MALARIA IN INDIA: THE SYNTHETIC DRUGS AND THE RELAPSE RATE.

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AND

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(Continued from p. 17).

SEASONAL INCIDENCE OF MALARIA CASES.

It might possibly be argued that the low relapse rate shown in the preceding tables is not a new occurrence: that the decline in the number of admissions for malaria which has taken place in the last few years is due to other causes such as the cumulative effect of anti-mosquito work, exceptionally favourable climatic conditions, etc.; and that the apparently low percentage of relapses is incidental to the general improvement which has taken place.

To test this argument, the question has been approached from another angle.

As a general rule, most primary attacks of malaria occur in the second half of the year (July to December) and relapses from these cases occur in the following January to June. It is fully realized that there are exceptions to this arbitrary rule. Relapses can, and do, occur in the July to December period. Similarly, "delayed" cases not infrequently make their first appearance in the early months of the year; and fresh cases are common in June, are occasionally found in May, and in certain parts of India may occur all the year round. Nevertheless, a comparison of the relative numbers of cases occurring in the July to December period and in the January to June period affords valuable information.

If the reduction in malarial incidence is due to the general causes mentioned above, and not to a decline in the relapse rate, the relative proportion of the cases occurring in the July to December and the January to June periods will remain unaltered, irrespective of the total number of cases. If, however, the decline is related to a decreased number of relapses, the January to June cases will form a smaller proportion of the July to December cases than in former years. This decrease will not, of course, indicate the true reduction in the relapse rate, partly because of the inaccuracies in the comparison which have already been mentioned, and partly because the July to December figure will also be lowered by the occurrence of fewer "short term" relapses.

The figures from which the following tables and graphs are compiled
are taken from the monthly returns of hospitals which, for this purpose, are both convenient and sufficiently accurate.

Graph II shows the incidence of malaria, by months, for the last ten years. All admissions to hospital for every type of malaria, whether fresh or relapse, are included.

This graph shows a tendency towards a lowering of the peak of the annual wave, which feature is, however, by no means constant. For example, in 1933 there is, in October, a higher admission ratio than in any one month since 1926. This was the consequence of unavoidable exposure to infection during the Mohmand-Bajaur operations; and was also related to an epidemic wave of malaria which occurred among the civil population of the north-western quadrant of India at that time. Nor are the low peaks of 1932 and 1934 in any way unique, as a still lower level was reached in 1928.

Of recent years, the most important difference is to be seen in the trough of the wave. This difference can best be appreciated by comparing the trough which followed the climatically “good” year, 1928, with that which followed the climatically “indifferent” year, 1934. (See Graph III.)

In 1928, as the result of severe and widespread drought, mosquito breeding was reduced to an extent which is never likely to be equalled by the usual routine anti-malaria field measures. In 1934 climatic conditions, averaged over the whole of India, were more or less inimical to a good malaria year.
From the superimposed curves of these two periods, it will be seen that, in 1928-29, there was a fairly rapid rise, a sustained peak, a moderately rapid defervescence, and a well-marked rise from April running into the fresh cases of 1929: while in 1934-35 the peak was much sharper, the defervescence more rapid and to a lower level, and there was a much flatter curve until the rise began in July. A decrease in the relapse rate in 1934-35, as compared with that of 1928-29, would afford a complete and satisfactory explanation of the difference of the two curves, and there can be little reasonable doubt that this is, in fact, the correct explanation. The standard treatment in 1928 was quinine alone, given in intensive and protracted courses.

In Table XII the same facts are shown in a slightly different way. An admission ratio is calculated for the July to December period of one year, TABLE XII.—MALARIA CASES OF THE JANUARY TO JUNE PERIOD SHOWN AS A PERCENTAGE OF THE CASES OCCURRING IN THE PREVIOUS JULY TO DECEMBER.

