4	0
" 9	Bantonding	5	0
" 10	<i>Fatotenda</i>	17	1
" 15	Kanube	23	0
" 22	Sallikene	36	0
		1,043	6

* These were natives, complaining of various ailments, who, from time to time, came to us to ask for medicine.

Cases were found both near the sea and some two hundred and fifty miles up the river at practically the end of British territory. There was no evidence to show that natives living in any particular type of locality were especially subject to infection, neither do we find that the disease occurs in well-marked zones or 'belts.' For instance, although three cases of infection were found in thirty-five natives examined at Lammin, the natives of similarly situated villages, both neighbouring and distant, have been searched in a far more thorough manner and have failed to disclose a single parasite. We have found cases in low-lying, river-side villages surrounded by mangrove swamps; we have seen them in towns built on high ground and far from water, and observed them both near the sea coast and as far inland as Fatotenda. How far the disease extends beyond the colony of the Gambia we are unable to say, since only twice have we been able to examine natives in immediately adjacent French territory.*

PREVALENCE

We saw altogether six cases of trypanosomiasis in the native, and once found the parasite in the blood of a quadroon, who can almost be considered a European, since his boyhood and a large part of his life were spent in England.

We examined altogether one thousand and forty-three persons in the Gambia. The percentage of infection given by these figures is, we are convinced, smaller than is actually the case.

Our reasons for holding this opinion are the following:—

(1) Many cases have certainly escaped identification both because of the scanty numbers in which the parasites are ordinarily seen in human beings and because of the periodicity with which even these are absent from the peripheral circulation.^{14, 15, 50}

(2) The majority of the natives examined were apparently healthy children and young adults, who could be persuaded by small bribes to allow their fingers to be pricked. It was in many places extremely difficult to obtain blood from older persons who are possibly more frequently infected. In any case the total number of examinations is too small to permit one to make a dogmatic assertion as to the percentage of infection existing among the Gambian natives.

SYMPTOMS

Since in both East^s and West Africa natives infected with trypanosomes have shown few or no symptoms; and since a few Europeans in whose blood the parasites have been demonstrated have all suffered in roughly the same way, we think that it will be well to describe the cases observed by us in the Gambia according to the race of the individual affected.

* We examined the blood of some two hundred and thirty individuals while at Dakar and St. Louis. Seventy-five were hospital patients who had come from various parts of Senegal and the Soudan; the remainder were children—the great majority of them inhabitants of St. Louis. Twenty-eight of the hospital patients were white, the remainder Africans. The European cases, usually invalided for malaria, were examined with especial care. In the fresh preparations from these cases no trypanosomes were seen. The stained films taken from each individual have not yet been searched.

CLINICAL DESCRIPTIONS OF TWO CASES OF TRYPANOSOMIASIS IN EUROPEANS

Notes by Dr. H. E. Annett

Case 1.—H.K., an Englishman, aged forty-two, for six years in the Government employ as a master of the Government steamer plying up the Gambia River, on the West Coast of Africa.

The illness dated back from May, 1901, when, after a period of heavy duties, he completely broke down and was admitted into hospital at Bathurst. There was a previous history of occasional attacks of malarial fever. It was during his stay in hospital at Bathurst that peculiar worm-like bodies were seen in specimens of blood, the nature of which Dr. FORDE was unable to recognize.*

The illness was at first sight diagnosed as malarial fever, but, proving quite resistant to the administration of quinine, and presenting other peculiar characters, this diagnosis was abandoned. The peculiar symptoms were: ‘irregular patches of a congested or cyanosed character appearing on different parts of the body, the colour slowly returning after pressure. An oedematous condition . . . most marked on the face below the eyes, varying in degree from a scarcely noticeable swelling to well-marked puffiness: a condition also noticed in the lower part of both legs and around the ankles, but only in a slight degree. The respirations always . . . from twenty to thirty, periodical accelerations occurring quite independently of any rise in temperature, and whilst the patient was quietly lying in bed. The pulse was also accelerated, varying from seventy to one hundred and twenty. There seemed to be no relation between the rate and the temperature. . . . The beat was always strong and regular. The appetite was bad all the time, and though a fair amount of liquid nourishment was taken daily, the patient gradually lost weight and colour and became very emaciated. The liver and spleen were practically normal in size and position. The temperature was irregularly intermittent with two or three days of normal or subnormal periods, and unaffected by any drugs.’

After three weeks in hospital, K. was invalided home to England, and arrived in Liverpool in a very weak condition. Until August 12 he remained at home under the care of his own medical attendant, and on that date was admitted into the Liverpool Royal Southern Hospital, under the care of Dr. MACALISTER. He complained of general weakness and lack of energy. Briefly, his chief symptoms and physical signs were:—‘Subnormal temperature; slight headache, but no pain; furred tongue; fair appetite; no vomiting; constipation; pulse (on admission), 120, falling to about 92, regular in time and force, low tension, fair volume; heart sounds weak and distant, otherwise normal; respiration (on admission), 32, falling later to 20; slight dyspnoea on exertion; lungs normal; weakness in legs; some wasting; knee jerk and plantar reflexes present and easily obtainable; sensation

* *Thompson Yates Laboratories Report, 1902, Vol. IV, Part ii.*

normal; urine normal in quantity, sp. gr. 1032, no albumen, no chlorides; liver slightly enlarged, but not tender; spleen normal in size; considerable tenderness over the splenic area.'

The patient remained in hospital for fourteen days, during which period the following further observations were noted:—The temperature curve* shewed three short periods of pyrexia, during which the temperature reached 101° to 102° F. in a few hours, and fell rapidly to normal, with intervals of about three days apyrexia. On one occasion the spleen was felt below the costal margin. The pulse and respiration were always frequent, and varied on exertion and with temperature. Generally, however, their rates were quicker than when in hospital at Bathurst. No malarial parasites were found in specimens of his blood during this time.

The patient remained in England recruiting his health for some months, and returned to Bathurst in December, 1901. On the voyage out he was very ill with what was diagnosed as pneumonia of an atypical character. The sputum was described by the ship's medical officer† as more of the nature of pure blood.

Immediately on his return to the Gambia, K. was placed on the sick list. He had not yet recovered from his attack on board ship, and was very thin and weak, and easily fatigued. On examination by Dr. FORDE and Dr. DUTTON, on December 18, his temperature was 100·4° F.; pulse, 96, respirations, 34.

His chief symptoms were weakness, marked loss of weight, inability to walk far without feeling very tired. Patient did not complain of any definite symptoms. There was no pain nor headache, but a little loss of appetite, and sleeplessness at times. On December 16 he had slight bleeding from the nose. There was no cough, but some dyspnoea on exertion. The general facial aspect, which had been remarked upon by his friends, was very striking. The face was puffy and congested. The eyes were sunken, the conjunctivae had a watery appearance, but were not congested; the most prominent feature was the puffiness of the lower lids, which were distinctly oedematous. On examining the body generally, one noticed that the skin appeared cyanotic, especially on the chest and thighs; pressure made with the hand caused a white mark which took some little time to disappear. There was some puffiness around the ankles, the skin pitting slightly on pressure; the skin was dry; no jaundice.

No cough, no expectoration. Respirations were increased in frequency. This frequency of the rhythm was very noticeable on the slightest exertion or excitement.

During the time of these observations, his respirations were never below 20 per minute, the usual being from 25 to 30; they were never laboured. Chest somewhat barrel-shaped; breath sounds, normal; no dulness; slight emphysema, otherwise the lungs seemed healthy. Pulse frequent, 96; regular in time and force; tension, normal; artery, normal. The pulse was always frequent and hardly ever

* The chart and details of the case are given in a 'Preliminary note upon a case of a Trypanosome occurring in the blood of man.' J. Everett Dutton. *Thompson Yaws Laboratory Report*, 1902, Vol. IV, Part ii.

† *British Medical Journal*, 1903, March 28.

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less than 90, even when the temperature remained low. Heart apex in fourth interspace, four inches from the middle line; impulse distinctly seen, not diffuse; cardiac dulness commenced about at the third rib and did not extend to the right beyond the midsternal line; cardiac sounds, normal, no adventitious sounds. Appetite fair, no diarrhoea; had to take an occasional aperient. No pain on abdominal palpation. Liver dulness, four-and-a-half inches in nipple line, extended just below costal margin. Slight bulging over the splenic area; dulness increased diagonally, measured seven inches; the edge of the spleen could be felt below the costal margin; no tenderness on palpation. In nervous system nothing abnormal could be detected. No definite enlargement of lymphatic glands. Urine normal in quantity, rather high-coloured; Sp. Gr., 1,020 acid; no albumen; no casts; some phosphates.

During the next few weeks, while the patient was still on the sick list, observation of his condition shewed a temperature, pulse, and respiration similar to what had previously been recorded in England. The temperature chart showed periods of slight pyrexia lasting three or four days, with intervals of four or five days in which the temperature remained below normal. During this time K. was never confined to his bed, and was able to take short walks; his appetite was distinctly good during the apyrexial periods, and the pain over the spleen disappeared.

On December 26, he was sent to Cape St. Mary, seven miles from Bathurst, on the sea coast, and here he quickly improved. He still, however, shewed a slight temperature nearly 100° F. at times, some puffiness about the eyes; slight injection of the conjunctivae; some weakness in legs; and slight oedema about the ankles. He was able to resume his ordinary duties, seeming to be much improved.

It was during this period that the true nature of the blood parasite was made out by Dr. DUTTON, and it was noted that during the pyrexial accesses the parasites were more numerous in the blood than during the apyrexial intervals, when occasionally none could be found during an ordinary examination.

'A blood count on December 18 (four hours after food) shewed red corpuscles, 3,850,000; white corpuscles, 12,000 per c.cm.; haemoglobin (GOWER'S apparatus), 76 per cent. A differential count of the white corpuscles made on several occasions, both when parasites were present and absent from the peripheral blood, shewed an increase of lymphocytes at the expense of the polynuclear cells, the relation being generally about fifty per cent. of the latter to forty per cent. of the former.'

On July 25, 1902, K. again arrived in England, invalided home from Bathurst. The chief points of his condition, taken from notes made at the time by Dr. DUTTON, were:—Although the temperature and pulse had not been regularly taken, K. described short periods of fever and of feeling out of sorts. He looked considerably thinner,

and said he had lost weight. He also complained of weakness of the legs. He had had occasional bleeding from the nose, and had noted some swelling about the ankles. The eyelids and nose were swollen and oedematous. Appetite fairly good. Some tendency to constipation. Respiration became troublesome and distressing on slight exertion.

On examination, the most noticeable feature was the presence of the erythematous-looking, raised rash, on the skin of the chest and back, thighs, and arms. There was marked dyspnoea. Pulse, 120, regular, poor tension. Respirations, 30. Heart slightly dilated. Urine, 1,016, acid; no sugar; slight trace of albumen; of normal quantity. Patient's weight, 158 pounds. Examination of blood: Sp. Gr., 1,057; red corpuscles, 4,400,000; white, 6,800. Haemoglobin, 90 per cent. Temperature varied only between 97° and 100° F. From one to three parasites could be detected in every cover-glass preparation of blood taken from the finger. It was noticed that tissue juice obtained from the swollen oedematous lower eye-lid, or from the raised erythematous patches contained a comparatively larger number of trypanosomes than the peripheral blood.

Up to December 29, the case continued to follow the chronic course, which apparently characterizes the disease in Europeans. The increased frequency of pulse and respiration, the peculiar congested areas of the skin, the local and fugitive oedemata, the enlargement of the spleen, and the irregular rises of temperature continued until the end. But under treatment, which, after the apparently useless administration of quinine and arsenic in large doses, consisted of the administration of urotropin in doses increasing from five to ten grains three times a day, the patient increased in weight (from 158 pounds to 168 pounds), and improved considerably in muscular strength. Slight rises of temperature, reaching sometimes 102° F., at intervals of from one to ten weeks, lasting only a few hours, and occasionally some increased difficulty in breathing produced some distress.

The patient was usually in fairly good spirits, although having some idea of the nature of his condition; but on rare occasions he manifested very considerable depression and indifference to living. There was always a tendency to constipation, corrected by regular use of aperient waters; and short, smart attacks of severe pain over the spleen at times caused him to remain indoors. Otherwise during this time he lived much in the open air, and practised the most regular and healthy habits.

During this time parasites were very scanty in the peripheral blood, and at some examinations appeared to be entirely absent, at others two or three only could be discovered after a long examination of several blood films. But the inoculation of animals (white rats and monkeys) demonstrated their presence.

On the morning of December 29, acute symptoms developed, ushered in by an indefinite rigor, the temperature being stated to be 99.6° F. In the

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evening the temperature was 104°, the pulse, 126, and respiration, 28. On the morning of the next day, the temperature, which had continued high during the day, was 105° F.; pulse, 130; respiration, 30. There was some delirium, and the patient recognized his friends with difficulty. The respiration became of CHEYNE-STOKES character, and there was some difficulty in utterance; the pupils appeared normal. An examination of the patient revealed nothing to account for the acuteness of the symptoms, which indicated some cerebral disturbance. The patient seemed to be *in extremis*, but rallied considerably during the next day, the temperature gradually falling to 102.5° F., while the pulse and respirations remained about the same; there was no delirium. On the morning of January 1, the temperature fell further to 101° F., but the respirations became still more frequent, reaching 60 to 70, until death occurred, the patient remaining conscious almost to the end. An autopsy was unobtainable.

Dec 28 - Jan 1 = 4

Case 2.—The second 'European' case observed by us was Mr. Q.

He was a quadroon and was educated in England, though born in Gambia, where he has been resident for the past twenty-four years as a trader. He spent a good deal of his time on or near the river and in swamps while occupied in getting out either logs of mahogany or loads of piassava fibre. For the two years before his death he had lived on the river's bank at his Sakuta factory, situated roughly one hundred and thirty-five miles from the sea. His house was built upon a bit of fairly dry ground about three feet above high tide. It was quite cut off from the main land by a huge swamp, which was impassable except in the middle of the dry season. Mosquitoes of all sorts bred and multiplied in this swamp, and they, with Tabani and innumerable Glossinae, were a continual pest.

During his rough life he very frequently lived for long periods in native villages, almost as a native. From these circumstances it can be gathered that he was very constantly exposed to, and very frequently bitten by all sorts of blood-sucking insects. In spite of this insanitary life Mr. Q. had, of late years at least, been very well, and had never suffered from 'fever,' although when he first came to the Colony he had had a good deal of 'malaria.' His habits were fairly regular. He smoked but little, and only occasionally indulged in too much alcohol. He was rather a heavily, sturdily-built man, aged fifty-six years. His height was five feet six inches, and his weight, in health, one hundred and ninety-four pounds. He was active in habits and robust in constitution, and, except during his periodic attacks of fever, felt quite well.

When we first saw him in October, 1902, he complained of not being well, and said that:—

- (1) He had lost flesh, weighing only one hundred and fifty-six pounds, instead of one hundred and ninety-four pounds as formerly. Weight

was lost gradually. Six months previously he had weighed one hundred and seventy-four, and two months before one hundred and sixty pounds.

- (2) He complained of constant accesses of intermittent slight fever (going up to 101° F.), which was not checked by quinine. He habitually used a clinical thermometer and took ten grains of quinine every two days; so this history of fever may be assumed to be correct.
- (3) He became easily fatigued. He lost breath easily. His legs seemed sometimes to be weak, and he occasionally had palpitation.
- (4) He had, from time to time, pain in his loins, particularly after exertion, and, twelve months previously, in the splenic region.

He attributed the commencement of his illness to the bite of some insect which he received on his thigh, a year before (October, 1901), while at closet in Bathurst. This bite became livid and tender, and swelled up in a very short space of time to the size of a walnut. It was followed six hours later by an attack of 'ague,' which developed into 'fever' and kept him in bed for some days. Since this illness he has never been himself.

Clinical Examination.—In October, 1902, he had all the general appearances of a healthy man—his figure was rotund, his face was not emaciated, and he certainly did not look like a man who had lost forty-two pounds during the preceding year. His friends, however, told us that he had lost a great deal of flesh and was much less stout than he formerly was. The exposed parts of his body were darkened from exposure, although his trunk, which was much marked by recent insect bites, was quite white. His legs were deeply scarred and pigmented by former 'bush boils and ulcers.' The blotchy erythemata seen^{15, 50, 49} in the first European cases were not present, but vasomotor reaction was easily excited and his skin was markedly dermatographic. Although persistent reddening of the skin was noticed in places pressed upon by clothing, as, for example, around the waist and on the chest, we never observed any of that doughiness of the skin or the local transitory maculae which have been such marked characteristics of the other European cases.

Slight pitting on pressure was observed on the shins and ankles. Mr. Q. was very certain that there had never been any puffiness of his eyelids.

No organic lesion of heart or lungs was detected, although the rapidity of both pulse and respiration during rest was increased. There was no history of haemorrhages. His appetite was good and his bowels regular. The liver extended 2 cm. below the costal margin but was not tender. The spleen extended 3.5 cm. below the costal margin and was rather tender on firm pressure.

It is most unfortunate that we were never able to obtain a specimen of urine for examination from this case. Sexual impotence which had been a feature in the first European case was absent here.

October 26 : Pulse, 99 ; respiration, 18 ; temperature, 99·4° F. (Taken while sitting). Four to seven trypanosomes were present in each coverslip preparation at this date.

Mr. Q. was again seen on January 19 at his Sakuta factory. He then felt much better. Shortness of breath and palpitation had left him, and he had gained flesh, his weight at this time being one hundred and seventy-five pounds. X

He had taken none of the solutions of arsenic which Dr. CHICHESTER had prescribed for him in October, 1902, and ascribed his improved condition to quinine and patent medicines which he had used rather freely. - One of the latter consisted of methylene blue tablets.

Objectively his condition was the same as it was three months previously. X

We again saw no macular erythemata nor marked oedema. There was still slight pitting over shins and ankles, slight temperature and frequency of pulse and respiration. Pulse, 104 ; respiration, 18 ; temperature, 98·2° F.

About five c.cm. of blood was centrifuged, in it nineteen trypanosomes were found. X

A blood count showed—

Red cells	-	-	-	-	4,350,000
White cells	-	-	-	-	8,000
Haemoglobin	-	-	-	-	96 per cent.

Differential counts of leucocytes made on preparations taken on two different dates were as follows :—

Blood count from five slides November 9, 1902, 1,100 cells counted. Stained with ROMANOWSKY, effect not good, as no granules are seen in mononuclears.

Neutrophiles	-	-	62·76 per cent.
Eosinophiles	-	-	6·45 per cent.
Mast cells	-	-	0·72 per cent.
Mononuclear, small cells,	19·54 per cent.—	Lymphocytes	- 19·36 per cent.
		„ with irregular nuclei,	0·18 per cent.
„ large cells	10·53 per cent.—	„ large,	6·81 per cent.
	—————	Large mononuclear,	0·18 per cent.
	100·00	Transitionals	- 3·54 per cent.

Blood count January 19, 1903, eight hundred cells from five slides were counted, ROMANOWSKY effect good in two of these.

Neutrophiles	-	-	53.26 per cent.
Eosinophiles	-	-	5.25 per cent.
Mast cells	-	-	1.25 per cent.
Mononuclear, small cells,	26.62 per cent.—	Lymphocytes	- 26.25 per cent.
		„ with irregular nuclei,	0.37 per cent.
„ large cells,	13.62 per cent.—	Large lymphocytes,	9.00 per cent.
		„ mononuclears,	0.25 per cent.
	100.00	Transitionals	- 4.37 per cent.

About four per cent. of lymphocytes contained granules.

„ eight „ of large lymphocytes contained granules.

On March 20 we received a message saying that Mr. Q. was very ill and asking us to go to him.

Unfortunately we reached Sakuta too late to see him alive. His native wife refused to allow an autopsy, and we were able to gather but a very meagre history of his last illness from those neighbouring traders who had seen him and from his native servant. We were told that his last illness had commenced with a 'cold and fever' on the 16th of March. Two days later he went to bed complaining of headache and pain over the spleen. On the 20th he was 'unconscious, and unable to speak or swallow, his throat seemed to pain him,' and he died during the early morning of the 22nd, three hours before we reached him. No more accurate information than this could be obtained from the more or less conflicting reports proffered us.

We were permitted to examine the body. P.-M. rigidity and hypostatic congestion of dependent parts were marked. There was the usual slight oedema of shins and ankles only. No fluid was found in the peritoneal or pleural cavities by aspiration. No parasites were found in blood taken with a syringe from the spleen and heart. A rat inoculated with heart's blood taken at this time has never become infected.

The original disease in this case commenced in October, 1901, so that its duration was almost the same as that of Mr. K.'s illness, viz., one-and-a-half years.

The regrettable incompleteness of our notes on this case is largely due to the patient's unfortunate objection to a systematic examination.

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X

CASES OF TRYPANOSOMIASIS OBSERVED AMONGST THE NATIVES OF GAMBIA

Case 1.—Woman, age thirty-five years, living at Lammin Village, on the Lammin Creek, Kommba.

History.—The woman was married and had one child seven years old. She was a native of the town.

She complained of feeling weak and ill, and asked for medicine. She said her illness commenced a month ago with a cough and fever, the fever at times, for a day or two, confining her to her hut. She related that during the rains she had been ill with fever and headache and that she had got much thinner.

Note.—History unreliable.

Clinical Examination.—Patient was a spare Mandingo woman, showing no signs of wasting and no skin lesions. There was a slight oedema around ankles, the skin ‘pitting.’ One lymphatic gland in the left axilla, though freely movable, was distinctly enlarged; all the other superficial glands were palpable.

The liver, spleen, heart, lungs, kidneys, and intestines were all normal.

Pulse, 93; respiration, 18; temperature not recorded.

Blood.—Coverslip preparations were examined on two successive days. The first observation showed one, the second five parasites to the coverslip.*

Differential Count of Leucocytes

Neutrophiles	-	-	24.5	per cent.
Eosinophiles	-	-	6.75	per cent.
Mast cells	-	-	0	per cent.
Mononuclear, small cells,	42.00	per cent.—	Lymphocytes	- 39.5 per cent.
			„ with irregular nuclei,	2.5 per cent.
Mononuclear, large cells,	26.75	per cent.—	Large lymphocytes,	19.25 per cent.
			Large mononuclear,	1.5 per cent.
			Transitionals	- 6.0 per cent.
		100.00	per cent.	

Four hundred cells from one slide were counted.

Case 2.—Boy, age nine years. Living at Lammin village.

History.—He had never been far away from the village. Though he was said by his parents to be weakly he had had no serious illness, and worked with other boys of his age on a ground-nut farm in the neighbourhood.

No definite history could be obtained.

Clinical Examination.—Patient was a rather poorly-developed lad. Nothing abnormal was detected, except the enlargement of the spleen usually seen in natives of this age. This boy could run and wrestle as well as other boys without any marked increase in the frequency of pulse or respiration.

* As previously stated three-quarter inch square coverslips were used throughout our work.

Blood.—Examined on one occasion showed one trypanosome to coverslip preparation.

Differential Count of Leucocytes

Neutrophiles	-	-	23.5	per cent.
Eosinophiles	-	-	5.75	per cent.
Mast cells	-	-	0.25	per cent.
Mononuclear, small cells,	57.50	per cent.	Lymphocytes	- 56.75 per cent.
			„	with irregular, abnormal nuclei, 0.75 per cent.
Mononuclear, large cells,	13.00	per cent.	Large lymphocytes	- 10 per cent.
			Large mononuclears,	0.25 per cent.
			Transitionals	- 2.75 per cent.
				100.00 per cent.

Four hundred cells from two slides were counted.

Case 3.—Girl, age fifteen years, living at Lammin.

History.—The girl was a water carrier and had never been far away from the village. She said she had never been ill in her life. She felt perfectly well and had had no fever.

Clinical Examination.—Revealed nothing abnormal, beyond a slight enlargement of the spleen. Patient was a very well-developed girl.

Pulse and respiration were normal, and there was no fever.

Blood.—Examined on one occasion showed twenty-three trypanosomes to coverslip.

Differential Count of Leucocytes

Neutrophiles	-	-	34	per cent.
Eosinophiles	-	-	10	per cent.
Mast cells	-	-	1.75	per cent.
Mononuclear, small cells,	44.25	per cent.	Lymphocytes	- 43.5 per cent.
			„	with irregular nuclei, 0.75 per cent.
Mononuclear, large cells,	10.00	per cent.	Large lymphocytes,	4.25 per cent.
			Large mononuclear,	1.0 per cent.
			Transitionals	- 4.75 per cent.
				100.00

Four hundred cells were counted from two slides.

Case 4.—Boy, age twelve years, living at Gunjur, a large town not far from the coast, in the Kombo district.

History.—This boy came to Gunjur last year (1901), before the rains, from near Rufisque, in Senegal. A very indefinite history of fever was given by his guardians, who mentioned that the boy suffered from enlarged glands in the neck, and would

soon have them cut out as a prevention against sleeping sickness.* The boy maintained that he was perfectly well.

Clinical Examination showed a sturdily built lad. All his organs were normal except the spleen, which could be felt 6.5 cm. below costal margin. There was no tenderness on palpation.

The axillary, inguinal, sternomastoid, and submaxillary lymphatic glands were all very much enlarged, not tender, hard, and freely movable. One in the left axilla was the size of a pigeon's egg. XXX

Pulse varied in frequency, was usually about 112; respiration, 26; temperature 101° F.

Blood.—Was examined on two consecutive days. The first observation showed one parasite to coverslip; at the second six were seen.

Differential count of Leucocytes

Neutrophiles	-	-	29.50 per cent.	
Eosinophiles	-	-	8.87 per cent.	
Mast cells	-	-	0.25 per cent.	
Mononuclear small cells,			46.26 per cent.—Lymphocytes	- 41.64 per cent.
			,, with irregular	
			nuclei,	4.62 per cent.
Mononuclear large cells,			15.12 per cent.—Large lymphocytes,	9.12 per cent.
			Large mononuclears,	0.50 per cent.
			Transitionals	- 5.5 per cent.
		—————		
		100.00		

Eight hundred cells were counted from two slides.

Case 5.—A Jollof Mohammedan man, age thirty-five years, living at Kuntur, a riverside village in McCarthy district.

History.—He was born at Bantanding, near Barra Point, but as a child lived in Bathurst.

He was at the age of fourteen years apprenticed as a sailor to one of the cutters plying on the Gambia River and worked up to be a captain.

For the past five years he had lived at Kuntur all the year round, working at one of the ground-nut 'factories.'

He had never had any serious illness, but at times had had what he called 'strong fever,' generally in the morning, lasting for three or four hours. Last rainy season he was laid up with headache and 'fever' for three days; he said he had never had headache before, and at the time noticed his 'eyes' were swollen. The swelling of the left eye persisted for two weeks.

* The Gambian natives recognize sleeping sickness, and assert that they can foretell its onset. They believe that an invariable prodrome is an enlargement of the lymphatic glands, particularly of the anterior cervical and sub-maxillary groups. The excision of enlarged glands, to prevent sleeping sickness, is one of their few surgical operations.

N.B.

He had had slight attacks of 'fever' during the past year, but they had not prevented him from working. Though he was a powerful man he said that he had lost strength during the past year and that he was not able to lift as heavy weights as usual. When at work and even when not working he sometimes 'breathed hard.' For about a month he had had pain in his right side and back, which he believed was due to a strain caused by lifting a heavy weight.

History reliable; an intelligent man.

Clinical Examination.—Patient was a very well-developed man. Muscles were good, appetite good, bowels regular. Respiratory system normal. Heart, slight accentuation of first sound at apex. Liver normal. Kidneys normal, urine could not be obtained for examination. Spleen extended 1.25 cm. below costal margin, not tender. All lymphatic glands were slightly enlarged, especially the inguinal groups, but freely movable. Some slight 'pitting' over tibiae and dorsa of feet, especially the left. No varicose veins. There were some slight scars on the shins and an old injury to the left little toe (crushed). Some slight puffiness of the lower eyelids, especially the left, was noticed.

Pulse, 82; respiration, 20; temperature, 99.8° F.

Blood.—One examination showed eleven trypanosomes to cover—

Red cells	-	-	-	-	4,600,000
White cells	-	-	-	-	7,500
Haemoglobin	-	-	-	-	77 per cent.

Differential Count of Leucocytes

Neutrophiles	-	-	43.75 per cent.
Eosinophiles	-	-	5.42 per cent.
Mast cells	-	-	0.71 per cent.
Mononuclear, small cells,	29.42 per cent.—	Lymphocytes	- 29.14 per cent.
		„ with irregular nuclei,	0.28 per cent.
Mononuclear, large cells,	20.7 per cent.—	Large lymphocytes,	13.42 per cent.
		Large mononuclears,	2.0 per cent.
	100.00	Transitionals	- 5.28 per cent.

Seven hundred cells were counted from five slides.

Case 6.—Mandingo youth, age twenty-two years, living at Fatotenda on the upper river.

History.—Was born at Medina, five days' march to the south from Fatotenda. He was a student, and although he had travelled a good deal in French territory to the south of the Gambia he had never, until he came to Fatotenda in January, 1903, either lived on the river or done any sort of manual labour.

He said he had never had any illness and that he was quite well and strong. He complained only of a small chronic ulcer on the left foot. He stated that for the last nine months he had occasionally had a slight nose bleeding, which sometimes lasted subcontinuously for two days.

Clinical Examination.—The patient was a well-developed lad. Muscles were good. Nothing abnormal was detected, with the exception of a slight enlargement of the spleen, which extended 1.5 cm. below the costal margin. All the glands were palpable, but none were markedly enlarged. There was a slight injection of the nasal mucous membrane, and the tonsils were slightly enlarged.

Urine, slightly acid, 1,007; colour, normal; no haemoglobin. Albumin and a small amount of pus were present.

April 10. Temperature, 100.1° F. - Pulse, 84 - Respiration, 20.
 „ 15. „ 99.4° F. - „ 104 - Respiration, 22.

Blood.—Examined on two occasions.

April 10. Two trypanosomes to cover were seen.
 „ 15. In three coverslip preparations, one trypanosome and two specimens of *F. perstans* were seen.

Red cells	-	-	-	-	3,950,000
White cells	-	-	-	-	8,000
Haemoglobin	-	-	-	-	68 per cent.

Differential Count of Leucocytes

Neutrophiles	-	-	40.6 per cent.
Eosinophiles	-	-	13.5 per cent.
Mast cells	-	-	0.3 per cent.
Mononuclear, small cells,	32.8 per cent.—	Lymphocytes	- 32.6 per cent.
		„ with irregular	
		nuclei,	0.2 per cent.
Mononuclear, large cells,	12.8 per cent.—	Large lymphocytes,	11.2 per cent.
		Large mononuclears,	1.1 per cent.
	100.0	Transitionals	- 0.5 per cent.

Two thousand five hundred cells from six slides were counted. Both EHRlich's tri-acid and ROMANOWSKY's stains were used.

DISCUSSION OF NATIVE CASES

Taken as a whole a consideration of the native cases reveals no marked constant characteristic.

Enlargement of the spleen was observed in the younger cases. But this is without significance since that organ is enlarged in nearly all Gambian native children.

Enlargement of lymphatic glands is also by no means unusual in native children.

