SOME OBSERVATIONS ON BLACKWATER FEVER.

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"The intensity of malaria displays itself in blackwater fever."—Axiom: Dr. J. W. W. Stephens.

It is with the utmost diffidence that I venture to offer any remarks on a subject that has engaged the earnest attention of medical men. At the same time, I plead that blackwater fever is a disease, or a complication of a disease according to the school of thought to which the individual observer may belong), whose etiology and even treatment are matters for marked differences of opinion; so much so, that an important School of Tropical Medicine is, at the moment of writing, about to send out a Commission to investigate, in the hope that their researches may throw some light and meaning on a page in the book of tropical medicine which at present is so vague as to be almost valueless. Under these circumstances, even in times of the most acute mental depression, one hopes that in a small mass of clinical evidence some point may be found that will add something to the general knowledge on the subject.

In my preliminary observations it is hardly necessary to note that blackwater fever is a disease that seldom comes under the notice of the general run of Royal Army Medical Corps officers. I cannot help thinking, however, that, once met with, the fascination inspired by this fell complaint is all-compelling. The suddenness (in many cases) of its onset, the almost secrecy of its ways, the apparent impossibility of guarding against it, must cause the tropical clinician to give it his closest attention and interest.

One feels that here

"The ball no question makes of Ayes and Noes,
But here and there, as strikes the player, goes."

It makes one think of cholera in the old dark days when vibrios did not wriggle and bacilli were unknown. I must also ask to be excused for reminding readers of some general points about this disease.

**DEFINITION.**

Blackwater fever is not a very difficult disease to define. It is an acute haemolysis, generally sudden in its onset, characterised
by pyrexia, persistent and distressing vomiting, marked jaundice, and the passage, usually in diminishing quantity, of black or brown urine.

**History.**

Manson says it was first described by a naval surgeon at Nossi Bé. Plehn asserts that it first began to be common in West Africa about 1850; but the first recorded death took place in 1832. Fisch, on the Gold Coast, is inclined to think that it is more common nowadays, though less virulent, or, rather, fatal, than it was thirty years ago. He points out that whereas the fatality was formerly always very high, it now averages only about 20 per cent. Certainly our own admission and discharge books show a higher frequency to-day than formerly. This may be more relative than absolute. We may flatter ourselves that nowadays our diagnoses are more accurate, or that our attention is more directed towards blackwater fever; or even, according to some, that owing to a proper appreciation of things, the habit of taking quinine is more general than it used to be! It may also be added that blackwater, as such, and as a separate entity, does not appear in the Nomenclature of Diseases. We enter it as "remittent" or "malarial fever" (blackwater).

**Etiology.**

*Geographical Distribution.*—In Europe it occurs sporadically, so to speak. It has been met with in Italy and Sicily, Greece and Spain, or, rather, it has been reported from these places. It has been found in Asia Minor and in Syria. In India it occurs in comparatively few places, viz., in the Terai, in the Duars, in Jeypore (Madras), in the Canara district (Bombay), in some parts of Assam and in Upper Burmah. In parenthesis, it may be remarked that all these are notoriously malarial spots. It is found in the Malay Archipelago, especially in Java and Sumatra. It is Africa, however, that seems to be the "place of election"; and it is with blackwater, as found on the African littoral, that I am concerned with in these notes. Broadly speaking, where malarial fevers have their highest "endemic index" there is blackwater. That is to say, on the West African Coast, and up the West African rivers, from 20° N. to 20° S., roughly, from Bathurst to Lobite Bay. On the East Coast, it is found through Portuguese East Africa, and in Zanzibar. It is found in Madagascar and in Nossi Bé. Cases
Some Observations on Blackwater Fever

have been reported from Somaliland, and Captain Ensor, D.S.O., R.A.M.C., reported in this Journal a series of cases he had met with in the Bahr-el-Ghazel. It is common in British Central Africa, around Blantyre, and the Great Lakes. It occurs in some of the Southern States of North America, in Central America, and in certain places in South America, notably in Brazil. It is found in certain of the West Indian Islands. It breaks out occasionally on board ship coming from endemic areas.

