UNUSUAL ASPECTS AND THERAPY IN AMŒBIC DYSENTERY

BY
Lieutenant-Colonel L. R. S. MACFARLANE, O.B.E., M.B., B.Ch., D.P.H.
Royal Army Medical Corps.

This paper is an account of amœbic dysentery in P.S.O. War taken by the Japanese—British, Australian, American and Non-Asiatic Dutch East Indian Army personnel. The experiences of the writer were gained in Malaya and Thailand.

The amœbic dysentery reached epidemic form. How much was of epidemic severity and how much due to the attendant circumstances of Prisoner of War life will later be discussed. Amœbic dysentery was shown in all its forms and in forms little seen, if ever, in peacetime conditions. Its behaviour without drugs and in its really acute form may add a page to the literature on this subject which may be of use—certainly it will probably never again fall to any writer to see so many cases, with so little facilities for studying and combating the disease.

EPIDEMIOLOGY AND ÆTILOGY.

On the capitulation of Singapore all British and Australian Troops were sent to the former military area of Changi.

Changi, normally, had buildings for some 3,000 men with their wives and families. Up to 6,000 were in the area at its most crowded periods.

Into this area went some 50,000 prisoners. The hygiene and sanitary conditions in Changi prior to capitulation were excellent. Amongst the troops in Malaya the only important diseases were venereal disease and malaria, the latter only contracted in the outlying island forts and some camps up-country. Occasional cases of amœbic dysentery and more rarely still, bacillary dysentery, were seen, and very occasionally typhus, cerebrospinal meningitis and enteric and diphtheria.

Infectious diseases amongst the civil native population were limited to typhus, enteric, malaria and venereal disease. Amœbic dysentery and more rarely bacillary dysentery were not seen frequently.

Changi as an entirely military area was even more favoured and its sanitation was excellent. Local natives were few and the only malaria was at the coastal and outer forts. The anti-malarial situation in the cantonment and for some distance around was excellent. The water supply was good and the sewage disposal by septic tanks adequate.

Just before capitulation Changi had been very badly bombed and mortared, the pumping station crippled and later purposely destroyed and the greater majority of the buildings damaged.

It was to this semi-derelict area that a defeated and weary Army was sent, marching the fifteen miles from Singapore in the heat of the day. Those that could crowded into buildings, those that could not, into shacks, tents, etc.
Many carried the germs of disease from the campaign up-country, many from the appalling conditions of the last days of Singapore—where all civil services including burial of the dead had broken down and the water had been cut off.

These men finding no sanitation and no water supply, dispirited and caring little, performed amazing feats of sanitary indiscretion before our medical services which were allowed to continue functioning were able to take charge.

Proper bore-hole and deep trench latrines were dug, chlorination of water commenced and general sanitation was attempted by using sea water for flushing building W.C.s. But the damage was done.

Bacillary dysentery which was just commencing during the last days of Singapore broke out in real earnest. An attempt to start a dysentery hospital was in the main successful but the Japanese decided to centralize all sick in one small barrack area, thus disseminating the dysentery and infecting the rest of the new hospital. After appalling conditions for a few weeks in this hospital permission was given to form a separate dysentery wing. To this wing the writer went in charge of the laboratory. In a few weeks there were 1,200 dysentery beds all full.

The first amoebic case appeared soon after the writer opened this laboratory and the disease continued at a steady rate of approximately eleven fresh cases per week for eight months. In short, just what might have been expected in the conditions prevailing, but quite different from what subsequently occurred. The bacillary dysentery did not abate till after November.

This may be said to be the genesis of the amoebic dysentery outbreak. We now pass to the aggravating factors.

Whilst a few troops went to Singapore, Saigon and the Japanese Empire as working parties the great bulk remained in Changi for eight months. As a punishment for “taking up arms against the Japanese” the rations issued for the first six months of captivity apart from being Asiatic were reduced to an outrageous scale, each man getting: Rice 8 oz., tea 1/6 oz., sugar 2/3 oz., milk 1/2 oz., salt 1/6 oz., butter 1/6 oz., flour 2 oz., cheese 1/2 oz., vegetables 1 oz.—daily.

After a protest the ration was raised in ten days’ time to: Rice 16 oz., sugar 3/4 oz., tea 1/5 oz., salt 1/6 oz., fruit 1/3 oz., milk 19/24 oz., M. & V. 17/24 oz. (from our own captured stocks!), Ghee 5/24 oz., flour 1/2 oz., fresh meat 1/4 oz.

This ration scale lasted with minor alterations and variations till August 16 when a certain amount of Red Cross stores were allowed in via Diplomatic Repatriation Ships.

It will be seen that apart from its low caloric value this ration scale was lamentably low in fats, vit. A, B, and C—especially so in B.

