NOTES ON BLACKWATER FEVER IN MACEDONIA.

BY COLONEL A. G. PHEAR, C.B.

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DURING the year ending October 31, 1918, 136 cases of blackwater fever were reported among the British troops in the Salonika Command. Of these cases thirty-six died, giving a case mortality of 26.5 per cent.

SEASONAL INCIDENCE.

Reference to the accompanying chart shows that 116 out of the total of 136 cases were reported during the months December to April inclusive, and that the case incidence reached its maximum during February, in which month 32 cases were reported. During the summer months from June onward no case of blackwater fever occurred until September, in which month nine cases were reported. The cause of this striking and unexpected recrudescence in late summer is not clear, but in point of time it was clearly associated with a period of exceptional fighting activity, the outbreak being limited to the three weeks immediately preceding the enemy capitulation. In the following month (October) only a single case was reported.

In the course of the winter (1917-18) there were three short spells of exceptionally rigorous weather. The first of these was in early December and coincided with the onset of the blackwater fever "season." The season was in the beginning of January and was followed by no material increase in the number of cases reported. The third was at the end of March, when a blizzard of exceptional severity was perhaps responsible for the check in the rapid fall of the blackwater fever curve which had already set in.
Notes on Blackwater Fever in Macedonia

It is of interest to note that, though the admissions from blackwater fever were considerably higher during February than in any other month of the year, there was no specially severe weather either in that month or in the latter half of the previous one. While the general rule holds good that blackwater fever is limited almost exclusively to the cooler months of the year, it would seem that temporary weather vicissitudes have but little effect on the incidence of the disease.

Nearly eight times as many cases of blackwater fever were recorded during the year under review as in the previous year. The actual figures are 136 for the season 1917-18, as compared with eighteen for 1916-17. The reason for this striking difference is not clear. It has been suggested that the tendency to blackwater is greater in those who are in their second season of malaria; but this is not sufficient explanation, as out of forty-seven cases in which precise information is available, in only ten did the first attack of malaria date back to twelve months or more before the
onset of blackwater; in twenty-four cases (roughly fifty per cent) the first attack of malaria fell within six months of the occurrence of blackwater fever.

**Relation to Malaria.**

In the aetiology of blackwater fever the outstanding feature is the constant association of malaria as an antecedent condition. In only one out of a series of seventy-eight cases was a history of malaria not forthcoming, and in view of the special prevalence of malaria in the command it may fairly be assumed that this solitary case was no more than an apparent exception to the rule. But apart from this general relationship, there has been found in the majority of cases no evidence of active malaria accompanying the actual attack of blackwater. Of fifty-eight cases in which careful search was made in sixteen only (twenty-seven per cent) were parasites found in the peripheral blood during the attack, and in nearly all of these the degree of infection as judged by the number of parasites present was a very slight one. Of these 16 positive cases 7 were examples of benign tertian, 4 of malignant tertian malaria, while in 5 the parasites were described as being of indeterminate type. In six cases in which repeated search for parasites was made during the actual attacks with consistently negative results, positive films had been obtained shortly before the attack at intervals varying from two to ten days. In one case there were two paroxysms of blackwater at four weeks' interval; in neither of the attacks were parasites found, but benign tertian rings in small numbers were present on one occasion during the interval.

These observations are in conformity with the view that some factor other than a direct malarial infection is effective in determining the actual explosion which constitutes the attack of blackwater fever—a view which gains further support from the consideration that the incidence of blackwater fever is at its height during the period of the year at which malarial activity is at its lowest ebb. The opposite would presumably be the case if blackwater fever were no more than a special manifestation of malaria.

**Quinine and Blackwater Fever.**

The experience of the past season has supplied no evidence in support of the hypothesis that the determining cause of an attack of blackwater is to be found in quinine administration. On the contrary, the facts are opposed to this hypothesis. Included in the series is a number of cases in which quinine as an exciting cause can be definitely excluded, as during a considerable period previous to the onset of blackwater no quinine had been given. On the other hand cases of severe blackwater fever occurred in which the condition cleared completely and rapidly notwithstanding the administration of quinine in large doses. With a few medical officers the view was favoured that blackwater was a symptom of a specially pernicious form of malaria, and acting on their convictions they were in the habit of
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treating cases of blackwater fever with full doses of quinine. Apart from
the question as to whether this was sound treatment, the cases thus
treated have provided clear proof that the administration of large doses of
quinine is no bar to the speedy and complete disappearance of haemoglobin
from the urine. These cases have at least done no worse than those in
which quinine has been at once stopped with the onset of haemoglobinuria.
In the light of the above facts it is impossible to regard quinine as a direct
causal agent in the production of blackwater fever, or even as a subsidiary
factor in maintaining or adding to an effect which is directly due to some
other agent. This conclusion is not invalidated by the well-known fact
that certain individuals, not necessarily the subjects of malaria, are
peculiarly susceptible to the effects of quinine, and that small quantities of
the drug may in these cases lead to haemolysis, with the production of
haemoglobinuria. An instance of the kind was met in the case of a Greek
boy, who had had malaria, and in whom a dose of quinine was followed by
haemoglobinuria with the development of an intense grade of anaemia. It
was thought the matter might be a coincidence, and accordingly, as there
were special reasons for quinine treatment, a further small dose of ten
grains was given after an interval of a week or two, but with the same
dramatic effect of an intense haemolysis with severe anaemia. Examples of
such idiosyncrasy are fortunately very rare.

