THESIS

ON

Blackwater Fever and Epidemic Cerebro-spinal Meningitis in Northern Nigeria

BY

Mr. G. R. TWOMEY, M.B. & C.M.

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P R E F A C E.

This Thesis consists of two parts, the first embodying the writer's experience of Blackwater Fever in Northern Nigeria, and a survey of the general history of the disease up to the present time; the second describing an epidemic of Cerebrospinal Meningitis which occurred in Zungeru, Northern Nigeria, in 1905. The first part of the Thesis is but just completed and has not been published. The second part, dealing with Cerebrospinal Meningitis was published in 1905 as portion of a Colonial Office Report.

G. R. T.

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Introductory.

Blackwater or Haemoglobinuric Fever has of late years become a subject of high interest to this country which is so largely employed in opening up tropical and subtropical lands to European influences and the benefits of Western civilization. It forms, with malaria, the principal barrier to the administration of numerous colonies and Protectorates on the West Coast of Africa, with which this thesis is chiefly concerned; and has long been recognised and rampant and a source of dread to Europeans in that part of the world. It is, more than any other disease responsible for death, and permanent invaliding among traders and government officials in Northern Nigeria, where the writer has gained his experience of the disease during the last seven years in the service of the Protectorate Government.

As this country was not effectively occupied

until 1900, when it was taken over from the Royal Niger Company, by the Imperial Government, the conditions influencing the occurrence of "blackwater" are as yet but little changed in many places, where Europeans live under more or less primitive conditions.

Definition.

Blackwater or Haemoglobinuric fever is an acute disease, characterised by the occurrence of fever, haemoglobinuria, jaundice, nausea or vomiting.

Recognition.

The disease appears to have been recognised first by some French Naval Surgeons who were stationed at Nossi Bé, a French settlement off the North Western Coast of Madagascar(1), and Bérenger Féraud is quoted by Plehn(2) as stating that it existed in West Africa as early as 1820. In British Territory Easmon described a case in 1885(3) In 1893 Sir, then Dr. Patrick Manson read a paper on the subject before the Epidemiological Society(4) which gave the study of "blackwater" a much needed fillip, and since then the literature of the disease has grown apace, and it has become one of the serious problems which the schools have to solve.

Geographical Distribution.

It is now known to be prevalent in West Africa, both on the Coast, in the Hinterlands even up to the 13th degree of latitude; in all the European possessions from the Congo to the Gambia. It also exists in East Africa, South Africa, and the tropics of North and South America; in India and the Malayan Archipelago, and of late has been described in Greece(5) and Sicily(6) and Calabria.(7)

Aetiology.

Although the consensus of opinion among those who have to treat cases of "blackwater" tends towards a malarial origin, there are still many who advocate the theory that it is a severe manifestation of malarial fever itself. The opinion that it is due to a specific organism is held by some and is strongly asserted by Dr. Sambon(8) as a very probable cause. And, finally, blackwater fever is ascribed to the toxic action of quinine on the red blood cells.

1. Although the malarial origin of the disease can hardly as yet be said to be established, there is a very large amount of evidence in favour of this view, and a careful consideration of the facts,

goes far to convince one of its soundness. Crosse, in an article in the Lancet 1899, unconsciously supports this view in holding that the disease is in some way due to the turning of soil as planting and building progressed in the Niger Territories. The turning up of soil, as is now pretty generally conceded, established favourable conditions for the propagation of Anophiles, and, consequently, the increased incidence of malaria. These conditions likewise appeared to favour the incidence of blackwater fever, and the bulk of cases seemed to occur on or near the plantations. Prior to this time Europeans lived on hulks moored in the Niger and the creeks which penetrate its delta. These boats could be kept in a comparatively sanitary condition and malaria was less common and severe than later when white men lived ashore under conditions of great hardship, and where they were being continuously infected by the swarms of gnats bred in the numerous pools of stagnant water around the houses and scattered through the plantations. One gathers that "blackwater" spread pari passu with the cultivation, and not in proportion to the increase of European population. Crosse also noticed that the disease was most prevalent when the dry season was hot and prolonged, and pools and lagoons dried up.(10)

Easmon stated that he had four cases of blackwater fever in 1881 at Quetta where the lagoon dried up.(11) Railway construction in the tropics, another potent cause of malarial outbreaks, always exacts its mead of human life through blackwater. In support of this one has only to recall the outbreak among the Chinese on the Congo Railway, and the long list of blackwater invalids for which the new Nigerian railways are responsible. One must not forget how the cutting of canals in malarious regions as Panama, and the Isthmus of Corinth gave rise to cases of this disease, and how the new tropical towns, except when made in very exceptional sites, are invariably unhealthy. I have many such in my mind as I write: Northern Nigerian settlements which for reasons of expediency, chiefly of a military nature, had to be placed on sites unfavourable to health, and where the aftermath of malaria was leavened with "blackwater". The chief town of N. Nigeria, Zungeru, is a case in point. It was, some eight years ago, when founded by us, a hotbed of malaria and had an unenviable reputation for cases of "blackwater", but as the town was drained and sanitation got the upper hand malaria became less severe and pari passu blackwater decreased until at present cases are rare, and, when they do

occur, are generally ascribable to outside causes. For purposes of comparison may be cited the cases of Hokito and Zaria, two stations built on very porous soils, where malaria is not uncommon but is not usually of a severe nature among Europeans. Here as one would expect, if the theory of malarial origin be correct, blackwater should be rare. And it is. In fact the only case which has occurred in these stations - Case 5, described later - was to the best of my belief imported, and was the patient's second attack.

It was thought formerly that malaria and "blackwater" geographical areas should coincide if this theory were to hold, and although much of the tropics still remains to be examined in this respect, it is being gradually shown that all intensely malarial regions produce cases of blackwater fever. Before the publication of the researches of Christopher and Bentley in the Duars in India, and the recognition of the same disease in Calabria and Sicily by Italian physicians, and in the Isthmus of Corinth by Greeks, one might feel doubts of the geographical coindidence of these diseases, but now there remains but little room for doubt.

It is also noticable that precautionary

measures such as the administration of quinine daily, tends to lessen the liability to contract "blackwater" fever. In support of this I may quote the opinions of Drs. Thompstone and Fagan, the Principal Medical Officer and Deputy Principal Medical Officer of N. Nigeria. Two of my own cases in the Zungeru European hospital confessed to carelessness in this respect, and two patients who died, one in Lokoja and one in Zungeru in 1908 were known to take quinine only very irregularly. All these patients suffered frequently from malarial fever. All these facts tend to impress one with the close connection between malaria and the disease under consideration. I have dwelt on the subject at some length, since, it seems to indicate that the best results in the prevention of "blackwater" will follow a vigorous antimalarial campaign.

2. The second view, that hæmoglobinuric fever is an intense attack of malaria does not appeal strongly to anybody who has seen much of the former. The symptoms of "blackwater" and malaria are so different that one can hardly conceive that they indicate the same form of illness. There is not really any more in common between them than pyrexia and nausea which though often absent in malaria is always

present in blackwater. A pyrexia is not unknown in the latter, but I believe a case of malaria without a rise of temperature has yet to be recorded. Undoubtedly the parasite of malaria is frequently found in these cases, and its presence or absence is a guide to treatment, but "blackwater" having set in, malarial symptoms appear to be completely superseded, and parasites rapidly disappear from the blood even where it is thought advisable to administer quinine.

3. Another theory is that the disease is due to the toxic effects of quinine; and this view seems to have a fascination for non-medical Europeans, in Northern Nigeria at all events. So much so that it is occasionally a source of anxiety to those who are responsible for the efficiency of the quinine prophylaxis of malaria. In this respect my remarks, with reference to the neglect of the precautionary doses of quinine, already alluded to, as practised by Europeans who have contracted blackwater subsequently, may here be repeated. And, it is further noted that in the converse case, of Europeans strictly observing prophylactic precautions by taking 5 grains of quinine daily, "blackwater" is of rare occurrence, and it is then probably due to exposure to unusually intense malarious conditions.

This was, I believe, the cause of death in the case of a Major M. who told me that he never omitted to take his daily 5 grains. He died, on February 10, up the River Benue where malarious conditions and "blackwater" are very prevalent. If one considered that the administration of quinine brought on an attack of blackwater one would hesitate to use it even in cases where parasites are found in the blood. But, on the contrary, I think the usual practice is to give quinine in small doses in these circumstances, and, in my own experience, at any rate, with good results.

Whether or not quinine can cause hæmoglobinuria is apparently still a moot point, and I should hesitate to say that large doses may not have this effect in certain persons. Thus Manson's case of a patient in the Seamen's Hospital, who undoubtedly had an attack of this nature on the exhibition of quinine even in small doses,(12) and similarly a mild attack of Hæmoglobinuria which Dr. M. -(Senior Medical Officer N. Nigeria) described to me as following the exhibition of 40 grains of quinine bisulphate in a few hours, when he tried to abort an attack of malaria so as to keep up with a column of troops,

seem to point to some such possibility. As against these may be mentioned the case of a Ward Boy who died from the toxic effects of some 200 grains of quinine sulphate which he mistook for magnesium sulpahte.(13) Even this large dose did not cause hæmoglobinuria.

The subject of the action of quinine as a hæmolysin is dealt with below under The Origin of Hæmoglobinuria.

4. The last hypothesis, which is supported so strongly by Sambon, is the possibility of this disease being due to a specific micro-organism: of the nature of a piro-plasmosis.(14) He also holds that it is epidemic among natives in Central Africa. Its epidemicity would point to such a specific origin, but this can be equally well explained by the presence of very malarious conditions. Blackwater in N. Nigeria can never be said to be epidemic among Europeans, and I believe cases among natives have not so far beenrecorded. Certain stations have certainly gained an evil reputation for the frequency with which the disease occurs, but these are intensely malarious places, and the disease does not seem to spread to other stations as one would expect in an epidemic, more especially as no special precautions are taken

to check its progress, beyond ordinary antimalarial measures. Sambon further described certain pear shaped bodies appearing in the red blood corpuscles in cases of "blackwater". If the recognition of these bodies were general the "specific" theory would become a subject for serious consideration, but as only a few observers have seen them, and a host of competent observers including Christophers and Bentley, who searched so carefully for piroplasma like organisms in the Duars,(15) have found none, one can only conclude that their presence is in no way related to blackwater fever.