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<tbody>
<tr>
<td>All-India</td>
<td>31·8</td>
<td>24·7</td>
<td>28·4</td>
<td>24·1</td>
<td>92·9</td>
<td>16·8</td>
<td>16·9</td>
</tr>
<tr>
<td>Northern Command</td>
<td>29·1</td>
<td>24·6</td>
<td>23·6</td>
<td>20·3</td>
<td>21·3</td>
<td>9·8</td>
<td>10·9</td>
</tr>
<tr>
<td>Rawalpindi District</td>
<td>32·8</td>
<td>19·5</td>
<td>21·6</td>
<td>21·9</td>
<td>20·6</td>
<td>14·8</td>
<td>6·3</td>
</tr>
<tr>
<td>Peshawar District</td>
<td>19·9</td>
<td>18·5</td>
<td>24·8</td>
<td>18·8</td>
<td>14·9</td>
<td>6·3</td>
<td>3·7</td>
</tr>
<tr>
<td>Western Command</td>
<td>22·3</td>
<td>9·3</td>
<td>44·5</td>
<td>29·4</td>
<td>22·0</td>
<td>26·2</td>
<td>13·5</td>
</tr>
<tr>
<td>Eastern Command</td>
<td>38·3</td>
<td>24·2</td>
<td>27·9</td>
<td>21·6</td>
<td>17·0</td>
<td>24·9</td>
<td>18·9</td>
</tr>
<tr>
<td>Southern Command</td>
<td>40·9</td>
<td>29·0</td>
<td>38·0</td>
<td>31·5</td>
<td>33·0</td>
<td>25·7</td>
<td>27·4</td>
</tr>
</tbody>
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and another for the January to June period of the subsequent year. The latter is then shown as a percentage of the former.

Of these figures the most reliable are those relating to "All-India." Where smaller numbers are involved, errors of considerable magnitude can be introduced by the departure during the troop season of heavily infected regiments, and their replacement by others fresh from home. A similar fallacy arises through the transfer of units in different Commands from malarious to non-malarious stations and vice versa. In the "All-India" figure these factors are more or less constant from year to year.

It will be seen that, until 1932-33, the figures showed little variation; but in 1933-34 and 1934-35 there was a well-marked decrease. It was from the beginning of the malaria season of 1933 that standard treatment with atabrine-plasmoquine or quinine-plasmoquine became universal.

The figures of the Northern Command are similar to those of "All-India," but the drop from 1933 onwards is more marked. Owing to climatic conditions, malaria in Rawalpindi and Peshawar Districts follows the seasonal incidence postulated above more closely than elsewhere in India and a very marked decrease in the ratio has taken place. In the last two years, the exchange of units in these two districts has not been such as to affect the incidence of attacks in the January to June period.

In the Southern and Eastern Commands a similar tendency can be seen; but in both of these areas (particularly Southern Command) primary cases are fairly common in May and June, and the comparison is, therefore, somewhat vitiated. The position in the Eastern Command is further complicated by the fact that experimental courses of treatment, as described above, and not the standard courses, were used in three important stations which provide almost a half of the malaria cases in the Command.

In the Western Command the number of troops is approximately that of a district. The effect of changing over a regiment is considerable, and can be seen here and there—as, for example, in 1929-30, when a heavily infected regiment departed and was replaced by one fresh from home. Here, also, primary cases are relatively numerous in May and June in those years when climatic conditions are favourable. Hence, the drop in 1934-35 is of no particular significance, except for the fact that it coincides with a similar state of affairs elsewhere in India.

Taken as a whole, and in spite of various fallacies, this comparison of the January to June incidence of malaria with that of the preceding six months affords strong corroborative evidence of a decline in relapses in the last two years.²

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¹ The same applies to Kohat and Waziristan Districts; but the number of British troops in these districts is too small to give reliable figures.
HISTORY OF THE MALARIA TREATMENT CENTRE.

