Papers and Originals

Imported Malaria in the United Kingdom

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British Medical Journal, 1969, 2, 781–785

Summary: Over 2,000 cases of imported malaria have been confirmed by blood examination. Ninety per cent. of cases from tropical Africa were infected with P. falciparum. Most of the patients were Caucasians and had primary infections. All developed fever within a month after arrival and most of them within two weeks of arrival. In some patients malaria parasites were seen in routine blood films.

Developing forms of P. falciparum were always present in the peripheral blood of patients suffering from a primary attack which was not diagnosed or treated until a week or more after the onset of fever.

All deaths investigated were caused by P. falciparum and were primary infections.

In not one of the P. falciparum infections did the victim continue taking prophylactic drugs for more than a few days after leaving the endemic area. Had drugs been continued for one month probably not a single overt case of P. falciparum would have occurred.

A primary attack of P. falciparum malaria is seldom, if ever, classical in that the fever is never tertian and may resemble clinically many other diseases.

Children in boarding-schools returning from the tropics should be supplied with prophylactic tablets and instructions to the matron. If there is an epidemic of a fever any students who have recently returned from the tropics should have a blood film examined for malaria.

The risk of contracting malaria among drug addicts is considerable, especially with P. falciparum.

Introduction

This paper is a commentary on over 2,000 cases of imported malaria, which became a notifiable disease in the United Kingdom in 1919 under the Malaria and Dysentery Regulations. These regulations still operate.

In 1919 thousands of Service men from the first world war who were potential relapsing cases of malaria returned to this country for demobilization. Most of them came from Salonika, but many came from other parts of the world, including East and West Africa, Mesopotamia, and India. Even in 1917 hundreds of persistently relapsing cases, mostly due to *Plasmodium vivax*, were sent home from Salonika under the "Y" scheme to relieve hospital congestion in the field. Many of these patients relapsed at least once a month despite heroic doses of quinine, and where numbers of them were concentrated in convalescent camps situated where the English *Anopheles maculipennis* density was high, especially in rural districts in the south-east coastal corner of England, some of the local population became infected. There were over 400 cases of indigenous malaria in Kent and Essex and nearly 100 in other counties, and one or more cases occurred in over half the counties of England.

To prevent a further outbreak of indigenous malaria after the second world war a scheme was prepared by the Ministry of Health whereby those areas which were known to have a high anopheles density were avoided as demobilization depots for Servicemen returning from malarial war zones. A map showing these potentially dangerous areas was prepared for the medical departments of the three fighting Services, and we believe that this contributed to the lower incidence of indigenous malaria after the second world war. In any event only 34 cases of indigenous malaria occurred after the second world war as against 500 after the first (Shute, 1949). All except one of the indigenous cases following both world wars were infected with the species *P. vivax*. The exception was a patient who died from *P. falciparum* malaria and was infected in Liverpool. How the patient became infected is uncertain. Blacklock (1921) believed that he was infected by a mosquito bite, but Ross (1920) considered that the cause was more likely an infected syringe used by a dentist.

Imported cases of malaria have been occurring for centuries, but until the introduction of air travel most of them, at least those returning from tropical Africa and infected with *P. falciparum*, developed fever on ships before arrival. Now, however, infected patients frequently arrive before the end of the incubation period, even though in *P. falciparum* infection it may be as short as five days and is seldom longer than 14 days.

At this laboratory we began to take an active interest in imported malaria in 1954. We had learned of a death from *P. falciparum* malaria, and on examining blood films and tissue material from the patient it was found that the peripheral blood was swarming with parasites, not only with small "signet ring" forms (trophozoites), as is usual in *P. falciparum* malaria, but also with developing forms—evidence that infection had started at least eight days before the blood films were taken (Shute, 1968).

By arrangement with the Public Health Laboratory Service we are now informed each week of all cases of malaria notified to the Central Public Health laboratory. We write to the pathologists concerned and ask for the loan of the films on which the diagnoses were based, and also ask the pathologists to fill in questionaries. We receive generous co-operation, and this enables us not only to confirm the diagnoses but also, in some cases, to correct the identity of the species concerned. At first we confined our investigation to patients returning from tropical Africa, but later, with the large influx of immigrants from Asia and the Caribbean, etc., we extended our requests.