Then, as Christophers and Bentley remark, the symptoms of blackwater are likely to be mistaken for the symptoms of malignant jaundice in dogs, Redwater in cattle, and Carceag in sheep; so that the analogy with diseases of animals failing, the "specific" view must fall back on the possibility of the existence of an ultra-microscopic cause.

That Blackwater is due to the ubiquitous faba vulgaris need only be mentioned to be dismissed, since numerous cases have occurred among people who are known not to be bean eaters.

Predisposing Factors.

1. Race. In Northern Nigeria we have to deal with Europeans, East Indians, and natives of West Africa. In this Protectorate the disease is chiefly confined to the first two races, and cases have occurred less frequently among non-natives of N. Nigeria, i.e. natives of the Coast Colonies. The largest proportion of cases occur among Europeans. The Indians are chiefly naiks and artisans who are employed in the Transport department, and clerks who were formerly much more numerous than now, and who are all Bengalis and are employed in Zungeru, the Government head-quarters. All the Blackwater among the Asiatics has occurred among those employed in the Transport department, and further only among such as follow the bullock wagons up and down country, and are more exposed to malarious influence than their fellow Indians. It is curious how the transplantation of a race renders it susceptible to malarious influences, because although these people confessed to frequent "fever and ague" in India, none of them had previously contracted "blackwater". The natives of N. Nigeria are not susceptible living as they are in their normal environment; but, as stated, natives from the coast are occasionally attacked. Similarly

about 1905 it broke out among Chinese labourers employed in the Congo, and Africans employed in the Panama works died from the same disease.

2. Sex. It is difficult to come to any conclusion on the relation of sex to blackwater, since European women are generally better housed and are less exposed to malarious influences than men. Further they do not generally remain more than a couple of tours, i.e. about two years if in the Government service; and if they are wives of officials and traders they are generally sent home to England when the rains set in. Women, further, are more careful in their methods of living, so it is extremely difficult to compare the susceptibilities of the two sexes. So far in N. Nigeria but one case of blackwater has been recorded among women - a fatal case, unfortunately. But I understand that the disease is by no means so uncommon among the female missionaries in Southern Nigeria, who live under very much less healthy conditions than white women in Northern Nigeria.

3. Age. It occurs at any age, roughly, between 20 and 45, which are about the limits of the ages of the bulk of those employed in the Protectorate.

and of these the younger members are engaged as clerks and assistants in trading firms, and employed by the Goverment on the new railway works. I travelled down the Niger this year with a young man of some 22 years who was being invalided on account of blackwater contracted on the railway line, where he was employed as foreman. There are many records in the hospitals of Zungeru and Lokoja of cases occurring between the ages of 25 and 30 years. Most cases occur in men between 30 and 35 years of age, which is probably accounted for by the fact that between these years the majority of officials are in their third tour, i.e. spending their third year in West Africa.

4. Length of Residence. Length of residence is an important factor in the determination of an attack, and in N. Nigeria Europeans are rarely attacked in their first tour - generally of twleve months. The majority after this period spend four or five months in England and then return for a second tour. It is generally towards the end of this second tour, or during the third that blackwater manifests itself in those who are attacked by it; that is to say, roughly, during the third year of residence in the tropics. Before and after this

period cases are fewer, and are generally traceable to residence in some malarious environment, other than N. Nigeria. Bérenger Féraud gives the following figures - Of 100 cases in Senegambia where conditions of life appear to be very similar to N. Nigeria 5.4 cases occurred during the first year of residence in the tropics, 22.5 during the second and 42.5 during the third, and a gradual decline is shown in the figures for the succeeding years. I have heard of but one case in N. Nigeria occurring in a person under one year's residence, viz. that of a young military officer, Lieut. F. who died of the disease when travelling from Zungeru to Sokoto at the end of the rains, in September 1909, notoriously the most unhealthy period of the whole year. This young officer had not been in the tropics before and had spent but some eight months in Nigeria. The journey alluded to was made for a great part up the "fadamas" or swampy cattle pastures, and no precautions could prevent continuous malarial infection.

Symptoms.

From a comparison of West African cases with those occurring in other countries the symptomatology appears to be fairly constant. The

case variation is very wide but there are certain manifestations which are present in every instance. These are nausea and vomiting of bright green bile, the presence of haemoglobin in the urine rendering it from a light red to porter colour, and a greenish yellow jaundice which persists for some days after other signs have disappeared.

The onset of the disease is rather abrupt though there is always a history of feverishness and malaise for at least a day before "blackwater" manifests itself, and in every case the writer has treated or seen there has been a rigor within the twenty four hours preceding the passage of haemoglobinous urine. The patient rarely believes after the fever and rigor that he is in for anything worse that one of his "goes" of malaria, and does not as a rule send for the doctor until the colour of his urine, or the growing yellow tinge of his skin arrests his attention. The first coloured urine passed is generally of a port wine colour viewed against a light, though it looks almost black lying in an opaque vessel. The icterus sets in some hours later and deepens to a median degree of intensity, never being so marked as in obstructive jaundice. Even on this first day the patient loses

strength rapidly. His face becomes thin, drawn and bloodless, and he seems hopeless and apathetic, refuses all nourishment and is worn from continuous retching and vomiting. The vomited matter after the stomach is cleared of food consists of a fluid coloured bright green by fine particles. This is the most distressing of all the symptoms, and generally the most difficult to deal with. The temperature on the evening of the first day usually rises to 104°F, and the first appearance of hæmoglobin is coincident with thisrise. He generally complains of pain and tenderness over the regions of the spleen and liver, both of which organs are generally, and the spleen always, enlarged. The presence of malarial parasites in the blood is sometimes determined, but as a rule they are absent. The blood itself after the first day becomes very thin and light coloured, and the corpuscles fall in number to about three millions per c.m.

On the second day the urine is a deep red in the morning, and begins to clear as the day passes. At the end of the second day it is usually nearly clear. If the urine does not begin to clear in 48 hours the prognosis is very grave indeed in Northern Nigeria experience. The temperature is

generally down to 100° or 101° in the morning and rises again in the evening to about 102° or 103°. The jaundice is now pronounced and the vomiting, sleeplessness, and blood destruction has reduced the patient to a low ebb - but as the day wears on the urine becomes clearer, the nausea tends to disappear and the sleep of exhaustion sets in, the patient waking flaccid and weak but on the road to recovery. The temperature afterwards subsides gradually, the jaundice slowly disappears, and in about a week he may be considered convalescent.

That is a picture of a mild case of "blackwater," and many such occur in N. Nigeria; but frequently relapses occur, and the hæmoglobinuria and other symptoms recur once or twice with an interval of a day or more between the various recrudescences. In these cases the continuous strain tends to a fatal issue on account of blood destruction, insomnia, and the prostration caused by constant vomiting. Such cases generally become delirious with the first relapse.

Rarer, fortunately, are those cases of another type where the hæmoglobinuria persists after the second day, and becomes gradually more scanty and deeper in colour, altering in consistency

until it is syrupy, and finally ceasing. Here the patient rapidly sinks into a typhoid state, hiccoughs painfully and breathes with difficulty. His blood count may reveal but one million corpuscles per cm. and the last urine passed is so laden with solids that after heating it will not fall out of an inverted test tube. The patient gradually sinks into a comatose state, and dies without recovering consciousness.

Finally may be mentioned cases in which the patient recovers from his blackwater and later succumbs to some complication such as nephritis and cases in which some concurrent disease, such as anæmia, hepatic or cardiac trouble, complicates the treatment and lessens the patient's chances of recovery.

Of these four types of hæmoglobinuric fever the second is the commonest in Northern Nigeria and from what I can gather - no statistics of the types being published - the third is the rarest.

The Urine. The urine passed just prior to the doctor's visit is generally of a characteristic port wine colour when viewed through a large test tube or Nessler's glass against the light. There is no mistaking the colour for that

of simple febrile urine. It may at times during the progress of the disease become light and transparent, or dark and opaque like porter. A spectroscope aids early diagnosis in revealing the bands of hæmoglobin long before it can be detected by the unaided eye.

If the urine be allowed to stand for some hours a brown precipitate of a granular nature is deposited, consisting of hæmoglobinous and hyaline tube casts, a few red blood corpuscles sometimes, and granular débris. If it be heated a brown coarsely granular precipitate is formed which settling down may occupy from 10% to 80% of the volume of urine, and in some cases completely replaces it so that it will not fall out on inversion of the tube. Treated with nitric acid in the cold a ropy light brown precipitate is formed, which as in the case of boiling may occupy from about 10% to the whole volume of the urine. In this and the preceding case a pale supernatant urine is left after the precipitate settles.

It is of interest to note that the quantity of this albuminous deposit does not seem to bear any relation to the severity of the disease. I have seen a deposit of 80% frequently in cases

which showed no unusual symptoms, and were of a common uncomplicated nature.

The Quantity of the Urine. The quantity of urine passed in an ordinary case is generally about the normal, and, indeed, is frequently above forty ounces daily.

In certain cases, however, it tends to diminish temporarily. It may fall to 10 or 12 ozs. in a day, and then the flow may be re-established at the normal figure.

In cases, which are nearly always fatal, it becomes gradually less, and of a syrupy or gummy consistency and if the patient live long enough may cease altogether.

Origin of Hæmoglobinuria.

A large number of theories have been put forward to account for the hæmoglobinuria which may be recounted for the sake of completeness though I do not propose to dwell on them in a treatise dealing more particularly with the clinical aspects of the disease in West Africa.

1. The earliest theories were concerned with the excessive hæmoglobinæmia where the liver being incompetent to deal with all, part was

excreted by the kidneys.

2. Again, degeneration of the liver cells caused a similar result.

3. Daniels suggests, and Manson supports tentatively the possibility of a peculiarly virulent kind of malarial parasite[18] being the cause, having as its host a special variety of mosquito.

4. Grocco thinks that malarial toxins may be formed in excess - under stimulation by quinine - and cause liberation of hæmoglobin.[19]

5. Stephens and Christophers thought that solution of quinine above certain strengths might hæmolyze red blood corpuscles. This is 0.001 to 1 c.m. of red blood corpuscles at 37°C., and 0.008 to the same in blackwater blood., the salt used being neutral quinine hydrochloride.

6. Marchoux holds that quinine is not excreted by the kidneys until blackwater is subsiding, and its cumulative effect may cause hæmolysis.