The evidence afforded by the history of the Malaria Treatment Centre has already been cited elsewhere, but will bear repetition, as it is a matter independent of statistics and of the criticisms to which statistics are always open.

In 1924 a Malaria Treatment Centre, with hospital accommodation for 30 cases and ordinary accommodation for 200 convalescents, was opened at Kasauli with the double object: (a) Of allowing relapsing cases of malaria to recuperate in a salubrious climate; and (b) of concentrating intractable malaria cases in a station where there was no chance of reinfection and where, in consequence, the results of various forms of treatment could be accurately assessed. It was at the Malaria Treatment Centre, after various trials, that the present quinine-plasmoquine and atebritin-plasmoquine courses were worked out.

The number of cases admitted to the Malaria Treatment Centre is as follows:—

<table>
<thead>
<tr>
<th>Year</th>
<th>Cases</th>
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<tbody>
<tr>
<td>1928</td>
<td>243</td>
</tr>
<tr>
<td>1929</td>
<td>248</td>
</tr>
<tr>
<td>1930</td>
<td>241</td>
</tr>
<tr>
<td>1931</td>
<td>126</td>
</tr>
<tr>
<td>1932</td>
<td>63</td>
</tr>
<tr>
<td>1933</td>
<td>51</td>
</tr>
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</table>

During 1932 and 1933 circulars were issued urging hospitals to send all suitable cases to Kasauli, as there was no wish to close the Centre but, on the contrary, a desire to investigate certain further lines of treatment. These measures were of no avail for the simple reason that suitable cases did not exist, and it was reluctantly decided to close the Malaria Treatment Centre in March, 1934.

It can be seen from Tables V and VIII that the decrease in chronic relapsing cases has continued.

RELATIVE PROPORTIONS OF BENIGN AND MALIGNANT TERTIAN MALARIA.

A decrease in relapses might arise from a change in the variety of malaria, i.e., fewer benign tertian and more malignant tertian cases—the latter being much less liable than the former to relapse between January and June. As can be seen in Table XIII—in which the incidence of malignant tertian malaria is shown as a percentage of the total benign and malignant tertian cases reported in the same year—annual fluctuations have occurred, but no progressive change has taken place.

<table>
<thead>
<tr>
<th>TABLE XIII.—MALIGNANT TERTIAN MALARIA SHOWN AS A PERCENTAGE OF THE TOTAL BENIGN AND MALIGNANT TERTIAN MALARIA OCCURRING IN THE SAME YEAR.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Year</td>
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<tr>
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<td>%</td>
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</tbody>
</table>
Malaria in India: Synthetic Drugs and the Relapse Rate

DISCUSSION.

From the evidence contained in the foregoing paragraphs, we consider there can be no reasonable doubt that, in recent years, there has been a decline in the relapse rate which has improved the malaria figures: directly, by lessening the actual number of admissions; and indirectly and cumulatively, by reducing the number of cases of the type best adapted for maintaining the chain of infection. In India, however, there is in the civil population such a bottomless reservoir of infection, that the importance of the latter factor is minimised.

The decrease in relapses may be due either: (a) To a change in the type of malaria prevalent in India; or (b) to the use of the new synthetic drugs.

(a) Time will decide whether or not, in the last few years, the type of malaria has undergone a change, presumably through some alteration in the properties of the prevailing parasite. As far as the evidence at present available goes, there is no suggestion of any such change. There is no significant variation in the relative proportions of benign and malignant tertian malaria; and there is nothing to suggest that a material alteration has appeared in the disease as it occurs in the civil population. The same applies to malaria amongst Indian troops, of which the annual admission ratio is shown in Graph IV.