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Malignant Malaria

Not surprisingly, over 90% of positive films from tropical Africa showed *P. falciparum* parasites. Many of the patients developed fever and parasitaemia a few days after arrival by air. Some developed symptoms a week or two after arrival, but none as long as a month later. Maegraith (1963) wrote: "Flying enables people to move vast distances within the limits of any incubation period, no matter how short." *In not a single case which we investigated did the patient continue taking prophylactic drugs, proguanil (Paludrine) or pyrimethamine (Daraprim) for one month after arrival in this country.* Had these patients done so it is unlikely that there would have been any overt cases. We were usually told that prophylactic drugs had been discontinued, either on the day of leaving Africa or within a few days after arrival, and that the patients had never taken the drug for more than a week after they arrived. It is possible that these patients were infected only a few days before leaving Africa, and therefore the prophylactic was not taken long enough to destroy the parasites before completing their development in the parenchymal cells of the liver.

B.O.A.C. has a large poster showing an anopheles mosquito, and the caption reads: "Malaria is a disabling and deadly disease, but you'll be quite safe if you take Paludrine every day while in a danger area and continue taking it for 30 days after you leave."

B.O.A.C. carry large stocks of Paludrine tablets on all aircraft going to the tropics, but very few passengers ask for any. Travellers to places such as the Falkland Islands, where there is no malaria, may easily become infected en route. On this journey the aeroplane may refuel at airports in malarious regions, and if the passengers sit about in the open air after dark they could easily be bitten by infected mosquitoes.

Deaths from Malignant Malaria

From 1954 to March 1969 there have been 58 deaths. We have investigated eight of them. Two of these are worthy of note.

(1) An air steward was switched from the North American route for only one trip to West Africa. He spent only one night in Africa, took no prophylactic drugs, and developed fever a week after his return. He died of malignant malaria a few hours after admission to hospital.

(2) A woman passenger on board ship called at a West African port and spent only *four hours* there and then returned to the United Kingdom. Two weeks later she developed fever and died of malignant malaria six hours after admission to hospital. The blood film showed 50% of the erythrocytes parasitized with "ring" forms and developing trophozoites.

In all eight cases fever and death occurred within one month of the patient's arrival in this country. In every case, in addition to "ring" forms, developing trophozoites were present in the peripheral circulation. All were primary infections. None of the patients had taken prophylactic drugs after leaving Africa and all except two had returned by air.

In some of these patients a mixed infection had been diagnosed, because developing forms of *P. falciparum* were present in the blood film, which is unusual for this species. We have pointed out on many occasions that developing forms of *P. falciparum* trophozoites show only one or two large lumps of pigment in the cytoplasm, whereas all other species of parasite contain 20 or more small grains of pigment. This is a very useful guide to species diagnosis, and of considerable importance (Shute, 1965).

Risk of Primary Attack of P. falciparum

Blood films often show developing forms of the parasite. In nearly every case these forms are associated with a heavy parasitaemia; sometimes 40% of the erythrocytes are parasitized. Investigations have shown that the patients had been febrile for at least seven days before the film was taken and also that the attacks were primary. Many years ago, when malaria therapy was the treatment of choice for general paresis, at this hospital we infected about a thousand cases with strains of *P. falciparum* from Africa, Europe, India, and Malaya (James *et al.*, 1932). It was found that if a patient was allowed to have more than seven days of fever in the primary attack before being given small doses of an antimalarial drug, developing forms invariably appeared in the peripheral blood and were associated with rigors. If, however, after five or six days of fever subcurative doses of antimalarial drugs were given, after three or four recrudescences tolerance was established and developing forms were never seen, even though the attacks of fever were clinically severe. There were no deaths.

It is rather surprising that patients who have been in malarious areas often do not consult their doctors for several days after fever develops. Many of them have stated that they did not feel ill enough to go to see a doctor and thought they had an attack of influenza. The absence of rigors in *P. falciparum* malaria, except in advanced primary infections, is perhaps why this is so. It is also one reason why malaria is more unlikely to be suspected. It is acknowledged that the first three or four days of an attack due to *P. falciparum* are less distressing to a patient than an attack due to *P. vivax*. Our personal experience bears this out, as during the past 25 to 40 years both of us have been infected many times with all four species, both in the field and in the laboratory.

Several cases of primary *P. falciparum* malaria have been discovered accidentally during a differential leucocyte count or a blood examination for anaemia.

Some strains of *P. falciparum* in South-east Asia and some parts of South America have developed chloroquine resistance. So far as is known, no resistance to chloroquine has been proved to occur in strains from tropical Africa, but repeated warnings are being given of the need for continuous vigilance. In primary infections of *P. falciparum* malaria, any delay in effective treatment, even for a day or two, may endanger the life of the patient. Many authorities are therefore now advising that in severe cases of *P. falciparum* malaria quinine (parenterally) should be given initially.