7. Bensaude thinks quinine hæmolyzes infected red cells, and that the Hæmoglobin become methæmoglobin and is excreted as a foreign substance.

8. Some French writers think that reduction of sodium chloride - "dimineralization of the plasma" - is destructive to red blood corpuscles.

9. McCay found that after a single dose of quinine sulphate the hæmolozic value of the plasma was reduced owing, he says, to the action of the acid radical, and hæmolysis resulted.

10. Christophers and Bentley as a result of their investigations in India consider that a condition which they call lysæmia is produced by the action of a special hæmolysin produced in some way in connection with malaria. They do not think it to be a toxin produced by the malarial parasites but in some way the result of the long continued phagocytosis, and storing up in the organs of the body of infected red cells.

This would account for the influence of length of residence in determining the production of blackwater fever, and the seeming paradox of apparently healthy and robust Europeans being occasionally attacked, because being "able to make good the loss of his red cells the actual infection by parasites affects the well fed European or better class native but moderately, and the very power of reaction which we know to be associated with healthy

rather than diseased conditions may be his undoing".(20)

11. This brings us up to the latest investigations regarding the origin and method of production of Hæmoglobinuria, the researches of Barratt and Yorke in Nyassaland.(21) They decided that quinine as the alkaloid, and in the form of the bihydrochloride; and also hydrochloric acid and sodium hydrate can all cause hæmolysis of red cells, producing hæmoglobinæmia and hæmoglobinuria. But they also state that quinine owing to its toxicity cannot be so concentrated in the body as to cause hæmolysis, and thus there are no great changes in the blood as resulting from quinine in blackwater fever if doses do not exceed 15½ grains - a very important fact from a therapeutic aspect.

They further discovered that the hæmolysin concerned is not in the blood plasma.

The existence of Hæmoglobinæmia - a matter of some doubt - was a further subject for study though Ponfick stated that when $\frac{1}{60}$ of all the hæmoglobin in the blood was set free this condition was established. If less than one-sixtieth was set free the liver was competent to deal with, and causes its excretion as bile.(22)

Barratt and Yorke arrived at the conclusions that there is hæmoglobinæmia in health,

and that when hæmoglobinæmia occurs in rabbits hæmoglobinuria rapidly succeeds, and the percentage of hæmoglobin in the urine rapidly exceeds that in the plasma, and falls in amount as the latter is diminished. They hold then that they have established the fact that hæmoglobinuria is dependent on hæmoglobinæmia.

The increase in the percentage of Hæmoglobin in the urine may possibly be due to the fact that urine can break up hæmoglobin whether in red cells and in solution and that occasionally blood cells are found in the urine in blackwater fever.

These are the main facts which have been worked out regarding the production of hæmoglobinuria, and I have recorded them at some length because it seems probable that by working on these lines the true cause of the disease will be discovered.

Blood Changes.

Investigations into the microscopic character of the blood changes in Blackwater Fever may still be said to be in an early stage. New forms such as shadow cells large and small, faintly defined bodies appearing soon after the onset and

during the convalescent stages are seen. Polychromatic changes are seen, said to be indicative of regeneration. Cells of perfect outline and smaller than red blood corpuscles, called spherocytes are described. Blood plaques containing what appear to be broken down corpuscles, or masses of hæmoglobin are found in the blood of the splenic, portal, and renal veins. But so far no definite conclusions have been drawn from all these facts, and they do not indicate any basis for rational treatment of the disease.

Recurrences of Blackwater.

Persons who have contracted Blackwater Fever are extremely liable to recurrent attacks. These may occur some months or years after the first, and even after long residence in England after leaving the tropics. These recurrences must not be confused with relapses, which occur in the course of one attack. Both relapses and recurrences occurred in the persons whose cases are shown as 4 and 5 below. Recurrent attacks tend to become more and more severe, but one occasionally meets persons who have suffered frequently from a very mild variety; and seem to enjoy a certain immunity against severe

forms. It is usual in practice in West Africa to invalid permanently persons who have suffered from a severe attack of Blackwater Fever.

Treatment of Blackwater Fever.

Many people have conceived fatalistic notions concerning blackwater, and consider that treatment has little influence on the prognosis. The scope for rational therapeutics is certainly narrow, but I am convinced that a great many cases are saved by symptomatic treatment, and undoubtedly even in fatal cases the patients can be spared a great deal of discomfort.

Plain uncomplicated cases of the disease it is generally conceded, recover; and if treatment is directed towards the prevention of complications primarily it should improve the patient's chances.

The causes of death are suppression of urine from occlusion of the renal tubules by hæmoglobin, acute nephritis, hyperpyrexia, cardiac failure, blood destruction, and exhaustion. These are the commonest causes.

The guiding principles in treatment should be -

1. Unremitting care in watching and conserving the heart's vigour;
2. The avoidance of chilling influences;
3. The efficient action of the skin, bowels and kidneys;
4. And the support of the patient's strength by the administration of suitable food, in proper quantities, and at appropriate times.

1. Thus absolute rest in bed is essential and freedom from all sources of worry, such as business, noise, strong light, and insect pests. As these cases, in West Africa, occur usually in places where trained nurses are not available, the selection of persons who are agreeable to the patient, to assist in the nursing I have frequently seen to be wise, by observing the results of haphazard selection.

The patient becomes so bloodless in blackwater that it is desirable to give enemata of normal saline solution as early as possible, and to administer some bland fluid such as barley water at frequent intervals if it does not markedly increase the vomiting; or, if diarrhœa and vomiting are severe, the saline fluid can be introduced into the

cellular tissue of the axilla, or flanks. This latter operation should be done very slowly, as otherwise it causes much pain. An enema of one pint given three times daily, and containing about 20 minims of laudanum if not retained easily, should suffice. Of course this presupposes the non-existence of heart or kidney diseases. About 1½ pints may be introduced into the cellular tissue in two sittings.

The pulse has to be watched carefully and occasionally when it becomes rapid and thready 1/100th of a grain of digitalin in tabloid form, or a combination of digitalin and strychnine, by the mouth will not cause vomiting, and will sustain the heart. Brandy is useful but it is apt to cause vomiting. If there is but little vomiting a dose containing 3 minims of the liquor of strychnine, and 3 minims of the tincture of digitalis might be given thrice daily. Hot bottles at the feet will also help to sustain the circulation of the blood.

2. To prevent chill the bed should be out of a draught, and sufficient bed and body clothing to prevent loss of heat, but not to incommode the movements of the patient, should be applied. A roll ofGamgee tissue with tapes for tying around the trunk and two passing over the shoulders is a

good protector for the body and not easily shaken off.

3. Perspiration may be induced by warm clothing, and hot drinks of milk and water, weak tea, or barley water, as well as by placing hot water bottles about the body and limbs. The bowels should be opened by calomel, about 3 grains,- cascara sagrada tabloids, or some such concentrated purgatives, and it is wise to clear out the rectum by an enema as soon as possible. Large initial doses of calomel do not seem to be in such vogue now as formerly.

The purgation can be kept up by daily small doses of sodium or magnesium sulphates .

The free action of the kidneys is all important owing to the tendency of the tubules to become choked with hæmoglobin, and plenty of bland fluid given by the mouth constantly in small quantities and the routine injection of saline enemata will help to sustain the renal functions.

4. Feeding is a matter of difficulty in small stations where food is not in great variety. It is safe to begin with diluted milk in small quantities given every couple of hours, and follow with Benger's Food, junkets, and similar semi-solid forms

of nourishment. It may be necessary to give more concentrated foods owing to the patient's weakness, and then a teaspoonful of Brand's essence occasionally will not exaggerate the emesis. On the third day one can generally give a little chicken soup in uncomplicated cases, and on the next day some milk pudding in addition, and then pass gradually from fish to eggs, chicken, and finally red meat. No routine dietary can be adopted since one patient will tolerate foods which cause emesis in others, Sometimes if the patient cannot take food by the mouth nutrient enemata must be given, and a convenient method is to mix the food such as extract of meat, beef tea, etc. with the saline enema. They may object to being fed per rectum if they know of it, but take the saline enema as part of their treatment. It seems a great error to keep blackwater patients on very low diet as their condition is so adynamic, and there is no definite reason for doing so in an uncomplicated case. These are the broad lines of treatment in ordinary attacks, and special symptoms have to be taken as they arise.

Vomiting. Thus, vomiting is often benefited by sinapisms on the epigastrium, sipping of iced champagne, sucking ice, etc. Some are in favour of the extract of Cassia Bearana. I cannot

say that it has controlled vomiting where I have used it. Creosote and sulphate of morphia in 2 minims and $\frac{1}{64}$ grains respectively make auseful mixture in some cases. Unless the patient is very exhausted it seems doubtful practice to control vomiting completely, and it is usually most severe soon after the onset of the disease when he is relatively strong.

Pyrexia usually yields to the general treatment which promotes the action of the skin, bowels and kidneys. Cold sponging of the limbs may be necessary in some cases.

Headache is sometimes severe, and is best relieved by cold applications to the head. Citrate of caffeine is a useful drug in these cases. Phenacetin and similar drugs are not safe.

Splenic and hepatic pains cause much discomfort occasionally and may be accompanied by aching in the corresponding shoulders. Sinapisms, or strong Liquor of Iodine applied over the hepatic and splenic regions have been found most efficient, and liniment of belladonna rubbed on the shoulders, and covered with cotton wool and a bandage soon relieves those joints.

Hiccough in blackwater is a most dif-

ficult symptom to treat and is of ominous portent. One has to ring the changes on the various drugs used for this condition, allowing for the peculiarities of the disease. Nothing does much good.

Suppression of urine is a very grave symptom but there seems a chance that a patient may recover in spite of it, e.g. Blackwater Fever Case/2 where partial suppression took place but the flow of urine was re-established to some extent. It used to be the practice to poultice or foment over the kidneys from the outset of blackwater, hoping thereby to prevent suppression. However this practice did not seem to influence the quantity of urine, and caused the patient a good deal of inconvenience. Latterly I believe it is more usual to swathe the region of the kidneys, spleen and liver in wool from the beginning, and poultice or foment if suppression threatens. Puncture of the renal capsules, and stripping off of the capsule are measures which might be tried as last resources. The free expansion of a much infarcted kidney might possibly allow a rehabilitation of function.

Quinine in Blackwater Fever.