The history of possible loss of weight and strength with fever was given in four cases; definitely in only two.

The remaining cases believed themselves to be perfectly healthy. Two boasted of their extraordinary vigour, and neither of the adult males were impotent. In appraising the value of the past history of these cases, we must remember that a native does not reckon time or observe himself so accurately as does a European. In addition, he will very often say what he imagines will please his questioner. Even if his story of past fevers and loss of weight, during the rainy season, is correct, it must not be forgotten that there are many other diseases which produce like symptoms.

We must, therefore, rely altogether on our objective examination, and this, unfortunately, was necessarily curtailed because travelling made it impossible to keep each case under observation for any length of time.

Taking all our facts into consideration, we believe the disease, as it occurs in natives, to be a peculiarly mild one, and that it is at present impossible to recognize it clinically.

The accelerated pulse and respiration rate, slight rise of temperature and past history of fever occurred not unfrequently amongst many of those in whom no trypanosomes were found.*

The abnormal results of the blood counts must not be assumed to be specific. An enormous percentage of these natives were infected, in varying degree, with filariae, and all may be assumed to have had intestinal parasites. The quantitative estimation of blood cells and haemoglobin shows a constant diminution of these elements as judged by the European standard. What the normal for an African may be we have no means of determining.

The differential leucocyte counts show a constant increase of eosinophiles, mast cells, and mononuclear elements. The proportional increase being greatest in the cases of the eosinophiles and mononuclear large cells.

In slides coloured by the previously mentioned modification of ROMANOWSKY'S stain many of the mononuclear cells were seen to contain deep, rich lake-coloured granules. The number of granules present in each cell ranged from, perhaps, twenty to eight or ten or even less. Their size and shape varied as much as their number. They varied from regular oblongs, as long as eosinophile granules, to mere points of colour. A small unstained area was often seen to partially surround these granules.

* A series of pulse rates taken at random from apparently healthy children, in whom no trypanosomes were found, showed that an increased pulse rate, ninety to one hundred, was not at all uncommon.

The granules occurred on an average in thirty to fifty per cent. of the large mononuclear.

” ” ” ” two to seven per cent. of the small lymphocytes.

” ” ” ” eight to twelve per cent. of the large lymphocytes.

In specimens stained by EHRLICH's or ROMANOWSKY's methods and fixed by any of the three previously mentioned methods, circular, refractile, non-staining, and sharply circumscribed areas were not infrequently seen in both the cytoplasm and nucleus of the leucocytes.

Their most usual situation was, however, in the cytoplasm close to the nucleus. They varied in diameter greatly, being usually about 0.5 to 1.0 μ in width. They occurred in about two per cent. of all leucocytes, but were, however, more numerous in the mononuclear elements.

The slight virulence of this trypanosome and the consequent lack of symptoms has frequently suggested to us the possibility that the natives in this disease may bear the same relation to the European as does the wild game of Central Africa to domestic animals in the tsetse fly disease.

We have had no opportunity of investigating the course of the disease—if we can call it a disease—in the native, but the ages of those infected indicate that there can be no absolute acquired immunity. It would be rather curious if the woman at Lammin or the man at Kuntur had only recently become infected for the first time after having been exposed to infection from childhood. We think that the varying ages of the various cases is another point in favour of our belief that the parasite frequently occurs in the natives, but appears in a periodic manner in their peripheral circulation.

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III. EQUINE TRYPANOSOMIASIS

The first trypanosome which we found while in the Gambia was in the blood of a horse, and this is the only animal in which we have seen a certainly *pathogenic* trypanosome.

Out of thirty-six horses examined, ten were found to be infected. The parasites derived from three of these animals have been, and are still being, studied. They, as far as we can see, are all of the same variety, and do not correspond in every respect with any of the described trypanosomata, although in a general way they resemble those species already described as pathogenic to horses.

Pending a further study of their properties, we shall describe them as one species, under the style of 'The Gambian Horse Trypanosome.'

DISTRIBUTION AND PREVALENCE

One of our first cares in the Gambia was to obtain data showing the amount of live stock of all sorts in the country, and to learn in exactly what localities the various animals throve or not.

To aid us to this end, Sir GEORGE DENTON, K.C.M.G., the governor of the Gambia, very kindly sent out a circular letter, containing questions on the subject, to all the District Commissioners.

From the information thus acquired, together with that obtained from traders and natives and supplemented by our own observations, we have drawn the following conclusions:—

1. Equine trypanosomiasis in the Gambia is a very chronic disease.
2. It occurs throughout the colony, and is possibly more prevalent near the river, especially during the rainy season.
3. Cattle and other domestic animals are not known to suffer from the disease.

The total number of horses in the Gambia is not large, probably not more than 1,000 altogether. In some districts there are very few, in others there are absolutely no horses nor, indeed, cattle. The natives explain this by their poverty, saying that they cannot afford to buy horses and cows. Others, again, are fishermen or pagan agriculturists and carriers, *e.g.*, the Jolahs; and do not keep live stock other than goats, which thrive everywhere. There are more horses on the northern bank of the Gambia, particularly of the upper river than on the southern.

The latter is said to be more unhealthy for horses than the district to the north of the river. In Fogni and Kombo there are very few horses.

We believe that the scarcity of horses in these two latter districts is due to trypanosomiasis of such a chronic course that the natives do not recognize that their horses are ill. They say that horses usually die by becoming gradually thin and weak, though eating heartily, until they die through sheer inanition. This is precisely the course of horse trypanosomiasis as we have seen it.

If the disease were more acute it would be recognized, for the natives are not altogether unobservant. They recognize various types of disease in their animals, and consequently practice rude veterinary medicine.

They describe two horse sicknesses (we have seen neither), characterized the one by 'swollen stomachs and legs and, rarely, by loss of hair from the neck,' and the other by 'pain in the head, running from the eyes and nostrils, and lack of appetite.' The latter is called 'Jukundo,' and about fifteen to twenty years ago it caused an epidemic, during which a very large number of horses died.

Herdsmen recognize diseases among their herds; in one instance a Fulah cattle owner asserted that it was *Jolofin Jolo* (*Glossina palpalis*) which killed his cattle. The native is far from blind to the well-being of his stock, and the chronicity of the horse disease will be still better appreciated when it is remembered that he fails to recognize it.

A large number of horses, perhaps three hundred or more, are yearly imported into the Gambia from towns in French territory. They come from all parts of Senegal, principally from Galum, Fotatora, and Niani. We have been told that many of them die within a year after reaching English territory, particularly if they have not been sent away from the river's bank for the rains, as is the usual native custom. During the wet season all horses, donkeys, and cattle are sent to higher ground several miles distant from the river. Whether this is done altogether with the idea of removing the animals from an unhealthy neighbourhood or partially to obtain better grazing we cannot say. Certain it is that during the rains many natives absolutely refuse to bring their live stock to the river. The tall red Soudan sheep will not live on the river, and invariably die within three or four months. Sheep brought to the river are, for this reason, always slaughtered at once. While in the Gambia we examined the blood of many domestic animals, goats, cattle, dogs, donkeys, and sheep for trypanosomes, but always with negative results.

The number of horses which were examined was very small. It was often much easier to persuade a native to give a drop of his own blood than to allow his animals to be pricked. In addition, there are never more than two or three horses in a village. We did not travel a great deal, and so saw comparatively few horses.

The following table shows the number of horses examined and the number found to be infected in each locality :—

TABLE SHOWING FREQUENCY WITH WHICH NATIVE HORSES ARE INFECTED WITH HORSE TRYPANOSOME IN THE COLONY OF THE GAMBIA

Origin of Horses	Number Examined	Number Infected	Cases
Owned by natives, Bakau and Cape St. Mary District -	6	5	i, ii, iii, iv, v
Officers of West African Frontier Force, Bathurst -	4	2	vi, ix
Governor Denton, Bathurst - - - - -	3	1	x
At Maka, French Territory, French Commandant, Monsieur H. Porthes - - - - -	8	2	vii, viii
Sallikene - - - - -	1	0	
Sakuta, Kommo - - - - -	2	0	
Lammin - - - - -	2	0	
Birkama, Kommo - - - - -	4	0	
Vintang - - - - -	3	0	
Bathurst Trading Co., Bathurst - - - - -	2	0	
McCarthy Island - - - - -	1	0	
	36	10	

The total number examined is again too small to permit of a final conclusion as to the prevalence of horse trypanosomiasis in the Gambia. That a large percentage of horses are affected there is, we believe, no doubt. We were frequently told by Europeans of horses which died (while in the Gambia) with symptoms of disease.*

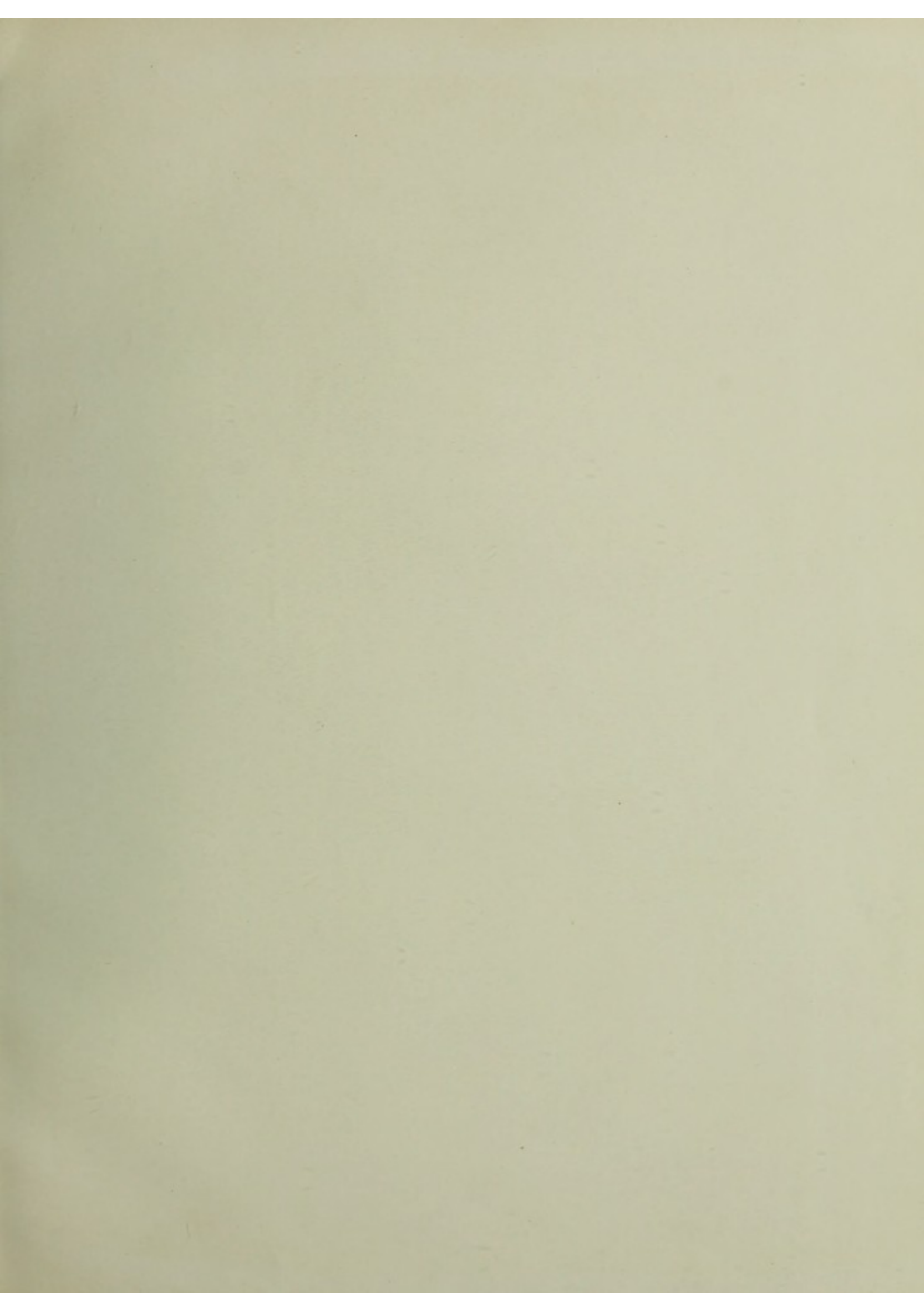
A glance at the names of the towns in which infected animals were found shews that, like human trypanosomiasis, the disease extends from the sea coast to the end of British territory. Judging from the scarcity of horses in the Kommo district, and the larger percentage infected, one would imagine that it is there that the disease is most common. Nevertheless it seems improbable that it obtains in Bathurst itself.†

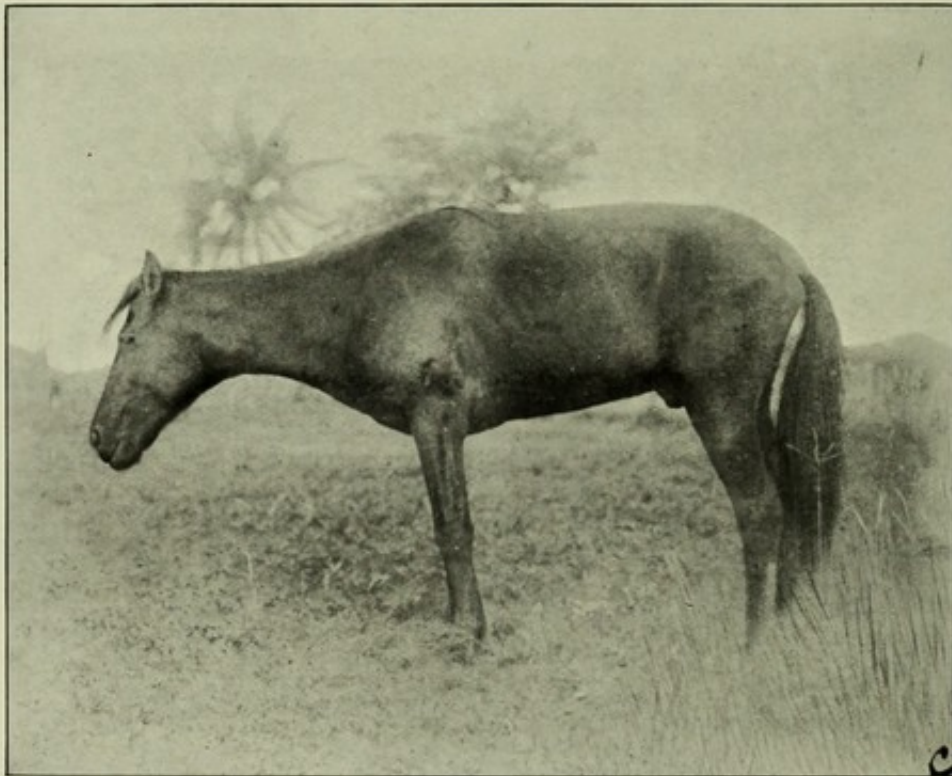
* Mr. A. K. Withers, a Travelling Commissioner, described to us a disease of which three horses out of twenty, owned at Quinela, had died during the month of December, 1902. His description very strongly suggests that these animals were suffering from trypanosomiasis.

† One of the two horses examined at the Bathurst Trading Company, coming originally from Barra Point, had lived for over twenty years at Bathurst, and had always been, and still is, in excellent condition. During all this time it had never been further away from Bathurst than Cape St. Mary. Bathurst is believed locally to be not unhealthy for horses.

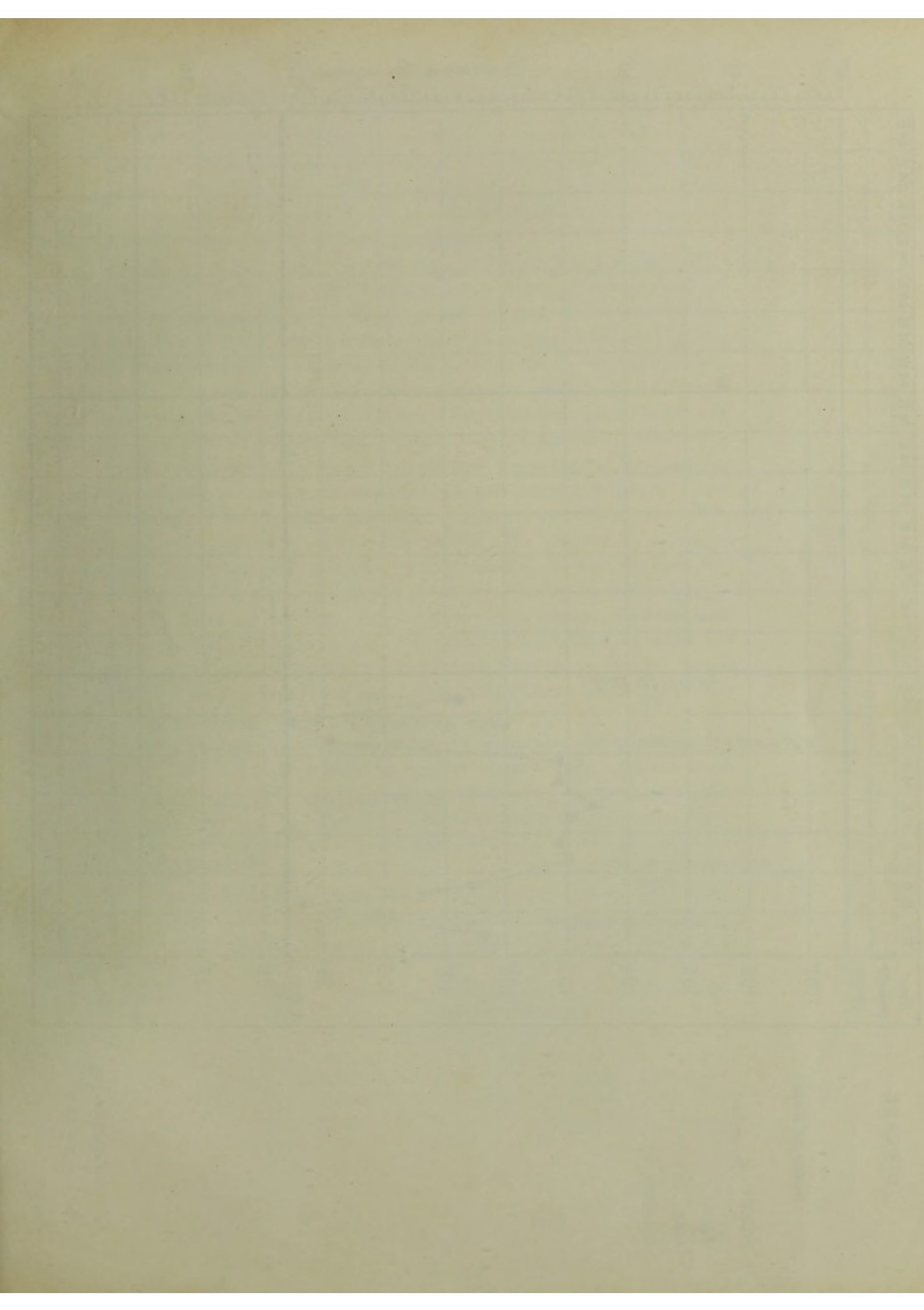
We were also told while in Senegal, both by Government officials and traders, that in the Soudan, at Koli-chor and in the neighbourhood of Kaolakh, horses died from a disease producing symptoms closely resembling those observed in cases of equine trypanosomiasis in the Gambia.

While at St. Louis and Dakar we examined, with negative results, the blood of a few horses, mules, cattle, and camels which were not in the best of condition, and had been in districts where this disease was said to occur.





Horse, Case I, *October 13, 1902*



DISEASE.

Notes of Case.

Name { Horse No. 1.

Age

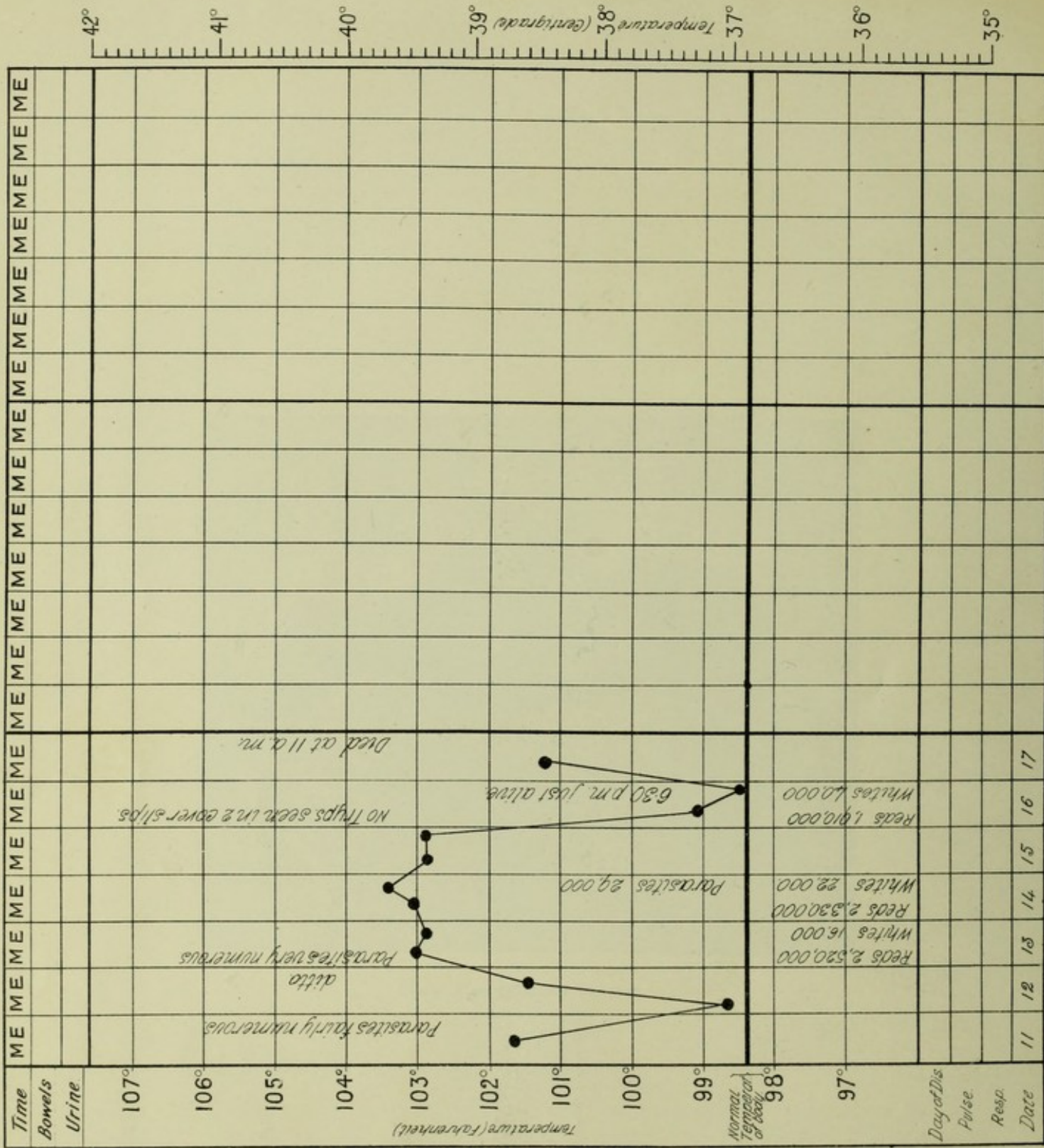
Diet

Case Book No.

Date of admission.

October 11th 1902.

Result



CASES OF EQUINE TRYPANOSOMIASIS OBSERVED IN THE GAMBIA.

Case I.—Stallion, age about seven years, in charge of a native at Cape St. Mary.

History.—The origin of the horse was unknown. It had been in British Kombo for about six months, and, according to the native who was in charge, it had been in the state in which we found it for only twenty days.

Clinical Examination.—When we first saw this horse it was *in extremis*, and it died six days later. We found a small dark-grey native stallion greatly emaciated and exhausted. He lay upon his side in the sun entirely apathetic to his surroundings. His skin was drenched with sweat and his breathing laboured and extremely frequent. Weakness was extreme, and it was not until after several attempts that the horse was able to stand. The loins and hind legs seemed weakest, it was their lack of strength which made the animal unable to rise. When at last the animal gained his feet he stood with legs apart and drooping head, a picture of utter weakness and inanition.

He was far too weak to protect himself from the swarms of flies hovering about him, and, as a consequence, his sheath and hide were dotted with 'bot fly' larvae holes. If the animal were forced to move, the hindquarters once more seemed to be weakest, they were not paralyzed but only paretic. We observed no oedemata, haemorrhages, or copious discharges from eyes or nostrils. Though the horse was ungroomed there was no 'staring' of the coat or loss of hair. The scrotum and sheath were relaxed. Mucous membranes and conjunctivae were anaemic, the latter were yellowish and showed no haemorrhages. The appetite was good and the animal fed and drank freely. It became progressively weaker, its temperature dropped, and it died on October 17 with markedly spasmodic movements almost amounting to convulsions. The temperature varied; it was usually about 103° F.

Blood.—The blood counts made showed a progressive diminution in the number of red cells, with a corresponding increase in white cells. A count made twenty-four hours before death gave only 1,910,000 red cells and 40,000 white cells. The number of parasites in this horse's blood three days before death was large, 2,900 per cubic mm., a day before death their numbers decreased to two parasites to a coverslip preparation.

The accompanying chart shows the temperature curve of this case during the short time it was under observation, and demonstrates in a striking manner an attack of fever associated with an increase in the number of parasites present in the peripheral circulation.

Autopsy.—Performed immediately. The coat was not staring. The mucous membranes were anaemic and rather yellow. On section, a yellowish gelatinous oedema of the subcutaneous connective tissue over the abdomen, particularly near the genitals and forehead, was noticed. It was not present on the legs or thorax. It must be remembered that this oedema was sharply localized to the areas mentioned. It was not at all general and was not distinguishable ante-mortem. The muscles were rather firm, dark in colour, and not oedematous or cloudy. On opening the abdomen, the fat throughout was of a marked canary-yellow colour. The intestines were seen to be full of faeces. There was no peritonitis nor

peritoneal fluid. The unwounded intestines were thrown free of the body, and among their free coils, as well as in the peritoneal cavity, several free adult male and female filariae were found. (These were preserved for examination).

Thorax.—There was no pleurisy nor increase in pleural fluid. About two hundred c.c. of clear amber-coloured, quickly coagulating fluid was found in the pericardium. There were no signs of pericarditis.

Heart.—Weight, 1,360 gm. The connective tissue round the great vessels at the base of the heart was much infiltrated with a yellowish, quickly coagulating fluid, similar to that seen in the subcutaneous tissues: right side collapsed; left, full of blood. Valves, normal. Muscle, firm and rather dark; no ante-mortem clots. The organ contained no filariae.

The aorta was normal and contained no filariae.

An aneurism about the size of a walnut was situated on the superior mesenteric artery, 3.75 cm. from its origin. On being opened it was found to contain numerous sclerostomata. The walls of the aneurism were in many places partially broken down. Their thickness was about 1.9 cm.

Right Lung.—Weight, 810 gm., was dark throughout and contained many small pneumonic areas; it was everywhere congested. There was slight bronchitis. Vessels contained no clots.

Left Lung.—Weight, 1,113 gm. The colour of the lung was almost a canary yellow. It had not the appearance of acute degeneration. There was no pneumonia.

Abdomen.—There were no signs of peritonitis, neither were there petechiae, subperitoneal or mucosal.

Intestines.—The contents of the upper three-fourths of the canal were much bile stained. The large gut was filled with hard faeces. No intestinal parasites were seen.

Right Kidney.—Weight, 607 gm. Its colour was dark, but there were no haemorrhages. It was firm in consistency, and the capsule peeled with ease. Section shewed nothing abnormal.

Left Kidney.—Weight, 405 gm. Same appearance as right.

Subrenals.—Seemed to be normal; there were no haemorrhages.

Spleen.—Weight, 810 gm. The substance was very dark and rather diffuent. The capsule slightly thickened, and the trabeculae well marked. They were particularly well seen after washing away the semi-fluid pulp. No infarcts.

Liver.—Weight, 3,645 gm. Everywhere over the capsule small, hard, yellowish nodules of about the size of a millet seed were seen. These were seen also throughout the substance of the liver; they seemed not to be calcareous, yet were too firm to be crushed between the thumb nails. The vessels were normal.

Pancreas.—Weight, 405 gm.; normal.

Lymphatic system.—Many of the mesenteric glands were enlarged to the size of a large walnut or pigeon's egg. These were seen on section to be soft, almost fluid in places, and yellow. They exuded a watery liquid strongly resembling the yellow fluid which has been described as infiltrating the connective tissues round the great vessels at the heart's base and the abdominal subcutaneous tissues. Other mesenteric glands were only slightly enlarged, many of them contained small haemorrhagic areas, principally in the cortex. Some of the pelvic glands were very dark in colour and uniformly haemorrhagic. Many small, markedly haemorrhagic glands were seen in the mesentery. On the left side, beneath the parietal peritoneum, was a large, dilated, turgid, lymphatic vessel, about 63 mm. in diameter. It was filled with pale yellow, easily coagulable fluid. It contained no adult filariae.

Brain and its membranes, particularly the pia mater, were congested.

Bone Marrow.—The marrow of the long bones was yellow in colour and apparently normal.

Coverslip preparations were made and examined from all the organs and from blood taken from different parts of the body. They were also made from the oedema fluid taken from the connective tissues, from the pericardial fluid, and from the

dilated abdominal lymphatics and glands. In none of them were any living trypanosomes found. Neither were parasites seen in the diluted vena cava blood which was used for injecting animals (successfully). Several c.c. of this diluted blood were centrifuged and examined, with negative results, for living trypanosomes.

Cases II and III.—Were both young stallions owned by natives of Bakau.

The previous history of neither was known. One had been in the village for only a year, the other longer. When seen, both were very thin, and one had a slight whitish discharge from the eyes. Temperature of each about 101° F. The blood of both horses contained trypanosomes (five to cover) similar in appearance to those seen in the remaining cases. The horse which had been in Bakau for only a year was said to have commenced to grow thin and weak in August, 1902. No oedemata, haemorrhages, discharges, nor marked paresis were seen in either of these horses. Mucous membranes were anaemic. Coats were glossy and not staring. One of these horses was living in May, 1903, the other died during December, 1902.

Case IV.—A stallion, two years old, owned by a native at Bakau.

History.—The horse was born in Bakau. Illness commenced a month previous to our examination on October 15. It had always previously been healthy. The mother of this colt had died of an illness which presented similar symptoms.

Clinical Examination.—The animal was very thin, ribs, ilia, and vertebrae being very prominent. There were no signs of oedema, nor of discharges from eyes or nostrils, of haemorrhages nor of paresis. The coat was glossy and smooth.

Temperature was 101.6° F.

A blood examination showed four trypanosomes to a coverslip preparation.

The emaciation continued, became extreme, and the horse died during the night of November 4. There was no autopsy.

Case V.—A young two-year-old stallion, bought from a native trader at Jesshuon (near Bakau), where the horse was born. It was first seen on October 19, 1902, when it was found to be infected with trypanosomes, two to a coverslip preparation. Temperature at this time was 103.4° F.