GRAPH IV
A GRAPH SHOWING THE ANNUAL INCIDENCE OF MALARIA (NUMBER OF ADMISSIONS PER 1000 OF STRENGTH) AMONGST INDIAN TROOPS (OTHER RANKS) FROM 1919 TO 1935

This graph bears little resemblance to that in respect of British troops (see Graph I). There is no gradual decline in incidence and, from 1929 onwards, owing to various factors (of which the occupation by a complete Indian Brigade of the very malarious station of Wana in Waziristan is one) the figures run at a considerably higher level than in the preceding
years. The high incidence in 1933 is caused by infection contracted during Frontier operations in the malaria season of a year in which the disease was epidemic amongst the civil population of this area.

The absence of improvement in the figures of Indian troops—as compared with those of British troops—is related to the leave which is granted each year during the malaria season to approximately a third of the Indian Army. The men go off to their villages, where they are infected or reinfected with malaria, and are treated by “indigenous” methods. They return uncured to their units, and usually relapse a short time after rejoining. This is a universal experience which is commented on annually in the hygiene reports received from commands and districts. There is no reasonable doubt that this affords an explanation of the lack of improvement in the total incidence of malaria in Indian troops; and it goes to show that inadequately treated malaria is just as liable to relapse as ever it was. On the other hand, it is very interesting to note that the ratio of January-June to the preceding July-December cases corresponds very closely with that of British troops (see Table XIV) and shows a very similar drop, which coincides with the introduction of standard quinine-plasmoquine and atebrin-plasmoquine treatment in 1933.

It seems probable that, given a few years with no abnormal cause of heavy infection—such as active operations during the malaria season—an appreciable fall in the incidence of malaria in Indian troops will ensue, although the endemic level will remain higher than in British troops because of periods of leave spent in areas of high infectivity.

<table>
<thead>
<tr>
<th>TABLE XIV.—MALARIA CASES OF THE JANUARY TO JUNE PERIOD SHOWN AS A PERCENTAGE OF THE CASES OCCURRING IN THE PREVIOUS JULY TO DECEMBER (BRITISH AND INDIAN TROOPS).</th>
</tr>
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<tbody>
<tr>
<td>British troops</td>
</tr>
<tr>
<td>Indian troops</td>
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No evidence, therefore, is forthcoming to show that the decline in the relapse rate can be explained by a change in the type of malaria.

(b) As regards the second possible cause, there is a close relationship between the decrease in relapsing malaria among British troops and the gradual introduction of plasmoquine.

In 1929 and 1930 this drug was used in an experimental way in certain selected hospitals.

In 1931 a limited quantity of plasmoquine was purchased and issued to hospitals all over India, but the quantity available fell short of requirements. The first indication of improvement is seen in the reduction of the number of cases sent to the Malaria Treatment Centre, to approximately one-half its previous level.
In 1932 plasmoquine was available for general use on an "as required" scale, but—in the absence of instructions enjoining its administration in all uncomplicated cases—was not employed as freely as it might have been, and in many cases was given in inadequate doses. Despite this, and without being dependent on unusually favourable climatic conditions, the "All-India" admission ratio fell to a record low level, and admissions to the Malaria Treatment Centre to approximately 25 per cent of what they were in 1930.

In 1933 instructions were issued that standard courses of quinine-plasmoquine or atebrin-plasmoquine (atebrin having now been authorized for general use) should be given to all cases, unless there were direct indications to the contrary. In the autumn of that year, during the course of a malaria epidemic among the civil population, operations took place in the Mohmand-Bajaur country. As a result, high admission rates were returned in September, October, and November; but, nevertheless, the "All-India" figure was remarkably good.

Admissions to the Malaria Treatment Centre fell still further, and it became necessary to close down the Centre.

In the January to June period of 1934 admissions as compared with those of the previous six months showed a lower ratio than formerly had been recorded, and the figures for the whole year—again without being helped by unduly favourable climatic conditions—were 19·7 per cent under those of the record year, 1932.

