REFRESHER COURSE FOR GENERAL PRACTITIONERS**MALARIA: ITS RECOGNITION, TREATMENT, AND PREVENTION**

BY

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There are four recognized species of human malaria parasite—namely, *Plasmodium vivax*, the parasite of benign tertian malaria; *P. falciparum*, the parasite of malignant tertian malaria; *P. ovale*, which gives rise to a particularly mild type of tertian malaria; and *P. malariae*, the parasite of quartan malaria. *P. vivax* has the most extensive distribution, being the predominant infection in most malarious areas within the temperate zone, and also widespread throughout the Tropics and subtropics. *P. falciparum* is common in most tropical and subtropical countries, and is associated especially with hyperendemic malaria, and with the great regional epidemics which occur from time to time in North-west India, Ceylon, and elsewhere. *P. malariae* occurs in most malarious regions, but is less common than either *P. vivax* or *P. falciparum*, whilst *P. ovale* has the most restricted distribution of all. The only fully authenticated records about the last-named species are those of infections acquired in East, Central, or West Africa. Cases have been reported from time to time from other places, but these are generally considered to be of doubtful validity.

Benign Tertian (vivax) Malaria

An overt attack of vivax malaria is in most cases preceded by prodromal manifestations such as headache, lassitude, anorexia, and possibly some gastrointestinal upset. In the initial stage of a primary attack the fever is remittent in type, rarely exceeding 103° F. (39.4° C.) and is not accompanied by rigors. At this stage parasites are extremely scanty in the peripheral circulation. In the fully developed stage the fever becomes intermittent and the peaks of temperature become higher (often exceeding 105° F.—40.6° C.), each paroxysm being accompanied by a rigor which may last for one hour or more. Numerous asexual parasites in all stages of development from young rings to mature schizonts are now present in the peripheral blood. There follows a hot stage of about four hours' duration, during which the skin is flushed and headache and vomiting are severe; this is succeeded by a sweating stage, when the temperature falls to normal and all the symptoms are relieved. In a primary attack the fever in the developed stage is almost always quotidian.

In a relapse the temperature curve differs from that recorded in a primary attack in that the fever usually shows definite tertian periodicity. Another striking difference is that asexual parasites, all generally in the same stage of development, are present in the peripheral circulation in large numbers on the first day of fever.

Ovale Malaria

Ovale malaria differs from that caused by *P. vivax* in that the disease is in most respects considerably milder. There is an initial remittent stage in less than

50% of cases, rigors are less frequent than in vivax infections, and spontaneous recovery in the absence of specific treatment is comparatively common. It has been observed that febrile paroxysms often begin late in the evening, whereas in vivax malaria they more often occur at about midday.

Malignant Tertian (falciparum) Malaria

Falciparum malaria differs clinically from other forms of the disease in several respects. One of the most striking is the infrequency of rigors at any stage. The primary attack begins with a sensation of chilliness, which is followed by a prolonged hot stage and a moderate amount of sweating. Severe aching pains in the back, legs, and joints are usually a prominent feature, and nausea and vomiting, diarrhoea, and abdominal pain and tenderness are often present. The fever is irregularly remittent, with numerous peaks representing the activity of different groups of parasites. Even in the most severe cases the temperature may not exceed 103 or 104° F. (39.4 or 40° C.), and it may not even reach as high as this. Prostration is greater than in other forms of the disease; the headache is more severe; and vomiting, mental confusion, and torpor are more common.

There is no known disease which may simulate so many other ailments as falciparum malaria, among them being heat-stroke, cerebral haemorrhage, thrombosis or embolism, uraemia, pneumonia, dysentery, and acute abdominal conditions such as acute pancreatitis, appendicitis, and gall-stone or renal colic. The graver forms of falciparum malaria are commonly grouped under such headings as cerebral, algid, dysenteric, or bilious remittent. The onset of the cerebral form may be gradual, beginning with headache, or sudden, simulating heat-stroke or apoplexy. Algid malaria is characterized by sudden collapse, often associated with the vomiting of blood, cramps, suppression of urine, and the passage of blood and mucus in the stools. Bilious remittent malaria is accompanied by nausea, vomiting, jaundice, liver tenderness, and persistent hiccup.

