EUROPEAN INTO COOLIE.
Ps.o.W. Adapt Themselves to the Tropical Villagers' Diseases

BY

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It is well known that the mortality and the disease rate among Ps.o.W. in the Far East were very high. We may have lost 30 per cent in the three and a half years. This article is a general survey of the health of the troops as seen by one medical officer. I was not in Siam in the first half of 1943 when the bulk of the deaths occurred. During the rest of the time, we were living as it were on the edge of a precipice. A high proportion of us would show minor or even grave symptoms of one or more potentially serious diseases; we had neither the diet nor the drugs to treat our patients adequately; only in a few places were laboratory facilities available. Yet the threatened calamities did not—except in 1943—occur; the diseases we experienced became instead part of a picture of general ill-health; we became stabilized at a level of health not uncommon in the oriental villager or coolie whose life we were—in effect living.

After the capitulation in February, 1942, the Japanese concentrated about 45,000 of us in Changi barracks at the east end of Singapore Island. They told us to feed ourselves for the first ten days, after which the rice diet started. Stores of tinned food continued to eke out the rice and lasted until the Japanese re-organized the cold stores in Singapore and issued some meat. The electric supply of these stores had not been out of action quite long enough to destroy the meat. The meat supply lasted till about November when the first Red Cross ship arrived. Our hopes that the shortage was over came to nothing for, instead of regular supplies, we had hardly any more until the end.

The first half of 1943, besides being the time of the cholera and the run of deaths from malaria, beri-beri and phagedenic ulcer in Siam, whither most of us had been transported, was a time of very bad feeding at Changi. Rice and a little fish were practically all we had.

In June, 1943, I was sent to join the rest in the bamboo jungles of Siam. We travelled in railway trucks. It was a week's journey, yet we had not enough room to lie down. We arrived to find the remnants of the labour force, Ps.o.W. and coolies, being transferred back to Malaya. Everybody was sick at some time; most had some disease all the time. Food supplies which had been hopelessly inadequate were improving; in some camps, we had actually more meat than we could eat.

With this introduction, let us glance over the total results of this ill-health as I saw it.
Beri-beri.—Though we had cases of neuritic beri-beri in alcoholics at the beginning, precipitated by the 15 mile march to our first camp; though we had deaths from cardiac beri-beri quite early; yet a continued deficiency of B, brought us less and less disease. One of the cardiac cases was three times sent to a specialist and twice admitted. He had no physical signs, and was allowed to help in the ward; moving some furniture, he fell dead. This did not encourage us to despise oedematous beri-beri or treat it lightly. Rarely, however, did we have aneurin to treat it. The rice later in the imprisonment was less highly polished. Rice polishings were available, usually only for frank cases; it was so unpalatable that it was difficult, even when there was plenty, to get it eaten prophylactically. Yeast preparations, including tablets, were used; and latterly a few tablets and injections of 300 to 500 units. Green peas of the kind used for sprouting (toungay) were issued as a ration in small quantities and sometimes supplemented from Siamese Red Cross stores.

So harmless and variable did the beri-beri appear that we remained in some doubt as to the possibility of confusion with protein deprivation oedema. This was expected much later than beri-beri which hit us as calculated after three months.

Only when peace was declared were we able to clear up this question. For it was then that the Japanese released some American Red Cross supplies. Among the drugs were ampoules of aneurin. I gave one of my worst oedema cases 100,000 units a day intravenously. The rapid relief of the oedema confirmed the diagnoses of avitaminosis.

Pellagra.—Within five months of the capitulation of Singapore a colleague and I found unmistakable signs of early pellagra in 13 per cent of several hundred men. Most had evidence of shortage of both nicotinic acid and riboflavin. Minor evidence was even more common. These men had had much improved rations for two months after a bad three months.

Naturally we were alarmed. The next run of symptoms, the painful feet mentioned as a rarity in the literature of pellagra, came as a veritable epidemic; there were soon hundreds of men pacing the roads of our spacious camp all night. We took this to be a manifestation of spinal cord involvement, and expected still worse signs. They came. Numerous spastic cases were admitted to hospital. There was a series of cases with brisk reflexes, ankle clonuses and up-going toes, which interested our specialists and will doubtless be well reported.