Connection with Malaria.—That it has a close connection with malaria is a point upon which all who have met with the disease are agreed. Whether malaria, that is one single attack or a series of attacks, can per se bring about an attack of blackwater is a point upon which the future must come to some decision. Consequently, it is safer at the present time to enter malaria as a predisposing, rather than as an exciting cause. All, therefore, that can be said is that intense malaria is associated with the presence of blackwater. But tropical medicine is becoming day by day less empirical, and the man who, without absolutely knowing, states anything, is very likely to be called on almost the very next day to explain. In support of this, I would remind my readers of the views about kala-azar that were held only a few years ago, when some of the men—great authorities they were—made absolute statements about it; they must be sorry now they were ever persuaded to speak at all. At the same time, no one can contradict Stephens, when I requote his aphorism: "The intensity of malaria displays itself in blackwater fever." Further, this authority says: "It can only occur in those who are suffering from, or have been recently infected with, malaria." I have not been able to find, in the literature I have availed myself of, any record of a case without the patient having given a history of at least one attack of malaria. Sometimes, as in two or three of my own cases, the attack has been so slight as to hardly make it worth the while of the patient to stop his daily work. In thus speaking of the relation of blackwater and malaria, one comes to the question of residence in malarial countries, and immunity.

Residence.—Blackwater rarely shows itself before at least six months have been spent under malarial conditions, and the longer one is exposed to malaria, and the longer one escapes having, at any rate, severe attacks, the less likely would it appear that one will contract blackwater. That this is a working rule is evidenced by the table given in Stephens' latest work, which he quotes from Beranger-Féraud, who gives the following statistics:—
The interpretation is, that either it is a case of the survival of the fittest, or that residence confers some degree of immunity. The notorious exceptions are, amongst others, the case recorded by Daniels, of a man who spent eleven years in British Central Africa and then contracted blackwater; and even this does not hold the record, for a case was reported in the British Medical Journal of a man who contracted the disease after no less than twenty years residence in malarial Africa.

Immunity.—The immunity, then, if any, is only relative. At the same time, it is a generally agreed upon fact on the West Coast that one does acquire some sort of immunity to malarial fever after a certain period of residence, and the figures of Beranger-Féraud seem to me to bear out this tradition; and there is this fact to take into consideration, that there are a great many men who come out to the Coast and do tour after tour, take no quinine, and complete their tours without getting any fever at all. Given, then, some sort of immunity against malaria, it is only reasonable to suppose that these immune persons are less likely to contract blackwater than the relatively new-comers.

Endemicity.—There seem to be certain spots in the blackwater countries where one can contract blackwater more readily than others. In Sierra Leone I have been struck by this fact. At Murree Town, about three miles out towards the promontory, a detachment of sappers were stationed to work the search-lights. It is a place that one would have put down as being fairly safe, as it is on the seashore, and fairly well removed from native dwellings; all round, the bush had also been fairly well cleared; yet in my series of cases all the Royal Engineer cases had been stationed at Murree Town. No Europeans are allowed to live there now, and what was then almost an epidemic appears to have entirely stopped since they were moved away. It may be added that these men took their quinine regularly and used their mosquito nets, but the nature of their work compelled them, and still compels them, to do their work at night at least once a week.

Sex is not supposed to have any influence. In my series of cases there are two women, with one death. For a medical officer in charge of not more than fifty women, this is a high incidence; and to judge
Some Observations on Blackwater Fever

by the way the last case succumbed, I do not put much faith in their powers of resistance. I have not yet met with a case in a child, although my duties give me abundant opportunities for observation. Children harbour the malarial parasite without showing signs of the disease. Whether they can do the same in blackwater I do not know; it is possible.

Susceptibility.—In West Africa, as would be expected, Europeans are the most susceptible, next come the West Indians of mixed blood, then West Indian negroes, and at the bottom of the scale the West African native. Observers on the East Coast note that Arabs, Eurasians and natives all suffer. It is the same in the case of Chinese and Malays, but to all practical purposes the pure “Bush boy” is immune. Plehn has observed an individual susceptibility. I myself have known an officer in the Preventive Service of the new Northern Territories who is alleged to have had eight attacks, and I have a West Indian orderly who is just entering on his third: but blackwater is said to be milder as attacks recur.