Benign Tertian Malaria

Whereas over 90% of all imported cases of *P. falciparum* malaria were contracted in tropical Africa, the majority of *P. vivax* infections were found in people from Pakistan. The explanation for this was probably an epidemic in and around Karachi which began about a year ago and is still continuing. Again, while 90% of *P. falciparum* infections were in Europeans, the majority of *P. vivax* infections were in Asians. In *P. falciparum* infections patients developed fever and parasites within a week or two after arrival in this country, but with *P. vivax* the incubation period varied between a few days after arrival and up to a year later.

Only very few of the Asians had taken prophylactic drugs. In one family of Pakistanis the father, mother, and child all developed fever six months after their arrival, and all within a few days of each other. These were obviously latent cases if the father's claim that they had not previously suffered from malaria was correct. There were no deaths due to *P. vivax*.

Plasmodium ovale

Because this species so closely resembles *P. vivax* morphologically, it is not surprising that most of the *P. ovale* blood films sent to us had been diagnosed as *P. vivax*, except those from the Tropical Diseases Hospitals in London and Liverpool.

It seems not to be realized that *P. vivax* is non-existent in most parts of West Africa. Though *P. ovale* has been found in one or two countries other than Africa, it is comparatively rare. That *P. ovale* infection is often mistaken for that of *P. vivax* is of no clinical importance. It is in fact a milder disease and usually clears up spontaneously after seven or eight attacks of fever, but high temperatures (104–105° F.; 40–40·6° C) may be recorded for the first few peaks. Relapses are rare, but, like those with *P. vivax,* they may occur several months after the primary attack. Latency of the primary attack is also a feature of *P. ovale,* and this may be as long as one year after infection (Shute, 1946). In 200 cases of general paresis infected with *P. ovale* by mosquito bites at this hospital the disease disappeared spontaneously and there was only one relapse.

Plasmodium malariae

Of all the four species of human malaria *P. malariae* is least common among imported cases; only 40 cases have come to our notice over a period of 14 years. The outstanding feature of *P. malariae* is its longevity in the human host, and it seems doubtful whether it is possible to say whether a patient is ever cured. Relapses have been reported 50 years after patients had left an endemic area, and at this laboratory we have seen relapses occurring after 10, 12, 15, and 21 years (Shute, 1944; Duggan and Shute, 1961).

The percentage of patients infected with *P. malariae* who relapse is unknown, but we have reason for believing that it is not high. In the practice of malariotherapy, *P. malariae* infections transmitted by mosquito bites were fewer than with the other species. It was extremely difficult to infect mosquitoes owing to the very low gametogony in the human host (Shute and Maryon, 1955). Only 19 cases have been successfully infected by us through the agency of mosquitoes, and none has so far relapsed. This includes both of us, one of whom was infected 25 years ago and the other three years ago. Of the 40 cases of *P. malariae* imported into this country two had mixed infections and all were from tropical Africa. Parasitaemia is never very high in *P. malariae*; in fact, it invariably has the lowest rate of parasitaemia of all four species, and a long search of several thin films is necessary before excluding it as the cause of an attack of fever.

As with *P. vivax,* in a primary attack of *P. malariae* the fever is often quotidian, owing to several stages of growth of the parasite in the blood at the same time and an absence of acquired premunition or tolerance; it is in relapses that the fever is the classical tertian or quartan, according to the species of parasite involved.

Examination of Thin Blood Films

In clinical malaria, with the possible exception of *P. malariae,* only thin blood films need be examined, because parasites are usually numerous enough to find after searching a film for a very short time. The host cell is helpful in clinching the diagnosis, and if the staining is good erythrocytic stippling, or its absence, in parasitized cells is helpful for species diagnosis. The thick film is useful for survey work and when species diagnosis is not of major importance.

A well-prepared thin film should have the erythrocytes almost touching but not overlapping each other. This is an essential requirement, but it is seldom practised by technicians who prepare films for haematological examination. Malaria parasites are most conspicuous if the distilled water has a *p*H of 7·0 or 7·2. If the distilled water is acid the cytoplasm of the parasite will be only faintly coloured or even uncoloured. All distilled water quickly becomes acid when exposed to CO_2, and it is good practice to use only distilled water which has been buffered to neutral or *p*H 7·2. Both Leishman and Giemsa stains, when diluted with neutral or very slightly alkaline distilled water, will always bring out prominently the coarse stippling of *P. vivax*-infected cells (Schüffner's dots) and so make species diagnosis easy and certain. In *P. falciparum* malaria, particularly in primary infections, the tiny hair-like rings, often occupying only about one-sixth of the host cell, show clearly the very fine wisp of deep blue cytoplasm and the pinkish mauve dot of chromatin. In the host cells of these minute parasites stippling is never seen; it is in relapses and chronic infections of *P. falciparum,* when many of the ring forms of the parasite are quite large, often occupying one-third of the host cell, that stippling (Maurer's dots) is seen.