Except for a small “amenity grant” of a few cents a day which started in June no pay was issued except to those working in Singapore until August 16. Then Officers were given 30 dollars a month. Hence except for those who had money and could buy “black market” food smuggled in at outrageous prices, no extra food could be obtained except coconuts. When the amenity grant started peanuts and an occasional egg could be added.
Men suffered considerably from the climate—an endurable one only in civilized conditions. Damp, muggy, and often with torrential rain. In spite of the presence of the sea, bathing was severely restricted after the first few days and even washing of clothes was infrequent. No fans or ice were available and little or no light at night, except in the hospital after September, when electric lights and some fans were mended and connected up to the Singapore power. Owing to the limited sanitary arrangements flies were a nuisance and with the cessation of malaria control, mosquitoes commenced their attentions.

The “Punishment Period” officially ended on August 16 with the departure of our General Officers to the Japanese Empire. Conditions were slightly ameliorated, pay as mentioned, light in the hospital, better entertainment facilities and the arrival of some Red Cross supplies. These were, however, only a prelude to the general exodus to the Thai-Burma Railway.

In addition there occurred during the first few days of September an incident which greatly reduced general health and increased the spread of disease, though paradoxically increasing morale. This incident now famous under the name of the “Selarang Tea Party” consisted in the herding into a space comprised by a Barrack Block for 800 men and a Parade Ground, of 17,000 men. This was the penalty for refusal to sign a parole not to try and escape. A further threat to move the 3,000 bedded hospital (including dysenteries) into this space also, led to the men having to give in—the Japanese however were forced to concede and put it on record that the parole was under duress—which of course rendered it non-binding—but they failed to appreciate this. Incidentally the punishment for attempted escape being death, generally bayoneting, there was little incentive to try it. Nevertheless holding out to this degree did a lot to restore morale and instilled into our enemies an unwilling respect.

The overcrowding, wetting and general strain of a tense situation did a lot of harm to general health besides encouraging the propagation of disease. The writer was present and can vouch that conditions baffled description—men crowding the buildings, roofs and Barrack Square—the centre of which had perforce to be dug into a vast open latrine.

During the period February to November with these factors in operation it is little wonder that disease had its inevitable way. Besides bacillary and amoebic dysentery already mentioned we had slowly increasing malaria and a few cases of scrub typhus. But the great fear of the physicians was soon to be realized—deficiency diseases. There is some disagreement as to the exact genesis of this and here it is discussed briefly.

**B. Deficiency.**—The very first cases noted were a selection of peripheral neuritis patients; at first they appeared to be beriberi, with absent knee-jerks, glove and stocking anaesthesia, hyperaesthesia of calves and paralysis. But as they appeared in men whose habits either admitted or suggested a high alcoholic intake, viz. planters, old soldiers who had risen to commissioned rank via the Quartermaster route, or remained Quartermaster Sergeants or Warrant Officers, and persons who were known to be heavy drinkers (as opposed to drunkards, in case the above statement should give rise to a wrong impression), it can be fairly
safely assumed that the cases were alcoholic neuritis, especially as a $B_1$ deficiency encourages the onset of this.

Beriberi itself followed soon after, viz. in about three and a half months from capitulation. The neuritic form was seen first and later the cardiac and wet type. This is to be distinguished from nutritional oedema which also made its appearance about this time. Beriberi was also seen as encephalopathy, death following definite mental signs, loss of memory, wanderings, etc., and with a post-mortem picture of Wernicke's sign (haemorrhages into the corpora mammillaria). Cases of retrobulbar neuritis were also seen.

$B_1$ Deficiency.—Pellagra next showed itself, with sore tongue, angular stomatitis, skin rashes aggravated by the sun, aching feet and in the worst cases, diarrhoea, mental symptoms and spastic paraplegia.

$B_2$ Deficiency.—Ariboflavinosis appeared at the same time, characterized by red, dry, itchy scrotum, later becoming weeping and raw. These were originally thought to be $B_1$ but nicotinic acid and its derivatives did not help, whereas riboflavine administration did, thereby fixing the condition as $B_2$ and $B_6$ deficiency.

$A$ Deficiency.—Although corneal ulceration and xerophthalmia commenced to appear at the end of the Changi period and also in Thailand, one must consider that it was present somewhat earlier as evidenced by the total lack of resistance to bacillary dysentery, and also to diphtheria which attacked the troops in August. Several cases of night blindness were noted in a mild form.

$C$ Deficiency.—Scurvy did not occur, nor did it occur in Thailand or Changi throughout the internment period—sufficient vegetables luckily were always available.

$D$ Deficiency.—Needless to say with the tropical sun this was absent.