Seasonable Prevalence.—Blackwater should present some relation to the malarial curve, and indeed, I find that it does; for I note during the past few years that the greatest number of cases occur in July and August, whereas the highest malarial ratio is in July. These months, it may be noted, are also the coldest months of the year, and consequently those in which a paludic is more likely to take a chill.

Incidental Causes.—The only incidental cause I feel disposed to admit of is change of climate, which again is possibly a question of “catching a chill,” already alluded to above. I consider it to be a real danger to send a patient home who is just getting over the last of a series of attacks of malaria. The homeward voyage from the Coast is very different to the one homeward from India. Cape Blanco on the West African Coast has an evil reputation in this respect. One meets here the full force of the cold north-east trade winds, then, given some previous fever, your patient develops a “chill on the kidneys,” and the ship slows down to drop him overboard. Thus Africa, who is difficult to rob of her children, claims her own. Hasty invaliding for malarial fever is worse than panic.

Summing up, then, the various etiological factors, one finds one’s self, amongst other things, face to face with the fact that, in spite of the malarial world being so large, the distribution of blackwater is, after all, very small and very local. Why does one only find it in certain parts of the malarial world? I have wondered myself why I got so many cases from Murree Town. One explanation
commonly given is that the distribution corresponds more especially with the distribution of the malignant tertian type of malaria. It is suggested that the parasite found in paludics in India is usually the benign tertian. This, however, hardly holds good if one goes about the world with a microscope. Personally, I have met malignant tertian in other parts of the world besides the West Coast, and in places, too, where there seemed to be just the same other etiological factors. For instance, I may mention the Mutwal district near Colombo, and the dense jungle country in the Eastern Province of Ceylon; in both these districts the type of malarial fever was the same malignant tertian variety that I meet with in Sierra Leone; yet in two years in Ceylon I never saw a case of blackwater fever or heard of one. It was the same at Koomati Poort, the same pernicious variety of malaria, but no blackwater. It seems, therefore, that one must fall back on some other actual determining factor, and herein lies the real fascination of the disease. And so one is obliged to look to "authority," and I must therefore recall some of the opinions and theories of the various experts.

Koch, in 1898, went out to the Kamerun, and, as the result of his investigations, came to the conclusion that the necessary formula was: malaria + quinine = blackwater. Then Koch and Tomaselli both quoted cases where the taking of quinine was actually followed by an attack of blackwater. These are classical instances. The news that quinine caused blackwater spread amongst laymen like wildfire, and, it must be admitted, has done a large amount of harm. One seeks for almost any excuse to avoid taking quinine, and a misunderstanding on the part of laymen on the teaching of the "quinine school" gave the very excuse which new- and old-comers were alike looking for.

In 1900, Stephens and Christophers came to the conclusions:
(a) That blackwater fever was malarial in origin; (b) that quinine in the great majority of cases was the proximate cause; (c) that there was no evidence of any special parasite.

Daniels, writing some years ago, noted that blackwater occurred in people taking quinine, but that it also occurred independently.

Crosse, who, be it said, has probably seen as much blackwater as any man, says: "I have not come across a single case in which people who have taken quinine regularly as a prophylactic have been attacked with blackwater." This is an opinion which I am afraid only a few medical men can endorse to-day; but all the same, the point may lie in the fact that those people who do contract
blackwater after religiously taking quinine as a prophylactic, have either not taken enough or else too much. Besides, no one can positively say that quinine will entirely prevent malaria, hence it is not an absolute prophylactic against blackwater.

H. Ziemann, in a long article, has recently reviewed the various theories; he asks, finally: Is blackwater (a) a disease *sui generis*? (b) a grave form of paludism? (c) or a simple quinine intoxication? And he comes to the conclusion that there seem to be two main causes: (1) A predisposition, resulting from former paludism, especially if no quinine has been taken; and (2) excessive treatment for malaria itself.

F. Plehn records twenty-four cases out of forty, where blackwater broke out after the administration of quinine, and A. Plehn gives forty-eight out of fifty-five, which he considers were directly attributable to quinine.