The procedure generally adopted in

N. Nigeria is I believe to give quinine in small doses should malarial parasites be found in the blood; otherwise, to avoid it. If quinine has been taken before the attack no more is given unless parasites are found. After albumin has disappeared from the urine small doses of quinine are given daily.

Sternberg's Formula.

Sternberg's mixture of corrosive sublimate and the bicarbonate of soda has been tried in all cases described, but others who do not use it get very similar results. It is therefore of doubtful value.

CASES OF

BLACKWATER FEVER.

CASES OF BLACKWATER FEVER.

The five cases here described were treated by the writer in the years 1905, 1906 and 1909 whilst he was in charge of the European and Native Hospitals at Zungeru, or in medical charge of Sokoto station. He has in addition had opportunities of seeing several other cases undergoing treatment by colleagues in his own service - the West African Medical Staff.

The cases described serve to show how widely the clinical manifestations of the disease may vary in different cases. The general treatment adopted is indicated more fully in the section of the Thesis dealing with that subject, special measures being dealt with under the cases. The fact is also borne out that Blackwater Fever per se does not appear to be the cause of death. In the first it appeared to be due to the accumulation of waste products during partial suppression of urine together with exhaustion; and in the second to acute nephritis operatingin extreme debility. The remaining cases comprise a simple uncomplicated one, and two showing relapses. The fourth case is peculiar in that the

relapses occurred at equidistant intervals, and shows that quinine was given without causing relapse which could be ascribed to its action. The last case is I believe the only one treated in the country where relapses are so numerous. I have therefore included the temperature chart, showing in red at the bottom, the duration of each attack of haemoglobinuria.

BLACKWATER FEVER CASE I.

Sergeant S., a European N.C.O. stationed at Zungeru. Aged about 32. Admitted to European Hospital, Zungeru on November 20th, 1905.

Previous History.

This patient was a hardy but not robust man, in his second tour of duty in the country.

He had been in India previously, and had also suffered occasionally from malaria in N. Nigeria. His quarters in Zungeru were low-lying close to the River Dago. Took quinine irregularly, and 10 grs. before admission. He had had a rigor before admission, and on arrival passed some portwine coloured urine.

On admission.

His temperature was 104°, and his complexion sallow. After a few hours jaundice became established. He complained of pain in the hepatic and splenic areas, and of nausea.

Course of disease.

He soon began to vomit bright green matter of granular appearance, and showed signs of exhaustion towards the evening. The urine continued a deep red all day, and his temperature remained up.

The next day no change for the better occurred and he began to hiccough towards the evening. The urine passed on the second day was very dark, and somewhat below the normal quantity.

On the third day his temperature fell slightly and he did not pass more than ten ounces of urine, and these of porter colour. He seemed very weak and vomited frequently whilst the hiccough became very troublesome.

Treatment.

He had so far been treated by jacket poultices over the loins, and was given Sternberg's mixture of sodium bicarbonate, and perchloride of mercury. Cassia Bearanahad been tried but, if anything, was found to increase the vomiting so was discontinued.

Normal saline solution was now injected into the cellular tissue of the axillae

and flanks, and on the fourth day no signs of improvement setting in it was decided to needle the kidneys. This operation was performed on one kidney, a fine aspirating needle being passed through the capsule in various directions, and with every precaution against sepsis.

After the needling it was satisfactory to note an increase in the quantity of urine passed, though it came in small instalments. However the general condition of the patient did not improve, and the urine remained very dark. His temperature was now fluctuating about 100°. Hiccough and vomiting continued in spite of various sedatives administered, and periods of muttering delirium alternated with periods of dazed consciousness.

It was decided then as a last resource to cut down and strip off the capsule of one kidney under ether, as had been done in cases of Bright's disease hoping that the removal of pressure might allow a freer secretion. However, the patient rapidly became so much worse that the idea was abandoned.

He died comatose on the 9th day of the disease.

Blood.

The blood was examined from time to time for malarial parasites, but none were found, and no quinine was given.

Urine.

The urine varied in daily quantity from 10 to 40 oz. Nitric acid in the cold caused a ropy brown precipitate. Boiling caused some 50% of the urine to solidify. The urine on standing deposited a brown granular substance consisting of débris, tube casts, and towards the end of the illness a few red blood corpuscles.

Post Mortem-

The spleen, liver and kidneys were enlarged. Both kidneys were much congested, and the sitesof the punctures were not discoverable by the naked eye.

BLACKWATER FEVER CASE II.

Mr. R. a European foreman of works, employed by the Public Works Department. Aged about 35 years. Admitted to Zungeru European Hospital on December 27th, 1905.

Previous History.

He was in his second tour of duty in N. Nigeria, and was of sturdy, thick-set build. He occupied quarters in a low lying district near the River Dago.

He had taken ten grains of quinine on the day of admission, and had had a rigor on the previous day. Some urine passed at his quarters just before admission was deep red in colour.

He had not been in the tropics before he came to N. Nigeria, but had had several attacks of "fever" in that country. He appeared to have taken quinine fairly regularly.

On admission.

His temperature was a little over 103° and he was slightly jaundiced. He complained of

slight splenic and hepatic pain. The spleen and liver were both enlarged, and tender. He vomited bright green bilious material on admission and continued to during the afternoon, whilst the jaundice deepened considerably, and the temperature rose above 104^{0}.

Course of Disease.

His temperature fell to 102^{0} after a restless night; urine continued a deep red; and vomiting of green matter continued. His temperature rose again towards evening above 104^{0}, and the total amount of urine for the day was less than normal.

On the third day his urine still continued a deep red, and the temperature did not remit whilst other symptoms continued unabated.

Treatment.

Up to this stage the treatment consisted of poulticing the loins, purging by vegetable laxatives, the administration of Sternberg's mixture whilst Cassia Bearana - which was not a success - morphia, creosote, etc., were used to check the vomiting, with various degrees of success. Now

normal saline solution was injected per rectum to the extent of about 3 pints a day. Intracellular injections were tried but caused so much distress to the patient that they were discontinued.

The urine began to clear on the third evening after admission, and the temperature came down to 102°. The amount of urine passed daily after this was from 10 to 20 ounces, and its sediment showed under the microscope a considerable number of red blood corpuscles and hyaline and hæmoglobinous tube casts and some endothelial débris. The course of the illness from the fifth to the eleventh day of the disease showed a gradual loss of strength, and the gradual substitution of acute nephritis for hæmoglobinuria, for the urine finally became a red transparent solution unlike the ordinary "water" of blackwater fever. During the last three days the patient became very restless, and at times violently delirious, struggling to get out of bed, and attempting to assault those who tried to restrain him. A few hours before death he became quieter and passed from muttering delirium into a coma which lasted until the end on the 6th January 1906.

Blood.

No malarial parasites were found in the blood, and no quinine was given.

Urine.

The urine was under 20 ozs. in 24 hours after fourth day of the disease., became a light red in colour, and the deposit in the cold became considerable - about 1/8 of the bulk of urine examined. The supernatent urine remained reddish, and always gave the blue ring with tincture of guaiac and ozonic ether. The deposit contained a large quantity of blood corpuscles, as well as hyaline, and hæmoglobinous casts, and endothelial débris.

On boiling, and on tretament with nitric acid in the cold, deposits similar to those described in the previous case were thrown down, and varied from 20 to 50% of the bulk of urine taken.

Post-mortem.

The liver and spleen were both enlarged, and dark on section.

The kidneys were both enlarged, dark and somewhat congested. On section the pyramids

were seen to be dark red in colour. Both capsules stripped easily. Nothing unusual was noted in the aspect of the brain.

BLACKWATER FEVER CASE III.

Dr. M. an European Medical Missionary aged about 40 years. Admitted to the European Hospital Zungeru, on 4th February, 1906.

Previous History.

This patient came to N. Nigeria about five years prior to the attack, and had spent about four of these in the country chiefly in a large native town up country, where the sanitary conditions leave much to be desired. He had occasionally had attacks of malarial fever, and had been in the habit of taking quinine in ten grain doses twice weekly.

I found him in his quarters in Government House, Zungeru, where he had arrived the previous day. He had a temperature of 104.5° and had taken ten grains of quinine earlier in the day, and ten the day before when he had a sharp rigor on the march.

Soon after my arrival he passed about 20 ozs. of dark red urine, and complained of nausea. He was - as he always is - sallow but not jaundiced. He is by no means of robust physique.

On admission.

He began to develop jaundice, and vomited occasionally during the afternoon. His liver was not enlarged, and the spleen but slightly so. No malarial parasites were discovered in his blood.

Progress of disease.

He passed another 10 ozs. of urine during the afternoon, and complained of splenic pain. In the evening his temperature rose to 105° and he spent a restless, wakeful night.

Next day his morning temperature was 103.2° and the urine still deep red in colour, and continued so all day. He vomited at intervals of two or three hours during the day, and the jaundice was well marked. His temperature was 104° in the evening, but he spent a quieter night.

Next morning his urine began to clear and at one o'clock was quite clear though it contained about 20% of albumin. His temperature was 101°, and he did not vomit again. His evening temperature was a few points above 101°, and next morning was subnormal. From then onward he made an uninterrupted recovery, and was discharged on

the 12th of February.

Treatment.

The loins were poulticed and later swathed in wool. Sternberg's mixture was administered, and Cassia Bearana and various sedatives were used to control the vomiting. Sulphonal was given on the second night. Saline enemata were injected twice daily in this as in all the preceding cases.

Blood.

No malarial parasites being found no quinine was given until the albumin disappeared from the urine, when 3 grain doses of the hydrochloride were given daily.

Urine.

The usual brown deposit came down on standing, and the brownish with heating. Nitric acid gave a light brown ropy precipitate. No corpuscles were seen at any time in the deposit. Although the course of this attack was not severe the precipitate after boiling was about 75% of the urine taken.

Note.

This patient was readmitted to hospital with a sharp attack of malarial fever a week after his discharge.

BLACKWATER FEVER CASE IV.

R. a native of India aged about 27 years employed as a Naik or bullock driver in the Transport department. Admitted to the Native Hospital Zungeru on September 23rd, 1906 suffering from "blackwater fever".

Previous history.

He had occasionally suffered from fever in India but had not had any "blackwater". He took quinine fitfully as a prophylactic; and had been treated twice before in N. Nigeria for malarial fever. Quinine is issued free to Indians, but though the Bengali clerks take it pretty regularly the transport Indians, Sikhs, Hindoos, etc. are neglectful in this respect.