Quartan (malariae) Malaria

The clinical course of quartan malaria resembles that seen in vivax infections, except that an initial remittent stage is less frequent and rigors are less often observed. The temperature curve usually shows evidence of more than one group of parasites, and the fever may be quartan, double quartan, or triple quartan—that is, quotidian—in character. In the primary attack parasites are less numerous in the peripheral circulation than in vivax and falciparum infections, and in relapses they may be so few that they are unlikely to be discovered other than by the thick film method, and then only after prolonged search.

Diagnosis

The only certain means of diagnosing malaria is the demonstration of the causative plasmodium by microscopical examination. In malarious areas the examination of a blood smear for malaria parasites should be a routine procedure in medical practice, whatever may be the symptoms or primary diagnosis. Though the clinical picture may not suggest malaria, it should always be borne in mind that in such regions it is often a complicating factor of other diseases, just as some other infection may complicate malaria. These considerations apply with equal force to patients who have returned from residence in, or from visiting, malarious countries, even though there may be no history of a previous malarial attack.

For the preparation of blood films it is essential that the slides used be scrupulously clean, free from any trace of grease, and without scratches, and that the film whilst drying be protected from dust and flies. The patient's finger should be cleaned with alcohol and allowed to dry completely before being pricked with a cutting needle. The latter may be conveniently mounted in a rubber cork and kept in a small glass tube containing alcohol, but must be allowed to dry before use.

To make a thick film, squeeze the finger until a large globule of blood exudes. Touch this with the surface of a perfectly dry slide and with a circular movement in the plane of the surface make a film about the size of a shilling. Spread the blood evenly with the corner of another slide or with the needle, and keep the film horizontal while drying. The film should be of such thickness that newsprint can just be seen through it when wet.

To make a thin film, the globule of blood should not be larger than the head of a large pin. Apply the end of a spreader (which may be either another glass slide with a perfectly smooth edge or a rectangular cover-slip) to the first slide in front of the drop of blood at an angle of 45 degrees, bring it back till it touches the drop, and allow the blood to spread evenly along the edge. Push the spreader forward along the slide, keeping it at the same angle, and dry the film quickly by waving the slide vigorously in the air. A properly made film should consist of an unbroken sheet one cell thick, the cells almost touching one another but not overlapping.

Films may be stained with either Leishman or Giemsa stain, the latter being generally preferred for use in tropical climates. A rapid method of staining thick films with buffered isotonic Romanowsky stain, introduced by Field, is now widely used. A modification of this method, the J.S.B. (Jaswant Singh and Bhattacharji) stain, has the advantage that it can be used for both thick and thin films. One of the most useful descriptions of these methods and of the techniques employed in the preparation of blood films and in the diagnosis of malaria parasites is that given by Field in 1948.*

There is usually a marked leucopenia within a few hours of the first rise of temperature, though there may be moderate leucocytosis during the early stages of a rigor. The number of polymorphonuclear leucocytes generally falls to about 50%, and the mononuclears rise to about 45%. The greatest number of mononuclears are seen between paroxysms, when they may constitute as much as 80% of the total white cells. The rapid onset of anaemia, hypochromic in type, is an important diagnostic sign, and in an acute attack the reduction in the number of red cells may be as much as

one million or more per c.mm. of blood within as short a period as 24 hours.

Another suggestive sign is enlargement and tenderness of the spleen, and there may be liver tenderness also. The spleen is unlikely to be palpable in the first few days of the primary attack, but there is often splenic tenderness before the organ can actually be felt.

Should the fever, or any other symptom, recur with regularity at intervals of approximately 48 or 72 hours, the disease is almost certainly malaria. As noted above, this is scarcely ever the case in a primary attack, but it may be an important diagnostic point in the case of a relapse.

It is important to recognize that parasites may be present in such small numbers in the peripheral circulation during the first few days of an attack that they are not detectable in blood smears, even after prolonged search, particularly when these have been taken at the height of the fever, when they are least likely to be found. In the event of a negative finding, further smears should be examined twice daily for at least a week before ruling out the possibility of malarial infection.

Mode of Action of Antimalarial Drugs

The demonstration of a pre-erythrocytic phase of *Plasmodium cynomolgi* in the liver of the monkey, and of a similar phase of *P. vivax* and *P. falciparum* in man, has shed new light on the chemotherapy of malaria. In theory, a drug may act on the malaria parasite in five possible ways: (1) against the sporozoites when first they enter the human body; (2) against the primary tissue or pre-erythrocytic phase; (3) against the asexual parasites in their erythrocytic phase; (4) against the sexual parasites in their erythrocytic phase; and (5) against the late exo-erythrocytic or secondary tissue phase, to which the phenomena of latency and late relapse in vivax malaria have been attributed. There is convincing evidence against the existence of the last-named phase in falciparum malaria.

