prevent anophelines from infiltrating from untreated areas, was selected for a pilot larvicide scheme. If the results in this district proved to be satisfactory it was decided to extend the scheme to the whole island in the winter of 1951.

At the end of the epidemic first five months of 1951 it was possible to conclude from results in Flacq that the use of the larvicide scheme on a larger scale would be warranted. Not only was breeding of *A. gambiae* reduced by more than 97% (as compared with other untreated areas of previously similar breeding intensity), but this mosquito remained confined to a narrow coastal strip throughout the summer and did not penetrate inland, as was previously the case during the rains. Accordingly, practice areas were set up in all the other districts for the training of personnel in the new methods, and the island-wide campaign began during the dry month of September, 1951.

Conclusion

Future policy must, of course, depend largely upon the reaction of *A. gambiae* to this second form of attack. The house-spraying, using a D.D.T. preparation, was repeated during the winter months of 1951, in the hope that the combined effect of the two methods would prove to be a potent weapon against this evasive species. However successful the scheme may be, it would be rash to relax vigilance for many years to come; for a small missed breeding-pocket might gradually re-establish transmission of the disease.

During the three years that the scheme has been in progress the total annual cost (including capital expenditure, running costs, salaries of United Kingdom and local personnel, and the laboratories with their equipment) has been the equivalent of a little over 3s. per head of population in Mauritius. It has been encouraging to see how rapidly house-spraying with a residual insecticide on a large scale can reduce the transmission of malaria in a community even when the vectors are as efficient carriers of the disease as *A. gambiae* and *A. funestus*.

NOTE.—Further information about the scheme, covering the period since this article was written, appears in a letter from the author on page 339.

I would like to express my thanks to Dr. A. Rankine, until recently Director of Medical Services in Mauritius, and to his successor, Dr. R. Lavoipierre; to Dr. P. C. C. Garnham, chairman of the Malaria Subcommittee of the Colonial Insecticides Committee; and to Mr. C. B. Symes, officer in charge of research to the Committee, for all their advice and assistance given during the course of the past three years.

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Writing on the hospital services in *The Fortnightly* (July), Professor A. P. Thomson, dean of the faculty of medicine at Birmingham University, says: "We have in the Midlands some first-rate hospitals, but in 1948 the regional board took over from a county authority, and immediately closed, a hospital for chronic patients, of whom there were 16. There was no indoor sanitation, and the only water supply was from a stand-pipe in the yard. Ten of the patients were bedridden. That was the worst example, but there were several larger hospitals that were not much better. I was so astonished at what I saw that I got photographs of several of these places, and when I am reproached that hospital development is slow I get comfort from looking at the pictures, because we have nothing like them now."

THE SUPERIORITY OF "CAMOQUIN" OVER OTHER ANTIMALARIALS

BY

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The rational objectives of antimalarial therapy have always been (1) to prevent deterioration in the patient's health by treating an attack vigorously and bringing it to a speedy end; (2) to prevent relapses; and (3) in the absence of world-wide mosquito control to reduce the patient's infectivity to the mosquito. Since the advent of chloroquine, proguanil ("paludrine"), and mepacrine ("quinacrine") the use of quinine has rapidly receded into the background. These three drugs have been very effective in eradicating M.T. malaria, although exceptions in the case of proguanil have been met with. None of them tried singly, however, cures B.T. malaria, but pentaquine and combinations of quinine and pamaquin, proguanil and pamaquin, and quinine and pentaquine have considerably lowered the relapse rate. In treating the acute attack mepacrine is often superior to proguanil, but both are inferior to chloroquine. In some respects "camoquin," which has been recently introduced, seems to be superior to chloroquine. We record here our observations on its use in comparison with some of the above drugs.