Clinical History.—The colt was small, and when first examined was in fairly good condition. Although thin, it was not emaciated, and was constantly ridden by its owner. Its appetite was excellent, but, in spite of a good supply of food, it became steadily thinner, and commenced to breathe rapidly and in a laboured manner even while at rest. Its coat was rough.

On November 28 it had become considerably thinner, and a scanty whitish mucoid discharge was noticed from eyes and nostrils. Emaciation and weakness progressed, and on December 18 the animal died suddenly. Towards the end of the disease, loss of power became a prominent symptom. The hind legs, particularly, were noticed to drag and to be weak.

The parasites in this case were never very numerous and were constantly absent for three weeks before death. The temperature was usually between 101° and 103° F. On two occasions, however, there were sudden rises to 106° F. Quantitative blood examination shewed a marked decrease of haemoglobin, on Nov. 27, thirty per cent., and a slighter decrease of erythrocytes; red cells, 4,700,000; white cells, 19,000; parasites, fairly numerous.

No oedemata nor yellowish tint of the anaemic conjunctivae and mucous membranes were ever noticed in this case.

Autopsy.—This animal died on December 8, 1902, and the *post-mortem* examination was commenced fifteen minutes after death. *Rigor mortis* had not set in. Body was much emaciated. Marked congestion of the conjunctivae and a few haemorrhagic points on the membrana nictitans and sclerotic. Mucous membranes, particularly of the gums, anaemic and of a slightly yellowish tinge. On section no yellowish oedema of the abdominal subcutaneous tissue, although there was some slight infiltration of a gelatinous straw-coloured fluid about the sheath of the rectus abdominis and about the penis. About 600 c.c. of straw-coloured liquid was taken from the peritoneal cavity. The lymphatic glands of the mesentery were boggy and permeated with a yellowish gelatinous, easily coagulable fluid, which oozed rapidly from them on section.

Spleen.—Weight, 506 gm. Slight thickness of the capsule together with an injection of its superficial vessels. On section the organ was dark in colour and slightly fibroid.

Right Kidney.—Weight, 354 gm. Normal.

Left Kidney.—Weight, 304 gm. Rather congested, otherwise normal; capsule of both peeled with ease.

Pancreas.—Weight, 202 gm. Normal.

Peritoneum.—Normal.

Thorax:—Pleurae.—Each contained about 100 c.c. of clear straw-coloured fluid, in which were floating small coagula of lymph, here and there attached to the parietes, particularly towards the diaphragm; no pleuritis. The pericardium contained about 55 c.c. of similar fluid; no pericarditis. The bronchial glands were soft, oedematous, and soaked with the clear yellow fluid so often mentioned. Some of these glands were injected and others quite pale.

Right Lung.—Weight, 709 gm. The superficial vessels were congested.

Left Lung.—Weight, 607 gm.

Heart.—Weight, 1,113 gm. As in the first case, the connective tissue round its base was filled with yellow coagulated oedema fluid. The muscle was very pale and friable and somewhat mottled. All the valves were normal.

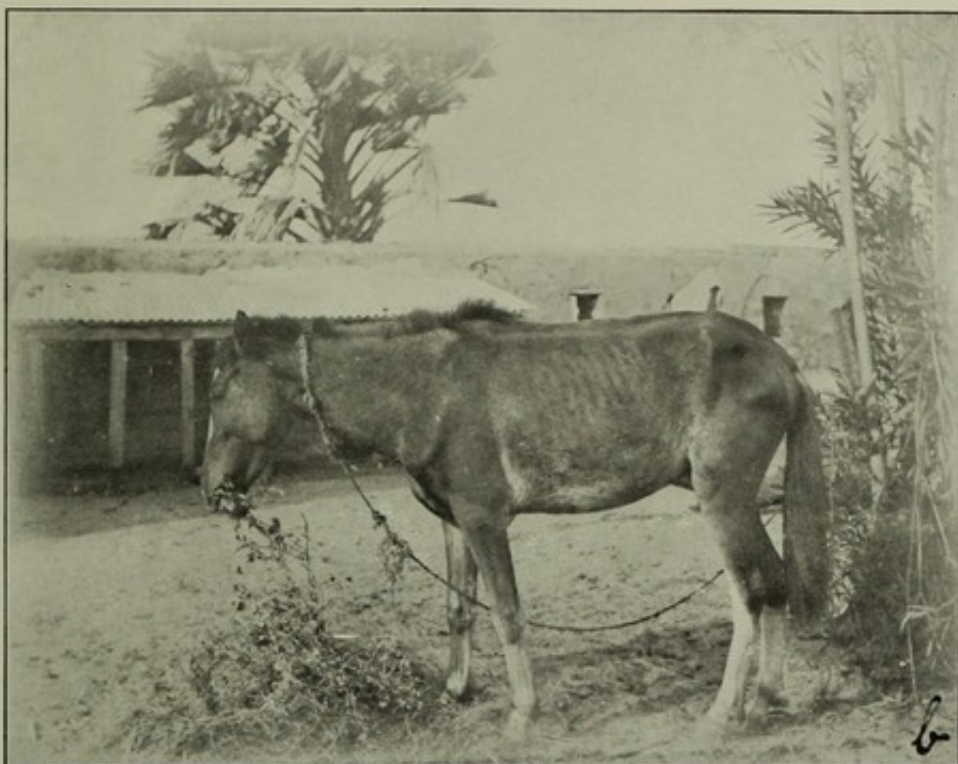
The *aorta* was normal. On the superior mesenteric artery there was an aneurism caused by sclerostoma similar to that seen in Case I.

The *alimentary canal* was normal; the intestines were not congested. A few bot-fly larvae were found in the stomach and also in the duodenum. The enlargement of the abdominal glands was not as marked as in Case I.

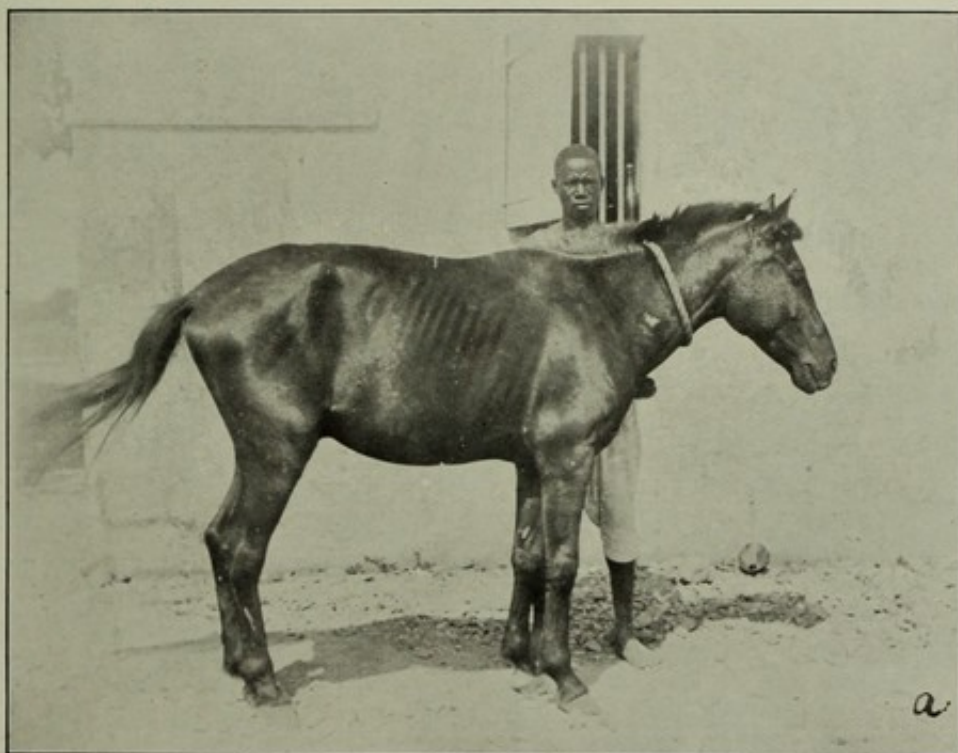
The marrow in all the bones, both long and short, was normal in appearance.

Films were made from all the organs. No parasites were seen in fresh preparations of blood taken from all the organs.

The accompanying chart of the morning and evening temperatures shows a fairly even temperature with rises at irregular intervals.



Horse, Case V, *November 28, 1902*

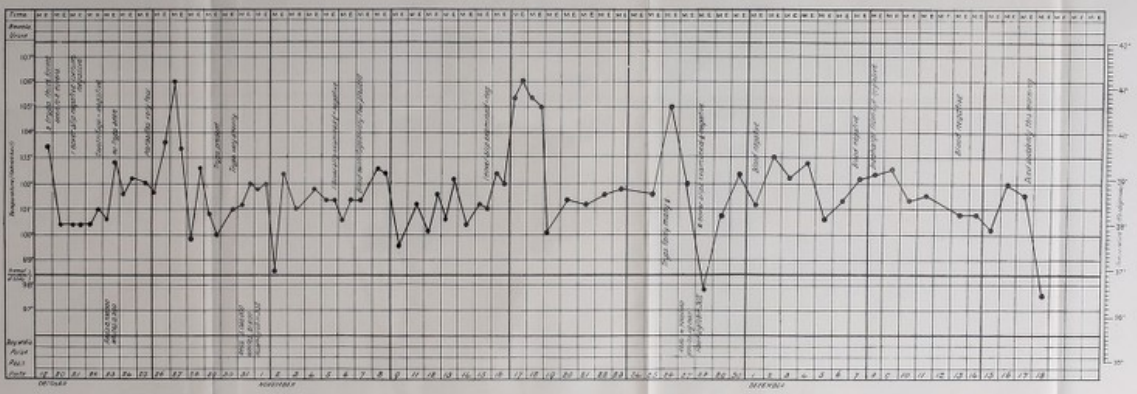


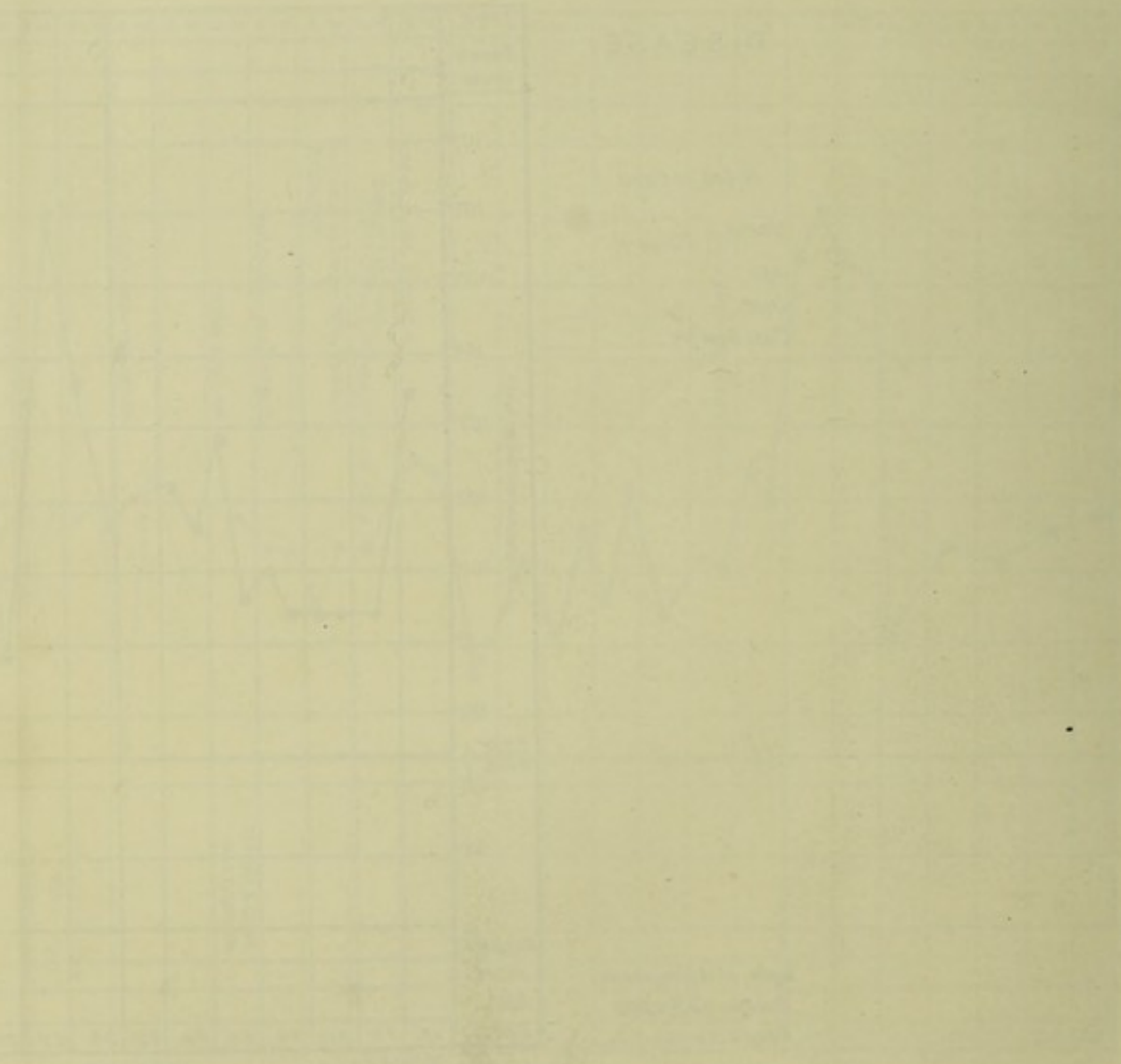
Horse, Case VI, *April, 1903*

DISEASE.

Name: _____
 Age: _____
 Sex: _____
 Case No. _____

Date of Admission: _____
 Discharge: _____
 Room: _____

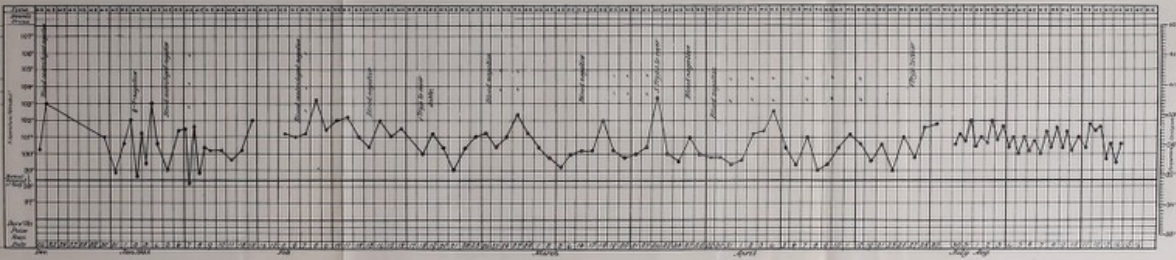




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No.
Date
City/State

Date of submission
DECEMBER 1911
Author



TO FACE PAGE 31

Case VI.—Small black stallion ; age eight years. Owned by an officer of the West African Frontier Force.

History.—This horse has been in Bathurst for four years, and during this time has always been well. Of its previous history nothing was known. At the beginning of October, 1902, he was taken into foreign Kombo, and on his return to Bathurst he had an attack of 'rheumatism' affecting his hind-quarters.

Clinical Examination.—He was examined on October 30, 1902, and found to be infected with trypanosomes. At this time he was sleek and in excellent condition. His owner stated, however, that the pony was not as vivacious as before the journey through Kombo, and was not so hardy as he had been a year previously.

To one seeing the pony for the first time there was absolutely nothing to suggest ill-health. There were no oedemata, no listlessness, and no emaciation. There was no perceptible dragging of the hind legs or slackness in gait. The temperature varied, being usually about 102° F. Parasites were only periodically seen in the peripheral blood.

We have never observed any oedemata, haemorrhages, or paralysis in this horse. We have seen slight discharges from his eyes, but this was probably due to an inter-current conjunctivitis. The genitals were relaxed, as is often the case, and conveyed the idea of enlargement without being really swollen.

Parasites were never very numerous in the blood of this horse and appeared periodically.

We purchased the animal towards the end of December, 1902, and have had it continuously under observation until the present date. The animal is still in apparently fair condition, and looks better than in March last when it was very thin and weak.

The temperature has been usually from 100° to 102° F., with periodic rises to 103° F., when parasites were often seen. The following chart gives the temperature curve of this case, and shows how frequently parasites were absent from the peripheral blood.

Urine was alkaline, Sp. gr., 1,042, and never contained albumen or blood. April 6.

Case VII.—Stallion, Joubert, aged eleven years, owned by French Government. Stationed at Maka.

*History.**—This horse came two years previously from Thiès. It had not been well for a year. 'Its feet, legs, and sheath had first become swollen.' This had been later followed by a fulness of the loins and by a general enlargement of the horse's belly.

When we saw this and the following case they were both apparently in excellent condition. We could detect no oedema, though the usual relaxation and apparent

* The histories of Cases VII and VIII, with the account of their illnesses was given to us by Monsieur Porthes. We take this opportunity of again thanking him for the aid which he gave us in our work.

enlargement of genitals was present. Their coats were smooth and glossy. There was no running from eyes or nostrils. Mucous membranes were rather pale. The appetite in each case was good. Their abdomens were rather distended. These animals were not grass fed. Temperature of Case VII, 100° to 101° F. Number of parasites, one to a coverslip preparation.

Case VIII.—Stallion, Jourdan, eight years old: it came two years previously from Thiès.

History.—This horse was taken ill at the same time as Case VII. The symptoms were the same in both cases. The animal had not been worked for some time when we saw it, and was in much better condition than Case VII.

The clinical examination has already been given. Temperature was 101° to 101.5° F. Trypanosomes were very scanty, one to a coverslip preparation, and were seen on but one occasion.

M. PORTHES recognized the illness from which these two horses were suffering. He believed that they acquired it while in Tenda, where forage is poor and water is scanty. He ascribed the first symptoms which he noticed, weakness and swelling of the cannons, to the poor food, and hard usage over stony ground which horses get while travelling in that district. He had seen other horses suffering from a similar affection, and described in them a fulness of the loins which alternated with distension of the abdomen and swelling of the sheath and scrotum. He described an accompanying anorexia, and stated that the course of the illness may vary, death following after a week or two to several months. Sometimes, indeed, animals may recover. He has never observed running from eyes or nose, haemorrhages from any part, or marked oedema in any of these cases. He had seen staring of the coat in some cases, never depilation. He had never seen the disease in an epidemic form.

Case IX.—A grey stallion, five years old.

The origin of the horse was not known. It had been owned for fifteen months by the officer who had it when we first saw it. Two years and a half previous to this time the horse became extremely emaciated and weak. It was expected to die, but recovered its strength after a prolonged rest. In July, 1902, it had lampus badly and became very thin. When the mouth was cured and the animal once more commenced to eat heartily, it rapidly regained the weight lost through lack of food. In January, 1903, the horse was taken on a tour through the country on the north bank of the Upper River, and, although it had lots of hard work, returned to the Lower River in apparently excellent health.

Clinical History.—When the animal was seen in April, 1903, its owner stated that a month and a half previously he had noticed that it was rather 'slack' and 'coughed' at night. It had since lost flesh, 'particularly about the chest,' and the abdomen had become 'bigger.' The horse stood with hanging head, in the same listless way as was noticed in the preceding cases. The scrotum and penis were

relaxed and apparently swollen, though neither about the genitals nor in any other part of the body could oedema be demonstrated. The temperature varied from 101.6° to 104.2° F. Trypanosomes were seen in the blood on two occasions during the two days the pony was under observation.

Case X.—A bay stallion belonging to the Governor of the Gambia. This was also a very early case. For only a few weeks had it been noticed that he was not as lively as usual. Parasites were demonstrated in his blood on two occasions. The symptoms were precisely those noted in the early stages of the preceding cases, save that there was very little loss of flesh. Temperature varied from 100° to 102° F. during the short time the animal was under observation. This case is the only one of this series in which treatment has been attempted. *Liquor arsenicalis* was used in gradually increasing doses until the horse was getting five grains of arsenic a day. The dose was kept at this for a week and then reduced. *Sir GEORGE DENTON*, in a letter dated June 23, says that the horse is 'decidedly better.'

With the aid of these cases we can construct a well-connected though composite picture of what we believe to be the usual course of the horse disease in Gambia and the immediately adjacent territories. Three of these cases were seen in the earliest stages of the disease, two during their last weeks, and the remainder at various periods during the course of their illness. One case we have had under continual observation since it was first noticed to be 'off colour,' and within a month of the date at which it was supposed to have been exposed to infection.

Sketch of Symptoms.—The first feature noticed is loss of accustomed vigour. There is not quite the same vivacity in harness nor usual power of much endurance. Still, the animal is apparently in perfect condition. He is fat and his coat is smooth. The temperature is only slightly above normal at this time (102.6° F.), and if the blood be examined but few parasites will be seen, perhaps as many as ten to a cover, often not so many. Even these may, for long periods together, be absent from the peripheral blood.

Two or three weeks later more marked signs of ill-health become manifest. The horse has commenced to grow thinner, his head is drooping, his eye is not so bright, and although he is well able to go under saddle, still the rider is conscious of the animal's weakness, which has now become patent.

At this period there are periodical rises of temperature, at which times the parasites will usually be found in the blood, although they may often be absent.

In another month emaciation is more marked. The ribs commence to shew. As a Frontier Force Officer pithily expressed it, 'the flesh seems to slip back from the horse's chest to his belly.' Although the abdomen becomes large it is not in any sense due to an oedema. We never observed pitting, and believe that the apparent enlargement is due rather to the atonicity which affects the muscles of the bowel in common with those of the trunk. The scrotum becomes relaxed and the testicles hang so

low that they seem at first sight to be oedematous. At times a slight watery discharge from the eyes may be observed. In none of the horses have we observed the marked oedema of abdomen, scrotum, and legs, nor the staring of the coat which is described in horses suffering from Nagana.

This last stage has lasted in one of our horses (Case VI) for ten months, and during this time on only four occasions have parasites been seen in his blood. On each occasion their presence was accompanied by a slight rise in temperature to 103° F., or thereabouts.

As the disease proceeds, emaciation becomes more and more marked. The ribs and ilia stand out prominently. The animal wears an apathetic chronically-tired expression which is most characteristic. No oedema is to be made out. A whitish discharge from the eyes, in small quantities, is often seen. Saddle galls and sores on the projecting hip bones are often present. No haemorrhage has been observed in any of the mucous membranes. No blood has been seen in the urine.

The parasites are now often almost continuously present in the blood and may reach very considerable numbers. Case V, however, presents a most notable contradiction to this generalization.

The temperature fluctuates, though it is generally raised, and may go up as high as 105° F.

Two horses have died under observation. One lingered for three days, scarcely able to rise from the ground, in a state of utter weakness. His breathing was very laboured, he sweated almost continuously, and just before death had a slight convulsive seizure (Case I).

The second animal died suddenly one day after being taken out a little distance to graze. In this animal a few conjunctival petechial haemorrhages were seen (Case V).

The most notable features observed in the autopsies of these two animals were the yellow gelatinous oedema fluid found round the sheath, and in the first case, on the abdomen, and the amber-coloured fluid with flakes of yellow gelatinous lymph seen in the peritoneum and in the pericardial and pleural cavities. There was a general enlargement of all lymphatic glands. Some were soft, amber-coloured, and watery, others had a chocolate-coloured centre, and still others shewed marked petechial haemorrhages; some were entirely haemorrhagic. Spleen was not enlarged. Lungs were congested. Liver shewed fatty change. In one case the heart shewed marked fatty degeneration (thrush heart). To the naked eye there was no change in the bone marrow.

Only in the very last stages of the disease does any marked alteration in the blood seem to take place, then both red cells and haemoglobin are diminished.

Duration of Disease.—How long the disease lasts we cannot at all say. We have now (November, 1903) under observation, a horse (Case VI) which we bought in November, 1902, in good condition (first stage). We believe that it contracted the disease at least a month before.

Just before leaving Bathurst we heard that a year-old colt examined and found to be infected in October last, was still alive. On the other hand, a similar young horse (Case V) in much the same condition, which was seen at the same time, died two months later.

Prognosis.—We think from what we have seen that it is not impossible that horses may occasionally recover.

The Diagnosis of Horse Trypanosomiasis.—Although a positive diagnosis of trypanosomiasis can only be made by a demonstration of the parasite, either by microscopical examination of suspected blood or by inoculation of that blood into experimental animals, we were in several cases enabled to diagnose the disease by the listless expression of the affected animal, and by the slight rise in temperature, which was always present. So constant is this latter symptom, that we came to regard with the greatest suspicion as a possible case of trypanosomiasis any horse whose temperature was over 101° F.

IV. MORPHOLOGICAL CHARACTERISTICS OF THE HUMAN AND HORSE TRYPANOSOME FOUND IN THE GAMBIA

We propose to give here short descriptions of the microscopical appearances of the human and horse trypanosomes as they are seen in the peripheral blood of their respective hosts and in experimentally infected tame rats.

We reserve a more detailed account of these parasites and a description of their appearances in various other animals inoculated for a future report.

The description of the morphological characteristics of a pathogenic trypanosome presents at the outset great difficulties; so varied are the forms seen, so minute are the differences distinguishing one form from another, and so many are the intermediate stages between two contrasted forms in any infected animal, that it requires some time and study before the characteristic adult form of parasite can be finally determined.

Though we could detect no constant morphological differences between the human and the horse parasite—more especially was this the case in inoculated animals—still the parasites usually seen in the blood of horses, examined in the early stage of their infection, presented marked differences in size and appearance from the parasite usually seen in the blood of the infected natives.

The Human Trypanosome.—Plate I, fig. 1, represents this parasite as usually seen in the native.

In stained preparations its length, including the flagellum, averages 20μ . Its width is 1.8μ to 2μ . The distance from micronucleus to the centre of the macronucleus is 5.9μ . The distance from the micronucleus to the tip of the posterior end of the body was found to vary, as a rule it measured 1.6μ , but parasites were often seen in which this distance was only 0.5μ .

This trypanosome, as seen in the native and quadroon cases, presented no features morphologically differentiating it from the one described by one of us in 1901, as occurring in the blood of a European and a native child in Gambia, and named *T. gambiense*.

The Horse Trypanosome.—The parasite seen in Senegambian horses was never encountered in any great numbers in the peripheral blood during the early stages of the infection. The blood of one horse, Case V, in which the disease proved fatal, never contained many parasites.

Plate I, fig. 6, represents the trypanosome as seen in the early stages of infection of the horse. The parasite is a very small organism, rather tadpole-shaped.

Its protoplasm stains deeply. Its posterior end is either sharply cut away or bluntly conical. The body tapers from the posterior end as a rule, and is prolonged anteriorly into a short fine flagellum.

The micronucleus and vacuole are placed almost at the extreme posterior end of the parasite, the former is a small dark crimson staining spot generally situated to one side, the latter varies much in size in different specimens. The macronucleus is generally rounded, granular, and does not quite extend across the short axis of the body. The blue staining body-protoplasm of some specimens contains a few isolated chromatic granules. The undulating membrane is narrow and is seen with difficulty in stained preparations.

The measurement of this type of parasite in stained preparations is as follows:—

Length, including flagellum, $11\ \mu$ to $13\ \mu$. Width, $0.8\ \mu$ to $1\ \mu$. Distance from micronucleus to centre of macronucleus, $4\ \mu$ to $6\ \mu$. Distance from micronucleus to posterior end of body, $0.3\ \mu$ to $0.5\ \mu$. Divisional forms of this small parasite are represented in Plate I, fig. 8.

In horses in the last stage of the disease and in rats inoculated with either the human or horse trypanosome, we were generally able to distinguish roughly two constant types of parasite. These types we called for convenience the 'long form' and the 'stumpy form' of the parasite.

A.—The long form (Plate I, figs. 9 and 10) is characterised by a long thin body and a long flagellum. The posterior end of the parasite may be long (pointed or square shaped) or stumpy and conical. The deeply staining macronucleus is ovoid in shape and lies longitudinally. It is this type of parasite that multiplies by longitudinal division. The length of such a parasite, including the flagellum, is from $26\ \mu$ to $30\ \mu$; width, $1.6\ \mu$ to $2\ \mu$. Distance from micronucleus to centre of macronucleus, $7\ \mu$ to $8\ \mu$. Distance from micronucleus to posterior end of body, $1.6\ \mu$ to $3.2\ \mu$.

This type can be recognized in fresh blood by its rapid movements and long flagellum, and is usually most numerous in the blood of an infected animal a few days before its death.

B.—The Stumpy form (Plate I, Fig. 7).

This type of parasite is more commonly seen in the blood when the disease in an infected animal is not far advanced. It is characterized by a short thick body and very short flagellum. The posterior end is short and conical, and generally quite close to the tip is situated the micronucleus and vacuole, the latter as a rule is well marked.

The macronucleus is either oval or circular in shape. If it is ovoid it is placed transversely in the body of the parasite. A few scattered chromatic granules may be seen in some specimens.

Longitudinal division of this type of parasite has not been observed.

Measurements in stained preparations: length varies from $16\ \mu$ to $19\ \mu$; width from $3\cdot4\ \mu$ to $3\cdot5\ \mu$. Distance from micronucleus to centre of macronucleus, $5\ \mu$ to $7\ \mu$. This type of parasite generally survives for a longer time in ordinary fresh preparations than the long form.

Though the above broadly divided types of parasite can be distinguished at a glance in stained preparations, yet in slides containing many trypanosomes one observes every gradation between the stumpy and the long forms, and between the stumpy type and a rounded form (Plate I, Fig. 5), which we have seen in the blood of horses and rats in the last stages of the disease, produced by infection with either the human or horse parasite.

The Human Parasite in Rats.—The parasite when first seen in the peripheral blood varies little in appearance from the usual type of parasite seen in the native, and, as in the native, few are seen in a preparation. Long and stumpy forms and longitudinally dividing parasites (Plate I, Fig. 3) are occasionally met with.

The long forms are generally slightly smaller than those seen in rats infected with the horse parasite, and their macronucleus is situated slightly more towards the flagellum-bearing end of the parasite. Compare Plate I, Figs. 2 and 10. A few of the parasites shew chromatic granules in the protoplasm in front of the macronucleus.

The appearance of the parasites differs to some extent from the above type in preparations which were taken, two or three weeks before death, from two rats which have died (Experiments LXIX and XXVII). The parasites are more numerous, and more long forms undergoing division are seen. In nearly all the parasites there is a marked chromatic stippling, produced by chromatic granules situated in the protoplasm, posterior as well as anterior to the macronucleus. The number of these granules varies greatly. In the parasites undergoing longitudinal division, generally few or none occur. In some of the parasites the chromatic granules are very large. They take on a deep blue purple colour with ROMANOWSKY'S stain which, though closely resembling the chromatic staining of the micro- and macronuclei, can be distinguished from it by its bluish tinge (Plate I, Figs. 2, 4, 5).

Besides the above types there are seen in rats, at this late stage of the disease, rounded forms (Plate I, Fig. 5). In them the macronucleus is round and, usually, the individual chromatin granules composing it can be made out. The macronucleus is situated either in the centre of the parasite or to one side. It does not occupy the whole width of the body. In forms more completely rounded than represented in Fig. V the posterior and anterior ends of the body cannot be made out, and the flagellum is often only attached to the parasite for a short distance. Some of these forms show two or more macro- and micronuclei. These probably correspond to PLIMMER and BRADFORD'S 'amoeboid forms.'