Quennac, in the Sudan, saw only one case of blackwater end fatally, and that in a doctor, who habitually took no quinine.

Scheube, in his “Diseases of Warm Climates,” though somewhat out of date now, reviews the matter, and thinks that the demand on the blood-forming organs in consequence of repeated attacks of malaria is excessive. A new invasion of parasites occurs, and a wholesale destruction of the red blood corpuscles follows, and thus blackwater originates. The hæmoglobin is partly carried to the liver, and there converted into bile pigment, and then partly excreted in the urine. In spite of the extraordinary figures originating from the Kamerun, this explanation certainly seems to accord with the ordinary clinical observations.

B. F. de Costa (St. Thomé) treats his cases by hypodermic injections of quinine in moderate doses and maintains that quinine is not the cause; while Ensor, in reviewing his series of cases, naively ascribes his only losing a small percentage of them to his refraining from giving them any quinine at all.

Plehn has suggested that some kidney lesion exists on top of the paludism, and his view is also suggested in a leading article in the *Indian Medical Gazette* for June, 1907. No doubt this view obtains credence from the fact that, coupled with the hæmoglobinuria, there is in almost every case a certain amount of albuminuria. I have never found any albumin in the urine, and what amount there is always clears up very quickly. None of my cases have ever been discharged from hospital as long as any albumin can be found in the urine. And it is surprising how soon this transitory
albuminuria clears up. This fact, on the face of it, does not look as if there were any primary kidney lesion.

Finally, the quinine school gets its mandate from Dr. J. J. W. Stephens, who says: "The etiology of blackwater fever may be summed up by saying that it is malarial in nature, that is, it can only occur in those who are suffering from, or have been recently affected with, malaria, and that the onset of blackwater is induced most commonly, though not invariably, by quinine.

In this edict there is not much theorising. It is for all practical purposes plain fact. Let it then be reiterated: (1) All who are familiar with blackwater admit the occurrence of previous paludism; (2) all now agree that individuals do exist who develop haemoglobinuria after taking quinine even in very small quantities; but (3) far from all agree that the most common cause of blackwater is quinine intoxication.

I came out to the Coast with opinions neither orthodox nor heterodox on this matter, but since I have had several cases of blackwater under my own care, and have had the opportunity of seeing other people's cases, I frankly confess I have come to consider the "quinine school" as the heterodox one. It seems to me that if one single case can be produced in which blackwater has occurred where quinine can be absolutely excluded (and these cases are well known and constantly met with), then the case for quinine as a proximate cause is considerably weakened. The converse, though, I cannot hold to be so true; since the relation between any drug's action and personal idiosyncrasy, is, as far as intoxication is concerned, and in our present state of knowledge, too uncertain a factor to be produced in argument.

Again, from figures worked out by Plehn, and quoted by Stephens, in connection with the incidence of the disease in quinine-takers and non-quinine-takers, I find the following:

<table>
<thead>
<tr>
<th>Quinine-takers</th>
<th>Non-quinine-takers</th>
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<tr>
<td>Attacks of malaria</td>
<td>30</td>
</tr>
<tr>
<td>Attacks of blackwater</td>
<td>6</td>
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<tr>
<td>Deaths from blackwater</td>
<td>0</td>
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To me it seems an extraordinary thing that the same drug acts in the one case as a partial protective, and in the other as a

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1 On the other hand, it must not be forgotten that malarial nephritis is by no means uncommon, though these cases bear no clinical resemblance to classical blackwater.
proximate cause. Further, it seems only likely, that if one treats an effect with a cause, except in the case of opsonic enthusiasts, the effect ought to be increased. Acting on my convictions, I have, as I shall show later, treated many of my cases with quinine, and with quinine in very large doses, with a view not to increase the effect, but to remove a cause by the specific action of quinine. I am quite prepared to admit that there are some people who are unable to take quinine without exhibiting the toxic effects of the drug. But these are people who, if they survive an attack of blackwater; should be invalided home and not allowed to come out again to any place that is malarial, or where they must take quinine to keep alive or in health.