On admission.

His temperature was 104.2° and the first water passed was of a deep portwine colour.

Progress of disease.

He vomited occasionally, and jaundice which was noticeable in the conjunctivæ, and even in

the dark skin deepened as the day progressed. After admission he had a rigor. The blood was examined and found to contain malarial parasites.

The symptoms did not alter on the second day, but on the morning of the third day the urine cleared and symptoms began to disappear. He was then given doses of 5 grains of quinine sulphate daily and seemed to be progressing satisfactorily, the urine being clear, and vomiting having ceased.

On the 8th day, however, his temperature ran up to 104.4° in the evening and all the symptoms of blackwater again became apparent. The symptomatic treatment adopted in the first onset was resumed, viz. poulticing over the splenic and hepatic areas, and later swathing in cotton wool, the administration of Sternburg's mixture, etc. but quinine was not given as I suspected its influence in causing a relapse. The course of this relapse was very similar to that of the first attack but left the patient weaker and eventually on the evening of the 10th day of the disease the urine cleared, vomiting ceased, and only the fading jaundice persisted. During the relapses no malarial parasites were found in the blood.

In spite of the fact that no quinine

was given, 8 days after the first relapse, i.e. the 16th of the disease, another rise of temperature to 104° coincided with another relapse, all symptoms being re-established as in the first attack. The same treatment was carried out, but I reverted to the use of quinine, giving the hydrochloride in three grain doses as soon as the water cleared which it did on the second day of this relapse, the 18th of the disease.

No further relapses occurred and the patient made a good recovery, and returned to his duties.

Blood.

Subtertian parasites were found in the first attack but not subsequently.

Urine.

The reactions with heat, and with nitric acid were as described in the other cases, but that the albumen which was not more than 50% of the bulk of urine taken at any time quite disappeared between the relapses.

Note.

This man prayed very earnestly to be allowed to remain in the country, and was allowed to do so at his own risk; unwisely as it proved because he died of blackwater fever about a year afterwards when engaged in bringing a convoy down country, medical aid not being at once available.

BLACKWATER FEVER. CASE V.

Armourer Sergeant F., aged 31, employed in repairing guns in various stations.

Previous History.

This N.C,O. was in his second tour of duty in N. Nigeria. Had been in China where he had suffered much from"fever". Had much fever and dysentery in his last tour, and was invalided on account of the latter. Had an attack of blackwater fever on the ocean boat going home, and subsequently an attack of dysentery in England. Came up to Sokoto towards the end of the rains by a very swampy road and spent about a fortnight en route at Birnin-kebbi a very malarious station on the Sokoto River. He took quinine irregularly. He had taken 10 grains when I saw him.

Admission.

He was first attended by me on the evening of the 31.8.09, and had been playing polo that afternoon. His temperature was 102.6°, and he said he had had rigors and "fever" for some days. He complained of epigastric pain, but not of pain in

the regions of the spleen ar liver. The former was enlarge d two inches below the ribs, but the latter could not be felt. He displayed a profound anaemia - too marked to be the result of his present attack. His blood was examined and found to contain no parasites. He was passing port wine coloured urine freely.

Course of the disease.

His skin soon acted, and his temperature fell about 12 p.m.

2nd day. He vomited a little in the morning and his temperature rose to 104 and he became jaundiced - more deeply as the day wore on. He passed a restless night, vomited frequently large quantities of green muc o-bilious matter. He became very weak, and his pulse thready and faint, but reacted well to 1/100th grain of digitalis.

3rd day. The urine began to clear about noon and was clear at 12 p.m. Jaundice also began to fade.

4th day. Nausea and extreme weakness were the chief symptoms. Passed a fairly good night.

5th day. Malarial parasites (subtertian) being found in the blood for the first time 5 grains of Quinine sulphate were given per oram, and later 5 grains per rectum. Towards midday his

temperature began to rise and he became delirious as it reached 103.6 later. His temperature began to fall at 6 and came down during the night.

6th day: He passed 20 oz. of urine between 12 p.m. and 6 a.m. and the last 6 ozs. was claret coloured, and contained haemoglobin. He became restless and rambled, and his pulse became rapid and thready so 1/100 grains of digitalin was given. When his clothes were being changed he had a sudden syncopal attack and stopped breathing, whilst his jaw "dropped". Some brandy was given at once, and it caused a paroxysm of coughing followed by vomiting. He seemed so prostrate that I gave him a hypodermic of strychnine. He slept then and woke improved, but became restless and incoherent soon after and remained so until 2 a.m. on the 7th day.

7th day: The urine was free from haemoglobin all day, but contained a little albumin. He complained of headache which was relieved, and then he slept most of the day and woke quite rational and apologetic at 2 a.m. on the 8th day.

9th day: The morning urine was clear, but later became haemoglobinous again, cleared, and again became haemoglobinous. The shade in both cases was less deep than former urines. He was

slightly delirious during the afternoon.

10th day: He complained of pain in the left shoulder, and numbness in both legs. The morning urine passed before 6 o'clock was clear, and that passed after 6 was haemoglobinous and contained a few red blood corpuscles.

11th day: He had severe pain in the region of the spleen and in the left shoulder. The spleen was about 4 in. below the costal margin. A little reddish urine containing haemoglobin was passed in the evening, and cleared after.

12th day: 10 ozs. of reddish urine passed in the afternoon, and 24 ozs of clear subsequently.

13th day. The spleen and shoulder were again troublesome.

14th day: Complained again of spleen and shoulder. Two relapses of haemoglobinuria occurred on this day, and these were the last manifestation of this symptom. After this he began to improve and his temperature gradually approached the normal line. Slight pyrexia continued until the 18th day which was no doubt due to the condition of the spleen. After the 18th day he made an uninterrupted recovery, and later was sent down country, accompanied by a recom-

mendation that he should not come to West Africa again.

Urine.

The urine varied in colour from light to deep red. It contained no albumin between the relapses, except once. There was a deposit after boiling, and after standing in the cold. The former was of a brown granular description and varied in quantity from 10 % to about 80 % of the urine taken. The latter deposit contained haemoglobinous and hyaline casts, and occasionally a few red cells.

Nitric acid caused a light brown ropy precipitate. On settling after boiling and after acid the deposit left an amber coloured supernatant urine. No bile pigment was found in the urine at any time.

Blood:

The thinness of the blood was very remarkable. It was examined daily, and as it flowed from the finger was a deep straw colour to the naked eye. There was marked diminution in the number of red cells, which fluctuated between 1 and 3 millions, whilst there was also a relative, and actual large mononuclear leucocytosis which varied from 9 to 20 per cent.

Urine began to clear at 330 on the 2nd.

Sounds began to go same morning.

Urine became deep red - more "porter" about.

Digitalis acted well. First sample of urine on 3rd = no albumen.

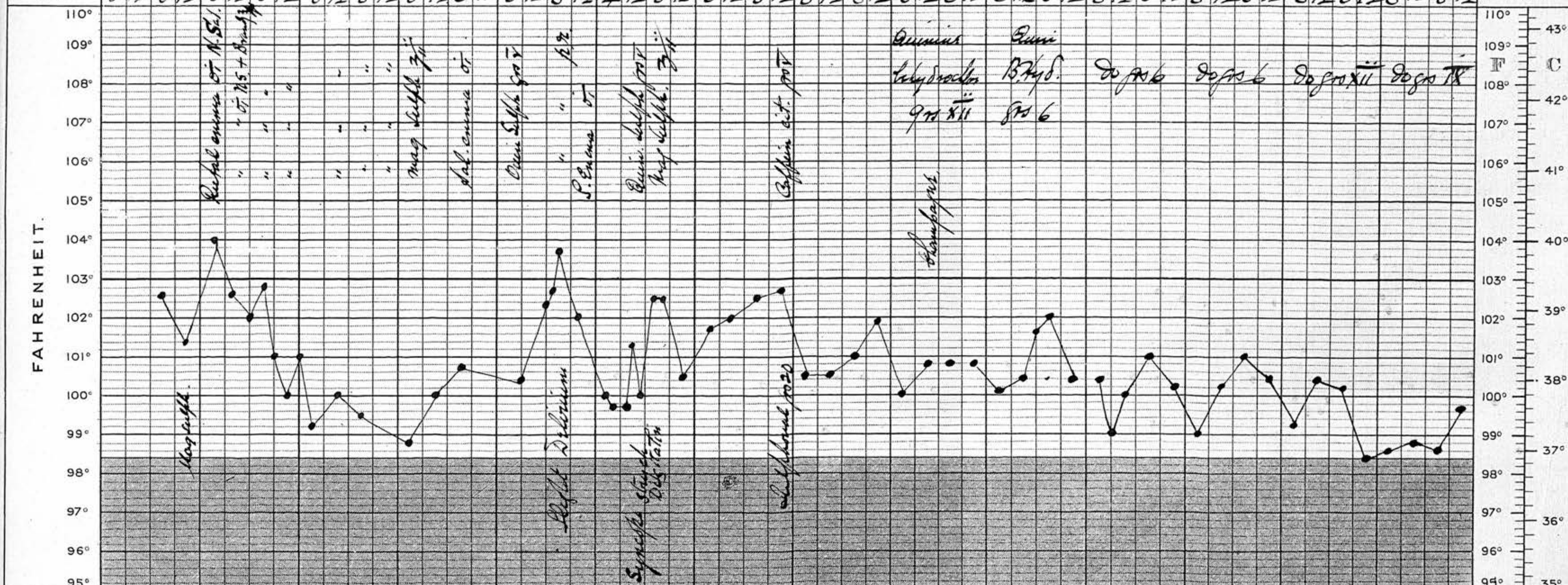
Copy 1

Blackwater Fever. Case 5

Medical Officer Dr G. R. Twomey Name of Patient Armourer Sergt Fidoe Age 31
Ward Sokoto (Quarters) Admitted 31. 8. 09 Discharged 5. 10. 09 Disease Haemoglobinuric Fever.