The Horse Parasite in Rats.—The parasites seen in the blood of rats infected

from Horse I were, from the commencement of the infection, precisely similar in appearance to those occurring in that horse. Stumpy forms were at first more frequently met with in these rats, but as the parasites increased in the blood more long and dividing forms were seen. About a week before death amoeboid forms were also seen in the peripheral blood of these rats. Chromatic stippling was only occasionally seen in the parasites.

The parasites which first appear in the blood of rats and mice inoculated from horses in an early stage of the disease are morphologically identical with the small forms seen in the horse (Plate I, Fig. 6). Many of these small forms are seen in all stages of longitudinal division (Plate I, Fig. 8). As the disease in the experimental animal progressed, long and stumpy forms were seen, and the smaller tadpole-shaped parasites tended to disappear. Rounded forms can be seen in the blood of many rats in a late stage of the disease.

Still another variety of parasite has been seen occasionally in the peripheral blood of rats inoculated with the horse trypanosome and also in the blood of horses either one, two, or three days before death.

In shape, like either the stumpy or long form, it differs from them by its very feebly staining protoplasm. This type corresponds very closely to the 'hyaline' forms described by PLIMMER and BRADFORD.

V. TRANSMISSION EXPERIMENTS

A tsetse fly, *Glossina palpalis*, is practically ubiquitous in the Gambia. It is particularly numerous among the mangrove swamps, and for this reason it is locally called a mangrove fly. It is, however, seen all along the river, far above the brackish water in which the mangroves grow. Though the flies, as a rule, seem to prefer the immediate neighbourhood of some large expanse of water, they have been seen and caught in the bush at some distance (two miles) from any collection of water. We have taken specimens just outside Bathurst, everywhere through British Kommo, and at many places along the river as far as Sunkunda. They were seen at Walia, and everywhere on the river itself they were a most pertinacious and disagreeable pest.*

It was with this fly naturally that we made our first attempts to transmit trypanosomiasis from infected to healthy animals.

We first allowed flies caught in British Kommo, where there was a good deal of equine trypanosomiasis, to feed upon healthy rats. Our results were always negative.

A detailed account of these experiments follows:—

Experiment 1.—A white rat was selected, and tsetse flies caught in the bush near Bakau were fed upon it in the following manner and on the following dates:—

Sept. 14, 1902	—	4	flies out of a batch of 6	fed.
„ 15	„	3	„ „ „	„
„ 15	„	4	of the flies caught on the fourteenth	also fed.
„ 16	„		a batch of 6	fed.
„ 17	„	3	flies caught on previous dates	fed.
„ 18	„		a batch of 4	fed.
„ 19	„	2	flies caught on previous dates	fed.
„ 23	„	4	flies out of a batch of 17	fed.
„ 24	„	3	freshly caught flies	fed.
Oct. 1	„	3	„ „ „	„
„ 3	„	8	„ „ „	„

Altogether, 44 flies (*Glossina palpalis*) caught from the bush at Oyster Creek, about three and a half miles from Bathurst, were fed on this rat at periods varying from

*In spite of a careful search, we never succeeded in finding *Glossina* in Senegal. No tsetse flies were seen in the mangrove swamps in the neighbourhood of St. Louis, which, in the Gambia, would certainly have sheltered myriads of them.

Traders and residents in St. Louis and Dakar failed to recognize the specimens of *Glossina* which were shewn to them, and officers on steamers plying on the river Senegal, between St. Louis and Kayes, had never seen the fly. We were, however, told that it occurred to the South, on the River Kaolakh.

The absence of *Glossina* from a district in which we failed to find equine trypanosomiasis forms, perhaps, a striking coincidence.

one to twelve hours after their capture. Only those flies which were seen to be gorged with blood were held to have fed. It may be assumed that all the flies which were given an opportunity to feed attempted to do so, and that the proboscis of a much larger number of tsetse flies than 44 pierced the rat's skin. The blood of this rat was examined constantly, always without success, for trypanosomes.

Experiment 1a.—Tsetse flies caught near Bakau, where five out of the six horses owned in the native village were infected with trypanosomes, were allowed to feed at once on an uninfected white rat.

Oct.	24—25	newly-caught flies fed well.
„	25—10	„ „ „
„	26— 2	„ „ „
„	28— 2	„ „ „
Nov.	14—30	„ „ „

This rat was constantly examined, always with negative results, until April 14, 1903, when it died from sunstroke.

Experiment XIX.—Flies freshly caught near Bakau were allowed to feed upon a young non-infected white rat.

Oct.	24— 2	flies fed.
„	25—10	„ „
„	26—10	„ „
„	28—20	„ „
„	29— 8	„ „
„	30—12	„ „

This rat was constantly examined without result until November 14, when it was killed.

Experiment VIII.—We also attempted to infect a tame rat by allowing it to be bitten by tsetse flies which had previously fed on a naturally infected horse (Case No I) and on an artificially infected rat (Experiment IX) in the blood of which there were a great many parasites.

A stock of flies, continually replenished by the addition of freshly-caught flies, was kept in a cage and fed on successive days alternately on the infected and non-infected animals in the following manner :—

INFECTED ANIMALS	UNINFECTED RAT
Oct. 11—12 flies put to feed 4 filled themselves	Oct. 12—5 flies fed well at 9 a.m.
„ 12— 6 flies fed on Horse I at 4 p.m. Horse died	„ 15—3 out of 6 flies fed, others attempted to feed at 9 a.m.

INFECTED ANIMALS—*contd.*UNINFECTED RAT—*contd.*

Oct. 21— 5 flies fed on rat, Experiment IX, 3 at 1 p.m., 2 at 6 p.m., many trypanosomes in blood.

„ 22— 5 flies fed on Experiment IX

„ 24—25 flies fed on Experiment IX this p.m.

„ 26— 2 flies fed on Experiment IX

„ 29— 6 flies fed on Experiment IX

Oct. 16—5 flies fed well.

„ 25—Many of 25 flies pierced the skin, but none succeeded in getting full meal.

„ 30—All flies fed on rat.

The conditions of this experiment were purposely made as broad as possible. The sole object of the experiment was to see whether *Glossina palpalis* would transmit the Gambian horse trypanosome in the same way as *Glossina morsitans* does *Trypanosoma brucei*.

With this end in view, flies which had fed at any previous period on either of the infected animals were allowed to feed indiscriminately on the test animal. This was examined constantly until April 14, when it was killed by sunstroke. Its blood was, as in all the transmission experiments, examined, at first, daily, both with and without the centrifuge, and always in vain. The autopsy revealed nothing abnormal.

Although tsetse flies could be found near McCarthy Island, where we made our headquarters from January to April, it was never possible to catch them in sufficient numbers to make the success of a repetition of these experiments probable.

It is maintained that the tsetse fly carries Nagana from animal to animal in a mechanical way, and that probably no biological change in the parasite intervenes. We, therefore, determined to repeat these experiments, using instead of *Glossina*, two varieties of *Stomoxys* (not yet identified), which were very plentiful in the Upper River, and easily caught.

Experiment CI.—During a period of eight days, a number of *Stomoxys*, about one hundred altogether, were caught from two horses and placed in the same cage. One of these horses (Horse No. VI) was a naturally infected animal, and at this time its blood contained on an average five parasites to a preparation. The other (Experiment LXXXVII) was a young native stallion which had been artificially infected with the human parasite from rat LXIX (Q. strain). The flies had an opportunity of feeding within twenty-four hours of the time at which they were caught, on a young, non-infected white rat, and they were allowed to feed daily on the same animal for a period of eight days. The rat has been under observation up to the present date and has never become infected.

Experiment C.—On March 18, 1902, a large number of *Stomoxys* were caught while feeding on a horse (Experiment LXXXVII) infected with the human trypanosome. They were daily fed upon this horse, whose blood contained five parasites to a preparation until March 24. On March 25 they were allowed to bite a young non-infected white rat. Twenty-four flies fed well.

The flies were then fed on alternate days on the horse and test rat as follows :—

March 26	—	Flies fed on	Horse LXXXVII.	Many fed.
„ 27	„	„	Rat.	„
„ 28	„	„	Horse LXXXVII.	
„ 29	„	„	Rat.	
„ 30	„	„	Horse LXXXVII.	
„ 31	„	„	Rat.	
April 1	„	„	Horse LXXXVII.	Very few fed.
„ 2	„	„	Rat.	Only one sucked blood.
„ 3	„	„	Rat.	None fed.
„ 4		Flies were not fed.		
„ 5	„	„	„	Many dead.
„ 6	„	„	„	All dead.

This rat has been under constant observation, and up to the present no trypanosomes have been demonstrated in its blood.

Experiment CVI.—Flies were caught from the same horses as in Experiment CI. They were placed together in a cage and allowed to feed upon Rat Experiment LXVIII, in whose blood there were at this time numerous ‘horse trypanosomes’ (derived originally from Horse I); twelve hours later they were allowed to feed upon a non-infected white rat. This was done on two occasions. Thirty-five flies were observed to feed well upon the non-infected animal.

The experiment was commenced on April 3, and, up to the present, parasites have never been seen in the rat’s blood.

If the natural mode of transmission of the Gambian trypanosomes is a simple mechanical transference of the parasite from an infected animal to a healthy one, which is infected by the prick of a proboscis containing parasites, it seems strange that our experiments should have always been unsuccessful, and our efforts to repeat BRUCE’s transmission experiments without result.

As has been previously said, if the parasite is transmitted in a purely mechanical manner, it seems probable that other insects, possessing biting parts similar to those of *Glossina*, should also be carriers of the disease.*

* Mr. E. E. Austen, dipterist to the British Museum, who is classifying and describing the biting-flies collected by the expedition in Senegambia, informs us that our collection contains five species of *tabanus*, one species of *glossina* (*palpalis*), two of *stomoxys*, and one of *lyperosia*.

In Senegal, *hippoboscidae* were very frequently seen, and at St. Louis we caught several blood-sucking flies which belong to a new genus near *Musca*.

Heads and probosces of the tsetse flies and *stomoxys* used in these experiments were frequently dissected. In none of them were trypanosomes, as they are observed in the blood, ever seen. The head parts of insects were examined *immediately* after they had fed upon animals in whose blood the parasites were exceedingly numerous, and never were they found to contain recognizable trypanosomes either living or dead.

Living actively motile parasites, not in the least degenerated, were, however, seen in the stomachs of these flies for many hours after their ingestion. Their peculiarities and the alterations which they later underwent will be considered in a further report.

Our fly experiments were done during the dry season. It is possible that they failed owing to the excessive lack of moisture which could not fail to have made it impossible for the parasites to live, for even a few hours, in the insect's proboscis.*

An observation, interesting in this connection and based upon a very careful diary, has been related to us by Mr. HEWBY, F.R.G.S., formerly a resident in Northern Nigeria.

There is on the north bank of the river Benue (Nigeria), opposite Ibi, a notorious tsetse fly belt where *Glossina* occurs and in which horses contract 'tsetse fly' disease.

Mr. HEWBY unhesitatingly picked out specimens of *Glossina palpalis* from our collections, and assured us that the tsetse fly of Ibi was practically identical with them. The symptoms and duration—three to (rarely) ten weeks—of the disease in horses (it also affects cattle) convinced us that what is called tsetse fly disease in Northern Nigeria is most probably Nagana.

On two occasions (1899-1900) all the horses sent by Mr. HEWBY and others through this particular 'belt' during the rainy season contracted 'fly disease' and died.

Twice during the dry season (1901-1902), although the tsetse flies—seen also during the wet season—were extremely numerous, have ponies gone through the same belt of fly-bush without suffering harm. Once a pony taken in the dry season (May 1902) through a bit of bush comparatively near the Ibi belt 'was covered for hours' with the fly and yet did not contract the disease.

Mr. HEWBY also informed us that, as in the Gambia, the neighbourhood of the river is believed in Nigeria to be unhealthy for cattle and horses during the rains. It is in addition generally believed that the fly is practically harmless during the dry season. It is interesting to note that Mr. HEWBY knows of no instance in which asses and sheep have suffered from 'fly-disease.'

* At McCarthy Island, during the period at which our *Stomoxys* experiments were done, the dry thermometer in our laboratory daily registered about 95° F., the wet bulb thermometer being usually from 15° to 20° lower.

Though the conditions were similar during the tsetse fly experiments done at Cape St. Mary the temperature was not so high nor was the daily difference between the wet and dry bulb thermometers so great.

VI. RESULTS OF INOCULATION OF THE GAMBIAN HUMAN AND EQUINE TRYPANOSOMES IN EXPERIMENTAL ANIMALS

INOCULATION EXPERIMENTS

The experiments have been divided into two sets of tables, Human Trypanosome and Equine Trypanosome. In these divisions all the inoculated animals of the same species have been arranged according to the passage of the parasite and, as far as possible, all those of the same strain have been grouped together. In every case the strain of the parasite inoculated has been noted. In all the inoculations the blood was mixed with sterile sodium citrate solution (page 2).

All the rats and mice used in the following experiments were tame ones with the exception of a few bush rats and some wild mice—in the tables these animals are specifically marked.

All the animals were carefully and repeatedly examined before inoculation, to ascertain if they were free from Trypanosomes.

RÉSUMÉ OF INOCULATION EXPERIMENTS

We think that it would be premature and unprofitable to enter upon a detailed discussion of the properties and identities of these two Gambian Trypanosomes, based upon the experiments which we present in this report.

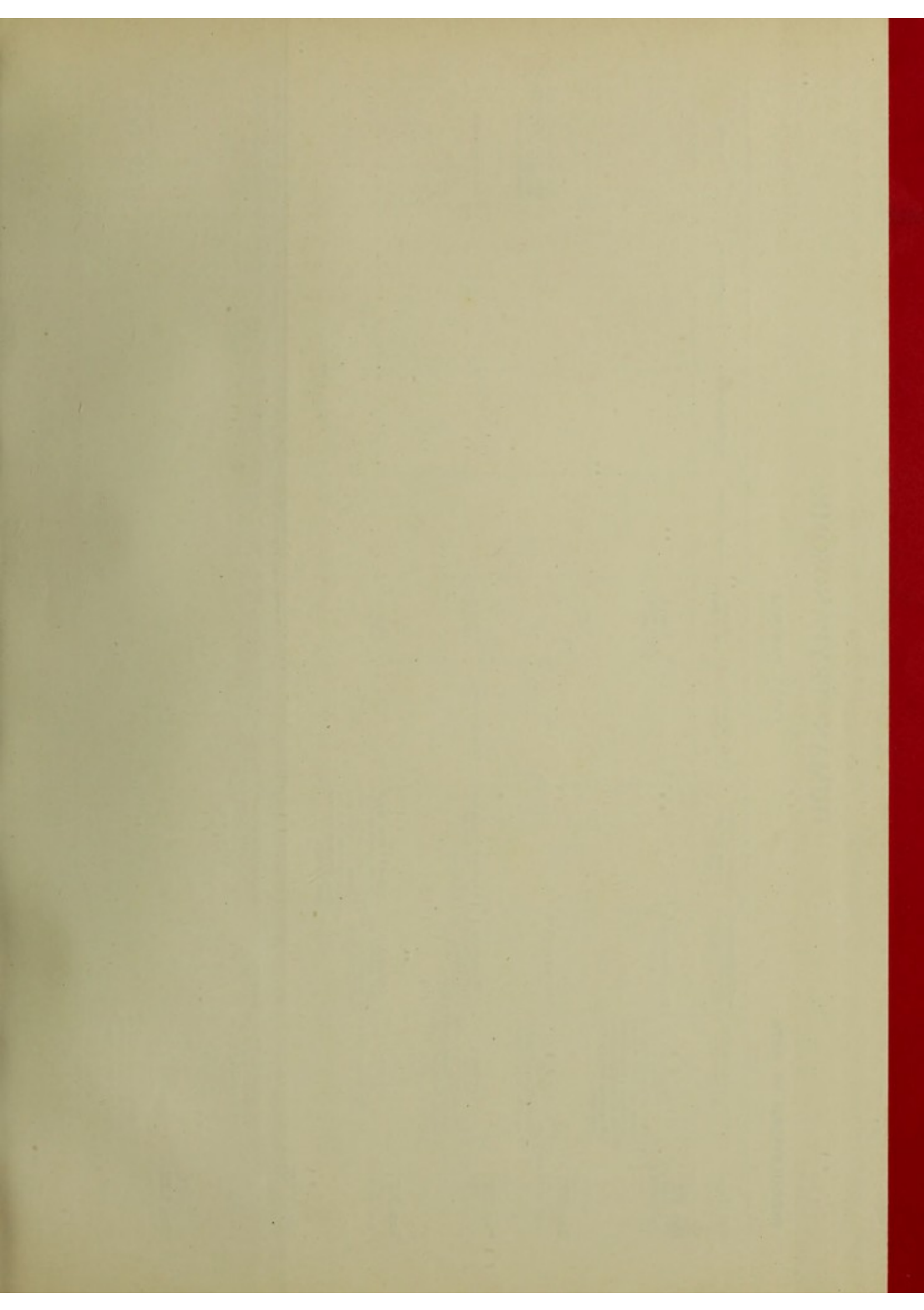
We fully recognize the importance of determining whether these two parasites are the same, or whether either is identical with any previously described pathogenic Trypanosome. At present we are inclined to believe that they are not the same species; our reasons for this opinion are:—

1. The more chronic course of the disease produced by the human parasite in all animals, perhaps with the exception of the goat.
2. Tame rats infected with the horse parasite certainly die from its effects, while, on the other hand, we have no evidence to show that the infection which has occurred in tame rats, inoculated with the human parasite, has been the only cause of death.
3. The modes in which the human and horse parasites appear and disappear in the blood of inoculated animals, during the course of the disease, is not the same; thus, in rats inoculated with the former parasite, there is a marked periodicity of entrance and exit of the parasite in the peripheral circulation, in the case of the horse parasite this periodicity is not a marked feature.

4. In most animals the enlargement of the spleen and the presence of haemorrhagic glands is a characteristic feature of infection with the horse parasite. In the case of the human disease, in the few rats that have died infected, the autopsies of these animals have not shewn haemorrhagic, nor even markedly enlarged glands.* On the other hand, we must remember that we have infected one horse with the human parasite, and it has produced a disease in that animal clinically indistinguishable from that seen in naturally infected horses. We know also that baboons (*cynocephalus sphinx*) have, up to the present, been insusceptible to both parasites.
5. We have already mentioned that the parasite found in horses, in the early stage of the disease, can be distinguished by its morphological characteristics from the human trypanosome found in the native.

The possibility of inoculating some of our animals now immune to the horse parasite with the human trypanosome, and other experiments of a like nature already in progress, will probably throw considerable light upon what we judge to be the most urgent problem requiring solution, namely, the identity or otherwise of these two parasites.

* In the case of a goat and a guinea-pig inoculated with the human parasite, haemorrhagic glands were seen at the post-mortem.



Animal	Date, Source, and Mode of Inoculation	Time of Appearance of Parasites in Peripheral Blood	Number of Parasites per Field	Type of Temperature Record	Death	Changes in Blood	Substitutions	P.M. Appearance	Remarks
Exp. IX Small black and white rat	October 14, Home 1 c.c. of blood. Parasites in peripheral blood.	October 15 No parasites in peripheral blood.	...	Not raised to appearance of parasites.	October 18 Dismembered.	Trace very dry, no enlargement of lymphatics.	...
Exp. IX Large black and white rat	October 14, Home 1 c.c. of blood. Parasites in peripheral blood.	October 15 No parasites in peripheral blood.	...	Even temperature, zero above 101° F.	November 4 Dismembered.	...	Exp. XVII Exp. XX Exp. XXI Exp. XXII	All lymphatic glands very much swollen. Spleen much enlarged and dark.	...
Exp. XIV Exp. XVII	October 14, Home 1 c.c. of blood. Parasites in peripheral blood.	October 14 No parasites in peripheral blood.	...	Irregular, up and down.	November 11 Dismembered.	Tablet, regularly attended with vomit. Spleen much enlarged. Spleen in cross-section normal in size. Spleen in longitudinal section 1 1/2 x 1/2 cm.	...
Exp. XVII White rat	October 14, Home 1 c.c. of blood. Parasites in peripheral blood.	October 14 No parasites in peripheral blood.	...	Very even temperature.	December 9 Dismembered.	Partially decomposed, enlarged lymphatic glands (spleen).	...
Exp. XXXI Large black rat	November 18, Home 1 c.c. of blood. Parasites in peripheral blood.	December 18 No parasites in peripheral blood.	...	Irregular, up and down.	December 25 Dismembered.	...	Exp. LIV	Spleen, f. s. 1 x 1/2 cm. 1 gland enlarged.	...
Exp. LXIV White rat	February 2, Home VI c.c. of blood. Parasites in peripheral blood.	February 7 No parasites in peripheral blood.	...	Very even temperature.	February 9 Dismembered.
Exp. LXXXIII White rat	February 2, Home VI c.c. of blood. Parasites in peripheral blood.	February 7 No parasites in peripheral blood.	...	Very even temperature.	February 9 Dismembered.
Exp. LXXXIII White rat	February 2, Home VI c.c. of blood. Parasites in peripheral blood.	February 7 No parasites in peripheral blood.	...	Very even temperature.	February 9 Dismembered.
Exp. CKLII Small white rat	July 15, Home VI c.c. of blood. Parasites in peripheral blood.	August 9 No parasites in peripheral blood.	...	Very even temperature.	August 11 Dismembered.
Exp. CKLIII Small white rat	July 15, Home VI c.c. of blood. Parasites in peripheral blood.	August 9 No parasites in peripheral blood.	...	Very even temperature.	August 11 Dismembered.
Exp. CKII Small white rat	April 12, Home IX c.c. of blood. Parasites in peripheral blood.	May 12 No parasites in peripheral blood.	...	Very even temperature.	May 14 Dismembered.

Animal	Date, Source, and Mode of Inoculation	Time of Appearance of Parasites in Peripheral Blood	Number of Parasites per Field	Type of Temperature Record	Death	Changes in Blood	Substitutions	P.M. Appearance	Remarks
Exp. XX Small pink rat	October 19, Home VI c.c. of blood. Parasites in peripheral blood.	Never became infected.	January 6 Dismembered.
Exp. XX Small pink rat	October 19, Home VI c.c. of blood. Parasites in peripheral blood.	Never became infected.	January 6 Dismembered.
Exp. XX Large white rat	November 1, Home VI c.c. of blood. Parasites in peripheral blood.	November 14 No parasites in peripheral blood.	...	Temperature zero.	August 18 Dismembered.
Exp. XX Small pink rat	November 1, Home VI c.c. of blood. Parasites in peripheral blood.	November 14 No parasites in peripheral blood.	August 18 Dismembered.
Exp. LXIX Black and white rat	January 19, Home VI c.c. of blood. Parasites in peripheral blood.	February 8 No parasites in peripheral blood.	February 24 Dismembered.

Animal	Date, Strain, and Mode of Inoculation	Days of Appearance of Pusicles on the Peritoneal Band	Number of Pusicles which were not peritoneal	Type of Pusicles	Date of Death	Changes in Blood	Histopathology	P.M. Appearance	Remarks
Exp. XXVIII Black rats, ♂	March 14 Inoculated with a suspension of pusicles taken from a rat after death, following the procedure of Latham, Case II (see page 10).	Never before observed.	August 11 1914.
Exp. XXVI Black rats, ♂	March 14 Inoculated with a suspension of pusicles taken from a rat after death, following the procedure of Latham, Case II (see page 10).	November 14 Two pusicles seen on the peritoneal band, one on the right, one on the left, about the same days.	August 11 1914.	..	Exp. XXXIX Exp. XL Exp. XLII Exp. XLIII Exp. XLIV Exp. XLV Exp. XLVI Exp. XLVII Exp. XLVIII Exp. XLIX Exp. L
Exp. XXVII Black rats, ♂	November 15 Inoculated with a suspension of pusicles taken from a rat after death, following the procedure of Latham, Case II (see page 10).	November 14 One pusicle seen on the peritoneal band, on the right, about the same days.	August 11 1914.	Hemoglobin and red cells, nearly none of last blood drawn.	Exp. XXXIII Exp. XXXIV Exp. XXXV Exp. XXXVI Exp. XXXVII Exp. XXXVIII Exp. XXXIX Exp. XL Exp. XLII Exp. XLIII Exp. XLIV Exp. XLV Exp. XLVI Exp. XLVII Exp. XLVIII Exp. XLIX Exp. L	See P.M. report, page 48.	The appearance of pusicles on the peritoneal band was noted after two weeks after death.
Exp. LXXI Tame rat, ♂	January 14, Case V Inoculated with a suspension of pusicles taken from a rat after death, following the procedure of Latham, Case II (see page 10).	Never before observed.	August 11 1914.	See Exp. CVIII.
Exp. CVII White rat, ♂	April 14, Case VI Inoculated with a suspension of pusicles taken from a rat after death, following the procedure of Latham, Case II (see page 10).	Pusicles had not been seen up to time of death, June 1.	See home.	Did not die until before the procedure of Latham, Case II (see page 10).
Exp. CIX Purkin rat	April 15 Inoculated with a suspension of pusicles taken from a rat after death, following the procedure of Latham, Case II (see page 10).	Pusicles have not been seen up to the time of death, August 11.	August 11 1914.	..	Exp. CLXXXIV.	..	Originally XXX.

Animal	Date, Source, and Mode of Inoculation	Date of Appearance of Parasites in the Peripheral Blood	Number of Parasites usually seen and periodicity	Type of Temperature Records	Result Date of Death	Changes in Blood	Subinoculations	P.-M. Appearances	Remarks
Exp. LXXIX Large black rat. ♂	February 5 Guinea-pig. Exp. XLIX Intraperitoneally 0.5 c.c. of blood, three parasites to field. Strain Lammin.	February 13 One to cover. Incubation, eight days.	Present until April 8, with three intervals of absence of five, six, and two days. Never more than one to field. July 21 absent; August 31 absent.	Not taken.	August 31 Still alive.	..	Exp. XCII.
Exp. LXXXI Large white rat. ♀	February 6 Bush rat. Exp. L Intraperitoneally 2 c.c. of blood, four parasites to field. Strain Lammin.	February 20 Incubation, fourteen days.	A few occasionally seen. Never seen after March 30.	Not taken.	April 10 From sunstroke on steamer.	Well nourished. Glands not enlarged. Spleen 3 x 0.8 cm. Lungs and right heart congested. No excess of peritoneal or pleural fluid.	Parasites not transmitted to young. Latter were susceptible to inoculation.
Exp. CII Black rat. ♀	March 27 Rabbit. Exp. XLII (P.-M.) Subcutaneously to c.c. of blood. Strain Lammin.	Never became infected.	August 31 Still alive.
Exp. CV Small piebald rat. ♀	April 3 Guinea-pig. Exp. XLIX (P.-M.) Subcutaneously heart blood. Strain Lammin.	Never became infected.	August 17 Duration, 136 days.	Spleen 3 x 0.5 x 0.3 cm. Bronchial and submaxillary glands enlarged. Lungs full of caseous material.	Previously inoculated as Exp. XX (P) from Mr. Q., but not infected.
Exp. CXLIX Bush rat. ♀	August 4 Rats. Exps. CXY, CXVI Intraperitoneally 1 c.c. of blood, two parasites to field. Strain Gunjur.	August 7 One to cover. Incubation, three days.	Present continuously, except on August 17. Very numerous, August 21.	..	August 26 Duration, twenty-two days.	Commencing decomposition. Spleen and glands not enlarged. Lungs healthy. Heart blood showed twenty to thirty active parasites to field.	..
Exp. CL Bush rat. ♂	August 4 Rats. Exps. CXY, CXVI Intraperitoneally 1 c.c. of blood, two parasites to field. Strain Gunjur.	August 8 Three to field. Incubation, four days.	Numerous until August 21. Never seen since.	..	August 31 Still alive.	..	Exp. CLXI Exp. CLXII	..	Bred from either rat LX or rat LXI.
Exp. XCV (A) Large brown rat. ♀	March 17 Horse. Exp. LXXXVII Intraperitoneally 0.5 c.c. of blood, two parasites to field. Strain Mr. Q.	March 25 Twelve to cover. Incubation, eight days.	Present for seven days. Subsequently present for a few days and absent for a few days alternately. Never more than one to field. August 31, one to cover.	Not taken.	August 31 Still alive.
Exp. XCV (B) Large brown rat. ♀	March 17 As in Exp. XCV (A).	..	Present April 21, 22, 29. Very few seen.	Not taken.	Lost on shipment home.

INOCULATIONS IN RATS (continued)

THIRD PASSAGE

IIIb

Animal	Date, Source, and Mode of Inoculation	Date of Appearance of Parasites in the Peripheral Blood	Number of Parasites usually seen and Periodicity	Type of Temperature Records	Result Date of Death	Changes in Blood	Subinoculations	P.-M. Appearances	Remarks
Exp. CXL White rat. ♀	July 30 Horse. Exp. LXXXVII Intraperitoneally 2.5 c.c. of blood. Strain Mr. Q.	August 14 Two to cover. Incubation, fifteen days.	Never seen since August 14.	..	August 31 Still alive.
Exp. CXLI White rat. ♂	July 30 Horse. Exp. LXXXVII Intraperitoneally 2.5 c.c. of blood. Strain Mr. Q.	August 14 One to cover. Incubation, fifteen days.	September 1, one to cover.	..	August 31 Still alive.

FOURTH PASSAGE

Exp. CXXIII Brown rat. ♂	July 14 Rat. Exp. LX Intraperitoneally 0.5 c.c. of blood, no parasites seen. Strain Lammén.	None seen.	July 21 Died.	Nothing abnormal. No parasites in blood. Spleen 3.5 X 0.7 X 0.3 cm.	This rat was born of a rat infected with human trypanosome.
Exp. CXXIV Brown rat. ♂	July 14 Rat. Exp. LX Intraperitoneally 0.2 c.c. of blood, no parasites seen. Strain Lammén.	August 4 Blood centrifuged. Few seen.	August 31 Still alive.
Exp. CLXI Piebald rat. ♂	August 10 Rat. Exp. CL Intraperitoneally 1 c.c. of blood, one parasite to field. Strain Gunjur.	August 15 Two to cover. Incubation, five days.	August 31 Still alive.
Exp. CLXII Piebald rat. ♀	Aug. 10 Rat. Exp. CL Intraperitoneally 1 c.c. of blood, one parasite to field. Strain Gunjur.	August 15 One to six fields. Incubation, five days.	August 31 Still alive.