I have noticed, too, amongst what I hope I may be pardoned for calling the rank and file of West African medical men, a majority in favour of quinine not being the exciting clause of blackwater; but when great "authorities," such as those I have already quoted, disagree, what then is the general practitioner to do? Imagine, he is away on some frontier, and his only white companion is down with blackwater, whilst the leading lights, sitting at home in professorial chairs, wrangle as to whether or not he is to give quinine to his dying patient. The situation is ridiculous and unworthy of modern medicine. An unfortunate point about the whole controversy is that so much is decided at home by "authority," and no one in the face of such authority cares for the responsibility of looking out for himself. One thus comes to the rather sad conclusion that, as far as blackwater is concerned, we know practically no more about it now than was known in 1898, when Dr. Crosse wrote his celebrated article on it in the Transactions of the Epidemiological Society of London.

No account of the various views held by authorities as to the proximate cause of blackwater would be complete without calling attention to the results that are known to occur after infection by various species of piroplasma. It will be remembered that the piroplasmata are probably closely related to the malarial parasite. They are generally placed in the class of Hæmosporida, which includes, of course, Plasmodia, Halteridia, Hæmogregarina, and the Piroplasmata. Piroplasmosis occurs in all sorts of animals, and the leading symptom in all its forms is hæmoglobinuria. In

1 Sir Patrick Manson's latest dictum appears to be: "Do not give quinine unless evidence of malaria exists." But that would only be in about 5 per cent. of cases.
D. S. Skelton

Dogs it is popularly called malignant jaundice. At the time of writing it is not generally agreed upon that there is a *Piroplasma hominis*, doubt having been thrown upon the piroplasmic origin of the spotted fever of the Rocky Mountains.

It may also be noted that certain forms of piroplasms under the microscope look almost exactly like the parasites of malignant tertian fever. The tick is the intermediate host of the piroplasma. But the tick does not infect any other animal directly; an infected tick gives rise to an infected progeny, so it is the next generation that is infective.

Before finishing with the etiology of blackwater, I may as well record here my own personal views on the subject. In my own mind, I see two conditions:—

1. Hæmoglobinuric paludism, which is what I personally mean when I speak of blackwater fever; (2) a quinine intoxication, supervening in many cases of paludism, which is what I personally do not understand by blackwater fever; and I am quite prepared to find that what I mean by blackwater fever will one day be found to be a disease *sui generis*, but a disease to which only paludics are susceptible.

**Symptoms.**

The classical symptoms are hæmoglobinuria, jaundice, pyrexia and vomiting. I do not remember to have seen any one symptom without the other three in any case of blackwater, no matter how slight.

*Hæmoglobinuria* is very often the first symptom that calls the patient’s attention to his condition. In colour the urine is in the great majority of cases almost exactly the colour of stout. Occasionally it is more reddish. Albumin is always present in small quantities. The hæmoglobin is present in the form of methæmoglobin. It may be recognised with the spectroscope. Blood cells are not usually present.

*Jaundice* is generally very deep. In spite of this, however, I have not met with a case where complaint was made of the itching, which is so troublesome in ordinary jaundice. I am inclined to think, however, that it does slow the pulse, as sometimes the pulse-count is out of all proportion to the condition of the patient. One may thus be deceived into a false security.

*Pyrexia.*—A great many patients, who afterwards develop blackwater, come into hospital looking very miserable, but with a temperature perhaps of only 99° F., and for a couple of days they
Some Observations on Blackwater Fever

remain in this state, when suddenly, they get a rigor, and the temperature rises to 104° F., or even higher. In my experience, cases of this sort are more likely to do badly than the type of case that was quite well "yesterday," and "to-day" has a high, bounding temperature, and marked jaundice. A point I think worth noting, is to watch that the temperature and the amount of urine do not fall together. I do not like to see a temperature of 99° F. and only 30 ounces of urine marked "up." I then look for a sudden suppression, and only too often it occurs. The vomiting calls for no particular comment, save that it is very distressing and persistent. It makes it well-nigh impossible to administer any drugs by the mouth, as they are all promptly returned. Patients usually complain of great pain in the back and limbs, and a severe headache is a constant symptom. I saw an erythematous rash in one case, but it disappeared very quickly. Personally, I gauge the efficacy of my treatment by the time the urine takes to become practically clear. This should happen any time after about forty-eight hours, but it often takes four or five days.