DATE	31				1				2				3				4				5				6			
PULSE				92	102	96	90	90	86	82	92		94	96	92		90	104	106	84	82	88	104	96	102	76	104	104
RESPIRATION				22	24	26	20	18	20	23	18		16	16	18		14	16	22	20	16	16	16	18		24	20	22
DAY OF DISEASE	1				2				3				4				5				6				7			
HOUR	6	12	6	12	6	12	6	12	6	12	6	12	6	12	6	12	6	12	6	12	4	12	6	12	6	12	6	12

DATE	7				8				9				10				11				12				13			
PULSE	99	100	102	100	100	96	113		100	100	104	96	101	100	88	96	90	100	104	100	96	104	96	100	100	88	90	100
RESPIRATION	14	18	16	20	16	15	18	20	16	28	25	3	18	20	24	22	24	20	24	24	20	24	16	24	20	16	16	
DAY OF DISEASE	8				9				10				11				12				13				14			
HOUR	6	12	6	12	6	12	6	12	6	12	6	12	6	12	6	12	6	12	6	12	6	12	6	12	6	12	6	12



	31		1		2		3		4		5		6	
SKIN			Jaundice		J	Less J	Slight		clear		Jaundice		clear	
URINE ℥		/25	10/16	0/10	20/0	12/0	12/12	20/18	0/28	0/0	20/0	12/5	12/0	6/0

	7		8		9		10		11		12		13	
SKIN	do		..		..		..		..		..		..	
URINE ℥	0/0	22 +9/18	18/12	12/0	17/12	18/12	18/0	18* +8/0	24/0	10/24	16/14	14/0	24/18	12/0

DATE	31				1				2				3				4				5				6			
BOWELS				3	1	0	0	0	1	1	2	0	1	0	0	0	0	0	0	1	0	0	2	0	0	1	1	0
WEIGHT																												
VOMITING				5	1	0	0	3	2	1	2	0	1	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0

DATE	7				8				9				10				11				12				13			
BOWELS	0	1	1	0	0	2	1	1	0	0	0	1	0	0	1	0	0	2	0	0	0	0	0	0				
WEIGHT																												
VOMITING	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	1	0

Blood in Urine: H C — clear ing — Clear — H b — clear

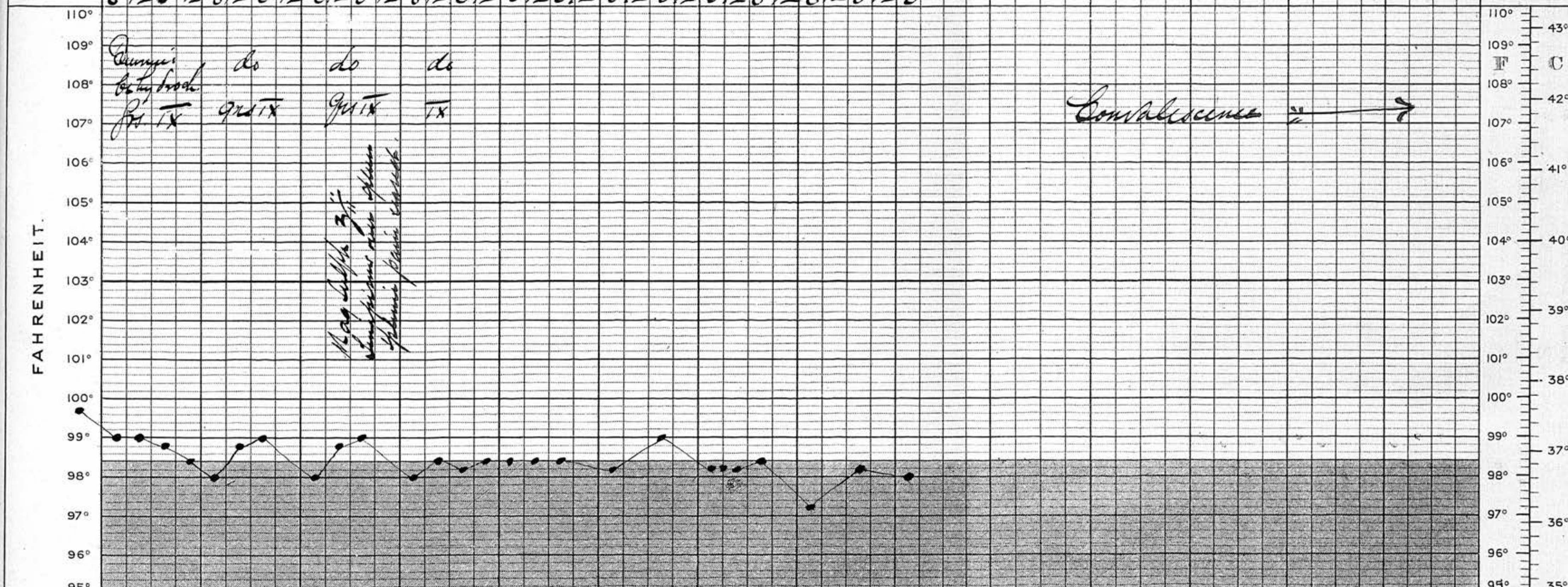
Haemoglobin in urine, thus:-

Blackwater Fever Case 5

Copy

Medical Officer Dr. G. R. Twomey Name of Patient Arm. Sgt. [illegible] Age 31
Ward Lokoja Admitted 31.8.09 Discharged 5-10-09 Disease Haemoglobinuric Fever

DATE	14	15	16	17	18	19	20	21	22
PULSE	88 92 88 88	88 88 88	88 88 84	74 90 92	90 96 92	80 88	80 84 84	74 76	80
RESPIRATION	16 24 16 16	16 24 15	15 18 16	18 18 15	16 15 16	15 14	14 14 16	14 14	14
DAY OF DISEASE	15	16	17	18	19	20	21	22	23
HOUR	6 12 6 12	6 12 6 12	6 12 6 12	6 12 6 12	6 12 6 12	6 12 6 12	6 12 6 12	6 12 6 12	6



SKIN														
URINE	0/18	14/16	24/15	18/14	18/6									
BOWELS	0	0	1	1	0	1	1	0	0	1	2	3	2	2
WEIGHT														
VOMITING	0	0	0	0	0	0	0	0	0	0	0			

No further Haemoglobinuria

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Part II.

EPIDEMIC CEREBROSPINAL MENINGITIS

Part II.

EPIDEMIC CEREBROSPINAL MENINGITIS IN ZUNGERU, NORTHERN NIGERIA in 1906.

PREFACE

The writer on his arrival in Zungeru at the end of January 1905 found that there was much speculation afoot concerning an epidemic disease among the natives of that town - the capital of Northern Nigeria. He was instructed by the Principal Medical Officer to make investigations with a view to ascertaining the nature of the outbreak. He remained about one month in Zungeru before proceeding on leave, and during that period was in charge of the Segregation Hospital for the reception of persons suffering from this disease.

The following is an account of the epidemic and was subsequently embodied in the Report of the Principal Medical Officer. It deals with seventeen cases which were under his care up to the 8th March 1905 when he proceeded on leave.

Introductory.

The increased mortality among the natives in and around Zungeru, and rumours of an excessive death-rate in Kano, Zaris, Ilorin, and several of the smaller villages attracted much attention about the middle of February. Small pox was epidemic at the time, and at first it was thought that the deaths were due to this disease, the natives themselves calling the sickness "small-pox fever." It was gathered that outbreaks such as this, though not of annual occurrence, were by no means uncommon during the first three months of the year, and were supposed to be due to the excessive dryness of the season. All evidence pointed to an enormous case mortality, and the natives stated that in some years cattle, in others men, and sometimes both cattle and men, were attacked.

At first no cases of the disease could be found, all the earlier cases running such a rapid course that death occurred within a few hours, and owing to the native's habit of hiding himself when ill, the first intimation usually received was that a dead body had been found, and no information was obtainable as to what had been the cause of death. Post mortems were made where possible, but nothing

was discovered but intense congestion of the vessels of the cerebral cortex.

A case of cerebro-spinal meningitis was admitted to the native hospital earlier in the month, and this was thought to be a sporadic one, but after the lapse of a week many nervous cases arrived, all corresponding more or less with the first, and on February 28th a native clerk was admitted with the classical symptoms of the disease. He died within twenty-four hours of admission, and the diagnosis was fully borne out at the autopsy. Epidemic cerebro-spinal meningitis was then decided on as a working hypothesis for the investigation of the epidemic. The health officer was instructed to search the native town, and send into hospital all cases of a doubtful nature for observation, and a house to house visitation was made.

Eight cases of the disease were discovered, which were placed in a temporary segregation hospital, and sanitary measures were taken to prevent its further spread as far as possible, these measures consisting in burning or disinfecting all houses from which cases had been taken, in opening up the more crowded portions of the town, and in isolating all fresh cases.

Etiology. - During the earlier part of the year many fires had occurred in the native town, destroying great numbers of the grass huts and driving the inhabitants into the remaining houses, with the result that portions of the town had become greatly overcrowded. This condition of affairs undoubtedly contributed to the spread of the disease after its introduction, but whether the disease is endemic in a mild form and occasionally becomes epidemic, or whether the disease has been introduced from without, there is no evidence to show. The former is probably the correct explanation, though no previous case has been recorded in Northern Nigeria.

Clinical Picture. - The following is a clinical picture of the average case founded on the signs and symptoms observed in the first seventeen cases which were admitted to the segregation hospital.

The patient when first seen is lying on his side with one or both knees slightly drawn up, his back is arched backwards, and his head retracted. He is generally only semi-conscious, but can be roused, and then moans and stares rather wildly, with an anxious expression on his face. If he can be induced to rise he does so in a characteristic manner, first

rising to his knees and then to his feet, keeping the head, neck and trunk rigid during the operation. If an effort is made to raise the head when he is recumbent the whole of the head, neck, and trunk is raised by the same action, and when having to enter a low doorway, he sinks gradually to his knees, and in this attitude shuffles in with head thrown back and spine rigid. When in the erect posture he sways and grasps at the nearest means of support. On being questioned he complains of severe pain at the back of the head, and of pain in the lumbar and dorsal regions. Pressure on the neck below the occiput on the posterior neck muscles and on the cervical spine is obviously painful, but tenderness of the dorsal and lumbar parts of the spine is not so marked, though generally present. Pains in the limbs are by no means uncommon. Kernig's sign could be elicited in nearly every case, and photophobia was a pretty constant symptom. Strabismus was absent, though curiously enough it was present in the first case observed, where it had existed from childhood. Vomiting was a pretty constant symptom in the earlier cases. The temperature is generally not high, running from 100° to 103°. Pulse about 60, and strong as a rule.