EQUINE TRYPANOSOME

IV

INOCULATIONS IN MICE

SECOND PASSAGE

Animal	Date, Source, and Mode of Inoculation	Date of Appearance of Peripheral Blood	Number of Parasites in Peripheral Blood	Type of Temperature Record	Remarks	Changes in Blood	Subinoculations	P. M. Appearance	Remarks
Exp. CLVII Black and white mouse	August 11. Rec. Exp. CLVII (100000) inoculated in 0.5 cc of blood from mouse VI.	August 11. Parasites first seen in peripheral blood.	August 11. Parasites first seen in peripheral blood.	August 14. Lethal.
Exp. CLVIII Black and white mouse	August 11. Rec. Exp. CLVII (100000) inoculated in 0.5 cc of blood from mouse VI.	August 11. Parasites first seen in peripheral blood.	August 11. Parasites first seen in peripheral blood.	August 14. Animal dead.
Exp. CLIX Black and white mouse	August 11. Rec. Exp. CLVII (100000) inoculated in 0.5 cc of blood from mouse VI.	August 11. Parasites first seen in peripheral blood.	August 11. Parasites first seen in peripheral blood.	August 14. Animal dead.
Exp. CLX Black and white mouse	August 11. Rec. Exp. CLVII (100000) inoculated in 0.5 cc of blood from mouse VI.	August 11. Parasites first seen in peripheral blood.	August 11. Parasites first seen in peripheral blood.	August 14. Animal dead.

INOCULATIONS IN WILD MICE

THIRD PASSAGE

Animal	Date, Source, and Mode of Inoculation	Date of Appearance of Peripheral Blood	Number of Parasites in Peripheral Blood	Type of Temperature Record	Remarks	Changes in Blood	Subinoculations	P. M. Appearance	Remarks
Exp. LXXXIV Black mouse	January 21. Rec. Exp. LXVII (100000) inoculated in 0.5 cc of blood from mouse VI.	January 21. Parasites first seen in peripheral blood.	January 21. Parasites first seen in peripheral blood.	Parasites first seen in peripheral blood.

SIXTH PASSAGE

Animal	Date, Source, and Mode of Inoculation	Date of Appearance of Peripheral Blood	Number of Parasites in Peripheral Blood	Type of Temperature Record	Remarks	Changes in Blood	Subinoculations	P. M. Appearance	Remarks
Exp. XCIV Black and white mouse	Rec. Exp. LXXXIV (100000) inoculated in 0.5 cc of blood from mouse VI.	Rec. Exp. LXXXIV (100000) inoculated in 0.5 cc of blood from mouse VI.	Rec. Exp. LXXXIV (100000) inoculated in 0.5 cc of blood from mouse VI.	As the majority of these mice die before the parasites are first seen in peripheral blood, the results are not recorded.

HUMAN TRYPANOSOME

INOCULATIONS IN MICE

SECOND PASSAGE

Animal	Date, Source, and Mode of Inoculation	Date of Appearance of Peripheral Blood	Number of Parasites in Peripheral Blood	Type of Temperature Record	Remarks	Changes in Blood	Subinoculations	P. M. Appearance	Remarks
Exp. CXXX Black and white mouse	July 14. Rec. Exp. XXVII (100000) inoculated in 0.5 cc of blood from mouse VI.	July 14. Parasites first seen in peripheral blood.	July 14. Parasites first seen in peripheral blood.	Wright, July 22, 23. Parasites first seen in peripheral blood.
Exp. CXXXI Black and white mouse	July 14. Rec. Exp. XXVII (100000) inoculated in 0.5 cc of blood from mouse VI.	July 14. Parasites first seen in peripheral blood.	July 14. Parasites first seen in peripheral blood.	Wright, July 22, 23. Parasites first seen in peripheral blood.
Exp. CXXXII Black and white mouse	July 14. Rec. Exp. XXVII (100000) inoculated in 0.5 cc of blood from mouse VI.	July 14. Parasites first seen in peripheral blood.	July 14. Parasites first seen in peripheral blood.	Wright, July 22, 23. Parasites first seen in peripheral blood.

INOCULATIONS IN MICE

THIRD PASSAGE

Animal	Date, Source, and Mode of Inoculation	Date of Appearance of Peripheral Blood	Number of Parasites in Peripheral Blood	Type of Temperature Record	Remarks	Changes in Blood	Subinoculations	P. M. Appearance	Remarks
Exp. CXXXV Black and white mouse	July 14. Rec. Exp. CXXXI (100000) inoculated in 0.5 cc of blood from mouse VI.	July 14. Parasites first seen in peripheral blood.	July 14. Parasites first seen in peripheral blood.	Wright, July 22, 23. Parasites first seen in peripheral blood.
Exp. CXXXVI Black and white mouse	July 14. Rec. Exp. CXXXI (100000) inoculated in 0.5 cc of blood from mouse VI.	July 14. Parasites first seen in peripheral blood.	July 14. Parasites first seen in peripheral blood.	Wright, July 22, 23. Parasites first seen in peripheral blood.

Animal	Date, Source, and Mode of Inoculation	Date of Appearance of Parasites in the Periplasmic Blood	Number of Parasites usually seen and periodicity	Type of Temperature Records	Result Date of Death	Changes in Blood	Subinoculations	P.-M. Appearances	Remarks
Exp. CXIX Exp. CXX Exp. CXXI Exp. CXXII White mice.	July 6. Goat. Exp. CXI (P.-M.) Intraperitoneally with 0.5 c.c. of blood. Strain Lamm.	Never infected.
Exp. CXXV White mouse.	July 14 Rat. Exp. LX Intraperitoneally with 0.2 c.c. of blood, showing no parasites. Strain Lamm.	August 3 Two parasites to preparation. Incubation, twenty days.	Parasites never observed again.	August 15 Duration, thirty-two days.	Body too decomposed.	..
Exp. CXXVI Black and white mouse.	July 14 As in Exp. CXXV.	August 21 Two parasites to stained preparation.	Parasites never observed again.	August 31 Duration, forty-eight days.	Body too decomposed.	..

INOCULATIONS IN WILD MICE

THIRD PASSAGE

Exp. LXXIII Wild house-mice from McCarthy Island. Four mice. Species?	January 10 Puppy. Exp. LVII Intraperitoneally each received 0.5 c.c. of blood, containing four trypanosomes to preparation. Strain Gunjur.	February 5 Two mice showed parasites. Incubation, six days.	Blood of these mice examined every other day through February and half of March. At most twelve parasites to field. Parasites were generally found to be present in the peripheral blood for four to six days, and then absent for two to four days.	..	One mouse died on March 10, another on the 12th, and the other two were killed by accident about a week later.	Autopsy immediately after death on mouse dead on April 12. No fluid in serous cavities, slight patchy congestion of lungs. Heart full of blood, showing four trypano- covers. Spleen congested, 1.7 x 0.5 x 0.2 cm. Liver congested, a hair-ball found in stomach. Intestines normal; abdominal glands and also mesenteric glands, slightly enlarged, not congested. Axillary and inguinal glands could not be made out.	Death probably hastened by injuries received in cage.
Exp. LXXXII Wild house-mice from McCarthy Island. Three mice. Species?	February 6 Bush rat. Exp. L Intraperitoneally with about 0.4 c.c. of blood, showing four parasites to field. Strain Lamm.	February 8 One mouse showed a few parasites to preparation. Incubation.	Parasites never very numerous in the blood, at the most five to a field. They never were constantly present in the blood; after disappearance two to four days would elapse before they were seen again. Observations were made up to April 28.	..	One mouse died in a week after inoculation, probably from injury; the other two on the passage home.	It was difficult to keep these house-mice in health in captivity. NOTE.—In two of the mice, on rare occasions, a flagellate organism was seen in the blood, having a long flagellum at one end.

INOCULATIONS IN WILD MICE

FOURTH PASSAGE

Exp. XCII McCarthy Island bush mice. Six mice. Species?	March 2 Rat. Exp. LXXXIX Intraperitoneally each received 0.5 c.c. of blood, containing one trypanosome to cover. Incubation. Strain Lamm.	March 4 Three mice had one parasite to cover; three with four parasites to cover. Incubation, two days.	Periodicity of parasites was noticed as in the wild house-mice up to April 28. Parasites never numerous in the peripheral blood.	..	Two mice died while away in bush; one on voyage home; one soon after arrival; one still alive, August 11.	No P.-M.	Mice difficult to keep healthy in captivity.
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EQUINE TRYPANOSOME

INOCULATIONS IN GUINEA-PIGS

FIRST PASSAGE

V

Animal	Date, Source, and Mode of Inoculation	Date of Appearance of Parasites in the Peripheral Blood	Number of Parasites usually seen and periodicity	Type of Temperature Records	Result Date of Death	Changes in Blood	Subinoculations	P.-M. Appearances	Remarks
Exp. XIII Guinea-pig. Weight 486 gm.	October 17 Horse 1 Intraperitoneally 10 c.c. of blood from vena cava at P.-M. No parasites seen.	October 25 Very few seen. Incubation, eight days.	Fairly constantly. Very numerous parasites towards death. 88,000 per c.mm.	Irregular. 102° to 105° F.	November 15 Died. Duration, twenty-nine days.	No diminution of red cells.	..	Some clear fluid in abdomen. No peritonitis. Spleen dark. Glands not enlarged. Slight parietal peritoneal ecchymosis opposite spleen.	Convulsions before death. Lardosis at death.

SECOND PASSAGE

Exp. XXII Guinea-pig.	October 28 Rat. Exp. IX (B) Intraperitoneally 0.5 c.c. of blood. Numerous parasites seen. Strain Horse I.	November 1 Ten parasites. Incubation, four days.	Parasites fairly constantly present. Varied between 25,000 and 50,000 per c.mm.	Persistently elevated. 103° to 104° F.	November 28 Died. Duration, thirty-one days.	No diminution of red cells.	Exp. XXXIII. Exp. XXXIV. Exp. XXXV.	Performed immediately after death. Old haematoma under skin over sternum, another near left inguinal glands. Spleen slightly enlarged, anterior end ruptured, blood in peritoneal cavity. Liver pale. Other organs healthy. Glands enlarged. Abdominal and mesenteric glands haemorrhagic.	Animal died after handling while taking blood.
Exp. CXIII Guinea-pig. ♂	May 14 Rat. Exp. CXII Intraperitoneally one drop of blood. Strain Horse IX.	Never became infected.	August 31 Still alive.	

HUMAN TRYPANOSOME

INOCULATIONS IN GUINEA-PIGS

SECOND PASSAGE

Animal	Date, Source, and Mode of Inoculation	Date of Appearance of Parasites in the Peripheral Blood	Number of Parasites usually seen and periodicity	Type of Temperature Records	Result Date of Death	Changes in Blood	Subinoculations	P.-M. Appearances	Remarks
Exp. XLIX Guinea-pig. Weight 453 gm.	December 15 Rat. Exp. XXVI Intraperitoneally 0.5 c.c. of blood. One parasite seen. Strain Lammim.	January 5 Blood centrifuged, a few seen. Incubation, twenty-one days.	January 21, one parasite. January 22 fairly numerous, almost constantly present to death. Numbers varied.	Irregular. 102° to 104° F.	April 5 Died. Duration, 111 days.	Nothing marked.	Exp. XLII. Exp. LXXVI. Exp. LXXXIII. Exp. LXXXIX. Exp. XCIX. Exp. CV.	See P.-M. report, page 47.	Became thin week before death.
Exp. CXIV Guinea-pig. ♀	May 11 Rat. Exp. XXVII Intraperitoneally 0.5 c.c. of blood. Strain Gunjar.	May 25 One parasite. Incubation, twelve days.	May 27, Four parasites present. Absent until August 31. Two to a field seen.	..	August 31 Still alive.
Exp. CXXXIII Guinea-pig.	July 20 Rat. Exp. XXVII Intraperitoneally 1.5 c.c. of blood. Many parasites seen. Strain Gunjar.	August 31 Blood centrifuged. Two parasites.	August 31 Still alive.	Weight 695 gm., July 21. Weight 857 gm., August 14.

INOCULATION IN RABBIT

FIRST PASSAGE

Animal	Date, Source, and Mode of Inoculation	Date of Appearance of Parasites in the Peripheral Blood	Number of Parasites usually seen and periodicity	Type of Temperature Records	Result Date of Death	Changes in Blood	Subinoculations	P.-M. Appearances	Remarks
Exp. IX White rabbit, ♀	October 12 Horse I Intraperitoneally. Numerous parasites in blood.	October 25 Numerous. Incubation, thirteen days.	Generally present and fairly numerous (2,000 to 5,000 c.mm.)	October 18, rose; October 19, 106°-80° F.; October 25, 105°-90° F. Remained high; November 20, 107° F.; November 23, 107° F.	November 24 Duration, fifty-three days.	Decomposition commencing. Testicles not swollen. No discharge from mucous membranes nor eyes. Spleen not enlarged, dark, diffident. Glands not enlarged. Peritoneum normal. Some fluid in pericardium. Heart muscle pale, flabby. Lungs congested, oedematous. Brain slightly congested.	Weight, October 12, 2,000 gms.; November 24, 1613½ gms.

HUMAN TRYPANOSOME

INOCULATIONS IN RABBITS

FIRST PASSAGE

Animal	Date, Source, and Mode of Inoculation	Date of Appearance of Parasites in the Peripheral Blood	Number of Parasites usually seen and periodicity	Type of Temperature Records	Result Date of Death	Changes in Blood	Subinoculations	P.-M. Appearances	Remarks
Exp. CVII Black rabbit, ♀	April 15 Boy at Farotenda. Case VI Intraperitoneally 1½ c.c. of blood.	Never became infected.	August 31 Still alive.

SECOND PASSAGE

Exp. LII Black rabbit, ♂	December 4 Rat. Exp. XXVI Intraperitoneally 1 c.c. of blood. Twelve parasites to cover.	Never became infected.	..	Steady, 103°-104° F. until January 9. Subsequently septic in type.	March 27	..	Exp. CII	Chronic abscess in spleen. Perisplenitis.	Suffered from suppurative orchitis for three weeks in February.
Reinoculation	February 5 Guinea-pig. Exp. XLIX 0.5 c.c. of blood. Two parasites to field.								
Reinoculation	March 3 Rat. Exp. XXVI Intraperitoneally 1.5 c.c. of blood. Eight parasites to cover. Strain Lammin.								
Exp. LI White and grey rabbit ♀	December 22 Rat. Exp. XXVI Intraperitoneally 0.5 c.c. of blood. One parasite to field. Strain Lammin.	January 12 Two to cover. Incubation, twenty-one days.	Present rarely up to Jan. 31. Present again April 27 and July 21.	Slightly raised when parasites present.	August 31 Still alive.	None.	Weight— December 22, 1587 gms March 31, 2040.6 gms.

EQUINE TRYPANOSOME

THIRD PASSAGE

Animal	Date, Source, and Mode of Inoculation	Date of Appearance of Parasites in the Peripheral Blood	Number of Parasites usually seen and periodicity	Type of Temperature Records	Result Date of Death	Changes in Blood	Subinoculations	P.-M. Appearances	Remarks
Exp. XXIII Small mangrove monkey. <i>Aryathrix</i> .	December 10 Rat. Exp. XVIII Subcutaneously 0.5 c.c. blood, 34,000 parasites to c.mm. Strain Horse I.	December 14 Four to cover. Incubation, four days.	Escaped during night, December 14.
Exp. LXIII Small baboon. <i>Cynopithecus sphinx</i> , ♂	January 10 Rat. Exp. LIV Subcutaneously 0.5 c.c. blood, 265,000 parasites to c.mm. Strain Horse V.	Not seen up to time of death.	January 24 Killed when on point of death.	..	Exp. LXXII.	Lungs, heart, and spleen normal. Liver showed marked fatty degeneration. Stomach contained altered blood, mucous membrane much congested.	Monkey ill three days before death, probably poisoned. Two rats inoculated with 3.5 c.c. heart blood directly after death; never infected.
Exp. LXXV Half-grown baboon. <i>Cynopithecus sphinx</i> , ♂	January 31 Rat. Exp. LXVII Intraperitoneally 0.5 c.c. blood, numerous parasites. Strain Horse I.	Never became infected.	February 25 From heatstroke.	No macroscopical lesions. Right heart gorged with blood.	Rectal temperature before death, 110° F.

SIXTH PASSAGE

Exp. XCIII Small baboon. <i>Cynopithecus sphinx</i> , ♀	March 3 Rat. Exp. LXXVII Intraperitoneally 0.5 c.c. blood, very numerous parasites. Strain Horse I.	Never became infected.	August 31 Still alive.	Formerly LXXVI (human trypanosome) q.v. Rat inoculated 3 c.c. blood, April 6; not infected.
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HUMAN TRYPANOSOME

INOCULATIONS IN MONKEYS

THIRD PASSAGE

Animal	Date, Source, and Mode of Inoculation	Date of Appearance of Parasites in the Peripheral Blood	Number of parasites usually seen and periodicity	Type of Temperature Records	Result Date of Death	Changes in Blood	Subinoculations	P.-M. Appearances	Remarks
Exp. XLIV Young baboon, <i>Cyncephalus sphinx</i> , ♀	December 8 Rat. Exp. XXXIX Subcutaneously 1 c.c. of blood. Nine parasites to field. Strain Lammin	Never became infected.	January 7
Exp. LXII Young baboon, <i>Cyncephalus sphinx</i> , ♀	January 10 Rat. Exp. XXXIX Subcutaneously 0.5 c.c. of blood. Numerous parasites. Strain Lammin.	Never became infected.	February 22 From sunstroke.
Exp. LXXVI Young baboon, <i>Cyncephalus sphinx</i> , ♂	January 31 Guinea-pig. Exp. XLIX Intraperitoneally 0.8 c.c. of blood. Numerous parasites. February 18 Same animal 1 c.c. of blood. Four parasites to field. Strain Lammin.	Never became infected.	August 31 Still alive.	March 3 Inoculated with horse parasite (Exp. XCIII) q.v.
Reinoculation									
Exp. XCIX Young baboon, <i>Cyncephalus sphinx</i> , ♂	March 24 Guinea-pig. Exp. XLIX Intraperitoneally 0.5 c.c. of blood. Numerous parasites. Strain Lammin.	Never became infected.	August 31 Still alive.

INOCULATION IN CHIMPANZEE

THIRD PASSAGE

Chimpanzee. ♀	July 24 Rats. Exps. CXV, CXVI Subcutaneously 2.5 c.c. of blood. Two parasites to field. Strain Ganjur.	August 3 One parasite seen and one <i>Filaria peritana</i> .	Present only occasionally and in small numbers.	Two periods of fever (103°-104° F.) a week apart in first fortnight when parasites present. Curve since then irregular. See Chart.	August 31 Still alive.	No decrease in red cells and Hb. No increase of white cells.*
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* DIFFERENTIAL LEUCOCYTE COUNT

	August 3	August 18
Neutrophiles
Eosinophiles
Small mononuclear
Large mononuclear
Transitionals
	50 per cent.	29 per cent.
	7 per cent.	7 per cent.
	27 per cent.	32 per cent.
	0.5 per cent.	0 per cent.
	10.5 per cent.	20 per cent.
	1 per cent.	6 per cent.
	4 per cent.	6 per cent.

EQUINE TRYPANOSOME

INOCULATIONS IN DOGS

FIRST PASSAGE

Animal	Date, Source, and Mode of Inoculation	Date of Appearance of Parasites in the Peripheral Blood	Number of Parasites usually seen and periodicity	Type of Temperature Records	Result Date of Death	Changes in Blood	Subinoculations	P.-M. Appearances	Remarks
Exp. XI Adult native bitch.	October 16 Horse I Intraperitoneally 3 c.c. of blood. One parasite seen.	October 25 A few seen. Incubation, nine days.	Numbers increased to October 31. Never numerous. Up to February very scanty, only occasionally seen. Absent up to date.	With appearance of parasites rose to 104° F. Rise to 102° F. coincided with each reappearance of parasites.	August 31 Still alive.	..	Exp. LXV. Exp. LXVI. Exp. CLIII.	..	No symptoms. Is strong and well. Rats, Exps. LXV, LXVI, became infected.
Exp. XII Native puppy, three weeks old.	October 16 Horse I Intraperitoneally 2 c.c. of blood. One parasite seen.	October 24 Numerous parasites. Incubation, eight days.	Parasites never absent, gradually increased up to death. November 15, 25,000 parasites per c.mm.	Remittent after parasites appeared.	November 17 Died. Duration, thirty-two days.	..	Exp. XXIV.	Subcutaneous tissue of abdomen and thorax markedly oedematous. Small abscesses of liver. Recent pericarditis, marked over heart. Spleen dark, diffuse. All abdominal glands enlarged. Mesenteric and omental, brown coloured, gelatinous. Chains of glands along aorta and iliac arteries ditto.	Bush rat, Exp. XXIV, infected.
Exp. XXX Native dog, half-grown.	November 28 Horse V Intraperitoneally 3 c.c. of blood. Few parasites seen.	December 9 Three parasites. Incubation, eleven days.	Never numerous, about three to six to preparation. For a week before death about 13,000 parasites per c.mm.	Remittent from three days before parasites appeared till death. Twice 102° F., usually 103° to 104° F.	January 3 Died. Duration, thirty-six days.	Six hours after death. Mucous membranes normal. About 280 c.c. straw-coloured fluid in abdomen. Spleen, 141 gm., congested, friable. Liver, 567 gm., congested. Duodenum and small bowel congested; latter contained many ankylostomata and taenias. Glands enlarged and oedematous; mesenteric and lumbar chocolate coloured; cervical haemorrhagic. Slight pericarditis, fluid in pericard. Fatty degeneration of heart. Patchy congestion of lungs. Brain slightly congested. Bone marrow, dark red.	..

SECOND PASSAGE

Exp. XVII Native puppy.	October 22 Rat. Exp. IX Intraperitoneally 1 c.c. of blood. Many parasites seen. Strain Horse I.	October 25 A few seen. Incubation, three days.	Never absent, numbers gradually increased up to death.	Remittent after appearance of parasites. Sudden fall from 100° to 98° F. before death.	November 10 Died. Duration, nineteen days.	Clear serous fluid in abdomen. Spleen few small recent superficial infarcts, otherwise normal. Peribronchial glands not enlarged, all other glands enlarged, oedematous. Medulla of all dark-brown. One mesenteric markedly haemorrhagic. Pericardium distended with blood-stained fluid. Some fluid in pleurae.	..
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EQUINE TRYPANOSOME

INOCULATIONS IN GOATS

FIRST PASSAGE

IX

Animal	Date, Source, and Mode of Inoculation	Date of Appearance of Parasites in the Periplasmal Blood	Number of Parasites usually seen and periodicity	Type of Temperature Records	Result Date of Death	Changes in Blood	Subinoculations	P.M. Appearances	Remarks
Exp. X Brown gelded goat about one year old.	October 15 Horse I. Intraperitoneally 5 c.c. of blood. Numerous parasites seen.	October 28 One parasite to coverslip. Incubation, three days.	Up to October 31. Parasites rarely seen and only in small numbers. Absent since October 31.	Rose on appearance of parasites, 105.2° F.; afterwards very irregular. Occasional pyrexia for one to two days, 106° F.	August 31 Still alive.	...	Exp. LXXVIII, Exp. LXXVIII, Exp. XCVI.	...	No signs of illness. Rat, Exp. XCVI, inoculated five months after appearance of parasites, became infected, as also Rat, Exp. LXVII.

THIRD PASSAGE

Exp. XXXV Grey goat, about one year old, ♂	November 28 Guinea-pig. Exp. XXIII Intraperitoneally blood and spleen juice. Many parasites seen. Strain Horse I.	December 1 One parasite to coverslip. Incubation, four days.	Present with intermissions for three and a half weeks after inoculation. Never numerous.	After first two months occasional pyrexia for one to two days.	August 31 Still alive.	...	Exp. XCVII.	...	Inoculated at P.M. of Guinea-pig, XXII. Rat, Exp. XCVII, became infected.
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HUMAN TRYPANOSOME

INOCULATIONS IN GOATS

SECOND PASSAGE

Animal	Date, Source, and Mode of Inoculation	Date of appearance of Parasites in the Peripheral Blood	Number of Parasites usually seen and periodicity	Type of Temperature Records	Result Date of Death	Changes in Blood	Subinoculations	P.-M. Appearances	Remarks
Exp. CXLIV Small goat, ♀	July 31 Rat, Exp. XXXVII Intraperitoneally 3 ² / ₅ c.c. of diluted blood. Ten parasites to field. Strain Gunjur.	Never became infected.	August 31 Still alive.	...	Exp. CLXXXV.

THIRD PASSAGE

Exp. XLVI Kid, about three months old.	December 8 Rat, Exp. XXXIX Intraperitoneally 0.5 c.c. of blood. Nine parasites seen. Strain Lammin.	January 7 Eight parasites seen in centrifuged blood. Up to De- cember 31 daily ex- aminations reveal- ed no parasites.	Parasites very scarce, only occasionally seen. Absent from February 26.	Rise of temperature to 106° F, usually coin- cided with appearance of parasites. March 29, 106° to 107° F.	April 12 Died. Duration, 125 days.	No obvious change.	...	No, P.-M., carcass too decom- posed.	Died while away. In- creased in weight for first two months. From March 29 became ema- ciated.
Exp. CXI Adult goat, ♀	April 25 Rat, Exp. XXXIX Subcutaneously 1 c.c. of blood. One parasite to three fields. Strain Lammin.	Absent up to April 29.	May (about third week) Died.	...	Exp. CXIX. Exp. CXX. Exp. CXXI. Exp. CXXII. At P.-M.	Spleen 60 gms. Organs nor- mal. Glands enlarged. Glands along oesophagus and root of neck soft with chocolate coloured centres; some small ones dark brown. In mesentery few small completely haemorrhagic glands, size of peas. Along iliac arteries large glands either haemorrhagic or chocolate col- oured. Brain slightly conges- ted.	Animal inoculated just before being sent to England. Died soon af- ter landing in Liverpool.
Exp. CX Adult goat, ♂	April 25 Horse, Exp. LXXXVII Subcutaneously 4 c.c. of blood. Two para- sites to field. Strain Mr. Q.	July 7 5 c.c. of blood centrifuged. Fair number seen.	August 31 Still alive.	...	Exp. CLXXXVI	...	Goat appeared to be thinner on July 7.

EQUINE TRYPANOSOME

INOCULATIONS IN BOVINES

SECOND PASSAGE

Animal	Date, Source, and Mode of Inoculation	Date of Appearance of Parasites in the Peripheral Blood	Number of Parasites usually seen and periodicity	Type of Temperature Records	Result Date of Death	Changes in Blood	Subinoculations	P.-M. Appearances	Remarks
Exp. XX Small native calf, ♂. Six months old.	October 28 Rat, Exp. IX 0.5 c.c. of blood. Numerous parasites seen. Strain Horse I.	November 6 Parasites seen after centrifuging blood. Incubation, nine days.	Very few	October 30, rose to 105° F.; fell next day. November 4 to 6, elevated. Again on November 8. Slight rise November 15.	November 17 Died. Duration twenty days.	...	Exp. XXV.	See P.-M. Report, page 48.	Very thin before death.

FOURTH PASSAGE

Exp. XLVIII Adult native bull, lock.	December 24 Rat, Exp. XXXVIII Subcutaneously 0.5 c.c. of blood. Many parasites seen. Strain Horse I.	January 5 Very few after centrifuging. Incubation, twelve days.	Very few. Usually 0.5 c.c. of blood contained only three parasites.	Up to January 22 never above 102° F.; January 22, 105° F., for two days.	February 2 Died. Duration forty days.	...	Exp. LXXVII.	See P.-M. Report, page 49.	Became thin towards end. No other symptoms observed.
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INOCULATIONS IN NATIVE SHEEP

THIRD PASSAGE

Exp. XXXVI Native sheep, ♂. Six months old.	November 30 Rat, Exp. XXVIII Intraperitoneally 0.5 c.c. of blood containing 6000 parasites per c.mm. Strain Horse I.	December 8 Eight parasites. Incubation, eight days.	Fairly numerous on Dec. 10, 11, 12. Few but constantly present to Jan. 7. Absent up to end of April.	Sharp initial rise to 107.5° F., coinciding with appearance of parasites. Later, very irregular; occasional pyrexia, 105° to 106° F.	May (third week) Died. Duration over 182 days.	Too decomposed.	Animal shipped to England, died on way. Up to end of April no symptoms observed.
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HUMAN TRYPANOSOME

INOCULATION IN HORSE

SECOND PASSAGE

Animal	Date, Source, and Mode of Inoculation	Date of Appearance of Parasites in the Peripheral Blood	Number of Parasites usually seen and periodicity	Type of Temperature Records	Result Date of Death	Changes in Blood	Subinoculations	P.-M. Appearances	Remarks
Exp. LXXXVII Young bay stallion.	February 14. Rat, Exp. LXIX. Subcutaneously in the cellular tissue at base of left ear with 0.5 c.c. of blood, showing four trypanosomes to field. <i>Note.</i> —Horse's blood previously examined by centrifuge and by inoculation into two rats which have never become infected. Strain Mr. Q.	Parasites were not seen up to March 4. After our return from Maka on March 16, blood showed one parasite to field. Infection between March 4 and March 16.	Parasites seen fairly constantly from March 16 up to the time horse was shipped home first week in May. They were never numerous. Absence of parasites sent from July to Aug. 31.	See Chart.	August 31 Still alive.	...	Exp. XCV (A), Exp. XCV (B), Exp. CX, Exp. CXL, Exp. CXXI.	...	On return from Maka horse looked unwell, it hung its head. There was noticed a slight opacity on left cornea and a watery discharge from both eyes with slight injection of conjunctival vessels. The opacity of cornea cleared up in about three weeks. Horse was decidedly thinner by the end of March, ribs showing slightly. No oedema ever seen. The animal improved on the voyage home; it is now in fine condition, though its blood is infective to rats.

EQUINE TRYPANOSOME

INOCULATIONS IN FOWLS

XI

Animal	Date, Source, and Mode of Inoculation	Date of Appearance of Parasites in the Peripheral Blood	Result Date of Death	Animal	Date, Source, and Mode of Inoculation	Date of Appearance of Parasites in the Peripheral Blood	Result Date of Death
Exp. XXI White hen.	October 28 Rat, Exp. IX Intraperitoneally 1 c.c. of blood containing numerous parasites. December 16 Rat, Exp. XXXVIII 1 c.c. of blood showing many parasites. February 17 Rat, Exp. LXVII 1 c.c. of blood containing numerous parasites. Strain Horse I.	Never became infected.	August 31 Still alive.	Exp. XXXIV Black hen.	October 28 Rat, Exp. IX Intraperitoneally 0.5 c.c. of blood rich in parasites. December 16 Rat, Exp. XXXVIII With 1 c.c. of blood. Strain Horse I.	Never became infected.	January Accidentally killed.
Reinoculation				Reinoculation			
Reinoculation							

Note by Dr. H. E. Annett

THE RESULTS OF INOCULATION OF BLOOD FROM THE EUROPEAN CASE, H.K.

(*vide p. 9*)

Tame Rats.—White, black and white. Injected intraperitoneally with about 0·2 c.c. peripheral blood diluted with normal saline solution; in some twenty-five per cent. only was it possible to demonstrate parasites. The parasites first appeared in the peripheral blood in from fifteen to twenty-five days after the inoculation, and their presence continued for three or four days, apparently without any significant rise in temperature. The animals lived for periods varying from two to four months or longer, and occasionally only during this time could parasites be found. Usually on *post-mortem* examination they also seemed to be absent, in occasional instances only were they present in the heart's blood.