In 1935 the cases of the first six months again formed a small proportion of those of the preceding July to December; but a further drop in the total admission ratio was frustrated by Frontier operations during the malaria season. In extent, these operations exceeded those of 1933: but in spite of this, the 1935 figure (calculated on monthly returns, and therefore subject to correction by the annual figure based on statistical cards) was only 0·5 per 1,000 higher than the record figure of 1934. Excluding the cases (and strengths) from the troops engaged in the above Frontier operations the 1935 malaria figure was 56 per 1,000, which is 17 per cent lower than that of 1934.

It was originally shown in India, in experiments carried out by Sinton and others at the Malaria Treatment Centre, that the addition of plasmoquine, in small doses, to quinine, in therapeutic doses, provided a form of treatment which had a well-marked action in preventing relapses in benign tertian malaria. This discovery was subsequently confirmed in larger experiments reported by Manifold (1931), and in special investigations such as those conducted by Dixon (1933). At a later date, the Malaria Treatment Centre tried out atebrin followed by plasmoquine, with very similar results.

There seems little reason to doubt, and good cause to believe, that these forms of treatment have been equally successful when applied on an All-India scale. By reducing the number of relapses, they are largely
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responsible for the fall in malaria admissions which for the last few years has been in progress.

We are aware, of course, that these conclusions are at variance with the opinion expressed by the Malaria Commission of the Health Organization of the League of Nations. In its report, "The Therapeutics of Malaria" (1933), the statement is made that doses of plasmoquine such as we recommend are "non-effective"; and it is considered that, while the administration of these quantities of plasmoquine in addition to therapeutic doses of quinine (presumably also of atebrin—although this drug is not specifically mentioned) may give better results than quinine (or atebrin) alone in preventing relapses, "the proposition can only be described as being paradoxical."

As far as can be gathered, this opinion is based chiefly on the fact that plasmoquine, when used alone, is not effective in the treatment of benign tertian malaria, unless dangerously large doses are exhibited; and on the failure of combined treatment to abolish relapses in a series of cases (number not stated) infected with the Madagascar strain of the parasite. Although it is not mentioned, it appears probable that these were artificially infected cases.

We have no experience with the Madagascar strain of Plasmodium, nor of the treatment of artificially infected individuals; but we feel confident that the evidence we have produced clearly shows that, as far as the current Indian strain of Plasmodium vivax is concerned, plasmoquine in these doses given with quinine or atebrin, does exert an influence in preventing relapses. We suggest that this treatment may have some specific action on that phase of the parasite (which we are not prepared to define) responsible for keeping the infection alive between attacks.

STANDARD COURSE OF TREATMENT FOR USE IN ALL TYPES OF MALARIA.

As the result of correspondence on this subject during the last three years with officers in all parts of India, of conversations and discussions during tours of inspection, and from a close perusal of large numbers of statistical cards, we have formed certain opinions in conformity with the present state of our knowledge regarding the best standard course of treatment for use in India. It is thought that these may be of sufficient interest to place on record.

The following is a brief résumé of the properties of the three drugs used in the treatment of malaria.

Quinine retains first place in the treatment of the acute phase of a malaria attack. Its action is certain and rapid, and it can be given either by the oral, intravenous or, rarely, by the intramuscular route, according to the circumstances of the case. It has certain disadvantages: its power to prevent relapses of benign tertian malaria is limited: it has little or no
action on the gametocytes of *P. falciparum*; and from the patients' point of view it is unpleasant to take, and even in moderate doses may give rise to disagreeable—if harmless—symptoms.

*Atebrin* is in many instances as efficacious as quinine in the treatment of the acute stage of malaria, but in a proportion of cases its action in preventing pyrexic attacks is somewhat delayed. This is a matter on which there are diverse opinions, but the volume of evidence to the above effect cannot be disregarded. On the other hand, the action of atebrin in destroying the parasites is, in the long run, probably more potent than that of quinine. Its relapse-preventing properties, as far as can be judged from a limited series of cases investigated at the Malaria Treatment Centre, are not of a high order; nor has it any specific action on gametocytes of *P. falciparum*. It is easy to take, and produces no unpleasant symptoms. The yellow coloration of the skin which occasionally results from its use is transient, and of no significance.