After about twenty-four hours, in cases run-

ning an unfavourable course, the stupor or somnolence gives place to delirium, the patient becomes very restless, noisy and sometimes violent. He cannot remain in one position for more than a few seconds. The temperature swings irregularly from perhaps 97° in the morning to 102° or 103° in the evening. Later, if he lives, relapses are noticed following hard on one another. Pulse generally slow but strong. Still later, about the third day the patient sinks into a stupor, gradually deepening into complete coma. The conjunctivæ become congested, and the face somewhat bloated. Herpetic eruptions may appear on the lips and face, the respiration becomes sighing, the teeth covered with sordes, and the tongue dry and brown, and the patient gradually sinks into an adynamic state which terminates in death. In most cases a yellowish discharge exuded from the mouth, nose, and eyes about twelve hours before death, and the breath sounds are obscured by loud moist râles which may be heard at some yards distance.

The malignity of the disease varies a good deal, death occurring sometimes within twenty-four hours, but the majority of deaths occurred after four or five days' illness.

The following table shows the occurrence of

the commoner symptoms in seventeen instances. The column showing the number of days each patient was sick before admission to hospital, though generally correct, cannot be vouched for in every instance, as information obtained on purely native evidence is not always accurate. (PAGE 78)

The three charts attached show the irregular character of the fever. (PAGE 79)

MORBID ANATOMY.

Details of the post-mortem examinations made during the epidemic are given below.

(1) James Mousa, aged 23, native clerk, died March 1st, 1905. This man died after three days' illness. The autopsy was made about twelve hours after death.

Head.- The vessels of the scalp were much injected. The pia-arachnoid was much congested. Patches of lymph and purulent exudate were scattered over the cortex and in the cortical sulci. A large quantity of purulent exudate was seen around the cerebellum and all over the base of the brain, bathing the emerging basal nerves. It also welled up around the divided cord, and where a portion of the spinal

column was removed in the dorsal region patches of purulent exudate were found chiefly on the posterior surface of the cord.

Thorax.- Large fibrinous clots were seen in the auricles of the heart extending through the mitral and tricuspid valves. The ventricles were nearly empty. The venæ cavæ were distended with blood. The heart weighed 11 ozs. The lungs were both slightly congested. The pleuræ and pericardium were normal.

Abdomen.- The stomach showed a good deal of congestion, and a few petechial hæmorrhages were noted on the mucous membrane. The liver weighed 46 ozs., the left kidney 5 ozs., the right kidney 5½ ozs., the the spleen 9 ozs.

(2) Mormoh, aged 23, horse boy, died March 2nd, 1905. This man died after four days' illness. The autopsy was made about three hours after death.

Head and Spine.- The pia-arachnoid was deeply injected. Patches of purulent exudation were found over the whole brain, markedly over the pons, cerebellum and medulla, and about the basal nerves. The brain substance was anæmic. A small quantity of purulent material was seen in the posterior cornua of the right lateral ventricle. Pus was found all

around the divided cord and a thick layer under the congested membranes of the lumbar cord behind, the exudate being less marked on the anterior surface.

Thorax.- The heart weighed 12 ozs. The left ventricle was hypertrophied, and the right dilated. The mitral valves showed thickening, the ventricles were empty. The auricles contained large fibrinous clots extending through the mitral and tricuspid valves, the venæ cavæ were engorged, the pericardium was congested and coated with patches of lymph. It contained about 2 ozs. of reddish-yellow fluid. Both lungs were much congested. The pleuræ were normal.

Abdomen.-The liver weighed 50 ozs. The stomach showed a good deal of congestion, and many petechial hæmorrhages on the mucous surface. It contained 4 ozs. of reddish fluid, mixed with bile. The rest of the alimentary tract was normal. The right kidney weighed 4½ ozs., the left 4 ozs. The spleen weighed 7 ozs.

(3) Djoah, aged 7, female, died March 1st, 1905. This child had been sick four days. The autopsy was made twelve hours after death. Post-mortem rigidity was absent.

Head and Spine.- The pia-arachnoid showed much

congestion. The whole surface of the cortex was covered with purulent exudate. The cerebellum and medulla were thickly coated and a thick layer was seen over the base of the brain and around the cut cord. Congestion of the membranes of the dorsal cord was likewise seen, as well as patches of exudation over the posterior surfaces, though the anterior surface was free from exudate.

Thorax.- The heart weighed 5½ ozs. The ventricles were empty. The right auricle contained a large clot, the left was empty. The venae cavæ were engorged with blood. The lungs, pleuræ, and pericardium were normal.

Abdomen.- Except that the stomach contained a coating of glairy mucus, the abdominal organs showed nothing worthy of note.

(4) Brimah, aged 18, horse boy, died March 1st, 1905. This boy had been ill since February the 15th. The autopsy was made three hours after death.

Head and Spine. - The brain was covered with patches of purulent exudate. The pia-arachnoid was injected, but not markedly. The basal nerves were bathed in the exudate, and it was seen in great

quantity over the pons, medulla, and upper portion of the cervical cord. A thick layer was seen on the posterior aspect of the lumbar cord, and a smaller quantity in patches on the anterior aspect.

Thorax.- The heart weighed 10 ozs. The right auricle contained a large fibrinous clot, the left a smaller clot. The venæ cavæ were full of blood. Pericarditis existed to a marked degree. The lungs were both congested, the right markedly.

Abdomen.- The stomach was coated with glairy mucus, and showed many small petechial hæmorrhages. There was also marked congestion of the inferior portion. The liver weighed 51 ozs., the right kidney 5½ ozs., and the left 5 ozs. The spleen weighed 10 ozs.

(5) Awudulai, aged 22, male pauper, died March 2nd, 1905. This man had been sick three days. Autopsy was made twelve hours after death.

Head and Spine.- A yellowish, thick fluid streamed from both eyes. Conjunctivitis was present on each side. The lips were covered with herpes, and a patch of facial herpes was seen on the left malar region. There was keratitis present in both cornea. The pia-arachnoid was much injected, and

patches of purulent exudate covered the sides and top of the brain, which rested in a bath of the same material. Both ventricles (lateral) contained a small quantity of the exudate. The exudate was found in smaller quantities around the divided cord, and covering the dorsal and upper lumbar portions on their posterior aspects.

Thorax.- The heart weighed 12 ozs. The auricles contained clots, the mitral valves showed thickening, the left ventricle hypertrophy. The venæ cavæ were engorged with blood. Pericarditis was very marked. Broncho-pneumonia was present in both lungs, and the posterior inferior portion of the left pleura was much congested.

Abdomen.- The stomach was coated with glairy mucus, and showed petechial hæmorrhage here and there in its lower part. It contained about 4 ozs. of reddish fluid. The liver wieghed 49 ozs., the spleen 6 ozs., right kidney 4½ ozs., and left 4 ozs.

(6) (a) Native, aged 45 approximately, male, died March 3rd, 1905. Full details of this and the two cases following could not be obtained. The man was said to have sickened and died in one day.

Autopsy was made about twelve hours after death.

Head and Spine.- The vessels of the scalp and pia-arachnoid were intensely injected, as also were the membranes of the dorsal cord. No purulent exudation was seen.

Thorax.- The heart weighed 10 ozs. The lungs were both congested, the right markedly. The pleuræ and pericardium were normal.

Abdomen.- The stomach showed a coating of glairy mucus and slight congestion. It contained a little reddish fluid. The liver weighed 50 ozs., the right kidney 5 ozs., the left 5 ozs., the spleen 9 ozs.

(b) Native, aged 24 approximately, male, died March 3rd, 1905. The man was said to have sickened and died in one day. Autopsy was made about thirteen hours after death.

Head.- The vessels of the head and pia-arachnoid showed the same intense congestion as seen in Case (a). The congestion of the membranes of the dorsal cord was less marked. No pus was found on the brain or cord.

Thorax.- The heart weighed 10 ozs. The right and left auricles contained clots; the venæ

cavæ were engorged. The lungs both showed slight congestion.

Abdomen.- The stomach showed a little congestion, and a coating of mucus. The liver weighed 50 ozs., the right kidney 5½ ozs., the left 5 ozs., the spleen 10 ozs.

(c) Native, aged 25 approximately, female, died March 3rd, 1905. This woman was said to have sickened and died in one day. The autopsy was made about twelve hours after death.

Head and Spine.- Like the other two cases, congestion of the scalp vessels and of the pia-arachnoid was very intense. The spinal membranes were also implicated, being very markedly congested.

Thorax.- The heart weighed 9 ozs. The auricles were distended with clots. The ventricles were empty. The great veins were engorged. The pericardium showed early congestion. Both lungs were congested.

Abdomen.- The liver weighed 45 ozs., the right kidney 4½ ozs., the left 4 ozs., the spleen 6 ozs. All the abdominal organs appeared normal in character.

(7) Salami, aged 22, pauper, died March 2nd, 1905. Duration of illness not known. Autopsy made twelve hours after death.

Head.- The pia-arachnoid was much congested. The whole of the brain was covered with purulent exudate, which extended into the sulci and the longitudinal fissure.. Pus was found in both lateral ventricles. The lumbar cord was found embedded in purulent exudate. Yellowish fluid exuded from the eyes.

Thorax.- The heart weighed 11 ozs. Large clots were found in the auricles. The venæ cavæ were full of blood. Pericarditis was markedly present. There was broncho-pneumonia in both lungs. The pleuræ were normal.

Abdomen. - The stomach contained about 2 ozs. of reddish fluid. The mucous membrane was coated with glairy mucus, and showed several small hæmorrhages. The lower portion showed marked congestion. The liver weighed 54 ozs., the right kidney 5½ ozs., the left 5 ozs., the spleen 10 ozs.

(8) Omamegi, aged 21, mason, died March 5th, 1905. This man was ill four days before death. The autopsy was made about twelve hours after death.

Head and Spine. Purulent exudate was found in patches over top and sides of brain. The pia-arachnoid was congested. The cerebellum, pons, medulla, and basal nerves were all bathed in the exudate. A thick coating of pus was found over the cord in the dorsal region. A yellowish, thick discharge streamed from the orbits.

Thorax. The heart weighed 10½ ozs. The auricles contained large clots. The ventricles were empty. The venae cavae were full of blood. Both lungs were congested. The pleurae and pericardium were normal.