Clinical Trials

Only pure infections of M.T. and B.T. malaria were selected. The patients were a heterogeneous crowd of soldiers aged between 16 and 35. They had acquired recent infection after a variable stay in endemic and hyperendemic zones in various parts of India. None of them had been on suppressive treatment or had had any antimalarial drug before treatment was started. All of them were treated in hospital under identical conditions. They had the usual febrile forms of the disease, and criteria for relapse were an enlarged spleen, history of malaria within the previous two years, and the presence of gametocytes in the blood on the first day of the disease.

Antimalarial therapy was given only to those who showed almost persistent parasitaemia for 24 hours. Parasite counts were done, being expressed as the number of infected cells per 1,000 R.B.C.s. Haemoglobin estimation, red-cell counts, total and differential leucocyte counts, serum-bilirubin estimations, tests for serum methaemoglobin, and routine urine examinations were done, and blood pressure was recorded before starting treatment in every case. Temperature was recorded four-hourly. Therapy was always begun at the onset of a rigor, to ensure accuracy and uniformity. Blood smears were continued six-hourly for parasite counts till they became persistently negative for 72 hours. In order to record any evidence of immediate or delayed toxicity other blood and urine examinations, as stated above, were made after 48 hours and again 10 days later, after starting treatment. Blood pressure was recorded daily. In the case of mepacrine and proguanil these investigations were confined to recording the duration of pyrexia and the period of asexual parasitaemia. In the case of camoquin only, the patients were followed up for the last 14 months for evidence of relapse.

Results

The results are mostly summarized in Table I. Other points are included in the discussion to avoid repetition. As we had only five relapses in the camoquin series and none in the chloroquine series, we record the results obtained by various treatments, except in the camoquin series, in fresh cases only. The results obtained with proguanil have been difficult to predict, as they were somewhat erratic; those with mepacrine were steadier and better, but did not compare favourably with those obtained with chloroquine and camoquin. A combination of proguanil and quinine was better than a combination of proguanil and mepacrine or mepacrine alone.

Discussion

Pharmacology.—Camoquin is chemically 4-(3'-diethyl-amino-methyl-4'-hydroxyanilino)-7-chloroquinoline, and is sold as the dihydrochloride dihydrate. It is a light-yellow crystalline powder, slightly soluble in water, giving a 5% solution at room temperature. The drug is rapidly absorbed from the gastro-intestinal tract and adequate blood concentrations are obtained about an hour after oral administration. Its concentration in the erythrocytes is double that in the plasma, but is many more times in the spleen, liver, kidneys, lungs, brain, and heart, the highest being in the spleen.

that the drug is very effective in the treatment of an acute attack and in diminishing the relapse rate (Penido *et al.*, 1948; Coggeshall, quoted by Payne *et al.*, 1949; Mein and Rosado, 1948; Simeons and Chhatre, 1947; Halawani *et al.*, 1947, 1948; Mein, Penido, and Deane, quoted by Ejercito and Duque, 1948; Chaudhuri and Chakravarty, 1948; Hoekenga, 1950). In most cases the drug terminated the attack and cleared the parasitaemia within 48 hours. The dosages were 1 g. six-hourly for three doses, or 1.2 g. daily for two days (Coggeshall), 0.25 g. 12-hourly for two doses (Halawani *et al.*, 1947), 0.5 g. single dose (Chaudhuri and Chakravarty, 1948), and 10 mg./kg. single dose (Simeons and Chhatre, 1947; Hoekenga, 1950).

Payne *et al.* (1949) obtained similar satisfactory results with two doses of 0.32 g. of the drug given intramuscularly at eight-hourly intervals without any local or systemic reaction.

In Table I the superiority of chloroquine over mepacrine and proguanil in terminating the attack and reducing parasitaemia is very significant. Similar results have been obtained by other workers using chloroquine against mepacrine and quinine (Most *et al.*, 1946), mepacrine (Pullman *et al.*, 1948), and proguanil (Canet, 1949) controls. Toxic symptoms have been rare with chloroquine, and a great advantage is that the treatment is completed in three days.