Tame Mice.—White, brown, black, etc. Parasites were never seen after injection.

Rabbits and Guinea-Pigs.—Seemed to be refractory.

Monkeys (Rhesus).—Seem to be all susceptible to intraperitoneal infection. Parasites can be demonstrated in the peripheral blood only after the twelfth day after infection, their first appearance being preceded by a rapid rise in temperature, followed by a more gradual fall during the next three days. The number of parasites per cover-glass preparation varied during this period from eight to ten in number, occasionally more, and then gradually disappeared. A similar phenomenon occurred at intervals of five to ten days, the animal gradually losing weight until death occurred in from six weeks to three months. In one case, *post-mortem*, numerous parasites were seen, but in others none were found.

Monkeys were inoculated at different periods during the time which K. spent in England, including two inoculations with blood two days before death, one of which died after three months' infection; the other shewed few parasites at times, and appeared very ill and emaciated on two occasions, but eventually recovered, and now, after a period of nine months, is healthy, and no parasites can be found in blood, either microscopically or by inoculation into rats.

In monkeys no such symptoms as are described in human cases occur, beyond the occasional rises in temperature (during which parasites are present in peripheral blood) and the emaciation.

NOTES ON POST-MORTEMS OF EXPERIMENTAL ANIMALS

Experiment XLIX—

POST-MORTEM OF GUINEA-PIG

Autopsy.—Died during the night. Body emaciated, some slight yellow-coloured fluid in pleural and peritoneal cavities, slightly turbid in the latter.

Heart.—Normal; right side contained a dark blood clot.

Lungs.—Showed patchy congestion marked in right lower lobe, otherwise normal.

Liver.—Large, and slightly congested.

Spleen.—3·5 x 1·5 x 0·4 cm. Slightly injected.

Stomach.—Some congestion along greater curvature.

The *duodenum* for about 3 cm. was much thickened and deeply congested; about 1 cm. from pylorus it was adherent to liver and a coil of small intestine. In this situation three ulcers were found, one large, with a sloughy base; this had almost perforated. In small gut Peyers patches were slightly thickened and inflamed.

Kidneys.—Slightly injected.

Lymphatic glands.—On both sides the superficial and deep inguinal glands, though only slightly enlarged, were crimson in colour; the smaller ones appeared like rounded bits of blood clot; in the larger ones, the cortex in places was pale. Similar glands were present in axilla and in neck, but were not so haemorrhagic. Mesenteric, lumbar, and pelvic glands were slightly enlarged, some smaller ones haemorrhagic. The larger ones were deeply injected in places. Bronchial and sternal glands were slightly enlarged and injected. No parasites seen in the blood from heart or lungs.

Experiment XXVII—

POST-MORTEM OF RAT.

Rat died during the night. The body was emaciated and covered with lice. Incisor teeth were absent from the upper jaw (decay). No fluid in pleural and peritoneal cavities.

Lungs.—Slightly congested, but perfectly healthy.

Heart.—Normal.

Liver.—Pale, studded with small white punctate spots.

Spleen.—Enlarged and congested, 6.5 × 1.7 × 1.0 cm. A small infarct present on outer surface three mm. in diameter, did not extend deeply into tissue.

Kidneys.—Apparently normal.

Lymphatic Glands.—Superficial inguinal and axillary slightly enlarged and congested. Bronchial glands were enlarged and deeply injected.

Abdominal glands small, not injected. Mesenteric slightly enlarged, not injected.

Brain.—Slight injection of pia.

No parasites seen in heart blood at P.-M.

Remarks.—The death of this rat was probably in a great measure due to old age, hastened by the loss of the incisor teeth.

Experiment XX—

POST-MORTEM OF NATIVE CALF

Autopsy.—Very slight subcutaneous oedema in tissues over thorax. Abdomen contained a small amount of slightly bile-tinged fluid.

Liver.—Dark, friable; gall bladder full of dark, viscid bile.

Spleen.—Enlarged; substance soft, congested.

Lymphatic glands.—Mesenteric glands immediately adjacent to small intestine much enlarged. Some the size of a pigeon's egg. On section medulla is dark brown in colour, glands surrounded by a gelatinous, almost colloid, oedema. In great mesentery, and also accompanying the above large glands, were other smaller completely haemorrhagic glands varying in size from a hemp seed to a pea. Some of these were so full of blood as to be diffuent. Iliac and lumbar glands much enlarged and soft, with surrounding oedematous tissue above described. Bronchial and cervical glands were similarly oedematous, many were completely haemorrhagic. Veins of kidneys, mesentery, and heart generally surrounded by yellow gelatinous oedema.

Marrow, of short bones, was normal in appearance; of long bones, yellowish and gelatinous, with here and there decidedly reddened areas.

Heart.—Muscle pale and firm.

Lungs.—Normal.

Experiment XLVIII

POST-MORTEM OF NATIVE BULLOCK*

Performed immediately after death. Some ten unidentified adult filariae were found in conjunctival sacs of both eyes. No sores nor discharge from nostrils. On section no oedema of abdominal wall. Stomachs enormously distended with food; small intestines empty.

Liver.—2·72 kilogrammes. Substance dark. On upper surface of right lobe was a superficial abscess the size of an acorn, which contained a small slough. The abscess was quite localized. Gall bladder full of light-green fluid bile. Dotted over surface of mucous membranes were small circular injected areas.

Spleen.—Weight, 793 grammes. Vessels of capsule injected, substance normal.

Kidneys.—Right, 510·2 grammes; left, 453·6 grammes. Appearance, normal. The perirenal connective tissue was infiltrated with a yellow gelatinous oedema. Pancreas and suprarenals apparently normal.

Lungs.—Left, upper lobe firmly bound down by old adhesions, substance normal. Right, lower and middle lobes deeply injected. On section patchy, recent consolidation present.

Heart.—Slight amount of straw-coloured fluid present in pericardial sac. Valves, normal. Muscle, normal. Some gelatinous oedema along coronary arteries. Fat, abundant.

Bone marrow of long bones, yellow; of short bones, dark red.

Brain.—Slight thickening and injection of the pia mater.

Lymphatic Glands.—The same appearances were seen as in P.-M. of Experiment XX.

Once more the most marked pathological changes observed were in the lymphatic glands. Many very small, entirely haemorrhagic glands, varying in size from a pea to a bean, occurred in the mesentery. All the lymphatic glands were enlarged and oedematous. The medullae of many were of a deep chocolate colour, and some of the retroperitoneal group were markedly haemorrhagic.

* The thanks of the Expedition are due to the Gambian Government, who kindly supplied the animal used in this experiment.

VII. OTHER FORMS OF FLAGELLATA FOUND IN SENEGAMBIA

In the course of our examination of a large number of small animals and birds, we observed some ten varieties of flagellates, for the most part belonging to the family *Trypanosomidae* (Doflein). These organisms occurred in the blood of frogs, small birds, tortoises, and in the African house mouse.

We were not able to make any serious attempt to ascertain the life history of these parasites. It is probable that a study of the trypanosomata, parasitic in lower animals, will throw light on obscure points in the life history of those species pathogenic to man and the higher animals. Such a study, in the case of other parasites, has met with most fruitful results, notably so in malaria. For this reason, we here present a short description of the forms we have seen in fresh blood, and of their appearance in film preparations, stained by Romanowsky's method.

TRYPANOSOMES IN FROGS

Trypanosoma sanguinis, Gruby,²⁵ 1843.

A parasite was frequently seen in the blood of African frogs corresponding to that first described by Gruby in 1843, and called by him *T. sanguinis*. Out of twenty-nine frogs examined, including *Rana trinodis* (?) and other species, this parasite was seen in the blood of fourteen. The parasites were never encountered in any great numbers in either the peripheral or central circulations of the infected frogs. At the most four to six were seen in a preparation.

This parasite has been described in the living state by Ray Lankester⁴³ as a minute pyriform sac, with the narrower end bent round on itself somewhat spirally, and the broader end spread out into a thin membrane which is produced on one side into a very long flagellum. The wall of the sac was striated coarsely as in *Opalina*, and the direction of the striae on the two sides of the sac, as seen one through the other, showed that the small end of the sac was twisted as well as bent over on itself. A pale clear nucleus and a very few granules were also seen. In life, the broad membrane undulates vigorously in a series of waves, the flagellum taking part in the movements. The series of waves of the undulating membrane are not incessantly in one and the same direction. In stained preparations (Romanowsky method) the simplest form of the parasite seen is represented in Plate II, fig. 3.

This parasite differs from other trypanosomes seen in frogs, in that there is no definite line of demarcation between the body proper and the undulating membrane. The protoplasm of the body takes on a deep bluish purple tint

and is arranged in bands in the long axis of the body. These bands commence at the blunted end of the organism as fine streaks which gradually increase in thickness towards their centre and then fade away into the undulating membrane. In the simplest form of this parasite there are one or two such bands. The posterior end (the end remote from the flagellum) is round and blunted, and is occupied usually by a comparatively large unstained area. The undulating membrane is broad; it commences $8\ \mu$ from the posterior end and extends along the base of the cone-shaped body, gradually decreasing in width. There is a general bluish mottling at the base of the undulating membrane. The flagellum runs along the undulating membrane, its free part extending for about $8\ \mu$ to $10\ \mu$ from the anterior extremity of the body. It is an extremely fine filament and does not stain well by ROMANOWSKY'S method. The micronucleus is seen as a crimson dot from which the flagellum arises; it is situated near the centre of the body and is often difficult to make out. By careful focussing with a $\frac{1}{12}$ " oil immersion a small, red-stained, rounded area can be made out either beneath or above the macronucleus, as the case may be. It is often obscured by the dark stained bands of the protoplasm above described.

Besides this simple form of the parasite there are other larger forms which differ from it in the following manner:—

(a) The posterior end of the organism is toothed. Three such ridges have been generally encountered in these forms.

(b) Instead of one or two there are generally three to four longitudinal bands of deeply staining protoplasm, which commence in these ridges and proceed, often in a spiral manner, towards the undulating membrane. Before their termination they generally present an unstained area. It would appear that these bands are slightly raised from the surface of the parasite. This may account for the striated appearance seen in the fresh state. In these larger forms the micronucleus and macronucleus are generally obscured by the darkly-stained bands.

In stained preparations the average length of the parasite, excluding the flagellum, is $27\ \mu$, its width opposite the micronucleus is $3.2\ \mu$, and the width of the undulating membrane at this point in specimens of the type shown in Fig. 3 is $4.5\ \mu$. LAVÉLAN and MESNIL* have already described this trypanosome as they observed it in stained specimens of blood taken from European green frogs.

Trypanosoma mega.—New species (Provisional), Plate II, fig. 4.

Many large trypanosomes, three to six in a coverslip preparation, were seen in the blood of a small frog, caught in a marsh at McCarthy Island, in January.

Progressive movement was rather sluggish in these organisms, and was evidently brought about by contraction of the body protoplasm, as well as of the undulating membrane. Both ends of the organism tapered for some distance into a long fine

* Laveran et Mesnil. Sur la structure du Trypanosome des grenouilles et sur l'extension du genre Trypanosoma. Gruby, *Compt. Rend. Soc. Biol.* Par. v. 53 (23), June 22, 1901.

process, which at one end was continued on as the free portion of the flagellum. The undulating membrane commenced at a highly refractile spot, surrounded by an area also refractile and situated about one-third of the body's length from the posterior non-flagellated end. The protoplasm has a coarsely granular appearance, and differed in structure in the anterior and posterior portions of the body.

The parasite progresses generally with the flagellum in advance. When stationary, waves of motion move in either direction up and down the undulating membrane.

In stained preparations, the parasite did not vary in form to any appreciable extent. The width in some few specimens was slightly less than in the majority. The shape of the body is seen in Plate II, fig. 4. The protoplasm stains very deeply blue with ROMANOWSKY'S stain. The portion (anterior two-thirds) of the body to which is attached the undulating membrane is longitudinally striated, to a marked degree, with dark and light bands. The spongioplasm of this portion is closely arranged, and its hyaloplasm stains more deeply than that of the posterior third of the organism. With careful focussing it is seen that the light unstained bands are raised from the surface of the parasite. If this part of the body of the organism is twisted on itself, a trellis-work appearance is brought about by the crossing of the light stripes. The anterior two-thirds of the body ends abruptly at the macronucleus, from which it is separated by a narrow unstained area, probably due to a retraction of the protoplasm caused by the drying of the film. The light bands are continued on past the nucleus to the base of the posterior portion. The protoplasm of the posterior portion of the body has a very spongy appearance, and presents a contrast to the anterior. This appearance becomes more marked as the posterior extremity of the parasite is reached. With careful focussing a fine superficial striation can be made out.

In the process of smearing the frog's blood on a slide some of these large parasites were injured. From a study of such specimens it can easily be made out that the body of the parasite is enclosed in an outer membranous covering, which on one side is prolonged as the undulating membrane,* along the free border of which the flagellum is attached. In one parasite this membrane was partially stripped off and stained a faint reddish purple colour. In it were seen fine pink staining lines, having a looped arrangement and apparently running round the short axis of the organism. The membranous envelope is much more easily seen in the anterior division of the parasite. The undulating membrane commences opposite the nucleus as a comparatively wide fringe which is thrown into folds and takes on a faint purple tint. It gradually narrows towards the anterior end. Along its free border is attached the flagellum, staining a reddish pink. The latter commences at the micronucleus and ends anteriorly as a free filament. The macronucleus is situated

* Wasielewski und Senn. Beiträge zur Kenntniss der Flagellaten des Rattenblutes. *Zeitschr. f. Hygiene, Bd. 33, 1900.*

about 28μ from the posterior end; it is finely granular, is coloured light crimson, and extends completely across the short axis of the parasite. The micronucleus is in close proximity to the macronucleus, though separated by a slight interval. Its position on an average is 26μ from the posterior end of the organism. It stains a deep crimson, is devoid of structure, and is surrounded by a zone staining light blue. The average length of a parasite as measured in stained preparations is 72μ . This excludes the free flagellum, which is from 10μ to 15μ long. The width of the parasite at the macronucleus is 8μ .

The frog infected was eventually killed, and preparations of blood taken from various parts of the body were examined. Parasites were seen in all, and were not found to be particularly numerous in the preparations from any one organ. No forms were seen in developmental stages. No parasites were seen in the peritoneal fluid. The organs of the frog appeared normal macroscopically.

Trypanosoma Karyozeukton.—New species (provisional).

In a stained (ROMANOWSKY) preparation of the blood of a frog (sp. ?) caught at Cape St. Mary, Gambia, October 23, in a fresh preparation of which *T. sanguinis* had been seen, one specimen of a large trypanosome (Plate II, fig. 5) was encountered presenting the following characteristics.

Length, excluding the flagellum, 67.2μ . Width at macronucleus, 6.4μ . Distance from micronucleus to centre of macronucleus, about 16μ . Distance from micronucleus to posterior extremity, 9.8μ . Length of free flagellum, 15.2μ . As in the preceding trypanosome, the body protoplasm posterior to the macronucleus presents a contrast to the anterior portion. The former does not stain so deeply and has a mottled appearance. The latter stains more deeply, has a much more closely arranged spongioplasm, and in it faint longitudinal striation can be made out. The micronucleus is ovoid, stains a dark crimson, and shows no structure. The macronucleus extends the whole width of the organism and takes on a light crimson colour. Its structure cannot be made out. Posterior to the macronucleus are scattered, for a short distance in the protoplasm, fairly large granules, staining a deep bluish-purple colour. Between the micronucleus and macronucleus runs a chain of small red oval granules, which are, apparently, of the same nature as chromatin. Examination with a $\frac{1}{12}$ " oil immersion has not enabled us to say definitely that there is a union between the macronucleus and micronucleus by means of this chain of chromatinic granules. The posterior end of the parasite gradually tapers to a fine filament. Posterior to the micronucleus the narrowing becomes more marked. The undulating membrane commences opposite the micronucleus and runs along one side of the organism as a narrow band. It is stained faintly pink.

Three other frogs were received at the same time as that which harboured the trypanosome just described; in the blood of two of these were seen an

actively motile, long and thin trypanosome. There were two to three of these parasites to a coverslip preparation. The movements of the parasite were so energetic that it was difficult to obtain an idea of its structure in the fresh state. The undulating membrane was seen to extend nearly the whole length of the parasite, and by its rapid screw-like movements to cause the organism to move in a spiral manner in and out among the red cells.

In stained preparations the average length of the parasite, excluding the free flagellum which is seen with difficulty in many specimens, is 56.8μ . Its width at the macronucleus is 3.5μ . The distance between the micronucleus and the centre of the macronucleus is on an average 6.5μ . Measurements taken from the tip of the posterior end of the body to the micronucleus averaged 7μ .

Though this parasite is much smaller than the larger parasite just described, the relation of the micronucleus to the macronucleus and the relative sizes of the flagellum bearing and posterior portions of the body lead us to believe that it belongs to the same species. The following resemblances are noteworthy with regard to the structure:—The smaller parasite has the same feebly staining macronucleus extending the whole width of the organism, and the same oval-shaped micronucleus as the larger form. The undulating membrane is comparatively narrow as in the larger parasite. The most noticeable structural difference between the two parasites is that in the smaller one it is the portion of the body anterior to the macronucleus which is more markedly striated. Other differences are as follows:—The striae in the smaller parasite appear as longitudinal white lines broken at fairly even intervals. It has no granules posterior to the macronucleus and no chromatinic dotted line between micro- and macronucleus. It is possible that the larger parasite may represent an early stage of reproduction.

TRYPANOSOMES IN TORTOISES

Two tortoises obtained from the marshes at Cape St. Mary, out of several examined, contained trypanosomes in their blood. Their blood was constantly examined during a period of three months, but only occasionally were parasites seen, and then only in small numbers. Two varieties were observed. One was a long thick form, the other, short and slender, possessed a comparatively broad undulating membrane which extended the whole length of the organism.

The few parasites seen in stained films of blood from these tortoises have not been sufficiently perfect to permit their detailed description. Our notes on their morphology are therefore reserved until further material is obtainable.

TRYPANOSOMES IN BIRDS

Trypanosoma johnstoni.—New species.

This trypanosome was found in the blood of a species of small bird (*Estrellda*

estrela) very common in Senegambia. One was found to be infected out of twenty-five birds of different species, for the most part belonging to genera *Estrela* and *Crithagra*, examined in the laboratory in August. These birds came from Bathurst, Gambia, whence they had been sent in May.

Only two to four parasites were seen in a coverslip preparation. In fresh preparations the parasite appears as a very actively moving spirillum-like body; so striking, indeed, is this resemblance, that at first sight it was thought to be a true spirillum. The undulating membrane is scarcely recognizable, and the parasite has no free flagellum. When its movements have become slower, the organism is seen to possess a long straight body pointed at both ends. At a point about one-third of its length distant from the posterior end of the parasite is seen a refractile spot, the micronucleus. A little further on, a slightly refractile area indicates the position of the macronucleus. When the parasite is stationary for a moment, waves of movement commencing at the anterior end travel down to this refractile area and there cease. The part of the body posterior to this spot remains perfectly quiescent. The parasite generally moves with the end remote from the refractile spot in front. Careful examination reveals the presence of a very narrow undulating membrane, commencing at the micronucleus and running along to the anterior end of the organism. No free flagellum could be made out in fresh specimens. The protoplasm of the organism was apparently homogeneous.

In stained preparations, Plate II, fig. 1, the above details are better seen. Along the free border of the undulating membrane runs the flagellum, stopping abruptly at the anterior end of the organism in a small red dot. The macronucleus is elongated, granular, and does not quite extend across the short axis of the parasite. The micronucleus is a small dot or oval spot of chromatin surrounded by a small halo. The protoplasm takes on a uniform blue colour. The length of the parasite varies from 36μ to 38μ , and its width at the macronucleus is 1.4μ to 1.6μ . The distance from the micronucleus to the posterior end is 10.4μ . Distance from the micronucleus to the centre of the macronucleus is 9μ to 10μ . No developmental or dividing forms were seen in any preparation.

Two larks were inoculated, each with 0.5 c.c. of blood from this bird, but so far parasites have not been seen in the blood of either.

Trypanosoma.—Spec. incert. This parasite was fairly common in the blood of species of *Crithagra* and *Estrela*, both at St. Louis and Bathurst, though they were never present in large numbers.*

At St. Louis out of fifteen birds examined seven were infected.

At the most three to six parasites were seen in a preparation. In the fresh film the parasite is a very stumpy organism which moves sluggishly in the blood.

* The popular name commonly given to these birds is 'millet eaters.' This name includes several varieties called Bec corail, cordon bleu, ventre rouge. The majority of the birds examined were of the first.

The width is great in proportion to the length of the body. The protoplasm has a very granular appearance. At one end is seen a highly refractile spot, at the other a fine, free flagellum. Both ends of the parasite are stumpy.

In stained preparations, the parasite generally assumes an oval shape, due to its stumpy anterior and posterior ends and to its great width. The protoplasm takes on a deep blue reaction, is longitudinally striated, and is marked with one or two lighter patches in front of the macronucleus. The micronucleus is a large crimson spot. It is situated at the posterior end of the parasite and is in close relation to a clear space, a vacuole. The macronucleus is situated centrally and extends across the middle of the body. It stains a pink red colour, the chromatin being diffuse. The undulating membrane is a very narrow band extending on one side of the parasite from the micronucleus to the anterior end of the parasite's body. In Plate II, fig. 2, the undulating membrane has the appearance of crossing over the body; this is due to the parasite being twisted on itself. The flagellum commences in the micronucleus, runs along the free border of the undulating membrane and ends as a fine free filament. Length of the parasite without free flagellum is 21.6μ . Its width opposite macronucleus is 8μ . The distance from micronucleus to centre of macronucleus is 9.6μ . The length of the free portion of the flagellum is about 10μ to 12μ . No divisional forms were seen.

Two pigeons and two larks have been inoculated with these parasites, but have not yet shown organisms in their blood. The pigeons were inoculated three months ago.

Dr. HANNA has very kindly shown us specimens of a trypanosome which he found in a pigeon in India, and we have also been able, through the kindness of Major Ross, to see one or two slides of blood taken from a blue jay in India, containing filariae and trypanosomes.

These will shortly be described and illustrated by Dr. HANNA; they present several morphological differences from the parasite we have seen in Africa. We feel certain that many more of these parasites will be found in birds and small animals both in India and Africa.

DANILEWSKY* has described two varieties of trypanosomes—*Trypanosoma major* and *Trypanosoma avium*—observed in the blood of birds. Unfortunately, we have not been able to obtain his original paper, and the reviews, which we have seen of his works, have not been sufficiently definite to enable us to determine whether our second avian trypanosome is the same as his or not. We have, therefore, left it unnamed for the present.

FLAGELLATA IN THE BLOOD OF A MOUSE

At McCarthy Island, we obtained a few house mice and some field mice (Spec. unknown).

* La parasitologie comparé du sang. *Nouvelles recherches sur les parasites du sang des oiseaux*, 1889.

Fourteen of the former in all were examined, and in the blood of three, flagellated protozoa were seen. Twenty field mice were examined, but none were found to be infected. The organism occurred infrequently in the blood of the mice infected, and we never saw more than one or two parasites in a fresh coverslip preparation.

The organism was striking, in that it presented characteristics which differentiated it, at a glance, from a trypanosome. It consisted of a long oval-shaped body with blunt rounded ends, to one of which a very long flagellum was attached. At this end the body tapered slightly. A glance showed that the long flagellum, slightly longer than the body, was the chief organ of locomotion. It acted as a tractellum, and obviously dragged the body of the parasite after it. On encountering an obstruction the flagellum lashed out in all directions, hurled the red cells behind it, and often twisted round, so that its tip reached past the posterior end of the body. On these occasions, it was easy to examine the structure and measure the length of the parasite, as the body was perfectly quiescent. The protoplasm has a slightly granular appearance and in its substance, placed a little in front of the centre and towards the flagellated end, is a collection of refractile granules. About 5μ from the anterior end is seen a highly refractile spot from which the flagellum takes origin. No suggestion of an undulatory membrane was seen. The length of the body in the living condition is 20.8μ , and its greatest width 3.2μ . The body of the parasite sometimes assumed an 'S'-shaped form, on account of external pressure or rapid changes of direction of movement, but no active contractions of the protoplasm were seen.

Unfortunately, two mice sent home infected with this parasite died on the voyage, and in the films made from the mice while in Gambia, no specimens have been seen. A small rat inoculated from one of the mice has not shown the parasite in its blood. No symptoms were observed in the mice infected.

This parasite resembles very closely *Herpetomonas (Leptomonas) Butschli* (S. Kent). The figures given of this organism correspond very closely with our flagellate, except that in the mouse parasite there is not the marked tapering of the non-flagellated end of the body. *Herpetomonas Butschli*, however, inhabits the intestinal tract of *Trilobus Gracilis*, and not a vertebrate circulatory system.

BIBLIOGRAPHY

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BIBLIOGRAPHY

Those who are interested in this subject will find in the *Emergency Report on Surra*, written by SALMON and STILES and issued by the Bureau of Animal Industry, Department of Agriculture, Washington, U.S.A., a most excellent bibliography by ALBERT HASSALL, M.R.C.V.S. It is a very complete list of the literature on the subject up to 1902.

We have added to our report therefore, a list of only some of the more recent publications. We hope that it may prove useful.

A

- 1 ADAMS, ALEX. MAXWELL, 1903. *Trypanosomiasis and its cause.* (Refers to case described by DUTTON.) *Brit. Med. Journ.*, March 28.

B

- 2 BRUCE, 1902. *Note on the discovery of a new Trypanosome (THEILERI).* *Lancet*, March 8.
- 3 BUTSCHLI. *Researches on flagellated infusoria and allied organisms.* *Zeitschrift f. wis. Zool.* XXX, *Quart. Journ. Micr. Sci.* 73 (p. 63, pl. vi, fig. 5).
- 4 BOYCE, ROSS, and SHERRINGTON, 1903. *The History of the Discovery of Trypanosomes in Man.* *Lancet*, February 21.
- 5 BAKER, C. J., 1903. *Three cases of trypanosome in man in Entebbe, Uganda.* *Brit. Med. Journ.*, May 30, No. 2213.
- 6 BLANCHARD, R., 1903. *Extraits de lettres de Blanchard à l'acad. de Méd.* *Bull. de la Séance*, Mars. 17, p. 368.
- 7 BRUMPT, 1903. *Bull. de l'acad. de Médecine*, Mars 17, T. XLIX, p. 372. *Bull. de l'Inst. Pasteur*, T. I, No. 5, p. 217.
- 8 BUFFARD, M. et SCHNEIDER, G., 1902. *Note sur l'existence en Algérie d'une trypanosome autre que la dourine.* *Rec. de Méd. Vét.*, No. 23, p. 721.

C

- 9 CASTELLANI, DR. ALDO, 1903. *Some observations on the Morphology of the Trypanosoma found in Sleeping Sickness.* *Brit. Med. Jour.*, June 20.
- 11 CASTELLANI, DR. ALDO, 1903. *Journal of Trop. Med.*, June 1.
- 12 CASTELLANI, DR. ALDO, 1903. *Untersuchungen über die Aetiologie der Schlafkrankheit.* *Archiv für Schiff- und Tropenhygiene*, Bd. VII, p. 382.
- 13 CASTELLANI, DR. ALDO, 1903. Also appeared in *Journal Trop. Med.*, No. 11.

D

- 14 DUTTON and TODD—note by ANNETT, 1903. *Preliminary Account of the Investigations of the Liverpool Expedition to Senegambia, 1902.* *British Medical Journal*, Feb. 7.
- 15 DUTTON, 1902. *Report of K.'s case in Brit. Med. Jour.* (Report to Association, July 29, 1902), *Brit. Med. Jour.*, September 20, 1902, Vol. XI, p. 88.

- 16 DUTTON, 1902. *Thompson Yates Laboratories Reports*, May 18.
 17 DOFLEIN, Dr. F. (München), 1901. *Die Protozoen als Parasiten und Krankheitserreger*, Verlag von GUSTAV FISCHER, Jena.

E

- 18 ELMASSIAN et MIGONE, 1903. *Sur le Mal de Caderas ou flagellose parésiente des équidés Sud-Américains. Annales de l'Inst. Pasteur*, No. 4.
 19 EBERTH, Dr. J. See LEUCHART. *The Parasites of Man*, translated by HCYLE, p. 248; describes *Trichomonas* in intestine of fowls and ducks.

F

- 20 FORDE, R. M., 1902. *Some clinical notes on a European patient in whose blood a trypanosoma was observed. Journ. Trop. Med.*, September 1.
 21 FORDE, R. M., 1902. *The discovery of the Human Trypanosome. Brit. Med. Journ.*, November 29.
 22 FRANCIS, EDWARD, 1903. *An experimental investigation of Trypanosoma Lewisii. Hygienic Lab. Bull.*, No. 11, U.S. Treasury Department.

G

- 23 GALLOWAY, 1903. *Erythemata as indicators of Disease. Brit. Med., Jour.*, July 18.
 24 GROTHUSEN, DR. —, 1903. *Über das Vorkommen der Tsetse Krankheit beim Zebra. Archiv für Schiffs- und Tropenhygiene*, Bd. VII, p. 387.
 25 GRUBY, —, 1843. *Compt. Rend. de l'Acad. d. Sci.*, November. Described *Trypanosoma Sanguinis*.

H

- 26 HASSALL, ALBERT, 1902. *Bibliography of Surra and Allied Trypanosomatic Diseases. Bull. No. 42, Bureau Animal Industry, U.S. Dept. Agriculture, Wash.*

J

- 27 JURGENS, —, 1902. *Beitrag zur Biologie der Rattentrypanosomen. Archiv f. Hygiene*, Bd. XLII, heft 3.

K

- 28 KERMORGANT, 1902. *Le Nagana au Chari (MOREL). Bull. de l'acad. de Médecine*, December 15, p. 574.
 29 KRUSE, 1903. *Ueber das trypanosoma Castellani den Erreger der Schlafkrankheit. Sitzungsberichten der Niederrhein Gesellschaft, f. Natur. und Heilkunde zu Bonn*, Mai.
 30 KOCH, R. *Experiments on attenuation of Nagana by passage through different species of animals. Deutsches Kolonialblatt*, No. 24, 1901.

L

- 31 LAVERAN et MESNIL, 1900. *Soc. de Biologie*, October 6.
 31A LAVERAN et MESNIL, 1900. *Sur le mode de multiplication du trypanosome du rat. Comp. rend. de la Soc. de Biol.*, T. III, No. 25.
 32 LAVERAN, 1902. *De l'action du sérum humain sur le trypanosome du Nagana. Acad. de Science*, April 1.
 33 LAVERAN et MESNIL, 1902. *Recherches sur le Traitement et la Prévention du Nagana. Annales de l'Institut Pasteur*, November.