Even in 1942 there was evidence of brain involvement, a few cases of mental pellagra. Knowing that 10 per cent of certified mental cases in the States are said to be of pellagrous origin, again we had reason to fear that a large proportion of us might go mad before long.

What happened far from justified our fears. An acute confusional insanity occurred in one of my patients with pellagra, who fortunately died a few hours after onset. But the painful feet cleared up within six weeks on 6 ounces of rice polishings and a little Marmite daily. A few resistant cases had
nicamid injections. Later it was quite common to diagnose incipient spastic paraplegia; painful feet became rare. Either seemed to clear up with a few injections of small doses of $B_2$, or even without.

Diarrhoea.—This was to be expected in connexion with pellagra. There is little doubt that a deficiency diarrhea delayed the cure of many of the bacillary dysenteries which were common at the same time. Months afterwards many of the chronic diarrhoeas were cured with grass juices, which would suggest their causation by shortage of minor $B_2$ vitamin. The Dutch forces, especially those brought up in tropical villages, were using the leaves and fruit of passion flower for the same purpose. Our comparative freedom from diarrhea in Siam may well be due to the discovery and use of jungle spinach of various species.

Scurvy.—This was expected in four and a half months, as there seemed to be no vitamin C in the food. None, to my knowledge, occurred. The eating of hibiscus leaves, which began early, may account for this.

Conditions of Doubtful Origin, probably Avitaminosis.—A spate of para-central scotoma, running to 20 per cent of the 5,000 sick left behind on Singapore Island in 1943, alarmed us and will be reported on by the Australian clinic, established for this; its work was past praise.

The point to note, however, is that despite our inability to satisfy ourselves that our treatment was removing the cause of the ensuing visual defects, very few cases were permanently blinded. Many improved and were sent to the Siam work camps where failure of vision was rarely noticed. Night blindness was not uncommon in some camps in Siam, but it cleared up in a few days on transfer to camps with only slightly better diet.

Bacillary dysentery was always with us. In 1942 it assumed epidemic proportions. Here, again, facilities for treatment were extremely poor. An admirable hospital was set up for 1,200 to 1,400 cases. But at one time the magnesium and sodium sulphate was down to 18 ounces for the whole hospital. There was no sulphaguanidine and little if any sulphanilamide. Facilities for spread of the disease were apparently unlimited. There were flies in abundance and personnel were largely untrained. Yet the epidemic had a very low mortality. Later sporadic cases—such as my own—likewise reacted well to the minimal treatment we had. One man whose stools were choleraic for thirty-six hours had 8 tablets of sulphaguanidine one evening. His morning dose was forgotten but on my visit he said he was well. He was soon on full diet and went to work a few days later at his own request.

Amoebiasis.—Amoebic dysentery was common. A minor research which fell to me demonstrated that the ameba was present at first only in those who had had close contact with native life before the capitulation of Singapore and in the native quarters which some of us had to occupy in Changi.

Again, the disease does not seem to have spread. The number of carriers was said to be high. Here it must be remembered that, when routine examinations were being made to select workers for Siam, amoebic stools were sold by
the infected to the malingerers. One man bought a specimen, divided it into two, and got his money back by selling half!

Apart from terminal infections in patients dying from other diseases—such as malnutrition, tropical ulcers and malaria—amoebic dysentery seems to have killed very few. Not that it was not alarming: there were quite a number of liver abscesses which in the absence of sufficient emetine had to be operated upon. Many of these had not had diarrhoea, let alone dysentery, so far as they could be trusted to remember.

In such circumstances, a jaundice in a known amoebiasis victim gives one pause. We did manage to supply a little emetine for such cases, perhaps a total of grains 3 1/2; but rarely for dysentery as such. One of my jaundice cases relapsed as a moderately severe dysentery. Treatment other than with sulphaguanidine was postponed because he was not going down hill, had not lost his appetite and was not being weakened or robbed of much sleep by going to stool too many times a day. He too survived till the days of peace and plenty—plenty of emetine, yatreil and stovarsol.