TABLE I.—Comparative Results Obtained with Various Antimalarial Drugs

Drug	Dose	Type of Malaria	No. of Cases	Duration of Fever in Hours			Duration of Asexual Parasitaemia in Hours		
				Av.	Max.	Min.	Av.	Max.	Min.
Camoquin	0.5 g. at onset of rigor and 6 hours later	B.T.F.	13	25	58	6	40	98	6
		B.T.R.	2	11	13	8	27	48	6
		M.T.F.	20	21	63	5	33	72	6
		M.T.R.	3	31	44	24	40	50	32
Chloroquine	1 g. initially, 0.5 g. 8, 24, and 48 hours later. Total 2.5 g.	B.T.F.	15	32	56	6	38	60	6
		M.T.F.	2	40	64	15	48	72	24
Proguanil	(a) 100 mg. t.d.s. for 10 days. Total 3 g.	B.T.F.	79	67	100	8	89	204	12
	(b) 200 mg. t.d.s. for 10 days. Total 6 g.	M.T.F.	10	72	96	8	58	84	12
Mepacrine	200 mg. t.d.s. for 2 days and 100 mg. t.d.s. for next 5 days. Total 2.7 g.	B.T.F.	132	46	84	6	70	120	12
		M.T.F.	39	46	72	12	58	72	6
Proguanil and quinine	Proguanil 100 mg. and quinine 10 gr. (0.65 g.) t.d.s. for 2 days and proguanil 100 mg. t.d.s. for next 8 days	B.T.F.	15	37	72	6	36	84	6
		M.T.F.	2	22	30	14	30	54	6
Proguanil	Proguanil 100 mg. and mepacrine 100 mg. t.d.s. for 2 days and proguanil 100 mg. for next 8 days	B.T.F.	5	48	96	13	62	108	12
		M.T.F.	2	60	84	36	75	96	54

Adequate concentrations are maintained for a week or more after a single dose. The leucocytes may contain it for 22 days after its administration. There is little or no discoloration of the skin. The parent drug is not excreted in the urine, but 10 to 20% of the daily dose is excreted as a degradation product, and the elimination can be increased by acidifying the urine.

Animal Studies.—In animals a dose of 5 to 10 mg./kg. orally or 2.5 to 5 mg./kg. parenterally produces no acute or cumulative clinical or histopathological effects. In rats a dose of 800 mg./kg. orally is lethal, and doses between 65 and 195 mg./kg. affect growth. Monkeys tolerate 25 mg./kg. daily for a month. In therapeutic doses in man no toxic symptoms have been recorded. Continuous heavy doses, which will never be used therapeutically, have given rise to lack of energy, insomnia, epigastric discomfort, and anorexia. The drug has been found six times as effective as quinine against *Plasmodium cathemerium* in the canary (Gingrich), 8 to 25 times against *P. gallinaceum* in the chick (Coatney; Porter), and five times against *P. lophurae* in the duck (Marshall).

Clinical Investigations

In trophozoite-induced malaria, using the McCoy strain of *P. vivax* and the McClendon strain of *P. falciparum*, the curative dose has been found to be 350 to 400 mg. in B.T. malaria, and about three times this in M.T. malaria.

Results obtained by various investigators in India, the Philippines, the Amazon valley, and elsewhere have shown

Duration of Fever

Chloroquine has now its rival in camoquin. Probably camoquin is the superior, but it is at present not readily available in the market, and a world-wide superiority has not yet been established for it. The period of treatment has been reduced to the minimum. A single dose is all that is required. The dosage and the cost are smaller than with chloroquine. In our series, although the maximum and minimum periods as regards duration of fever in B.T. malaria are almost identical with the two drugs, the average duration is in favour of camoquin, being 24 hours with camoquin and 32 hours with chloroquine, thus indicating that in a great number of cases fever is controlled earlier with camoquin. There is a slight difference in the rate of clearance of B.T. parasitaemia, the average period being 40 hours with camoquin and 38 hours with chloroquine. The minimum period is the same with both, but some cases have taken a day longer to clear up with camoquin. There has been, however, no relation between this and pyrexia, all the cases being afebrile during this period.