- 34 LAVERAN, 1902. *Sur l'épizootie de Surra qui a régné à l'île Maurice.* *Bull. de l'acad. de Méd.*, October 29.
- 35 LAVERAN et MESNIL, 1902. *Trypanosisme du Nagana ou Maladie de la mouche tsétsé.* *Annales de l'Institut Pasteur*, January 1.
- 36 LAVERAN, 1902. *Au sujet de deux Trypanosomes des Bovides du Transvaal.* *Compt. rend. de l'Acad. d. Science*, T. CXXXV, No. 18, p. 717.
- 37 LAVERAN, 1902. *Les Trypanosomes des poissons.* *Arch. f. Protistenkunde*, Bd. I, No. 3, p. 475.
- 38 LAVERAN, 1903. *Sur deux Hippobosques du Transvaal susceptibles de propager Trypanosoma Theileri.* *Compt. rend. Soc. Biol.*, T. LV, February 21.
- 39 LAVERAN, 1902. *Le Nagana et le mal de caderas sont deux entités morbides bien distinctes.* *Compt. rend. de l'acad. de Sci.*, T. CXXXV, No. 20, p. 838. *Rec. de Méd. Vét.*, January 15, 1903.
- 40 LAVERAN et MESNIL, 1902. *De l'évolution du Nagana et de sa variabilité suivant les espèces animales.* *Bull. de l'acad. de Méd.*, Juin 3.
- 41 LAVERAN et MESNIL, 1902. *Les Maladies à Trypanosomes leur Répartition à la Surface du Globe.* *Janus*, Mars, 15, 1902; Juli 15, Août 15, 1903.
- 42 LAVERAN et MESNIL, 1903. *Le Nagana, le Surra, et le Caderas, constituent trois entités morbides distinctes.* *Compt. rend. de l'Acad. des Sciences*, T. CXXXVI, p. 1529, January 22.
- 43 LANKESTER, Prof. RAY, 1871. *On Uudulina the type of a new group of Infusoria.* *Quart. Jour. Mic. Sci.*, XI, p. 387.
- 44 LEUCHART, 1879. *Parasites of Man*, translated by HOYLE, 1886.
- 45 LEISHMAN, 1903. *On the possibility of the occurrence of Trypanosomiasis in India.* *Brit. Med. Journ.*, May 30.
- 47 LIGNIERES. *Details of experiments on agglutination.* *Revista de la Societ. ad Medica, Argentina*, T. X, p. 481.
- 48 LIGNIERES, 1903. *Contribution à l'étude de la trypanosome des équidés Sud-Américains—Mal de Caderas.* *Rec. de Méd. Véter. (D'ALFORT)*, T. X, Ser. 8, Nos. 2, 4, 6.

M

- 49 MANSON, 1903. *Reviews work on Human Trypanosomiasis.* *Tropical Diseases.* CASSELL & Co., London.
- 50 MANSON & DANIELS, 1903. *Case of Trypanosomiasis.* *Brit. Med. Journ.* May 30, p. 1219.
- 51 MANSON, 1903. *Trypanosomiasis on the Congo.* *Brit. Med. Journ.* March 28, p. 720.
- 52 MANSON, 1903. *Opened discussion of Trop. Sec. at B.M.A. meeting, Swansea.* *Journ. Trop. Med.*, August, 15.
- 53 MANSON, DANIELS, & HABERSHON, 1902. *Described a case of Human Trypanosomiasis.* *Editorial, Brit. Med. Journ.*, Vol. II, p. 1452.
- 54 MOREL, 1903. *Existence de la tsétsé et du Nagana au Chari.* *Annales Hyg. et Méd. col.*, T. VI, No. 2, p. 264.
- 55 MARTINI, ERICK, 1903. *Ueber die Entwicklung der tsetse parasiten in Säugethieren.* *Zeitschrift. f. Hyg. u. Infekt.* T. XLII, No. 2.
- 56 MUSGRAVE, Dr. W. E., and WILLIAMS. *Preliminary Report on Trypanosomiasis of Horses in Phillipines.* Manila Biological Laboratory.
- 57 MOLLEREAU, 1888. *Maladies des Mulets au Tonkin* (report on work of Blanchard). *Bull. Soc., Centrale Méd. Vét.*, December 30, p. 684.

N

- 58 NOCARD, Ed. et Leclainche, 1903. *Les maladies microbiennes des animaux.* Third edition.
- 59 NOCARD, 1900. *Bull. de l'acad. de Méd.*, Juillet 31, p. 154.

P

- 60 PLIMMER, H. G., and BRADFORD, ROSE, 1899. *The Trypanosoma Brucei, the Organism found in Nagana or tsetse fly disease.* *Quart. Jour. Mic. Sci.*, Vol. 45, part 3, February, 1902, p. 449, and *Centrabl. f. Bakt, Abt. I*, T. XXVI, p. 440.

R

- 61 ROUGET, M. J, 1903. *Contribution à l'étude de la Dourine.* *Recueil de Méd. Vét. d'Alfort*, T. X., p. 82, Février.

S

- 62 SCHNEIDER et BUFFARD, 1900. } *Sur les rapports qui existent entre le Dourine et le Surra et le Nagana.*
 63 SCHNEIDER et BUFFARD, 1900. } *Archives de Parasit*, T. III, p. 124. *Compt. rend. Soc. Biol.*, Mai 4,
 1901, p. 464.
 64 SCHNEIDER et BUFFARD, 1900. *La dourine et son parasite.* *Recueil Méd. Vét.*, 8^e serie, T. VII,
 Février 15, Avril 15, pp. 157, 220.
 65 SAMBON, LOUIS W, 1903. *Sleeping Sickness in the light of recent knowledge.* *Jour. Trop. Med.*,
 July 1.
 66 SCHILLING, 1901. *Bericht über die Surrakrankheit der Pferde.* *Centralbl. f. Bakt. Abt. I*, XXX.
 October 20, p. 545.
 67 SIVORI, F. et LECLER, EN., 1902. *Le Surra Américain ou Mal de Caderas.* *Ann. del min. de Agri. sect.*
de Zootechnia, bact, etc., Buenos Aires.
 68 STILES, CH. E., 1902. *Trypanosomatic disease of domestic animals.* *Journ. Comp. Med.*, No. 9, p. 565.
 69 SANDER, 1902. *Beiträge zur afrikanischen Tsetse krankheit.* *Deutscher Kolonial-kongress*, Section II.
 Berlin.

Z

- 70 ZABALA, 1901. *Work on Dourine, description of parasites, and experiments in horse, rat, mouse, cattle, goat, sheep, cat, dog, guinea-pig.* *Annales du Departement National Hygiene de Buenos Aires*, Ano. IX, Noc.
 71 ZIEMANN, 1902. *Ueber ein neues Halteridium und ein Trypanosoma bei einer kleinen weissen Eule in Kamerun.* *Archiv. f. Schiff's-u. Tropen. Hyg.* Bd. VI, No. II.
 72 ZIEMANN, 1903. *Vorläufiger Bericht über das Vorkommen der Tsetse krankheit im Küstengebiet Kameruns, etc.* *Deutsche. Med. Woch.* Nos., 15, 16.

APPENDIX

A NEW CULICID FROM SENEGAL AND NOTES ON THE SPECIES OF MOSQUITOES, ETC.

BY F. V. THEOBALD, M.A.

Amongst a large collection of mosquitoes and other biting-mouthed diptera collected and bred by Drs. DUTTON and TODD during their recent expedition to Senegal, is a large series of a new Culicid, resembling in habits the *Deinocerites cancer*, THEOBALD, of the West Indies. It comes very near my genera *Stegomyia** and *Macleaya*,† but cannot be placed in either. Without an examination of the scale structure, one would certainly place it in *Culex*, but hasty microscopic examination shows it to come much nearer the two first-named genera. I propose for it the generic name *Catageomyia*, (κατάγειος, subterranean, and μύια, a fly).

GENUS *Catageomyia*. Nov. gen.

Head covered with rather irregular loosely-applied flat scales, a few narrow-curved ones on the nape in the ♀, spreading rather further on to the occiput in the ♂, also with numerous narrow, upright, forked scales. Palpi short in the ♀, composed of three segments, the last one as long as the two basal ones; in the ♂ the palpi are long, but not nearly as long as the proboscis, the two apical joints short, the apical one slightly shorter than the penultimate, the apex of the antepenultimate slightly expanded, dense hairs on each side of the penultimate and on one side of the apex of the antepenultimate; apex bristly. Thorax with narrow-curved scales on the mesonotum; small flat ones on the mid lobe of the scutellum; narrow-curved ones on the lateral lobes.

This genus thus differs from *Stegomyia* in (1) having narrow-curved scales on the back of the head, (2) narrow-curved scales on the lateral lobes of the scutellum; and from *Macleaya* in (1) having the flat cephalic scales more loosely applied, (2) in having the ♂ palpi much shorter than the proboscis.

In general appearance it resembles a *Culex* of the *fatigans* group, but the scale structure, palpi, etc., are quite distinct.

Catageomyia senegalensis. N. sp.

Thorax rich, deep brown, with small scattered golden scales, paler before the scutellum; numerous long dark bristles posteriorly. Abdomen black, with basal white bands, which spread out laterally; venter, with broad basal white bands; head ornamented, with grey and black; proboscis deep brown, unbanded; legs deep brown, unbanded, except the hind tibiae, which have an apical white band; venter of femora, white. Wings with brown-scaled veins. Male palpi shorter than proboscis.

♀. Head clothed with loosely applied flat scales all over, a few narrow curved ones behind, and narrow upright brown forked ones scattered about; the flat scales are in black and white areas, and the narrow curved ones are pale grey; there are dark brown bristles projecting in front; eyes purple when dead; palpi, brown, three-jointed, the last joint as long as the two basal ones; antennae, brown; basal joint pale testaceous, with grey sheen, and a few small dark scales internally; clypeus and proboscis, deep brown.

* *Mono. Culicidae*, Vol. I, 1901.

† *The Entomologist*, Vol. XXXVI, p. 154, 1903.

Thorax, deep rich brown with narrow-curved bronzy scales, amongst which are scattered narrow-curved bright golden ones, grey ones in front of the roots of the wings and in front of the scutellum; numerous long black bristles over the roots of the wings and on the posterior part of the mesonotum; prothoracic lobes brown with outstanding creamy narrow-curved scales, and a series of stout black orwardly projecting bristles. Scutellum, testaceous with narrow-curved grey scales wider than those of the mesonotum; on the side lobes, border bristles deep brown in two series; on the median lobe, six larger ones, and numerous smaller ones behind; the mid lobe covered with loose flat grey and dusky scales; pleurae, brown, with patches of grey scales; metanotum, testaceous brown. Abdomen covered with black scales, the segments with narrow white basal bands which spread out laterally to form spots; the first segment is pale testaceous at the base with a large apical median patch of black scales, and a few grey ones at the sides, and numerous long brown hairs; posterior borders of the segments with pale golden border bristles of two alternating sizes; venter with very broad grey basal bands and narrow apical dark ones.

Legs, deep brown; bases, paler; femora, white beneath; fore and mid legs without any trace of banding, but the hind tibiae have an apical white band and are longer than the hind metatarsi; femora, tibiae and metatarsi with bristles which are pale golden in some lights, brown in others. Fore and mid ungues equal, uniserrated; hind, equal and simple.

Wings with the veins clothed with brown scales, rather dense and large on the second long vein and also on the basal areas of all the veins, longish lateral ones on the hind and apices of the fourth and fifth; first submarginal cell longer and narrower than the second posterior cell, its stem about one-third the length of the cell, its base nearly level with the base of the second posterior cell; in the second posterior cell the stem is about two-thirds the length of the cell; posterior cross-vein about one and a half times its own length distant from the mid; halteres, pale ochraceous.

Length, 4 to 4.5 mm.

♂ Palpi brown, much shorter than the proboscis, the last two joints short, of nearly equal length, darker at their tips than at the base, apex of the antepenultimate joint slightly expanded, also darker than the rest; traces of a very narrow pale band; hair-tufts brown; proboscis deep brown, longer than the palpi; antennae with flaxen brown plumes, banded brown and grey; head like the ♀, only rather more narrow-curved scales running into the crown; thorax and abdomen with similar ornamentation; fore and mid legs with unequal ungues, the fore both uniserrated, the mid with the large simple, the smaller uniserrated, hind equal and simple; genitalia with the basal lobe very bristly; claspers slightly sinuous.

Length.—4 to 4.5 mm.

Habitat.—St. Louis, Senegal (Drs. DURRON and TODD).

Time of Capture.—May and September.

Observations.—Described from a large number of dry and spirit specimens. In habits it bears some resemblance to *Dernocerites cancer* (THEOBALD). They breed in crab holes, where the larvae were taken as deep down as three feet three inches. The adults were caught in and around the crab holes, in mangrove swamps, near the race course, at St. Louis, also in the bush by Oyster Creek, Bathurst, and in an old tub in a garden at St. Louis.

Culex duttoni.—From old sand garden pool. Sor St. Louis, May 21, 1903. Bred from pool dug in sand to obtain water for gardening purposes, about ten feet deep; diameter, twelve feet, now disused, now only three inches at bottom. Sor St. Louis, May 22, 1903. Garden tub. Bridge puddle. Bred in laboratory, June 2.

Culex luteolateralis, var. *pallida* (n.v.)—Caught in marsh behind Subenta, January 18.

Culex luteolateralis, var. *albithorax* (n.v.)—Caught in bush near Inkutu, January 18.

Culex tigripes.—Hatched out from larvae taken in tub, in garden, and in hospital. St. Louis, May 31, 1903. Tub in garden, Sor St. Louis.

New Crab-hole form, n.sp.—Mangrove Swamps racecourse; caught in bush oyster creek; crab-hole and old tub in garden, Sor crab-holes racecourse; taken from crab-hole, three feet three inches from surface, St. Louis.

Culex fatigans, WIED.—St. Louis, north end of island. Garden tubs, St. Louis; hospital garden water-tubs; Sor St. Louis leaky water main. All in May; bred from putrid water, mouth of drain, St. Louis; Sor, taken from pool near leaky water-pipe; bred from putrid water at mouth of old drain, St. Louis. May; Dakar, June 16; Sor, bred from larvae in old native well in sandy pool; from Gorce, June; bred from pupa in puddle, Halftei, September 21, 1902. Cult. F.N.

Pyretophorus costalis.—Var. Dakar, June 16, 1903; bred from sand pool, Sor., May 24; Dukai, June 16, 1903; leaky water-main, Sor, St. Louis, May 30; hatched out from discarded pool behind houses, in sand, Sor, St. Louis; hatched from tub and bridge puddle, Khor., June 1.

Stegomyia fasciata.—St. Louis, caught in our house, May 23, bit just as much at night as at day; Goree, June 15, 1903, numbers marked 31 all caught in house at St. Louis; hatched in tub, St. Louis, May 6, 1903; hatched from tub in hospital, St. Louis, McCarthy Island.

Uranotaenia amulata.—THEOB. Caught in dry marsh at Dakar, June 13, 1903.

Mansonia uniformis.—THEOB. McCarthy Island, June; caught in bush near Sukutu, January 18 (? June).

PLATE I

HUMAN TRYPANOSOME

- FIG. 1. *Trypanosoma gambiense* in Gambian native. $\times 2,000$.
FIG. 2. *Trypanosoma gambiense* in tame rat. 'Long form.' $\times 2,000$.
FIG. 3. *Trypanosoma gambiense* in tame rat, showing longitudinal division. $\times 2,000$.
FIG. 4. *Trypanosoma gambiense* in tame rat, from a specimen taken one week before death. 'Stumpy form,' showing chromatic granules. $\times 2,000$.
FIG. 5. *Trypanosoma gambiense* in tame rat, from a specimen taken one week before death. 'Round form,' showing granules. $\times 2,000$.

GAMBIAN HORSE TRYPANOSOME

- FIG. 6. Horse trypanosome. The small 'tadpole'-shaped parasite in the early stage of the disease. $\times 2,000$.
FIG. 7. Horse trypanosome. 'Stumpy form' in tame rat. $\times 2,000$.
FIG. 8. Horse trypanosome, showing longitudinal division of 'tadpole'-shaped parasite. $\times 2,000$.
FIG. 9. Horse trypanosome. 'Long form,' showing longitudinal division. $\times 2,000$.
FIG. 10. Horse trypanosome. 'Long form.' $\times 2,000$.

PLATE I



Fig. I.



Fig. II.



Fig. III.



Fig. IV.



Fig. V.



Fig. VI.



Fig. VII.



Fig. VIII.



Fig. IX.



Fig. X.

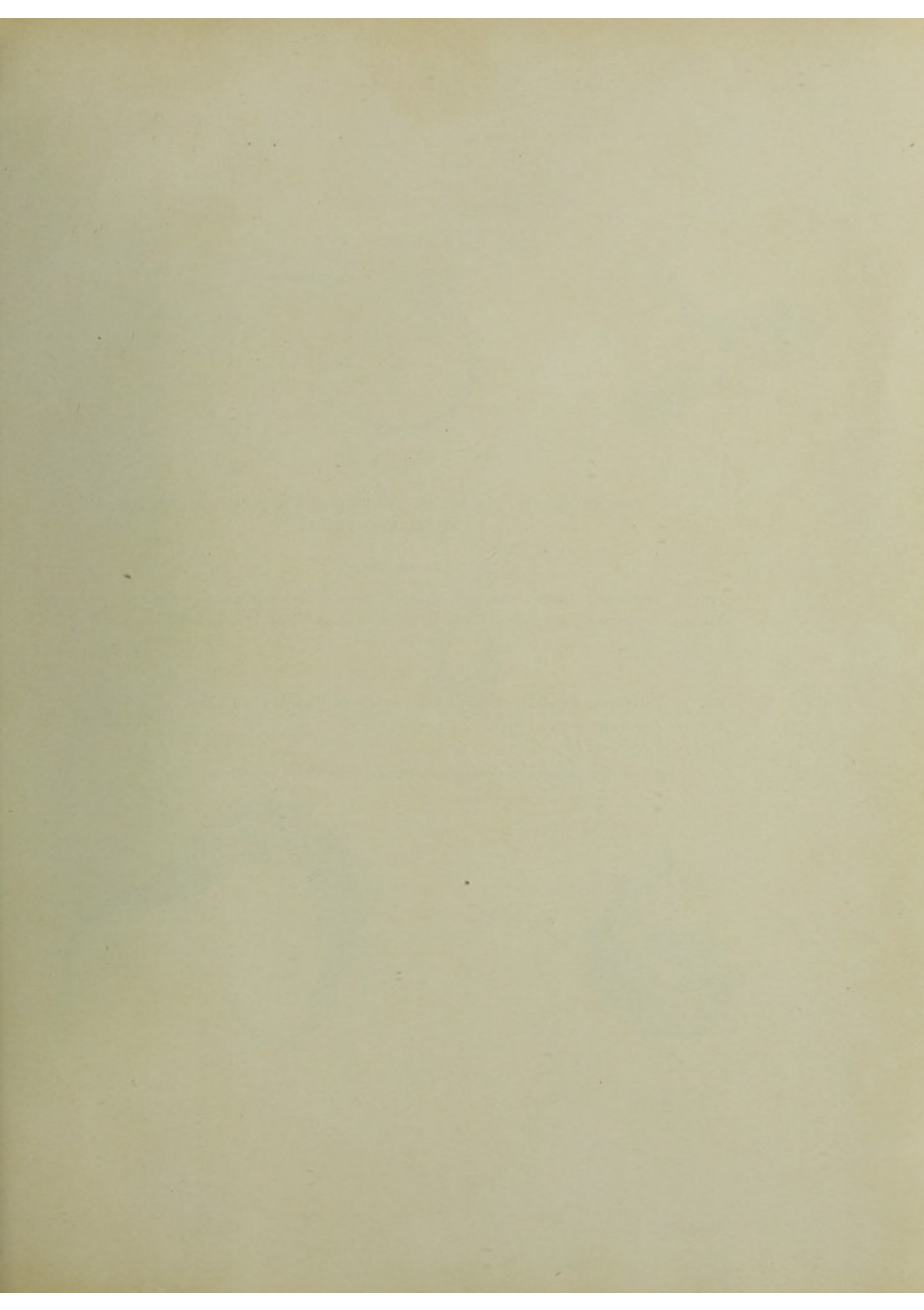


PLATE II

TRYPANOSOMES FOUND IN SENEGAMBIA IN THE BLOOD OF
BIRDS AND FROGS

IN BIRDS

- FIG. 1. *Trypanosoma johnstoni*. Nov. sp. From the blood of *Estrela estrela*. $\times 2,000$.
FIG. 2. *Trypanosoma*. Sp. incert. Found in the blood of small birds in Senegambia. $\times 2,000$.

IN FROGS

- FIG. 3. *Trypanosoma sanguinis*. GRUBY. The simple form of parasite. $\times 2,000$.
FIG. 4. *Trypanosoma mega*. Nov. sp. $\times 2,000$.
FIG. 5. *Trypanosoma karyozeukton*. $\times 2,000$.

Note the chromatinic chain of granules running from micro- to macronucleus.



Fig. 1.



Fig. 2.



Fig. 3.



Fig. 4.



Fig. 5.

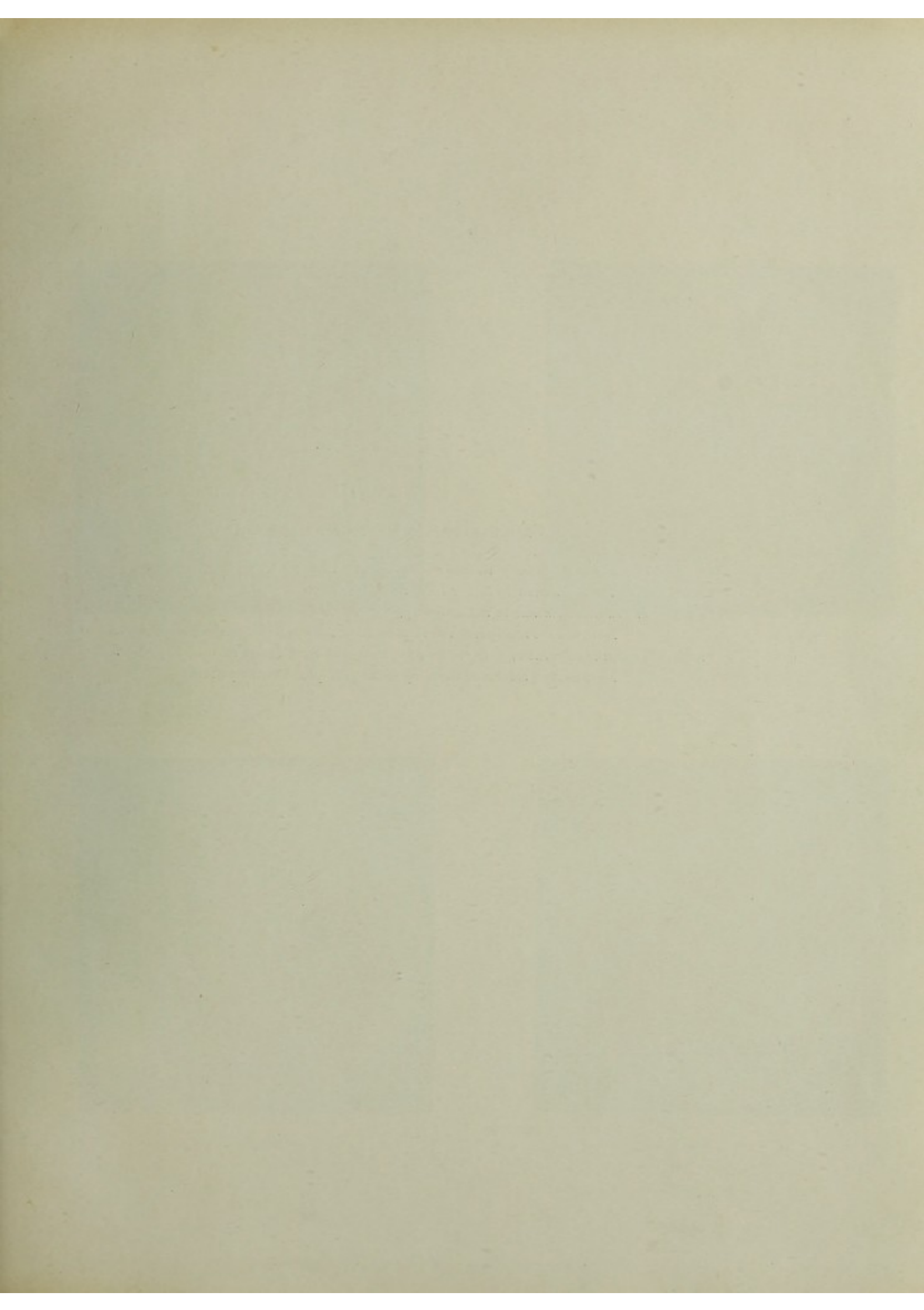


PLATE III

PHOTOMICROGRAPHS OF HUMAN TRYPANOSOMES

FIG. 1. *Trypanosoma gambiense* in the blood of a native. Case V. $\times 840$.

FIG. 2. *Trypanosoma gambiense* in rat. 'Long form.' $\times 840$.

FIG. 3. *Trypanosoma gambiense* in rat. 'Stumpy form.' $\times 840$.

Note few chromatic granules in parasite situated anterior to the macronucleus.

FIG. 4. *Trypanosoma gambiense*, from the blood of a rat, one week before death. Exp. XXVII. $\times 840$.

Note marked chromatic stippling of the protoplasm of the parasite.

PLATE III

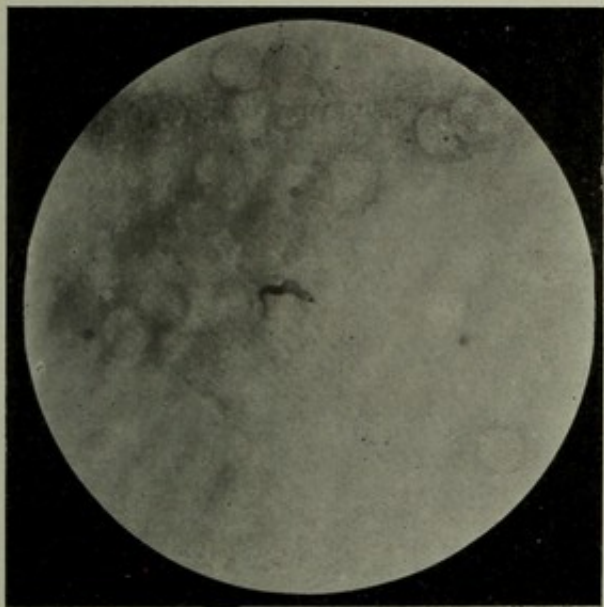


Fig. 1

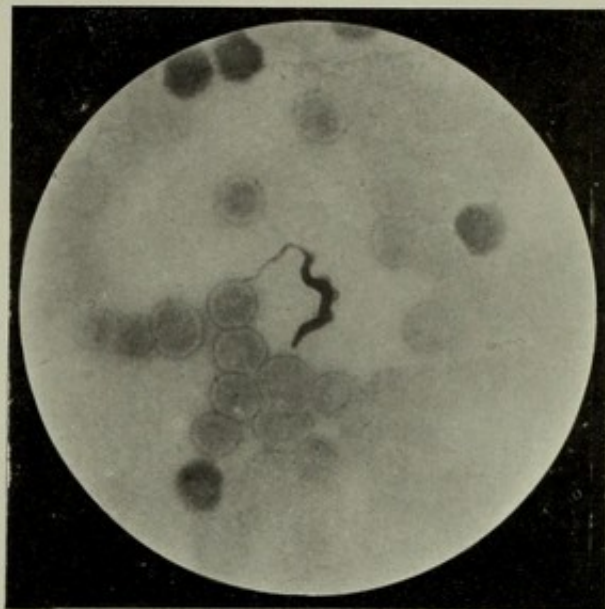


Fig. 2



Fig. 3

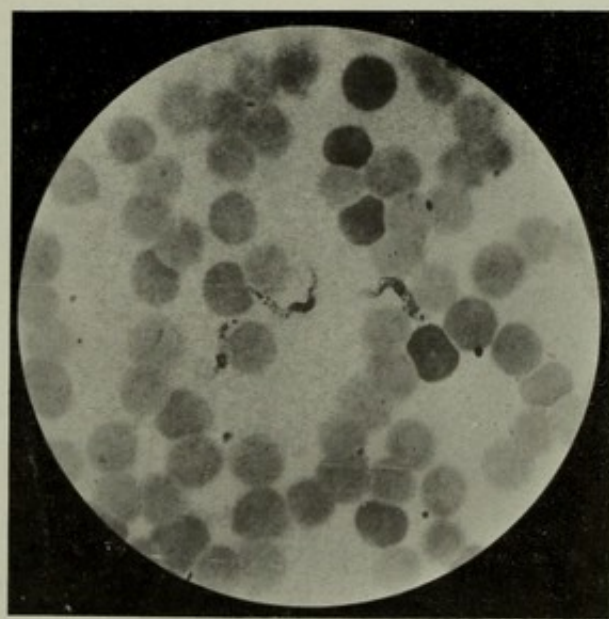
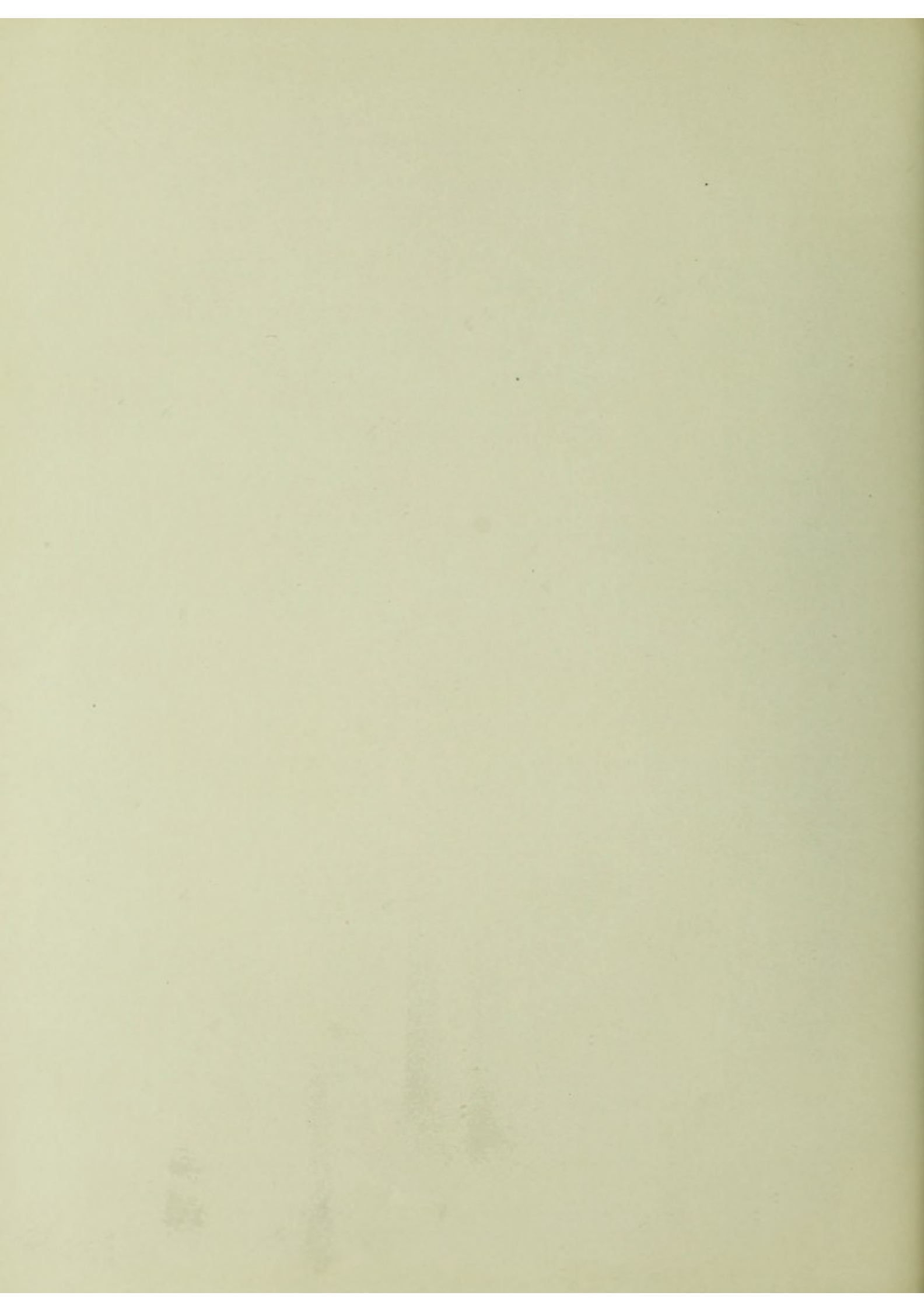