CLINICAL FEATURES.

From the clinical point of view the cases observed during the past
season have presented no exceptional features. The sudden haemolysis of
catastrophic severity, with a corresponding drop in the number of red
corpuscles to thirty per cent or even twenty per cent of the normal number,
the state of collapse with extreme pallor and small compressible pulse, the
dark coloured urine with characteristic spectroscopic phenomena, rapidly
clearing in the course of a few days, the pyrexia of sudden onset with head­
ache, shivering and pains in the loins all go to form a disease picture
which in the cases under review differs in no essential respect from other
epidemics.

The average time during which haemoglobin was present in the urine
was three days. The longest period was five days; the shortest was in the
case of a patient who on one occasion only passed dark red urine showing
characteristic absorption bands; all subsequent specimens were clear.
Recurrences were uncommon; in 6 cases only of a total of 78 (8 per
cent) was there a definite relapse of blackwater. In four of these cases
the second attack was relatively mild; the other two were fatal. In one
case of blackwater lasting for five days there were two occasions on which
perfectly clear urine free from blood was passed; subsequent urine was
depthly coloured with blood.

In almost every case there was enlargement and tenderness of the liver.
This was in many instances associated with vomiting and jaundice. The degree of jaundice varied from a slight icteric tinge to a deep coloration of the skin, and afforded a fairly reliable indication of the seriousness of the case. Of 30 fatal cases, in 13 there was well-marked jaundice, and in 17 the generalized icteric staining of the skin was intense.

Suppression of urine was an almost constant feature of the fatal cases. In 18 of 30 fatal cases the suppression was complete and of the remainder in all except 2 the excretion of urine was reduced to a few ounces per diem. In only two of the fatal cases was a fair excretion of urine maintained; in one of these death was due to cardiac failure, on the fifth day after the urine had become completely clear from hæmoglobin; in both the heart was found post mortem to be considerably dilated with pale and flabby muscle. The conclusion was justified that so long as urine is being freely passed the risk of a fatal event is almost negligible.

Repeated vomiting and uncontrollable hiccough were common symptoms in the cases with suppression of urine, but other manifestations of acute uræmia were not observed. In a few cases the symptoms resembled those of a calculous anuria, the patient remaining for several days with urinary suppression, but, until a few hours from the end, with symptoms so slight as to convey no indication of the extreme gravity of the condition.

The volume of urine secreted during the blackwater phase in non-fatal cases without suppression was very variable. Careful measurements were made in a series of twenty-five cases. In 9 cases the daily average quantity passed during the period of blackwater was higher than 60 ounces with a maximum of 85 ounces; in 16 cases the daily average was below 60 ounces with a minimum of 20 ounces. The average of the whole series was fifty-three ounces. When the large quantity of fluid which was being administered and absorbed in all these cases is considered, the somewhat excessive amount of urine secreted in a few instances is readily accounted for and there is no ground for assuming that hæmoglobin in its passage through the kidney had any specific action as a diuretic. In a few cases the presence of hæmoglobin in solution in the blood plasma was demonstrated. No extended observations were made on this point.

The number of cases in which a series of leucocyte counts was made is small, but the evidence so far as it goes is not in favour of a marked leucocytosis as a common feature of blackwater fever. Of 9 cases, in 1 only were there more than 10,000 leucocytes to the cubic millimetre; in this case the highest leucocyte count was 10,600 on the fourth day of blackwater; of the remaining 8 cases the highest count ranged from 3,600 to 8,800.

Uninterrupted convalescence was the rule with a rapid formation of new blood-elements even in cases which initially were of great severity; it was a common experience to find that in the course of three weeks the number of red corpuscles had been more than doubled. No hæmoglobin observations are recorded here as it was found that the instruments had suffered from the climate and were not reliable.
TREATMENT.