In M.T. malaria the average duration of fever with camoquin is about half that of chloroquine, although the maximum periods are almost identical. The minimum period is also less. M.T. parasitaemia also has cleared more rapidly with camoquin than with chloroquine, the average duration being 33 and 48 hours respectively. In M.T. malaria, therefore, camoquin seems to be definitely superior to chloroquine in controlling fever as well as parasitaemia.

Asexual Parasitaemia

When the last of the four samples of blood taken six-hourly before the administration of camoquin was negative it remained negative subsequently. It appears that erythrocytic forms do not develop from the exoerythrocytic forms when the blood contains camoquin. It would be interesting if this can be confirmed in *in vitro* cultures. It may be mentioned that only ring forms and amoeboid forms develop in culture under the influence of chloroquine. Failure of these early active forms to develop in numbers may account for the more rapid clearance of parasitaemia in camoquin-treated cases.

The rate of parasite clearance during the first 48 hours of treatment in M.T. or B.T. malaria was not found related to the initial parasite density. In fact some of the cases in which the density was greatest cleared up more rapidly than those in which the density was less. Presumably this indicates how powerful the drug is in its lethal action on asexual parasites. Schizonts did not develop in 12 out of 13 cases in which initially trophozoites were present.

In five cases the parasite density actually increased after the administration of camoquin, nearly doubling itself. In two of these the parasites almost completely disappeared within the next six hours. In the remaining three the increased density more or less persisted for 30 to 60 hours before rapid clearance. Clinically, however, there was no correlation between the duration of high density and fever, and the patients remained afebrile. The maximum parasite density in these cases was 11,000 and 17,000 per c.mm. of blood. We were unable to correlate this density with the concentration of the drug in the blood owing to lack of facilities. It is likely, however, that the delayed parasitocidal action may have resulted from a slower absorption of the drug from the gut. In such cases larger or parenteral doses would be more effective.

Sexual Parasitaemia

In cases in which gametocytes were initially absent before the administration of camoquin and remained so thereafter it may be concluded that asexual parasitocidal action was complete and any immature primary gametocytes which may have been present in the bone marrow or the spleen were entirely destroyed. Simeons and Chhatre (1947) obtained similar results in crescent-negative cases. They concluded that in this respect camoquin was superior to mepacrine. In crescent-negative cases mepacrine does not appear to have this occult gametocidal effect, although Rogan *et al.* (1944) found that in mepacrine-treated cases the incidence of crescents following one week after treatment was closely related to the mepacrine dosage, being less with larger doses.

In our series, where the gametocytes were already present in the blood, 90% cleared within six days on an average in B.T. cases, and about 60% during the same period in M.T. infections. The maximum period in one case of B.T. malaria was 14 days, and in a case of M.T. malaria 43 days. In Chaudhuri and Chakravarty's (1948) cases B.T. gametocytes disappeared in one to five days, counting from the day the drug was administered, while crescents took 7.8 days on an average to clear, the range being 1 to 26 days. Ejercito and Duque (1948) found that crescents persisted for four to nine days after treatment, but B.T. gametocytes showed marked reduction on the second and third days, and all disappeared by the fourth day.