Fig. 4



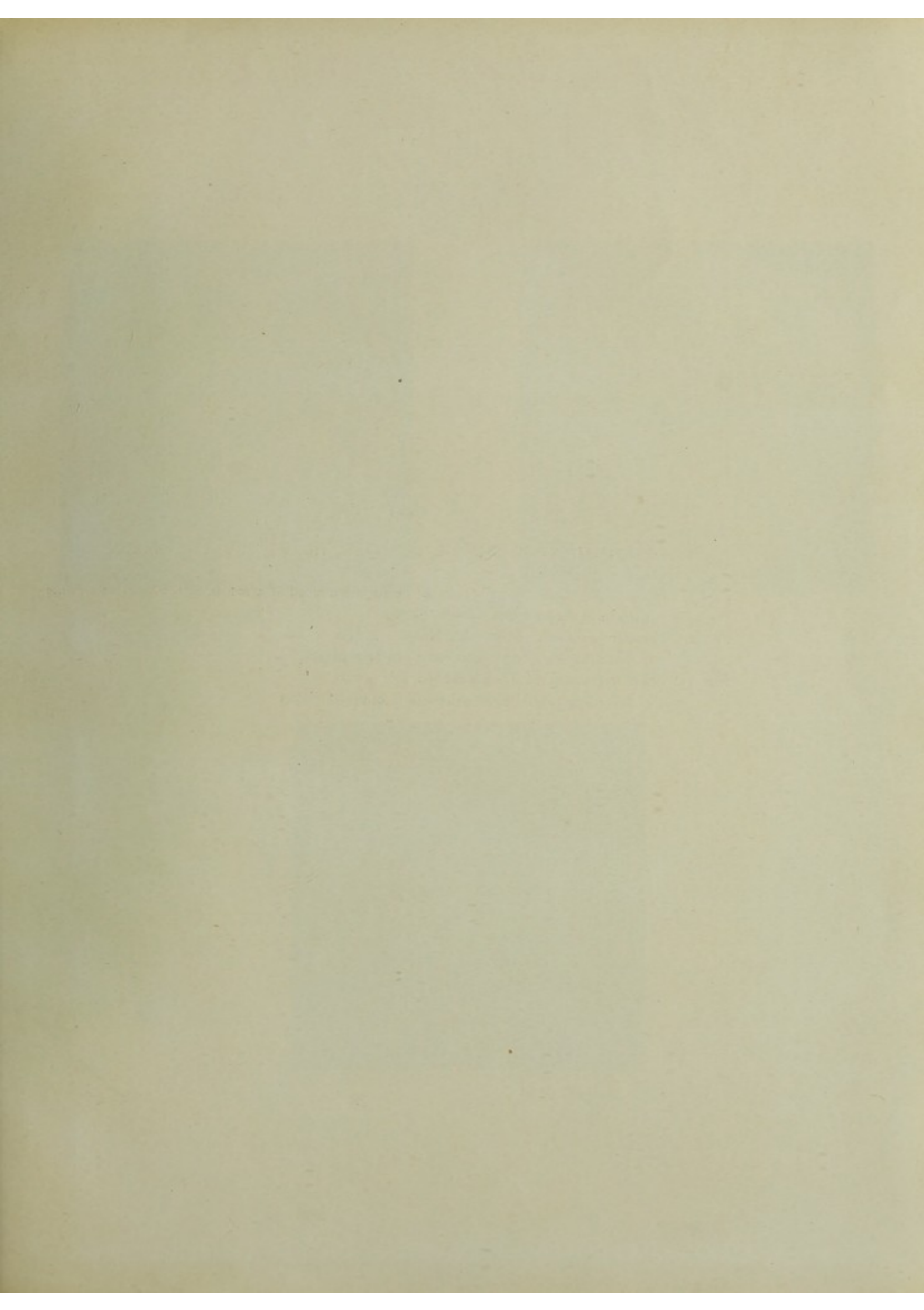


PLATE IV

PHOTOMICROGRAPHS OF GAMBIAN HORSE TRYPANOSOMES

- FIG. 1. Horse trypanosome. Small 'tadpole' forms in the blood of a mouse on the appearance of the parasite in the peripheral blood. $\times 840$
- FIG. 2. Horse trypanosome. From horse No. 1. $\times 840$.
Note the two stumpy parasites in centre of photo.
- FIG. 3. Horse trypanosome. From horse No. 1. $\times 840$
Note longitudinal divisional forms in centre of photo.

PLATE IV



Fig. 1

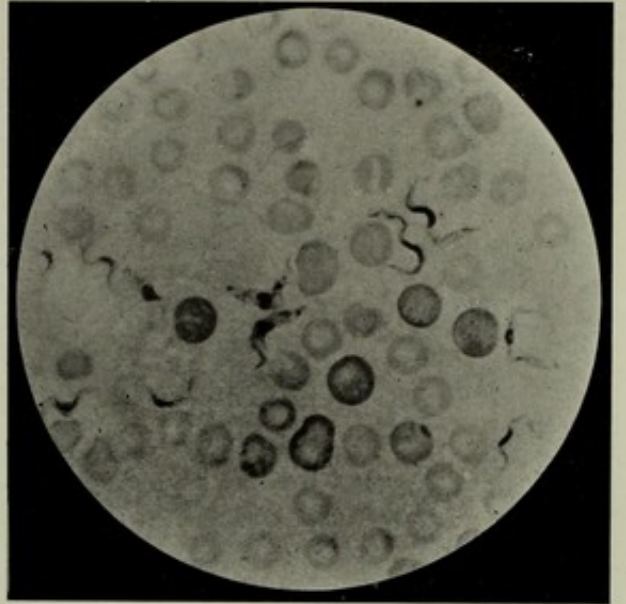


Fig. 2

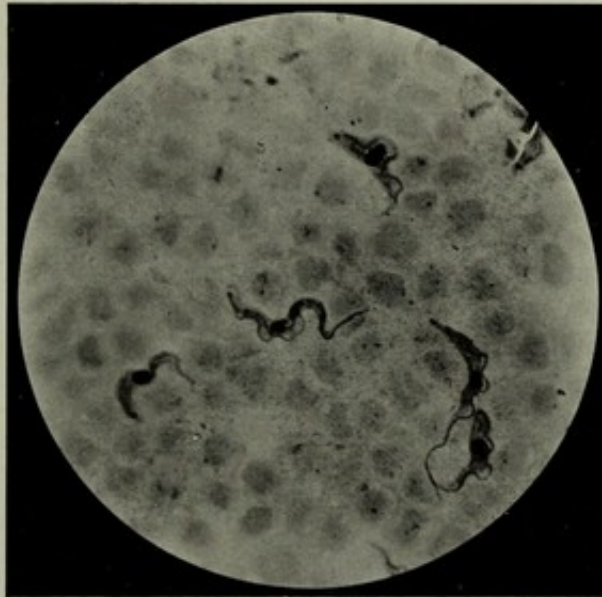
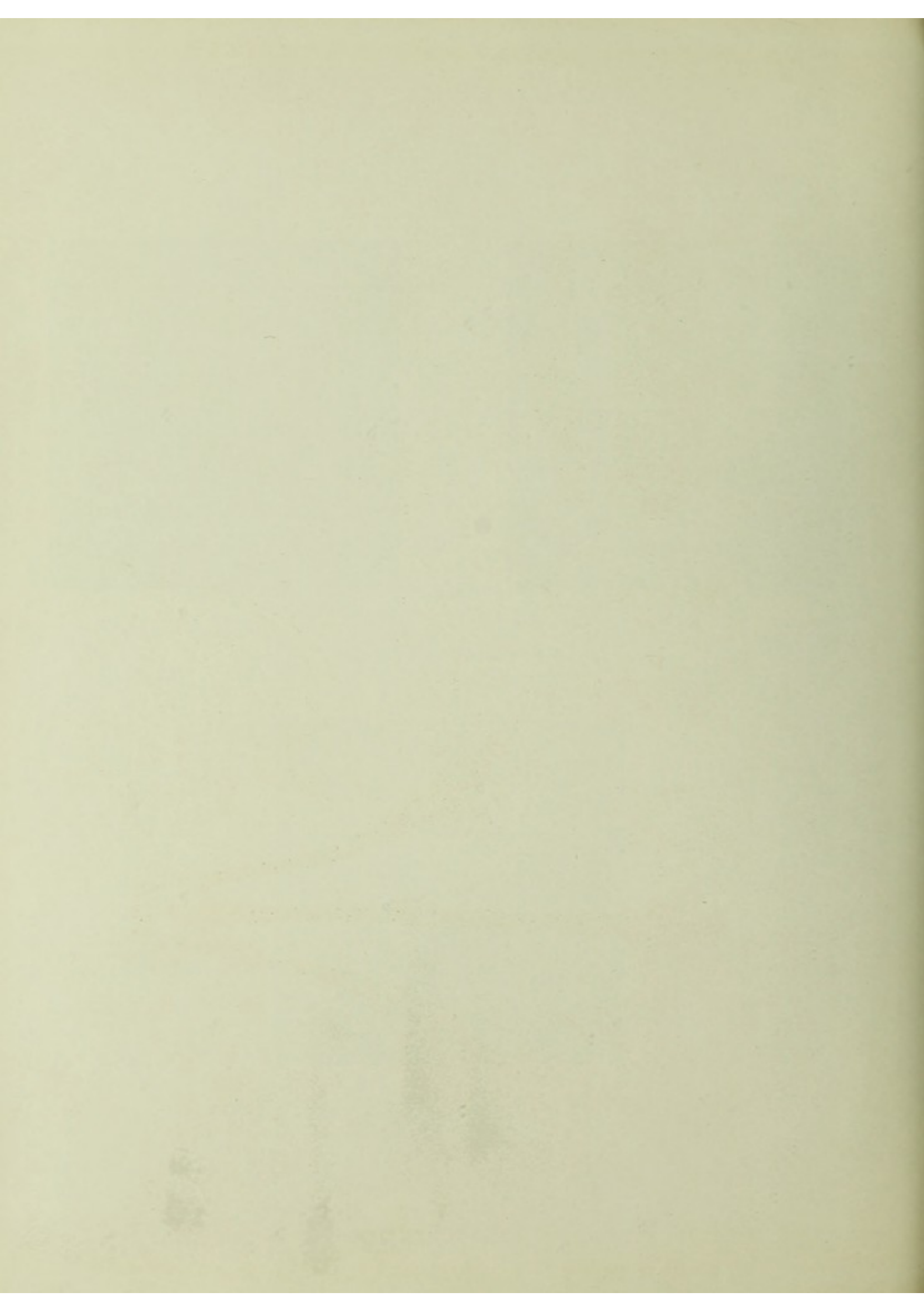


Fig. 3



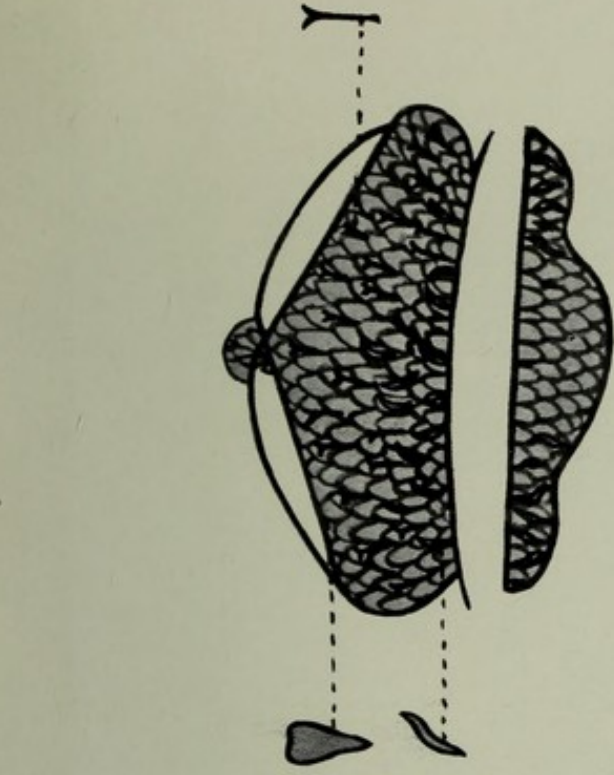


Fig. 1

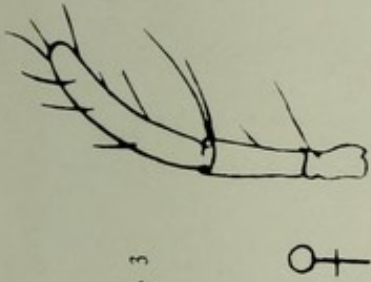


Fig. 3

♀



Fig. 2

♀

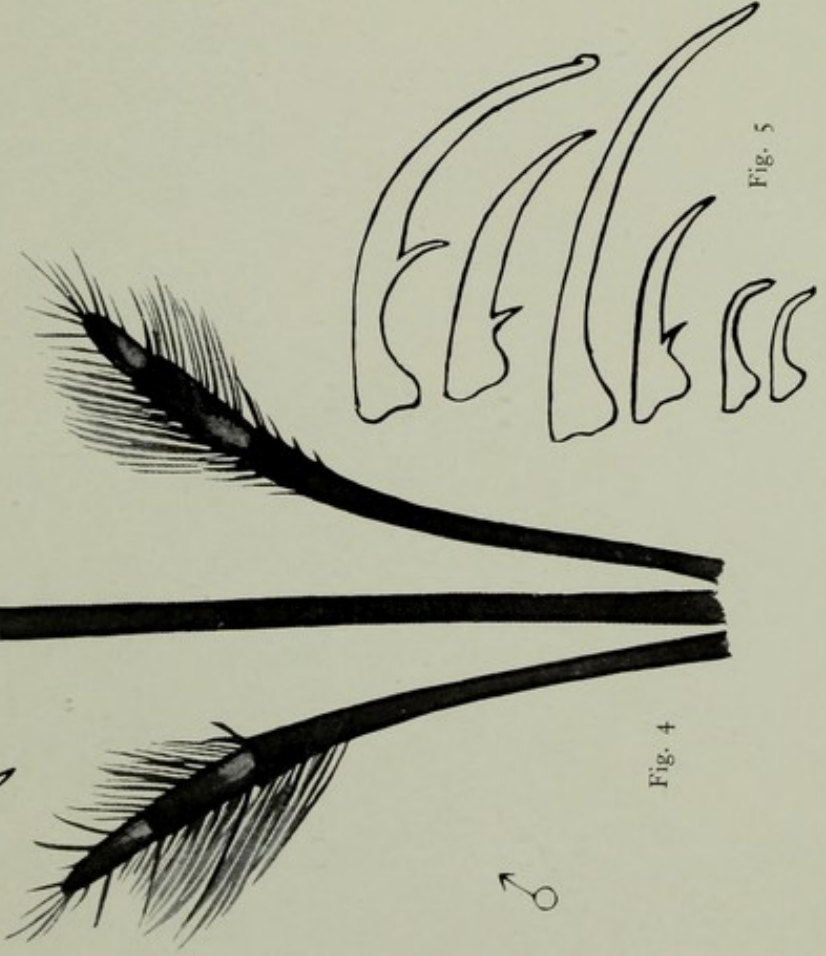
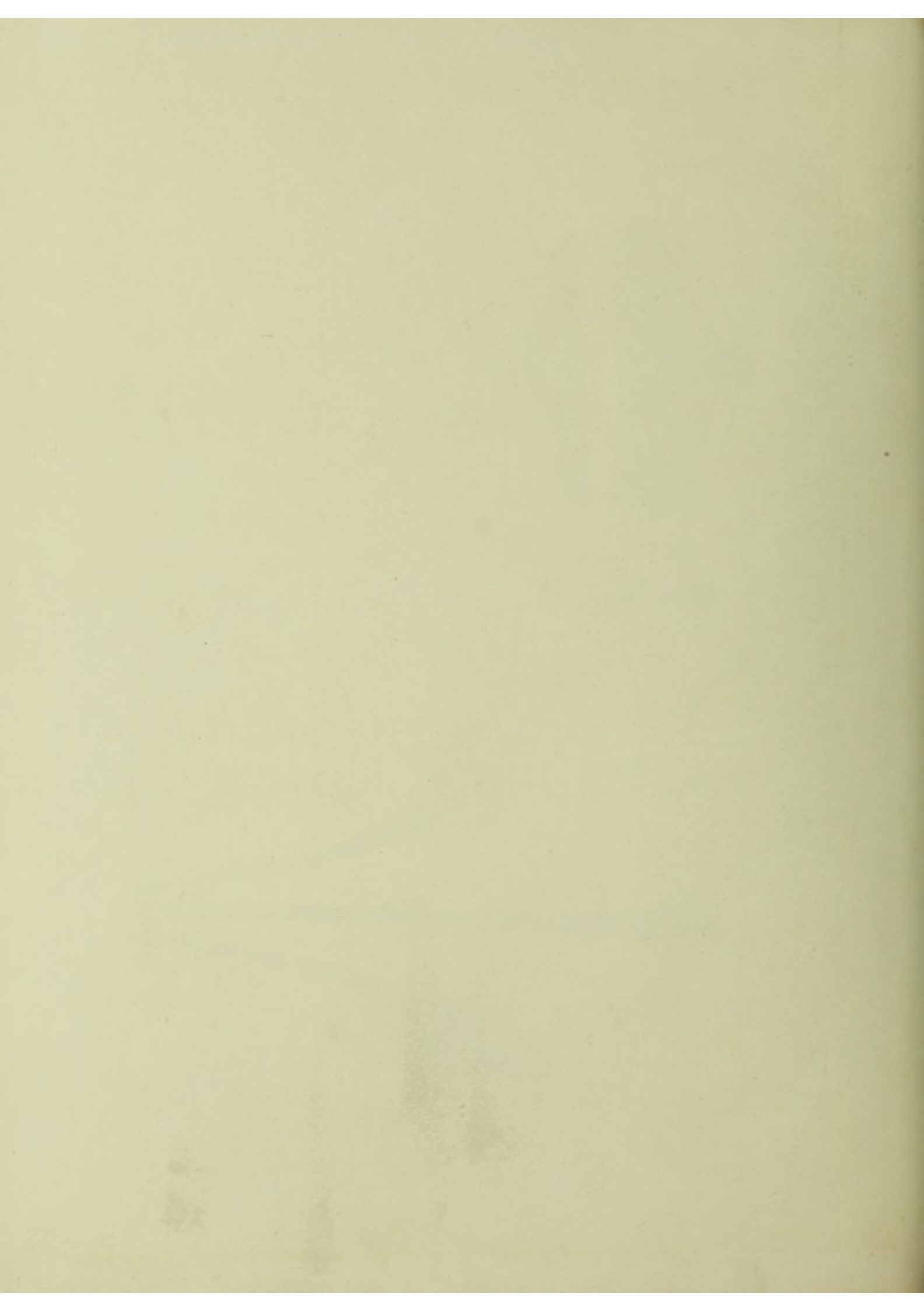


Fig. 4

♂

Fig. 5

Fig. 1. *Cataglyphis senegalensis* (head ♀). Fig. 3. Palpus ♀ × 60.
 Fig. 2. Ungues ♀. Fig. 4. Palpi ♂. × 80.
 Fig. 5. Ungues ♂.



DISEASE.

Exp. XXIV

Notes on Case

Name: Chapman

Age: 9

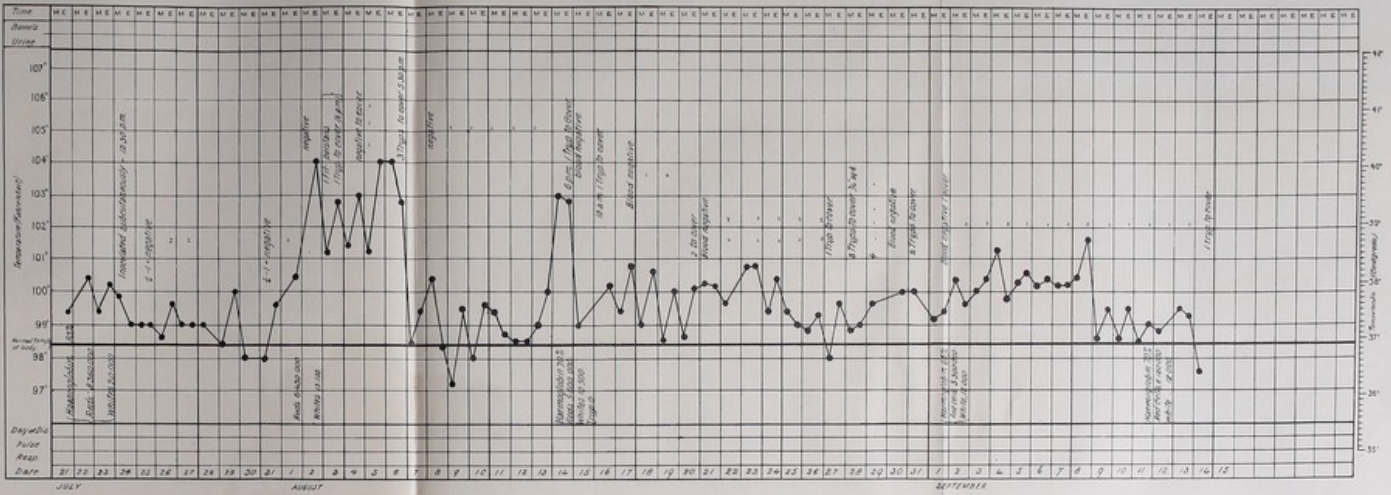
Sex: M

Case Book No.

Inoculated with 2 cc of blood from rats. Exp. CV & FV on July 22 & 23 subsequently. Note in case of blood inoculated in one cover / Trip to field

Date of Admission

Result



DISEASE.

Notes on Case

Name: EXP LXXXVII

Age: 10

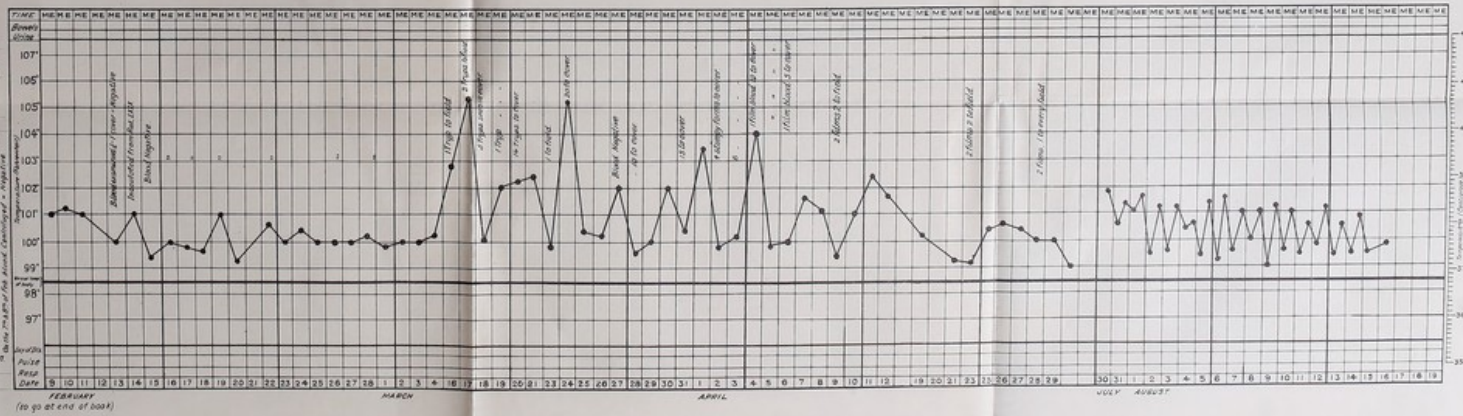
Sex: M

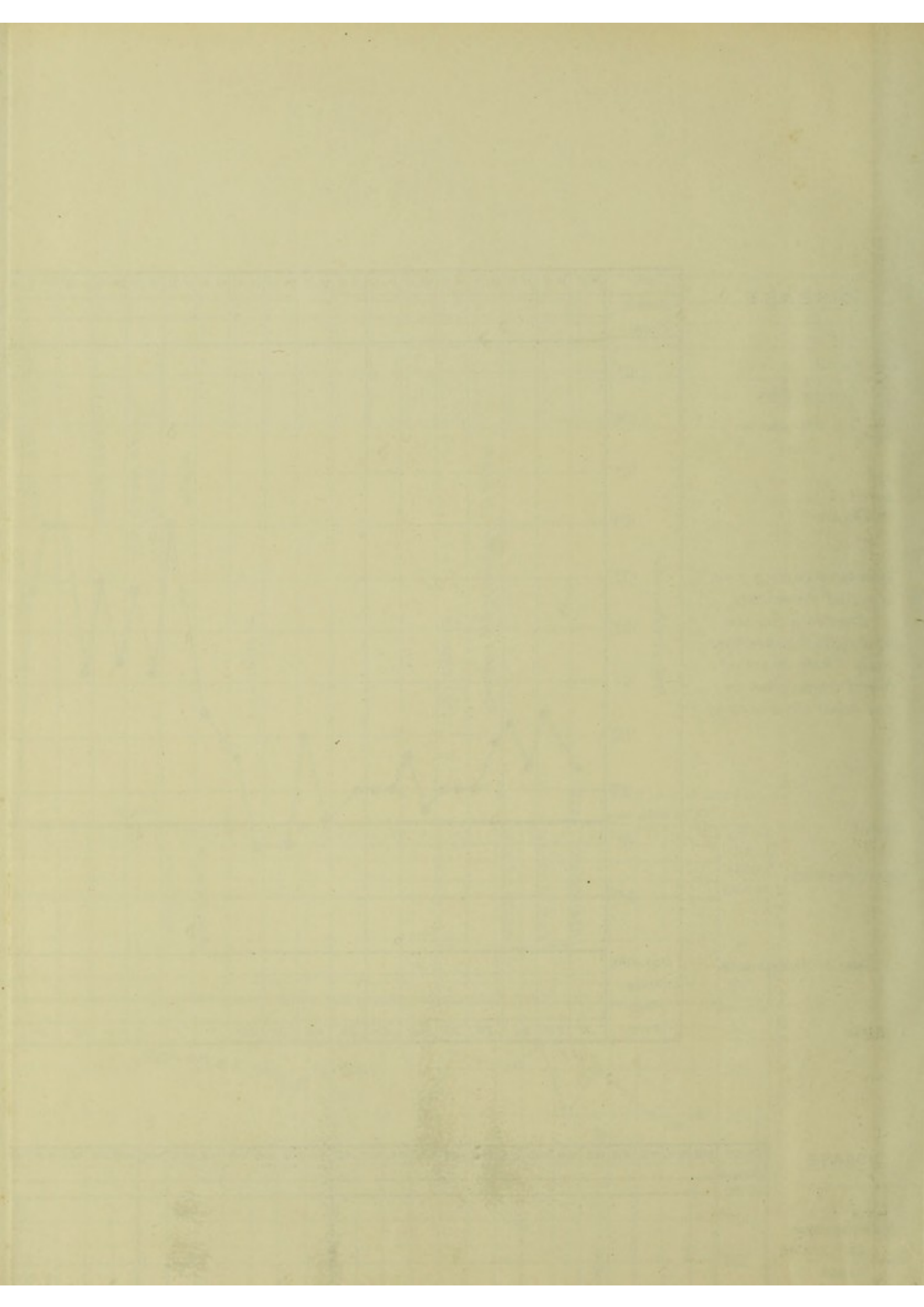
Case Book No.

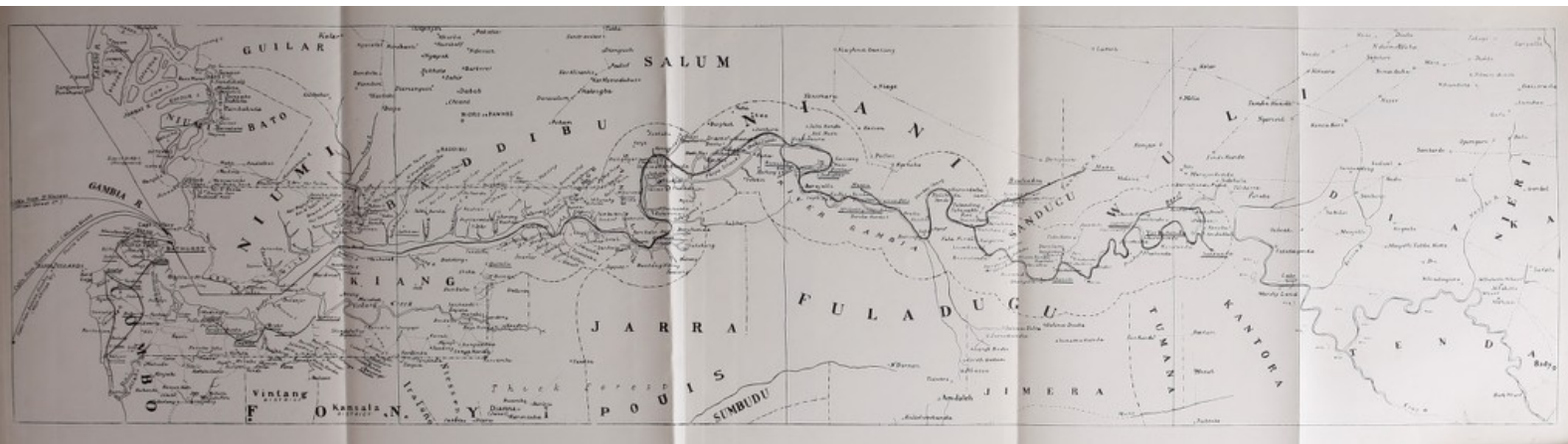
Inoculated lower cellular tissue of left ear with 2 cc. blood from rat on Feb 14

Date of Admission

Result







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