The classes of antimalarial drugs in common use are (a) the cinchona alkaloids (quinine, "total-quinine"); (b) the 9-aminoacridines (mepacrine); (c) the 4-aminoquinolines (chloroquine, camoquin); (d) the 8-aminoquinolines (pamaquin, primaquine); and (e) the biguanides (proguanil). Certain of the 2:4-diaminopyrimidines have also given promising results in experimental trials, but these are not yet available for general use.

No drug as yet tested has any demonstrable effect on the sporozoite stage of the malaria parasite. The cinchona alkaloids, the 9-aminoacridines, the 4-aminoquinolines, and the biguanides have a destructive action on the asexual forms of all four species of human malaria parasite in the erythrocytic phase. This was formerly designated "schizonticidal action," a term rendered obsolete by the discovery of pre-erythrocytic schizonts in the liver. The 8-aminoquinolines do not affect the asexual parasites in the erythrocytic phase, but have a powerful destructive action on the gametocytes. They also act on the late exo-erythrocytic forms of *P. vivax*. They have been shown to have an inhibitory effect on the pre-erythrocytic phase of both *P. falciparum* and *P. vivax*, but only when employed in dosage too high for routine use. The biguanides are

*Field, J. W. Studies from the Institute of Medical Research, Federation of Malaya, No. 23. 1948.

the only class of drugs which can be used with safety against this phase, and, so far as is known, they are effective only in falciparum infections. They have no demonstrable effect on the sexual forms of *P. falciparum* in the peripheral blood, but render them incapable of completing their development in the mosquito.

Specific Treatment of Clinical Malaria

It is sometimes stated that specific antimalarial drugs should be withheld until parasites have actually been seen in a blood smear. In principle this is obviously desirable, but in falciparum infections in non-immune subjects death has been known to occur while parasites were so scanty in the peripheral circulation as to be scarcely detectable even after prolonged search. There is, in fact, no safe parasite-density level in malignant tertian malaria. It is my opinion that in a region where falciparum infections are common, when a patient is seriously ill with symptoms which do not clearly indicate some other disease, the administration of a specific antimalarial drug should not necessarily be dependent on the microscopical demonstration of parasites. This applies particularly to patients in coma or with other grave cerebral manifestations, when delay in beginning treatment may prove disastrous.

The chief objects aimed at in the treatment of the clinical attack are the prompt alleviation of symptoms and the radical cure of the infection, with minimum risk of toxic side-effects. Sterilization of the sexual forms of the parasite, so that mosquitoes which may feed upon the patient are unable to transmit the disease to others, is also desirable, and in certain circumstances may be a matter of considerable importance.

Quinine sulphate or hydrochloride, given in a daily dosage of 20–30 gr. (1.3–2 g.), will usually bring about rapid termination of the clinical attack and, in falciparum infections, a high rate of radical cure. This drug is, however, comparatively ineffective against certain European strains of *P. falciparum*. It has no effect on the infectivity of gametocytes to mosquitoes, and in certain circumstances may act as a contributory factor in the precipitation of blackwater fever.

Mepacrine, when given with a loading dose of 600–900 mg. on the first day or first two days of treatment, followed by 300 mg. daily for the following five days, will usually effect a rapid termination of the clinical attack in all forms of malaria, and a high rate of cure in falciparum infections. It does not sterilize gametocytes, and toxic side-effects, though of rare occurrence, are by no means negligible. This applies particularly to the so-called mepacrine psychosis, which may be attended by symptoms of mental aberration or even of maniacal excitement. Another drawback is the yellow discoloration of the skin which sometimes follows the administration of this drug.

Chloroquine brings about rapid alleviation of clinical symptoms in all forms of malaria, and a high rate of cure in falciparum infections. It has the advantages over mepacrine of not tinting the skin and of being less likely to induce disagreeable intestinal or psychotic manifestations. The regimen usually recommended is an initial dose of 600 mg. of the base, followed by 300 mg. six hours later, and a single dose of 300 mg. on each of the next two days. Camoquin, whose action resembles that of chloroquine, has been given in doses

up to 400 mg. thrice daily for five days, and is said to be equally effective. Neither of these drugs renders gametocytes non-effective to mosquitoes.