Relapses

The use of heavy doses postpones the relapses. This was observed by Rogan *et al.* (1944) in mepacrine-treated cases, and Simeons and Chhatre (1947) obtained similar results with camoquin. With smaller doses M.T. cases relapsed more often than B.T. cases, as shown in Table II. These writers, however, have not clarified whether the cases treated had primary attacks or were relapsing cases, or whether they were pre-immunized subjects from endemic or hyper-

TABLE II.—*Relation of Camoquin Dosage to Rate of Relapses (Simeons and Chhatre, 1947)*

Dose mg./kg.	Type of Malaria	No. of Cases	No. of Relapses	Latent Period in Days
10	{ B.T. M.T.	39 11	Nil "	— —
7.5	{ B.T. M.T.	10 6	" 2	— 20 each
5	{ B.T. M.T.	8 10	2 7	28 and 5 10 to 30

endemic areas. The patients belonged to Kolhapur State Forces, in India, and were "mostly heavy infections."

Ejercito and Duque (1948) treated both primary and relapsing B.T. and M.T. infections with camoquin, and obtained results as in Table III. Thus relapsing B.T. cases relapsed earlier and with about three times the frequency of primary B.T. cases. Primary M.T. cases did not relapse within six months, and were therefore apparently cured. A relapsing M.T. case relapsed after 81 days, a period longer than any of the B.T. cases. It may be noted that their dose was smaller.

TABLE III.—*Frequency of B.T. and M.T. Relapses (Ejercito and Duque, 1948)*

Dose	Type of Malaria	No. of Cases	No. of Relapses	Latent Period in Days
0.4 g., single dose	{ B.T.F. B.T.R. M.T.F. M.T.R.	40 44 22 22	2 6 Nil 1	39 and 56 25 to 38 — 81

During a period of observation of 5 to 12 months the relapse in *all* cases was 7.8%. Ejercito and Duque have compared it with a rate of 5% obtained by Mein, Penido, and Deane, in Brazil, with a single dose of 0.5 g. of camoquin. The latter claim that this dose "would cure 95% of all malaria cases (vivax and falciparum, whether primary or relapse)."

Hoekenga (1950), in Panama, treated 30 B.T. and 20 M.T. cases of malaria (unqualified) with a single dose of 10 mg./kg. of the drug and recorded only one B.T. relapse during a period of 6 to 16 months' observation.

With our dosage, which works up to 16–20 mg./kg., the relapse rate during a period of 14 to 16 months' observation has been as shown in Table IV.

TABLE IV.—*Relapse Rate in Present Series*

Dose	Type of Malaria	No. of Cases	No. of Relapses	Latent Period in Months
0.5 g., repeated in 6 hours. Total 1 g.	{ B.T.R. B.T.F. M.T.R. M.T.F.	2 13 3 20	Nil 2 Nil "	— 10 to 12 — —

Making allowance for insufficient data in some of the above-quoted references and the comparatively small number of treated cases, one finds a marked diminution in the relapse rate with larger doses of camoquin. With one exception of M.T. relapse recorded by Ejercito and Duque (1948), the relapses, however, have all been in B.T. cases, although one would expect the opposite from the results of experiments with induced malaria (see above). The latter, however, explain to some extent why B.T. relapses are delayed and few. Apparently, therefore, the drug is superior in this respect to all other antimalarials and their various combinations. This is obvious from Table V, which gives results obtained by Most *et al.* (1946) in the treatment of *all* types of B.T. cases from the South-west Pacific zone.

Chloroquine definitely delays relapses, but after about four months it gives only slightly better results than mepacrine or quinine. A combination of quinine and pamaquin is definitely much superior. A combination of proguanil

TABLE V.—*Comparative Results with Various Antimalarials in Therapeutic Doses (Most et al., 1946)*

Drug	Latent Period in Days and Percentage of Relapses			
	30	40	50	120
Chloroquine ..	Nil	Less than 1%	11%	70%
Mepacrine ..	9%	28%	40%	80%
Quinine ..	54%	67%	72%	85%
Quinine and pamaquin ..	—	—	—	11%

and pamaquin has been found by Fairley (1946) in the same theatre almost to double the latent period obtained by a combination of quinine and pamaquin, although the percentage of relapses remained the same in both series. Also in the same theatre Pullman *et al.* (1948) found the relapse rate in severe B.T. infections to be four and a half times that in moderately severe infections with a combination of quinine and pentaquine, the superiority of which, therefore, still has to be proved.