Emetine treatment of some of the terminal cases was disappointing. Two coolies I was able to try it on both developed œdema immediately. It appears that the dysentery was draining a deficiency œdema. When the dysentery was checked, the œdema recurred.

The small doses of emetine recommended by de Langen and Lichtenstein served us well. This was no surprise to me. In 1934, I had been using such grain 2 1/2 courses in cases negative for amoebae, and finding it often had a marked tonic effect. My cases were Nigerian lepers, but a neighbouring M.O. was doing the same for Europeans with excellent results.

Malaria.—Experiences with minimal doses of quinine in men who had had numerous attacks of malaria did not alarm those of us who had treated malaria in hyperendemic areas like the Gold Coast. The experiment had not been tried on Europeans before, and it is to be hoped that some good papers will be forthcoming from camps with full laboratory facilities.

We came to regard both benign tertian and sub-tertian malaria as diseases of health. Often the actual illness was a matter of hours. At a college in Africa it had been difficult to prevent scholars from hiding their malaria attacks and staying at work. We rarely found that in Siam work-camps; but, when the Japanese insisted on return to work after three, four or five days, this rarely led to any harm or to early relapse. The shortest intermissions occurred in men who were doing more than twice a coolie's job, and who were often in the rain all day without clothing.

When quinine was not available, a run of fatalities sometimes occurred. At one camp at which I worked there had been two such spells. In the first there were a dozen deaths, in the second, there were none. This was possibly due to the use of a bark known to the Javanese; it seems to have staved off but not cured the malaria. One case ran a temperature of 105° F. daily for five days, but was not given the scanty reserve quinine. He survived.

A Dutch colleague in similar straits made up an injection. He obtained some used battery acid to dissolve his quinine sulphate and gave injections of
about grain 1 per case. During this incident, he had a cerebral malaria who survived on this treatment. He describes sixteen blackwater cases with only two deaths, if my memory serves me. One of his blackwater fever cases had a second attack one day when I was with him; the next day he had three rigors, each with a new discharge of haemoglobin; twenty-four hours later, he travelled with me by train to a base camp, twelve hours' journey away!

Finally, we can report a survival from epileptiform malaria. In that case we had the necessary intravenous quinine. Our staff set up a still for double-distilling water and we kept the patient going with intravenous salines during his thirty-six-hour coma. He had three or four typical epileptic attacks in this time.

In another respect, malaria as seen in Europeans who had had thirty or forty attacks corresponded to native cases in Africa; that is in respect to the spleen. This was palpable in a large proportion of those who resisted the disease best. It was either grossly enlarged or not palpable—in those who were really suffering from chronic malaria or had malarial cachexia. This is what I have found (but not yet recorded) in malarious children. It seems that the spleen aids immunity. If it fails to enlarge, or continues to enlarge because it is failing to overcome the parasite, the patient suffers. If it succeeds, it settles at about three or four times normal size, and the malaria becomes, as the League of Nations report on it says, a disease of health. Such diseases only cause occasional short interruptions of health and lead to no stunting or cachexia.

**Dengue.**—Even if we accept de Langen and Lichtenstein’s excellent classification of dengue-like fevers into epidemic and endemic dengue, and include the odd jungle fevers of various short durations under the latter head, it has to be admitted that dengue in Siam manifested itself as a mild disease. Some cases were typical and of the true epidemic type. But they were distinguished from malaria by slight differences in localization of pains and aches rather than by the severity of these; and by rapid recovery. The textbook description of a disease needing long convalescence seems to us not to be accurate. Perhaps under ideal conditions we should have sent these cases home to convalesce.

**Febrile jaundice** was far from rare in Siam, and the consensus of medical opinion, in the absence of laboratory confirmation, was in favour of a diagnosis of Weil’s disease; yet it accounted for no deaths to my knowledge.

**Prolonged Fevers.**—These were usually diagnosed as typhus. It seemed more probable that this was spread by lice than by ticks. The local eschar characteristic of scrub typhus was rarely seen. Again, there were very few deaths.