Proguanil, in the absence of reinforcement with some other drug, has a less rapid action on the clinical attack than the cinchona alkaloids, 9-aminoacridines, or 4-aminoquinolines; moreover, it cannot be relied upon to effect radical cure, even in falciparum infections. Both these disadvantages can, however, be overcome by reinforcing a five- to seven-day treatment of 600 mg. of proguanil daily with 600 mg. of mepacrine or chloroquine on the first day. Proguanil effectively sterilizes the gametocytes of *P. falciparum*, a property not possessed by mepacrine, chloroquine, camoquin, or quinine. It is, moreover, by far the least toxic of all known antimalarial drugs.

Whatever form of treatment may be adopted for the clinical attack, it is advisable to place the patient on a suppressive regime (see below) for the following eight weeks, to cover the period during which recrudescences are most likely to occur.

From what has been stated above, it will be realized that none of the drugs available fulfils all the requirements postulated for treatment of the clinical attack. Quinine has the disadvantage of its association with the precipitation of blackwater fever, and is comparatively ineffective against certain geographical strains of malaria parasites. The clinical response to mepacrine is rapid in all forms of malaria, but minor toxic manifestations and occasional psychoses of a more serious nature are definite drawbacks. Chloroquine and camoquin are probably the most effective agents for terminating the clinical attack, but a number of toxic manifestations have been recorded with each of them. None of these drugs renders gametocytes non-infective to mosquitoes. Proguanil has an extremely low degree of toxicity and effectively sterilizes gametocytes, but its action on the clinical attack is relatively slow, and it cannot be relied upon to effect radical cure of falciparum infections. Reinforcement with mepacrine or chloroquine, as noted above, overcomes both these defects, and such a combined treatment will in most cases ensure rapid alleviation of symptoms, radical cure of falciparum infections, and sterilization of gametocytes, with minimal risk of toxic side-effects. Neither this regime, however, nor any other of the treatments cited above will prevent the occurrence of late relapses in vivax malaria (see below).

For the dispensary treatment of indigenous populations of malarious countries, most of whom possess some degree of tolerance to malarial infection, a single dose of 300 mg. of proguanil or chloroquine base, or 600–1,000 mg. of camoquin, has proved effective. The extent to which each of these drugs is used will no doubt be very largely determined by their market price.

General Treatment of Malarial Attack

The patient should be confined to bed during an attack, and the bowels kept open by small doses of calomel, followed by salts in the morning if found necessary. During the height of the fever the diet should be restricted to fluids, glucose being given freely, if available. During a rigor, hot-water bottles and several blankets should be provided. Hyperpyrexia may necessitate cold sponging. The headache may be

relieved by aspirin, caffeine, codeine compound, or phenobarbitone. Arsenic in small doses combined with iron is useful in convalescence to combat anaemia.

Emergency Treatment

In the treatment of pernicious forms of falciparum malaria, whether cerebral, algid, or gastro-intestinal, oral administration of drugs is seldom practicable; and since prompt action is necessary to save the patient's life antimalarial drugs must be given parenterally. Mepacrine sulphionate ("atebrin musonate") may be given intramuscularly, 200 mg. being injected into each buttock. Good results have also been reported with intramuscular and intravenous injections of chloroquine. Quinine hydrobromide or dihydrochloride, 10 gr. (0.65 g.) in 20 ml. of normal saline, may be injected *very slowly* intravenously, and repeated in six to eight hours if necessary. The intramuscular injection of quinine, formerly widely practised, is apt to cause necrosis or abscess and is not recommended. There is also some evidence that this procedure predisposes to tetanus infection. As soon as the patient is able to take drugs by the mouth, all further medication should be administered orally. The action of proguanil is not rapid enough to warrant the use of this drug in cases of extreme urgency.

Blood transfusion is indicated in pernicious malaria accompanied by grave anaemia, preceded always by careful cross-matching of the donor's cells with the patient's serum and vice versa. Persistent vomiting may be relieved by injections of 5% glucose in 200–400 ml. of normal saline, repeated if necessary, with the addition of 1 mg. of thiamin hydrochloride for each 25 g. of glucose.