Toxicity

Blood and urine examinations which were carried out showed no changes, and camoquin was well tolerated by all patients without giving rise to any complaint. Toxic symptoms with mepacrine and proguanil were also very rare. A few patients complained of abdominal pain after proguanil, and some vomited after mepacrine.

Summary

Camoquin has been found definitely superior to proguanil and mepacrine in the treatment of malaria. In M.T. malaria it seems to be superior to chloroquine in controlling fever and asexual parasitaemia. In B.T. malaria, fever is controlled earlier with camoquin, but in a few cases there is a slight difference in the rate of clearance of parasitaemia in favour of chloroquine. Evidence suggests that with larger doses of camoquin relapses are few, and are delayed. Although further experience is required to establish a world-wide superiority, especially over chloroquine, the use of a single dose and the smaller cost are distinct advantages.

We are indebted to Major S. P. Mukerji, I.A.M.C., for his kind help in the management and care of the chloroquine series of cases; to Messrs. Parke, Davis and Co., Bombay, for the free supply of camoquin; and to Lieutenant-General D. R. Thapar, Director-General, Armed Forces Medical Services (India), for his permission to publish this paper.

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OBSERVATIONS ON METHODS OF MEASURING THE ERYTHROCYTE SEDIMENTATION RATE

BY

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The most popular methods of measuring the erythrocyte sedimentation rate (E.S.R.) are those described by Westergren (1921) and Wintrobe and Landsberg (1935); the latter is commonly known as the Wintrobe method. Technically, these procedures differ mainly in the nature of the anticoagulant and the length of the sedimentation tube; the bore of the tube is the same in both. Many investigators have compared the methods to assess their reliability, and Davis (1946), in a critical survey, concluded that the one of choice for general purposes is that of Westergren, because it is less sensitive to minor influences and more responsive when sedimentation is rapid. Recently, Gilmour and Sykes (1951) found, in a large series of cases, that a raised Wintrobe result was always accompanied by a raised Westergren; but a raised Westergren was not accompanied by a raised Wintrobe in 27% of female and 28% of male cases. They considered that the Westergren method is the more efficient as a pointer to organic disease.

In the course of routine haematological investigations we observed, within a short space of time, two patients in whom the E.S.R. by the Wintrobe method was normal whereas very high readings (over 100 mm. in one hour) were obtained by the Westergren. In both instances an abnormal protein was subsequently discovered in the blood, and if only the former method had been used an important diagnostic clue would have been lost. Because of these paradoxical results we decided to compare the two methods in a small series of cases. This investigation confirmed the confidence of Davis (1946) in the superiority of the Westergren method. It is, however, convenient to be able to perform a full haematological examination, including the E.S.R., on 5 ml. of oxalated blood as recommended by Wintrobe (1933). A series of experiments was therefore designed to explore the possibility of adapting the Westergren method, in which sodium citrate is the anticoagulant, to the use of oxalated blood.

Technical Methods

All needles and syringes were dry-heat sterilized and the sedimentation tubes were scrupulously cleaned and dried before use. Care was taken to ensure that the tubes were set in a strictly vertical position with air-bubbles excluded.

Blood samples for the Wintrobe and Westergren tests were taken by venepuncture. First, with the needle left *in situ*, 5 ml. of blood was delivered from a syringe into a glass bottle containing dry oxalate mixture (ammonium oxalate 6 mg., potassium oxalate 4 mg.). A second syringe containing 1 ml. of 3.8% sodium citrate was then attached to the needle and 4 ml. of blood withdrawn; this blood-citrate mixture was transferred to a clean dry test-tube. Both specimens were mixed by gentle shaking for approximately one minute. For straightforward testing the sedimentation tubes were charged immediately, the Wintrobe with the