Technique

A thin film is prepared with the erythrocytes almost touching but not overlapping (Shute, 1966; Shute and Maryon, 1966[1]).

Leishman Stain.—(1) Without previous fixation, put 7–8 drops of undiluted stain on the film. Leave for 20 seconds, *not more,* then add 12–15 drops of neutral or very slightly alkaline distilled water. (2) Mix by gentle rocking. (3) Leave to stain for 15–20 minutes, wash off with a flood of distilled water from an aspirator, drain, dry, and examine.

Giemsa Stain.—(1) Fix the films with one or two drops of methanol (Analar); these will evaporate in about 20 seconds. (2) Make up a 5–7% solution of stain with neutral or slightly alkaline distilled water as a diluent. (3) Pour the diluted stain on the blood film and leave to stain for 20–30 minutes. Wash off with a stream of distilled water, dry, and examine.

Malaria in Schoolchildren

Thousands of schoolchildren, nearly all from boarding-schools, go to Africa and other tropical areas to be with their parents during holidays; some go even twice a year. The parents usually see that their children take prophylactic drugs regularly while they are there, but make no provision for continuing prophylaxis after the child's return home. There have been a few cases of *P. falciparum* malaria in children after they have returned to school, and in some the symptoms were very severe (Shute, 1965).

Malaria is easily overlooked among children in boarding-schools, especially since a boy or girl may develop malaria at a time when there is an epidemic of some other fever such as influenza. Manson used to teach, "Malaria may simulate any disease known to medical science." He was, of course, referring to primary attacks of *P. falciparum* malaria, and not to relapses of the other three species, when the fever is nearly always classical tertian or quartan. In a previous paper (Shute, 1965) we suggested that children returning from the tropics should be provided with one month's supply of proguanil or pyrimethamine, and that the tablets should be handed to the matron of the school with instructions to see that the child took the drug regularly over a period of one month. In addition, the matron should be told that the student has been at risk to malaria and that, in the event of an attack of fever, arrangements should be made to have a blood film examined for malaria parasites.

Immigration and Indigenous Malaria

The question is frequently asked, "What is the risk of the spread of malaria as the result of the numerous cases of malaria among the immigrant population?" The answer is, "Very little." One reason is that few, if any, coloured immigrants seem to be settling in rural districts where anopheles are prevalent, and in industrial areas there is no mosquito problem.

[1] Reprints available on request.

Nevertheless, it cannot be stated dogmatically that there will be no indigenous cases in the future, even in large towns. In 1953 two indigenous cases of *P. vivax* occurred in Lambeth, London (Shute, 1954), and after a long search *A. plumbeus* was found breeding in a collection of water in the hollow of a plane tree close to the houses where the cases of malaria occurred. *A. plumbeus* is an efficient carrier of all four species of the human malaria parasites. In built-up areas where trees line the streets and harbour collections of water between large branches, forked trunks, and rot-holes this mosquito can often be found. It has also been collected from trees in parks. As the only blood meals available are usually those afforded by man, it is a remote possibility that this species may become infected. Fortunately it seldom enters houses and prefers feeding in the open and near to its breeding-grounds.

The indigenous malaria which began in 1917 in some coastal parts of south-east England soon showed that the carrier species of mosquito was *A. labranchiae atroparvus*. This mosquito prefers low-lying brackish water for breeding purposes, and the adults pass the whole of their lives in dark, ill-ventilated animal houses, human or otherwise, where blood meals are always available, and leave the dwelling only for egg-laying. Bionomically the females are both anthropophilic and zoophilic, and for every individual found in a human dwelling dozens or even hundreds can be found in horse stables, pigsties, byres, and even domestic rabbit hutches. In one village on the Kentish coast 50% of a population of 400 became infected with *P. vivax* between 1917 and 1920. In inland districts *A. labranchiae atroparvus* is comparatively rare. Its near relative *A. maculipennis messeae* is present in inland rural areas in very large numbers wherever there are suitable fresh-water breeding-grounds and nearby animal houses. The female adults are highly zoophilic and seldom bite man when domestic animals are available. Nevertheless, in the laboratory they readily become infected with malaria when they can be induced to feed on man. It is interesting to record that during 1917–22, while in villages of some coastal districts of Kent and Essex there were small epidemics, in inland rural areas there were never more than one or two cases at a time.