TREATMENT.

The exciting cause of blackwater is not merely a subject for academic discussion, but is a very vital point, as it must necessarily affect the treatment.

The lines upon which one may act are:—

(1) Anti-malarial, that is, according as one believes it to be a paludic hemoglobinuria, or vice versa; or (2) simply systematic, making no attempt at striking at any specific cause. This practically means a masterly inactivity. (1) of course will be combined with (2) as far as relieving distressing symptoms goes.

In a series of 20 cases, 8 have been treated with cassia bereana, i.e., practically under (2); 10 have been treated solely with quinine by injections; 2 have been treated with atoxyl by injection. Under systematic treatment there has been one death. Under atoxyl there was also one death. I have never lost a case that has been treated by quinine. This gives a total mortality of 10 per cent.

Under (1) the line I take up is as follows: I give a simple soap and water enema and get the rectum well washed out. I then slowly syphon into the rectum the following: quinine sulphate 50 grains, ac. hydrochlor., dil. q.s. to get it in solution, warm water 8 ounces. A small medicinal enema like this I have found is always well retained. What I have always wanted to find out is how much of this is really absorbed. But the technique for its
estimation is elaborate and beyond my resources. At the most I do not expect more than one-half is absorbed, if as much. I give quinine by the rectum, as I am convinced that it is the most comfortable way. If the hypodermic method is used, say, under the skin of the arm, more often than not the arm remains painful for some days, no matter what aseptic precautions be used. Very much the same happens if quinine be injected, say, into the muscles of the buttock. I have used this method frequently for treatment of ordinary malaria, but after having seen many cases suffer very severe pain at the site of injection, have discarded it. I repeat the quinine enema after twenty-four hours. Thus I consider I have, at any rate, removed the cause, and any stray parasite that may have escaped the first administration falls a victim to the second.

The most distressing symptom, as I have already mentioned, is vomiting. This must be relieved at the earliest. In the milder cases an effervescent mixture may relieve it, but this is useless in the more severe cases. For some years past at the Military Hospital at Tower Hill, Sierra Leone, trial has been made of the liquid extract of cassia bereana, a root that has acquired some notoriety in Zanzibar. It is claimed that this drug has some specific action as well. I have never seen it do any good, and the best that can be said of it is that it has never been known to do any harm. I had two West Indian negroes with blackwater both admitted to hospital the same day. Both had a high temperature, about 104° F., and both were passing black urine in fair quantities. I gave the one quinine enemata, and the other 40 minims of liquid extract of cassia bereana, every two hours. The quininised patient’s temperature dropped to normal in twelve hours, and did not rise again, and the following evening his urine was clear. The temperature of the other man came down by lysis, but his urine did not quite clear up for four days. I was greatly struck at the time by the contrast afforded by these two cases. The drug I rely on to stop the vomiting is morphine; I inject ½ grain and repeat it after six hours. I also apply hot fomentations to the stomach area. It has failed me only once, the case being Corporal F., who vomited almost incessantly for twenty-four hours. I neglect the fact that there may be an accompanying nephritis in blackwater cases. The vomiting has to be stopped, and morphine is the only drug that I know of that will do it quickly. I have seen no ill-effects from the use of this drug. As soon as it seems likely that the patient can retain anything in his stomach, I return to quinine, which
Some Observations on Blackwater Fever

I give in 5-grain doses every morning, accompanied with a tonic of iron and arsenic.

Those who do not believe in the efficacy of quinine, or rather those who believe it to be a positive danger, most usually fall back on the so-called "Sternberg treatment," as used for yellow fever. This consists in the administration of 10 grains of bicarbonate of soda, and 30 minims of liquid perchloride of mercury, to be taken every two hours, ice cold. The principle of the treatment in yellow fever is to "combat the acid diathesis."