Abdomen. The stomach was lined with mucus, and showed a few small haemorrhages. The liver weighed 46 ozs., the right kidney 5 ozs., the left kidney 5 ozs., the spleen 9½ ozs.

That some cases die of hyperpyrexia is probable as in one instance where a man was reported to have dropped dead, and a partial autopsy revealed the unmistakable presence of cerebro-spinal meningitis, the temperature of the brain substance was higher than 110°F., the limit of the scale on a clinical thermometer.

BACTERIOLOGY.

The bacteriological evidence was inconclusive in that all the evidence obtained was derived from the morphological characters of the organisms found.

Smears were taken or films made from the nasal discharge, the purulent exudate on the brain surface, and the cerebro-spinal fluid both in the living patient and the cadaver.

At least six specimens were made of each, and stained variously by carbol-fuchsin, Jenner's stain, and Gram.

In every case diplococci were found without difficulty, which were decolourised by Gram's method.

The exudation in the brain surface was found to contain fibrous material, polynuclear leucocytes, pus cells, and diplococci, which were usually seen within the leucocytes, though occasionally free.

The nasal discharge contained numerous diplococci, but also numerous other bacilli.

No culture media were available, so that no further investigation into the character of the organisms was possible.

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SYMPTOMATIC TABLE OF SEVENTEEN EPIDEMIC CASES.

No	Name	No. of days sick before admission	Date of admission sick	Date of discharge	Age	Sex	Occupation	Race	Headache	Retraction of Head	Tenderness of nape and neck	Tenderness of spine	Delirium	Coma	Kernig's sign.	Herpes	Photophobia	Conjunctivitis	Discharge from Eyes.	Vomiting	Remarks.
1	Jundi	2	14.2.05	..	21	M	Pauper	Yoruba	Yes	Yes	Yes	Yes	Yes	No	Yes	Herpes	Yes	No	No	Yes	Doing well
2	Ari Kukuwa	1	17.2.05	..	17	M	Recruit	..	,,	,,	,,	,,	,,	,,	,,	,,	,,	,,	,,	,,	" " but quite deaf
3	Kolo	1	27.2.05	..	22	M	Soldier	Yoruba	Yes	Yes	,,	,,	,,	,,	,,	,,	No	Yes	,,	,,	" " " "
4	Marmah	2	28.2.05	..	32	M	Interpreter	,,	,,	,,	,,	,,	,,	,,	,,	Labialis	Yes	,,	,,	No	" " " "
5	Balon Flingy	3	25.2.05	..	24	M	Policeman	,,	,,	,,	,,	,,	,,	Not deep	,,	No	,,	,,	,,	,,	" " " "
6	Omomeji	1	2.3.05	5.3.05	21	M	Mason	,,	,,	,,	,,	,,	,,	Yes	Not marked	Labialis	,,	,,	Yes	Yes	Died
7	Bamaiye	2	1.3.05	..	18	M	Horse boy	Housa	,,	,,	,,	,,	,,	,,	,,	Labialis & facialis	,,	,,	,,	,,	
8	Saba	2	2.3.05	..	20	..	Pauper	Nupe	,,	,,	,,	,,	,,	,,	,,	No	,,	,,	,,	,,	Sinking
9	Jemma	3	2.3.05	..	23	..	,,	Yoruba	,,	,,	,,	No	No	,,	Yes	,,	,,	,,	,,	No	
10	Yanna	2	2.3.05	7.3.05	35	F	Trader's Wife	Nupe	,,	,,	,,	Yes	Yes	,,	,,	,,	No	No	No	Yes	Died
11	Salami	..	2.3.05	2.3.05	22	..	Pauper	Yoruba	?	,,	?	?	No	,,	No	,,	?	Yes	Yes	,,	Admitted in extremis
12	Amadu	2	2.3.05	..	21	,,	,,	,,	Yes	,,	Yes	Yes	Yes	,,	Yes	Labialis	Yes	No	No	No	
13	Adawnyi	3	3.3.05	..	20	..	,,	,,	,,	,,	,,	,,	No	,,	,,	No	,,	,,	,,	Yes	Developed parotitis
14	Alabi	2	3.3.05	7.3.05	17	..	Steward	,,	,,	,,	,,	,,	Yes	,,	,,	,,	,,	,,	,,	,,	Died
15	Balali	2	3.3.05	..	20	..	Labourer	,,	,,	,,	,,	,,	,,	No	Not marked	,,	,,	Yes	,,	No	
16	Esa	2	5.3.05	7.3.05	23	..	Pauper	Nupe	..	,,	..	..	No	Deep	Yes	,,	..	,,	Yes	Yes	Died; admitted in extremis.
17	Magagi	1	6.3.05	..	25	..	Ex-slave	Housa	Yes	,,	Yes	Yes	No	No	Not marked	,,	No	No	No	No	Doing well

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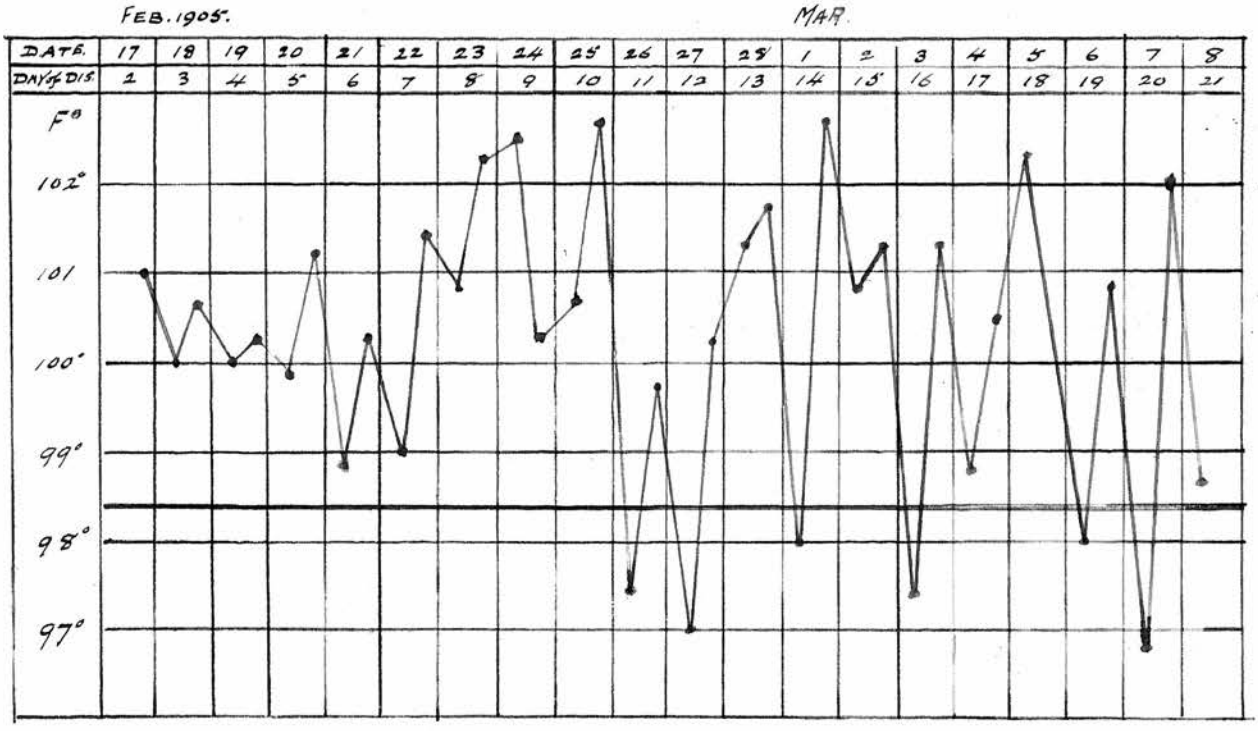

Temperature Chart of No. 2 in Symptomatic Table.

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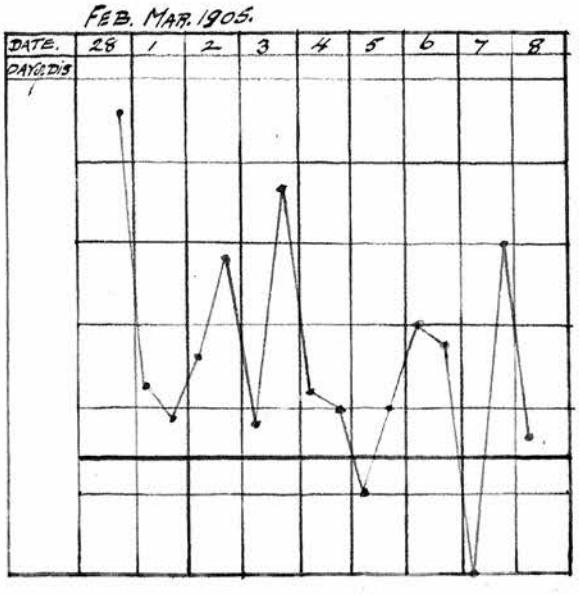

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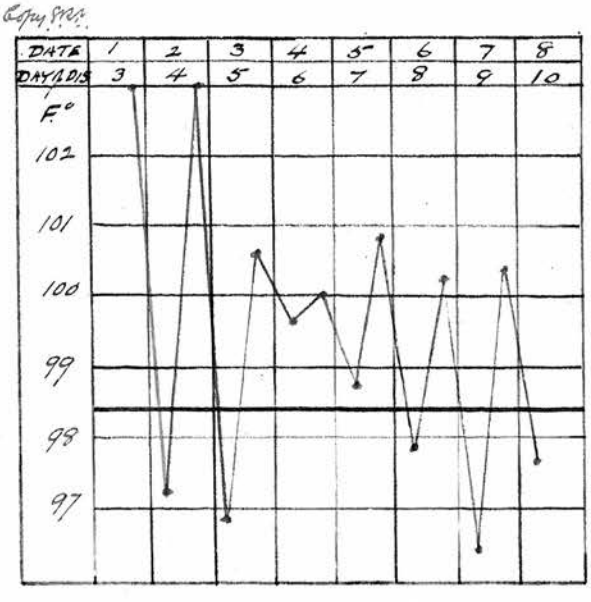

Temperature Chart of No. 5 in Symptomatic Table. Temperature Chart of No 7 in Symptomatic T.

C. R. Lowney. M.D.