The three essentials in the treatment of blackwater fever were found to be: (a) Rest: (b) protection against chill by suitable coverings, hot water bottles, etc.; (c) the introduction of large quantities of fluid into the system with the object of maintaining a free flow through the kidneys and thereby lessening the risk of suppression of urine. Whenever possible cases were treated in hutted hospitals; but in cases arising at a distance it was not considered justifiable to expose the patient to the risks involved in a journey to the base and such cases were treated at the nearest tented hospital or casualty clearing station. As far as practicable the requisite amount of fluid was given by mouth; but in cases of vomiting or when it was required to supplement the amount taken by mouth, the subcutaneous, rectal, or intravenous routes were utilized. The slow rate at which fluid is absorbed subcutaneously, or per rectum, rendered these channels preferable to the intravenous route. Rectal injections were usually given in quantities of fifteen to twenty ounces of normal saline at intervals of four or six hours. In a few cases by means of a Souttar's apparatus continuous rectal saline was administered up to five or six pints in the twenty-four hours with very satisfactory results; the fluid is absorbed as it flows into the rectum and the method is free from drawback other than the slight inconvenience caused by the presence of the rectal tube. Dry cupping over the loins appeared in some cases to be beneficial. For the treatment of vomiting and obstinate hiccough Sternberg's mixture containing sodium bicarbonate and perchloride of mercury was given in some cases, but without any marked degree of success. In a few instances these refractory symptoms were checked by hypodermic injections of hyoscin ($\frac{1}{10}$ grain) combined with morphia ($\frac{1}{10}$ grain).

The usual practice as regards quinine has been to withhold it in the absence of definite evidence of active malaria. The presence of parasites in the blood, or of tenderness over an enlarged spleen, have been taken as indications for quinine, which has then been given in effective doses until the malarial manifestations have been brought under control.

After the subsidence of acute symptoms a generous and varied diet has been found essential to the establishment of a satisfactory convalescence. Arsenical preparations are of great value in promoting blood formation. Arsenic has been given (a) by the mouth, either alone or in combination with iron; (b) subcutaneously in the form of sodium cacodylate; (c) intravenously in the form of galyl in doses of 0·2 or 0·3 gramme at intervals of four days; three doses are generally sufficient. In a few cases of extreme anaemia galyl has been used with remarkable benefit; in cases of anaemia of ordinary severity the subcutaneous and intravenous methods do not offer any special advantages over the method of oral administration except in cases where on account of gastric disturbance it is undesirable to push arsenic by the mouth. All convalescent cases were evacuated by hospital ship as soon as a convenient opportunity arose.
In illustration of the above remarks charts and short notes of fifteen cases are appended. The amounts of quinine are stated in grains; O signifies oral, IM intramuscular quinine. In estimating the percentage of red blood corpuscles, 5,000,000 is taken as the normal standard. In most cases the presence of haemoglobin in the urine was proved by spectroscopic examination; in some units no spectroscope was available, and microscopic examination was relied on for differentiating between haemoglobinuria and haematuria.

No. 67.—Aged 36. First attack of malaria in August, 1917; many relapses since; in hospital six times. Quinine, none for seven weeks prior to admission. Taken ill on February 13 with shivering, headache and vomiting. Slight jaundice, deepening later. Spleen large. Benign tertian parasites present at onset. (Chart 2.)

Note.—No quinine previous to blackwater. Rapid improvement while taking quinine.

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No. 52.—Aged 38. History of one attack of malaria only, in July 1917. No quinine since December. Noticed urine was "quite dark" on March 6 and 7; admitted on following day. On March 12 benign tertian parasites found in blood; previous films had been negative. (Chart 3.)

Note.—No quinine since December. No recurrence of blackwater during quinine treatment.

No. 22.—Aged 27. Five attacks of malaria, the last December 14, 1917. Very pale and ill on admission, with marked jaundice. Spleen and liver enlarged and tender. No parasites found. (Chart 4.)

Note.—Rapid clearing of blackwater during administration of considerable doses of quinine.
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Chart 3

Chart 4

Chart 5
No. 36.—Aged 25. Malaria, "two or three attacks." Spleen large and tender. Liver tender. Marked jaundice. (Chart 5.)

Note.—Satisfactory clearing of urine during quinine administration.

No. 42.—Aged 24. Malaria, eight attacks, date of first not stated. Admitted to base hospital on February 1, 1918, passing urine like blood, free in amount. Characteristic absorption bands. No parasites found in blood. (Chart 6.)

Note.—Clearing of blackwater while taking quinine. Rapid formation of new blood-elements.