In 1935 a new preparation of atebrin called *atebrin musonate* was placed on the market. This is a soluble drug which is suitable for intramuscular or intravenous administration, and is claimed to relieve all symptoms in a very short time. Trials have been made with this drug, and while preliminary reports are encouraging, our experience is too limited (only a few hundred military cases having been treated) and too short from the follow-up point of view, to permit us to express a definite opinion on the above claim.

*Plasmoquine* has been used by us only in conjunction with either quinine or atebrin, so that we are unable to comment on its action when given alone. Our experience shows that when given in combination with these other drugs, as laid down in the standard courses detailed above, it has a well-marked action in reducing the relapse rate in benign tertian malaria. Its property of destroying the gametocytes of *P. falciparum* is well known.

In the vast majority of cases plasmoquine produces no unpleasant symptoms when given in the dosage recommended (0.03 gramme per day for British troops). In the event of abdominal pain or cyanosis making its appearance, cessation of treatment for a day or two produces in most cases a speedy return to normal.

Hæmoglobinuria, and signs and symptoms to all intents and purposes indistinguishable from blackwater fever have, in rare instances, followed the use of plasmoquine among Indian troops; but whether *post hoc* or *propter hoc* is a moot point. A series of cases has already been reported by one of us,¹ and subsequent experience has confirmed the conclusions then formed, viz. that some unknown and unusual factor, acting in conjunction with plasmoquine in the presence of the malaria parasite, has

¹ *Journal of the Royal Army Medical Corps*, llii (1934), pp. 178, 269, and 318; *ibid.* livi (1935), p. 100.
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existed in each case. Since the adoption of the dosage of plasmoquine now recommended, only one fatal case has occurred in the British Army in India.

In our experience there is nothing to suggest that the toxic properties of plasmoquine are enhanced when it is administered after a course of atebrin.

Based on these observations, and on results obtained in certain hospitals, we recommend routine treatment as follows:

As a first step active purgation should be ensured, preferably by calomel followed next morning by Epsom salts.

Quinine should be used in the treatment of the initial febrile attack. It should be administered according to the circumstances of the case in the approved fashion, i.e. orally where practicable, and intravenously where immediate intervention is necessary. While the intramuscular route has its advocates in certain cases where the oral route is not practicable (e.g. in the presence of persistent vomiting), it is not a method which we recommend.

As soon as the initial febrile paroxysms have been controlled, quinine should be stopped and replaced by atebrin, 0.3 gramme daily for seven days. Thereafter, plasmoquine 0.03 gramme should be given daily for five days.

If any signs and symptoms of poisoning supervene, the drug concerned should be withheld until all traces of toxicity have disappeared.

It may be said that this is “blunderbuss” treatment, and unscientific in its conception. We do not think so. It is an established fact that the properties of the three drugs differ in detail, and the proposed course is designed to make the best use of the special qualities of each. The treatment has the advantage of being equally applicable to all types of malaria. It gives a free hand to the man-on-the-spot during the febrile stages of the disease when individual treatment is necessary, and imposes restraint only in respect of what may be regarded as “sterilizing treatment,” in which atebrin and plasmoquine are given to complete the destruction of the parasite and prevent subsequent relapses.