Treatment of Chronic Relapsing Vivax Malaria

The action of the 8-aminoquinolines on the secondary tissue phase of *P. vivax* has already been mentioned. The first of these to be used for the prevention of relapse was pamaquin, which was given in combination with quinine—for example, 0.01 g. of pamaquin concurrently with 10 gr. (0.65 g.) of quinine sulphate twice daily for 10 days. American workers have recently shown that certain other members of this group—for example, pentaquine, isopentaquine, and primaquine—are more effective than pamaquin and less toxic, primaquine being probably the best of the three in both respects. Close medical supervision is necessary, however, with all members of this group of drugs, chiefly because of the occasional unpredictable occurrence of acute intravascular haemolysis. The routine administration of these drugs in all cases of vivax malaria is not recommended. They should be reserved for the treatment of stubbornly relapsing cases, more especially in circumstances where there is no risk of reinfection.

Blackwater Fever

This condition is essentially an acute intravascular haemolysis, accompanied by haemoglobinaemia and haemoglobinuria, occurring in individuals who have experienced repeated and severe attacks of falciparum malaria. The onset is usually abrupt, with headache, nausea, vomiting of bile-stained fluid, severe pains in the loins, and marked prostration. There may be a rigor, and the temperature usually rises to 102 to 103° F. (38.9 to 39.4° C.) or higher, and may be either con-

tinuous or remittent. The urine is typically dark red to almost black from the outset. At first the pulse is rapid and the blood pressure low, but the latter rises later with the development of renal failure. The patient is now restless and anxious, with marked dyspnoea and collapse. Jaundice is a cardinal symptom, and may appear a few hours after the onset of the attack. Anuria may develop suddenly, presaging uraemia and an early fatal termination. The spleen is enlarged and tender, and there is usually some enlargement and tenderness of the liver also.

The red-cell count may fall by as much as 2,000,000 per c.mm. within a period of 24 hours. The parasitized erythrocytes are among the first to undergo haemolysis, and blood smears taken after the onset of the attack are therefore often negative. Patients who survive the primary manifestations of the disease remain for some days in a precarious condition owing to the intense blood destruction, and, though the urine may regain its normal appearance, heart failure may supervene with a fatal issue. Relapses are common, and severe anaemia of a macrocytic type is usually a prominent feature.

Treatment

Owing to the excessive blood destruction associated with blackwater fever there is always great risk of cardiac failure. Hence the first essentials in treatment are careful nursing and absolute rest. The patient should on no account be subjected to any form of exertion, and should not be allowed even to sit up in bed until the acute stage is past. To counteract the restlessness of mind and body parenteral phenobarbitone sodium has been recommended.

If malaria parasites are seen in the peripheral blood, a full course of treatment with a specific antimalarial drug should be given as described above. Neither pamaquin nor any other of the 8-aminoquinolines should be given under any circumstances, and quinine is best avoided. Vascular failure may be treated by intravenous plasma, followed if necessary by intravenous saline solution, which may be alternated with isotonic glucose. The fluid intake should not be less than 2 litres a day, and should be so adjusted that the urinary output does not fall below 1.2 litres.

The administration of alkalis in massive doses was formerly recommended, but it has been shown that a too vigorous alkali treatment may give rise to an alkalosis which itself may impair the excretory function of the kidneys. It is therefore recommended that not more than 20 g. of sodium bicarbonate be given in the 24 hours, and that once the urine has become alkaline only enough be given to maintain a slightly alkaline reaction. If used intravenously bicarbonate solution should never be sterilized by heat.

Should the red-cell count fall as low as 1,500,000 per c.mm., blood transfusion, preceded by very careful blood grouping, is indicated. Good results have been reported following the administration of washed packed red cells resuspended in normal saline solution. The patient should be kept strictly in bed for at least 10 days after the disappearance of haemoglobinuria. Liver extract is useful during convalescence.

Malaria Prophylaxis

As noted above, there is no known drug which acts on the sporozoite stage of the malaria parasite, but there

are two classes of drugs which may prevent the onset of an overt attack: (1) those which act on the pre-erythrocytic phase; and (2) those which act on the asexual erythrocytic phase. The biguanides are the only drugs which can achieve the former effect in non-toxic dosage, and their action in this respect is confined to falciparum malaria. Since, however, they act also on the asexual erythrocytic forms of all species of human plasmodia, their use in prophylaxis is by no means limited to falciparum infections. The dosage of proguanil recommended for prophylactic use is 100 mg. daily for non-immune individuals, and 300 mg. in a single dose once weekly for indigenous inhabitants of malarious areas. A great advantage of this drug is that it can be placed in the hands of laymen for distribution without risk of ill-effect, even should the prescribed dose be grossly exceeded.