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Chart 6.

No. 60. Aged 25. First attack of malaria July, 1917; twelve relapses. On admission vomiting, deep jaundice, spleen large and tender, benign tertian parasites in blood, urine dark brown with characteristic absorption bands. (Chart 7.)

Note.—Rapid clearing of urine while under quinine treatment.

No. 68.—Malaria first attack September, 1917, frequent relapses. On March 19, 1918, a malarial relapse; quinine thirty grains given this day and subsequently. An attack of blackwater five days later clears during continuance of quinine. (Chart 8.)

Note.—Blackwater runs its course apparently uninfluenced by quinine.
No. 29.—Aged 33. First attack of malaria June, 1916, two attacks since. Admitted for malarial relapse in December, 1917. (Chart 9.)

*Note.*—An attack of blackwater fever a few days subsequent to a relapse of malaria (clinical). Quinine was stopped with the onset of blackwater.

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**Chart 7.**

No. 57.—Aged 21. No notes as to malarial history. Has had quinine in ten- or twenty-grain doses daily during last month. Was ill three days before admission with vomiting and shivering, but noticed that urine was dark for the
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**Red blood corps.:** 63% 56% 34%
Notes on Blackwater Fever in Macedonia

first time on day of admission. Spleen and liver both enlarged and tender. Vomiting. Slight jaundice. No parasites found in blood on repeated examination. After two days of blackwater a perfectly clear sample of urine was passed containing no blood. This was followed by plentiful urine tinged red. Next day the urine was clear; on the following day a large amount of very dark haemoglobin containing urine was passed. (Chart 10.)

Note.—Interruptions in course of blackwater; rapid fall in number of red corpuscles.

No. 25.—Aged 31. Twelve attacks of malaria. No quinine for six weeks previous to onset of blackwater. Noted that urine was dark a few hours before admission. Vomiting and jaundice, marked symptoms. Liver enlarged and tender. Spleen tender. No quinine given. Almost complete suppression of urine; becomes clear two days before death. (Chart 11.)

Note.—A fatal case in which no quinine had been taken for six weeks previous to attack. Urine clears two days before death.

No. 63.—Aged 41. Ill three days with "malaria"; no previous history. Jaundice on admission; becomes intense. Vomiting. Spleen to umbilicus. Blood-films negative. Suppression of urine. Death. (Chart 12.)

No. 46.—Aged 23. Admitted to casualty clearing station on November 24 with history that he was taken ill on November 22 with headache, shivering, vomiting, and passing urine coloured like blood. On admission temperature was 105·2° F.; he was very anæmic, with large tender spleen and tender liver. Frequent vomiting and severe hiccough. From date of admission to his death five days later (on November 29) no urine was passed with the exception of a few cubic centimetres on November 25, porter-coloured and showing characteristic absorption bands. Hiccough and vomiting ceased two days before death. Blood
A. G. Phear

films were negative on repeated examination. The temperature chart of this case is unfortunately missing.

Note.—Suppression of urine, practically complete, for five days previous to death.

![Temperature chart](chart13.png)

**Chart 13.**

![Temperature chart](chart14.png)

**Chart 14.**

No 40.—Aged 24. First attack of malaria July, 1917; two or more attacks since; malignant tertian parasites found in September, 1917. On February 11, 1918, shivered and was sick and passed no urine after 11 a.m. On following day passed 1½ ounces of porter-coloured urine giving characteristic spectroscope bands. Marked jaundice. Liver and spleen—both enlarged and very tender.
Blood-film negative. On February 15 the urine was clear. Subsequently his general state rapidly became worse and he died on February 18, although the urine remained clear and had increased somewhat in quantity. (Chart 13.)

**Note.**—The clearing of urine and slight increase in quantity during the two or three days before death.

No. 62.—Aged 27. First attack of malaria July, 1917. Eleven relapses. Jaundice slight at onset; deepens and becomes intense with frequent vomiting and large tender liver. No parasites found. Death two days after urine had become clear. (Chart 14.)

**Note.**—Recurrence of blackwater; free secretion of urine to the end; rapid destruction of red corpuscles.

No. 18.—Aged 27. Malaria “frequent attacks.” Death from cardiac failure on fifth day after urine had become clear. (Chart 15.)

**Note.**—Abundant urine secreted up to date of death.

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**In conclusion** I wish to express my indebtedness to medical officers and officers in charge of divisions of hospitals, who by their careful observations made often under difficult circumstances have provided the data on which the above notes are based. I have to thank Lieutenant-Colonel J. A. Anderson, R.A.M.C., for the curve showing the seasonal incidence of the disease.