It may be asked why, instead of being replaced by atebrin, quinine should not be used throughout, thus making a course of seven days quinine followed by five days plasmoquine. There are two reasons. The first is that it has been found (see ante) that such a course is not so efficacious in preventing relapses as the atebrin-plasmoquine course. Possibly the administration of quinine over a longer period would have a better effect, but, other things being equal, it is obviously desirable to shorten the treatment as much as possible. The second reason is that atebrin is much more pleasant to take, both as regards the actual swallowing of the drug and the absence of after-effects. Many patients taking even moderate doses of quinine have ringing in the ears and a general feeling of being “below par.” Atebrin-treated patients have no such discomforts, and in consequence recuperate rapidly.
Again, it may be asked in what respect this course is preferable to the fourteen-day quinine-plasmoquine course used in certain areas. The answer lies in the fact that it is applicable to every case of malaria, while the quinine-plasmoquine course requires modification in all cases which are not quite straightforward. A further advantage is that it involves the use of considerably smaller doses of plasmoquine, the one drug of the three in which the margin between the therapeutic and the toxic dose is known to be small.

The omission of preliminary quinine is a permissible modification in certain mild cases, but it is very difficult to say which cases will be controlled readily by atebrin and which will not. On the other hand, we know that the vast majority of cases are quickly rendered afebrile by suitable quinine treatment.

Some clinicians recommend an interval of two to three days between the stoppage of atebrin and the beginning of plasmoquine treatment, the object being to ensure that most of the former is excreted before the latter is given. This idea is founded on the assumption that the toxicity of plasmoquine is enhanced in the presence of atebrin. We have been unable to find any confirmation of this assumption, and are of the opinion that the introduction of an interval lengthens the course unnecessarily.

Diet and nursing are of great importance. Diet should be fluid during the febrile stages, being increased after the fever subsides, but kept light and nutritious throughout. The cellulose-free diet once recommended to accompany atebrin treatment seems to be an unnecessary refinement. The patient should be confined to bed until the completion of the atebrin course—i.e. for eight to ten days after admission. During the plasmoquine course the patient should be allowed up for increasing periods, so that he can be discharged from hospital at the end of the course sufficiently fit to return to his unit. This régime involves a stay in hospital of at least a fortnight, which, in our opinion, is a minimum period. There is no doubt that thorough rest along these lines does much to render treatment more efficacious, and that it is short-sighted policy to reduce the stay in hospital.

In the final stages tonics such as iron and arsenic may be given if necessary.

In conclusion, we would emphasize that, as the statistics on which our findings are based are drawn from British troops in India, so the recommendations we make apply only to those who enjoy the same amenities, and do not apply to an impoverished population living in a malaria-ridden locality.

1 In this connection attention is drawn to the results recorded in an article entitled "A Study in Malarial Relapses in the United States Army," published in the American Journal of Hygiene (1933), xvii, No. 1.
SUMMARY AND CONCLUSIONS.

(1) There has been a decrease in admissions to hospital for malaria among British troops in India during the last few years.

(2) As far as can be determined this has no obvious direct relationship to favourable climatic conditions or to anti-malaria work or to any other factors of a like nature which affect the incidence of infection.

(3) Statistics are given showing that a decline in the relapse rate has occurred, and it is considered that this is the cause of the decrease in admissions.

(4) This decline runs pari passu with the introduction of plasmoquine.

(5) A relatively short atebrin-plasmoquine course of treatment has given very good results.

(6) A slightly modified standard course of treatment is outlined and recommended.

(7) These figures and conclusions refer to a specific community, namely British troops in India living under existing conditions.

We have to thank Major-General E. A. Walker, C.B., K.H.S., Director of Medical Services in India, for permission to make use of the statistical resources of the Medical Directorate and to send this article for publication.

Our thanks are also due to the clerical staff of the hygiene, pathology, and statistical section, and particularly to the Office Superintendent, Mr. G. A. Davies.

The use of the synthetic drugs in the Army in India was initiated by our predecessors, Colonel H. H. A. Emerson, D.S.O., and Major (now Brevet Colonel) J. A. Manifold, D.S.O.

REFERENCES.

[Note.—We have intentionally refrained from quoting any references other than those directly connected with the treatment of malaria in the Army in India.]

Idem. Ibid., 1929, 17, 725.
SINTON, J. A., SMITH, S., and PUTTINGER, D. Ibid., 1930, 18, 793.