Captain F. Harvey, R.A.M.C., has demonstrated that between soldiers in normal health and patients suffering from malaria there is no difference in the alkalinity of the blood. The same held good for a small series of blackwater cases that came under his notice. He then fed healthy natives on large quantities of carbonate of soda. It made not the slightest difference to their blood alkalinity. The theory of any acid diathesis in blackwater appears to be founded upon a coincidence of symptoms rather than upon scientific fact.

Treatment of Complications.—As long as the patient is secreting urine in fair quantity, that is, at least, 40 ounces per diem, I consider that he is progressing as well as may be expected, but when the quantity is diminishing, even if the temperature is falling, matters are not going so well. As I have remarked, this double fall is a kind of danger signal. I take it, there is a gradual mechanical blockage of the glomeruli in progress, and suppression is the next step. Two cases of practical total suppression I have met with yielded to a combination of a hot-air bath, 1 minim of croton oil, and the infusion of 4 pints of saline solution. In one case, by the time I had got the infusion apparatus ready, the patient was comatose and I had abandoned all hope of saving him. He rallied slightly after the infusion, and an hour afterwards had a most violent rigor; this was followed by profuse sweating, and he eventually passed 16 ounces of urine in the next twelve hours. An injection of pilocarpin is also an invaluable aid to the hot-air bath, &c.

Relapses are fairly common, and usually are a very serious complication. The mortality is generally high. Kohlstock, in the Kamerun, using no quinine, had no death in 8 cases. F. Plehn had 1 death out of 25 cases (4 per cent.), and A. Plehn lost 5 out of 53 (9.3 per cent.). These, apparently, were cases treated in hospital, for out of 53 treated outside 15 died (43 per cent.). The general average is stated to be about 20 per cent. Dr. Steuber,
D. S. Skelton

in his observations on the employment of European troops in the Tropics, puts the mortality from blackwater after cholera and before enteric. A. Broden, writing from Leopoldville, mentioned the case of a doctor who gave injections of quinine (gramme 1·50 to 1·80 a day), and lost 7 cases out of 12 (58 per cent.). Some other mortality statistics are Steudel (German East Africa), 16 to 17 per cent.; Reynolds (Gold Coast), 50 per cent.; Beranger-Feraud (Senegal), 23 to 24 per cent.; Koch (German East Africa), 21 per cent.; and Schellong (Malay Archipelago), 42 per cent.

Pathology.

It may be said that there are no very characteristic pathological changes to be found post-mortem. There are no marked changes in the stomach; this affords a differential point between blackwater and yellow fever, if any be needed. The spleen is enlarged, and melanin occurs in the splenic cells. One would expect to find considerable damage done to the kidney, but this is not the case post-mortem. The kidneys have never been described as presenting anything like the changes found in nephritis. Stephens describes some degenerative changes in the convoluted tubules. Blood examinations give various readings. In an ordinary case the red blood corpuscle count yields about 3·5 millions per centimetre, but it may be as low as 1·8 millions. On the Coast, provided that quinine has not been taken in any quantity before the attack, the parasite of malignant tertian may be found more often than not. And even if the parasite be absent, great stress is now laid on the mononuclear increase and the excess of pigmented leucocytes as evidence of a previous or present malarial infection. This was pointed out by Christophers and Stephens as long ago as 1901.

Experimental Pathology.—Major W. H. Grattan, R.A.M.C., has, I understand, tried some inoculation experiments on animals, but no results are reported up to date. Captain F. Harvey, R.A.M.C., has done the same, and he allows me to say that he has met with no definite results so far. Dr. Nabarro tells me he obtained a result in a monkey, but did not follow it up.

Later Note.—Captain F. Harvey, R.A.M.C., kindly allowed me to see many of the post-mortem examinations he made on dogs and other animals that were being tried with injections of perchloride of mercury and atoxyl. In every case there was extreme congestion of the kidneys, which was most likely due to the atoxyl. It appears to me, therefore, that atoxyl is not a drug that should be further tried in blackwater.
Some Observations on Blackwater Fever

D. S. Skelton

*J R Army Med Corps* 1908 10: 602-615
doi: 10.1136/jramc-10-06-03

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