Though immigrants who develop malaria after arrival in Britain do so with all four species of malaria parasite, only *P. vivax* is likely to give rise to indigenous cases. Immigrants or Europeans who develop *P. falciparum* in this country will never be a source of risk of the spread of this species of malaria. There are two main reasons why this is so:

(1) A constant atmospheric temperature of at least 75° F. (23·9° C.) is necessary for the completion of the parasite cycle in the mosquito, and at this temperature the completion of the cycle takes 12 to 20 days. The chances of an anopheles mosquito living this length of time and biting man twice during the period, once to acquire the infection and again to transmit the infective organism (sporozoites), are very slight. A temperature of 80° F. (26·7° C.) is optimum for *P. falciparum*.

(2) Intensive research some years ago proved conclusively that African, Indian, and Malayan strains of *P. falciparum* studied in this laboratory failed to develop in our indigenous anopheles, even though ripe gametocytes were numerous in the peripheral blood of the patients and ookinetes formed readily in the blood in the stomach of the mosquito. In other words, English anopheles are refractory to at least several tropical strains of the species. On the other hand, the same species of English anopheles is highly susceptible to eastern and central European strains of the same species (Shute, 1940). This research was carried out before malaria was eradicated from Europe, and at this laboratory our English anopheles became heavily infected when fed on suitable gametocyte carriers infected with *P. falciparum* strains from Rumania, Sardinia, and the Roman Campagna. *P. ovale* and *P. malariae* are so rare that they need not be considered as ever being likely to give rise to indigenous malaria in this country.

At one time malaria was endemic in some parts of England, but according to the Registrar General's report it began to recede about 1847, and by the end of the century it had disappeared completely, though no deliberate measures were taken to eradicate the disease. Why this was so has never been satisfactorily explained. Agricultural land drainage, the introduction of quinine which could be bought cheaply in chemists' shops around 1850, and the improved housing conditions all undoubtedly played a part (James, 1929). It was not only in England that malaria as an endemic disease began to recede in the latter half of the last century. It was the same in Denmark, Northern Italy, France, and other northern countries of Europe. James (1929) states: "The view is quite common that the downward trend of malaria in some countries is a natural phenomenon independent of human action and unexplainable."

Induced Malaria after Blood Transfusions

The regulations on the prevention of the spreading of induced malaria by blood donors are adequate, and the fact that there have been only five cases in the United Kingdom over the past 30 years confirms this (National Blood Transfusion Service, 1963). Only one case of *P. falciparum* malaria has occurred after a blood transfusion, and this was when an African student, aged 18 years, was inadvertently accepted as a donor (Grant *et al.*, 1960). The student had been in England only a few months and was a chronic carrier (parasites) without clinical symptoms. Very scanty parasites were found in his blood after a prolonged search.

Only one case due to *P. vivax* has occurred (Thomas *et al.*, 1936). This was in 1935, and is believed to be the first case in the United Kingdom. The other three patients were all infected with *P. malariae*. *P. vivax* and *P. ovale* may be considered as an entity because the two species are closely allied and have about the same life-span in the human host, which is about two years. Relapses of both species may occur up to one and a half years. Whether parasites circulate in the peripheral blood during this interval is unknown, but it is generally believed that this is not so. People infected with *P. vivax* or *P. ovale* may have a protracted incubation period as long as one year, but this is unknown to occur with *P. falciparum*. It is worth recording that blood-induced malaria of any of the four species does not relapse after a course of appropriate drug therapy.

Three of the five cases of malaria resulting from a blood transfusion were caused by *P. malariae*. In Eastern Europe, where transmission has been eradicated for nearly 10 years, malaria after blood transfusions still continues. In Rumania malaria was eradicated in 1963, yet during 1959–62 there were 73 cases of malaria after blood transfusions and all were with *P. malariae*. It is the same in other European countries where indigenous malaria has been eradicated (Lupaşco *et al.*, 1963). Relapses of *P. malariae* infection have occurred as long as 50 years after the individual has left an endemic area, and it is probable that a low-grade parasitaemia with this type of malaria may persist for life. Some years ago a man who had lived in Britain for 12 years acted as a donor for his infant son. Three weeks after transfusion the infant developed fever, and subsequently died. *P. malariae* parasites were found in post-mortem blood films (Nabarro and Edward, 1939). The father was an Englishman born in Ceylon and was not aware that he had ever been infected with malaria. On investigation after the child's death scanty *P. malariae* parasites were found in thick films of the donor. Parasites are nearly always very scanty in chronic carriers of *P. malariae*, and even in thick films prolonged search may be necessary to find one parasite.