The second group of prophylactic drugs, which exert their effect solely by their suppressive action on the asexual erythrocytic parasites, includes the cinchona alkaloids, the 9-aminoacridines, the 4-aminoquinolines, and the 2:4-diaminopyrimidines.

For the effective suppression of falciparum malaria, quinine has to be administered in doses as high as 10 gr. (0.65 g.) daily. Against New Guinea strains of *P. falciparum* even this dosage has proved insufficient. Apart from the unpleasant side-effects caused by the prolonged administration of this amount of quinine, the association of this drug with the precipitation of blackwater fever renders it unsuitable for use as a suppressant in areas where falciparum infections are prevalent.

Mepacrine, in a dosage of 100 mg. daily, is a very effective suppressant of all forms of malaria, provided that the drug is taken for 14 days before exposure to infection and continued for one month after leaving the endemic area, but it has certain disadvantages which militate against its routine use under peacetime conditions. A variable degree of nausea and abdominal discomfort is fairly common during the first fortnight of administration and these symptoms persist to some extent in a small proportion of cases. Headache, insomnia, and giddiness may also occur during this stage, but are less frequent. When taking the drug over long periods a proportion of individuals develop skin lesions, the most common of which is a lichenoid dermatitis affecting chiefly the hands, wrists, feet, and ankles. Yellow discoloration of the skin is also comparatively common.

Chloroquine is probably an even more powerful suppressant than mepacrine, the dosage usually prescribed being 300 mg. of the base (500 mg. of the diphosphate salt) once weekly. Its action resembles that of mepacrine, but it is considerably less toxic and does not tint the skin. The same advantages are claimed for camoquin, in a dosage of 500 mg. weekly.

At present the choice of drug for prophylaxis seems to lie between proguanil and either chloroquine or camoquin. Neither of the last two possesses the very low grade of toxicity which is so remarkable a property of proguanil, rendering this drug particularly suitable for widespread administration among labour forces or other employees recruited from indigenous populations of malarious regions. The comparatively low cost of proguanil and its inhibitory effects on gametocytes are other arguments in its favour. On the other hand, it has been shown that, in falciparum malaria at least,

resistance to proguanil may be brought about by irregular or insufficient dosage. It is important therefore to ensure that the drug is taken regularly and in the amount prescribed. It is also advisable in dealing with communities under proguanil prophylaxis to use some other drug, such as chloroquine or mepacrine, for the treatment of overt malarial attacks.

Conclusion

There are many examples of the wide variation in the response of different geographical strains of the same species of malaria parasite to particular drugs. For example, Italian strains of *P. falciparum* have been found relatively resistant to quinine, but readily susceptible to mepacrine. A strain of *P. falciparum* in New Guinea proved resistant to mepacrine, but highly susceptible to proguanil. The existence of strains which are naturally resistant or have acquired resistance to proguanil has also been reported. It is therefore unwise to be too dogmatic on the most suitable form of treatment of malaria in any particular circumstance, and it is fortunate that, owing largely to the enormous volume of intensive chemotherapeutic research carried out during the war, we now have at our disposal a number of specific antimalarial drugs, so that, should a strain of parasite prove resistant to any one, an alternative may be used.

FIVE YEARS OF PROGRESS IN THE HONG KONG HEALTH SERVICES*

BY

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Five years have now passed since the Japanese surrender, and it is possible, on looking back over what has been done, to get far enough away from the trees to see something of the wood.

When news filtered into Hong Kong's prisoner-of-war and internment camps in August, 1945, that the war was at an end, those responsible for the administration of the Colony, including the health services, made their way out of confinement and began a survey of the desolation and destruction left behind by the Japanese.

Nearly two-thirds of the European type of dwelling-houses and about one-fifth of the houses of the working-class Chinese were damaged or in ruins, and, in addition, many public buildings, such as schools, hospitals, dispensaries, and other similar institutions, were destroyed. Those responsible for the administration of the health services set to work without proper office equipment, with a complete absence of all records, and having to rely almost entirely on memories impaired by three and a half years of severe privation. The streets were largely deserted, and the whole Colony could boast of probably little more than 600,000 people, most of whom were suffering from a prolonged period of semi-starvation, and who, like those released from the camps, were unfit for any extended physical effort. There was no public transport, and private cars were a rarity. Antimalaria measures had been neglected for three and a half years,

*Written in 1950.