**Lice.**—Another surprise was the ease with which lice could be dealt with. In coolie work, there were times when we were all infested. Yet they rarely spread from mat to mat. My neighbour had them when we were lying on overlapping mats, having about 2 feet 3 inches by 6 feet space each to sleep and feed in. I had not a single louse.

One time, when I had them, a couple of boilings of two articles involved on successive days was all I could do. On the third day I picked an adult
louse from one of them, a sure sign the job of disinfesting was far from done. Despite the fact that I could not repeat the treatment, I had no more lice from that day on.

**Bedbugs.**—Of these we were never free except when we had separate beds. They bred freely and hardly anyone’s sleep was disturbed by less than several score of them. They, too, disappeared without routine disinfestation after our departure to Bangkok. Freer use of soap, which we found fatal to them in high dilution, may account for this.

**Scabies.**—Septic itch was an alarming concomitant of nearly all disease in Siam. We could get no sulphur at all for a long time. One of my colleagues tried 0.5 per cent cresol for it. We used gallons of this (which was poured neat by Japanese into latrines, and begrudged to us for this economical use). This had the most remarkable effect in clearing up the sepsis and relieving the irritation.

Beyond this, we found it unnecessary to go. A dispenser of mine in Sierra Leone once told me he always kept an area of scabies on his body, for fear of developing an all-over infection. The latter I have only seen in soldiers, and one African child, who had had no previous scabies. Our freedom from symptoms of scabies late in our P.o.W. life would suggest, as these other facts do, that the body learns to resist the ecto-parasites, so long as there are a few in residence in some area of the skin.

By this time, however, the Japanese had begun to supply an excellent sulphur suspension, known as sulphur-lye and smelling strongly of H₂S. This appeared to kill the mite when applied once or twice to the affected areas only. Wherever possible we also had clothing and bedding boiled, but it is doubtful whether this was necessary.

**Droplet Infections.**—Diphtheria came as an alarming epidemic in 1942. It was especially alarming because one of our first cases was only recognized when he died of heart failure from merely turning over in bed. We had no serum. Here must be recounted an accident by which we discovered that our carrier rate was enormous. It had been decided that as we could not deal with the carrier problem, we would not investigate it. By a misunderstanding, the staff of one (non-diphtheria) ward were tested. Some 25 to 35 per cent were positive on culture, if my memory serves me. This had to be hushed up, but the epidemic never assumed the terrible proportions one might have feared.

A corresponding observation made in tropical Africa is that even measles spreads but little there. This may well be due to the fact that in such warm climates, everyone who can sleeps with windows and doors wide open. As P.s.o.W. we had huts open to the air; walls were either non-existent or well short of the roof.

Finally, **Flies.**—The only type of latrine we could make in Siam work-camps depended, as Japanese Army latrines seem always to do, on the larvae of the bot-fly for disintegration of faeces. When the larvae were destroyed with cresol, the latrines filled up and stank. It went against the grain to use an
open trench every square millimetre of which was squirming with life, but it is evident that only a minute proportion of the larvae became adults.

Fly larvae, but not flies, are apparently essential to some sewage plants in Britain. This was stated in a Parliamentary reply which reached me (in the *British Medical Journal* of May 11, 1945) while I was writing these notes.

Thus the presence of millions of larvae and very few flies, as we had them in Siam, is not so remarkable as the other observations. Our freedom from dysenteries, when our meal inevitably attracted hundreds of green bot-flies from the latrines, struck us as a matter for surprise and gratitude.

This note would seem incomplete without mention of the mental changes of P.o.W. life. Psychoses were rare. Psychoneuroses and even psychosomatic diseases seemed to have been rarer than in civil life. There was a simplicity, a lack of inner conflict, which explains this; family relationships ceased to worry those who had been embroiled in them. Perhaps a further reason for lack of nervous illness was the frequency of parasitic and dietetic disease to take its place.

There can be little doubt that difficulties of adjustment between returned P.o.W. and their home folk will occur; but the resilience of the body in relation to health will as certainly be paralleled by that of the mind to its own health problems. We have shown we could take it, and we still can.