In countries where malaria is holoendemic and where all species of parasites are found it is generally considered that rarely more than 5% of an indigenous population is infected with *P. malariae*. Because of the extremely low parasitaemia in *P. malariae* infections, it is probable that 5% is a gross underestimation. Were it not for the longevity of *P. malariae* in man, people from the tropics could safely act as whole-blood

donors after residence in this country for four or five years. Because of the very low parasitaemia of this species, it would never be safe to use as a donor anyone who had resided in a malarious endemic area at any time. It is difficult to imagine that there have been undiagnosed cases of malaria resulting from blood transfusions. Accidental infection with *P. falciparum* would be fatal, and if the species were *P. vivax* the swinging temperature, rigors, and sweating would hardly fail to be recognized clinically. In death from malaria by a blood transfusion the enlarged slate-coloured and spongy spleen would pinpoint a diagnosis of malaria.

Risk of Contracting Malaria among Drug Addicts

There are several reports of malaria contracted by drug addicts. Many deaths from *P. falciparum* malaria have occurred in this way. In Egypt Biggam (1929) observed accidental hypodermic transmissions of malaria among heroin addicts, and many cases have been reported from the U.S.A. So far as is known no cases of malaria have occurred among drug addicts in this country. Now, however, with the large number of immigrants from endemic areas entering the United Kingdom there is always the possibility of cases occurring, especially when members of the indigenous population use a contaminated syringe. If a syringe contaminated with blood containing even a few parasites of *P. falciparum* malaria were used by a non-immune individual the result would be fatal, because it would be unlikely that malaria would be suspected, even if the addict reported sick. As the early stage of an illness due to *P. falciparum* is relatively mild, and rigors are absent, a correct diagnosis could easily be missed if a medical examination were restricted to clinical examination.

All species of malaria could be contracted by addicts who use infected syringes, especially if the narcotic drug is injected intravenously, but the disease would not be likely to end fatally, except with *P. falciparum*. In the event of the syringe being contaminated with parasites of *P. malariae* the infected addict could be a source of danger for many years and perhaps for the rest of his life.

Conclusion

While it is true that malaria has been eradicated from the whole of Europe, most of the Southern United States of America, and many islands, including some tropical ones, it is not so in any part of tropical Africa. The percentage of mosquitoes infected with malaria, and their actual numbers, is much the same as it was in the days of Livingstone and Lugard and is likely to remain so for a very long time.

If it were possible to persuade all people returning from the tropics, and especially from tropical Africa, to take regularly for one month prophylactic antimalarial drugs, proguanil, or pyrimethamine, it is likely that there would not be a single case of *P. falciparum* malaria among these returning travellers.

People returning from malarious areas who have been infected with *P. vivax, P. ovale,* or *P. malariae* may develop fever for the first time as long as one year after leaving an endemic area. Because rigors are a feature of these three species malaria is very much more likely to be suspected than when the infection is caused by *P. falciparum.*

Although there are five species of *Anopheles* indigenous to this country, only the domestic *A. labranchiae atroparvus* and *A. plumbeus* have been incriminated as carriers of malaria in nature.

An epidemic of malaria as the result of a large number of immigrants is unlikely. Indigenous cases of *P. falciparum* can be ruled out. (1) The atmospheric temperature is not high enough over a period of time for the parasite to complete itself in the insect host. (2) Our *A. atroparvus* is refractory to all the tropical strains of *P. falciparum* which have been tested in the laboratory. Any indigenous cases which may occur would be with the species *P. vivax.* There is no risk of contracting malaria by blood transfusion if the advice given by the National Blood Transfusion Service is followed.

We wish to thank the many pathologists who have kindly sent us blood films; the Director of the Public Health Laboratory Service for kindly notifying us of all reported cases; and Dr. Antony Duggan, Director of the Wellcome Museum of Medical Science, for his constructive comments.

The Malaria Reference Laboratory at Horton Hospital, Epsom, Surrey, is always glad to receive and examine thin blood films (preferably unfixed and unstained), either for the presence of malaria parasites or for species diagnosis.

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