$E$ Deficiency.—There was no means of determining this, but it is generally agreed that any impotence or sterility amongst returned prisoners of war is of psychiatric rather than dietetic origin.

Apart from avitaminosis and the dysenteries the remaining diseases in Changi were malaria, diphtheria and skin diseases. Malaria gradually increased pari passu with the cessation of malarial control outside the camp but it did not present a serious picture in the first nine months and quinine and atebrin was still plentiful. Diphtheria was seen occasionally in Changi before the Japanese occupation chiefly amongst children, but spasmodic cases commenced to appear amongst the troops immediately after capitulation. The disease reached epidemic form in August, 1942. Many severe cases were noted with many deaths. Antitoxin was hard to obtain and stocks scanty. In addition laboratory facilities were very limited and cultural examination could only be undertaken in the Australian section laboratory. Even then, proper Loeffler's medium was not available, an egg medium being substituted when eggs could be obtained. Consequently diagnosis was unreliable and many doubtful cases occurred. Skin diphtheria occurred, especially on the weeping scrotum of $B_2$ deficiency, the heat and sticky serious exudate making no doubt an excellent medium for Coryne bacteria. Skin disease was brought on inevitably by difficulty in washing and laudering and by vermin and sweating.
At the end of the month of October, 1942, came the great move of prisoners to Thailand. Conditions in Thailand were unknown and the nature of the work to be undertaken, if any, unknown. Food was heard to be plentiful in Thailand, which was true up to a point as will be seen later. Consequently it was not known exactly whom to send and whom not—unfortunately many men were sent totally unfit for the conditions. Indeed the whole prisoner population had undermined constitutions from the events laid down above. Also, owing to calls for more men, many post-amœbics without sufficient clearance tests were sent up; this may on first sight appear careless on the part of the medical authorities at Changi but it must be remembered that many thought this move merely a security one and that specially prepared camps and hospitals would be provided. Men continued going up-country until May, 1943, and others came from Java, Sumatra and other Dutch East Indian islands, including many Dutch and Dutch Eurasians.

The railway journey was appalling—five days in the train, 25 to 31 men being crowded into metal goods wagons without room to stand or lie down. Those that went by sea had an even worse time. On these journeys old dysenteries relapsed, sanitation was completely absent and conditions may be imagined.

Thus came to Thailand an already debilitated force after a most arduous journey, to find torrents of rain and the most primitive accommodation provided—Coolie huts of the worst type—no hospitals—no comfort of any description; the only bright spot was the food which was plentiful and nourishing.

The writer arrived as Pathologist with the Base Hospital Staff—a very limited laboratory staff and equipment accompanied him.

It was then learned that the work was the building of a railway from Bangkok to Burma (Moulmein) through virgin jungle.

The base hospital was to serve all the troops up the river along which the line was to be built—this hospital was only begun in November and was designed for 1,200 beds—if wooden platforms can be styled beds. Our captors evidently imagined our sick would not be more; it is difficult to understand how any organization could make such a gross underestimation. It is, however, now known that Dr. Saito responsible for these medical arrangements has been arraigned as a War Criminal.

As men arrived they were taken by barge, lorry and lastly on foot straight up-country—the nourishing food of the Thailand hinterland was not to be theirs for long.

History will tell of the sufferings of these men and the mortality, 1 in every 3½, speaks for itself. Conditions up the river were appalling; as this was pioneer work men had to march through jungle and camp in the open, often in torrential rain and in the winter months considerable cold. Floods, difficulty of transport, etc., prevented a steady supply of food and men were often for days on rice, pumpkin and water. The work was hard, especially to a debilitated population, and increasing numbers fell sick. Their places had to be taken at once as the railway had to go on. More men were constantly being sent up including Tamils from Malaya—even so the work lagged and even sick men had to work.
Evacuation of serious sick was terribly difficult—the worst cases generally dying on the way. Drugs up-country were almost non-existent and doctors and orderlies few and far between.

The Base Hospital soon became full and quasi-hospitals sprang up everywhere—some becoming bigger than the Base hospital itself. The Base hospital had most of the drugs sent up but even that was a totally inadequate amount. The other hospitals had even less. In addition sick got separated from their own "groups"—which were the Japanese system of accounting for prisoners—and were constantly being shifted from place to place to rejoin their respective "group" hospitals quite without consideration of their condition.

The railway was finished in August, 1943. Not till January, 1944, however, was a really big hospital started for the accommodation of the victims of the railway and it was not completed until July, 1944. This was the Allied P.o.W. Hospital, Nakom-Paton, near Bangkok and it cleared sick from Thailand and Burma. By the time it was fully functioning the original need for it had ceased, viz. the cause—the building of the railway.

Conditions were improving, the dead were dead and could not be brought to life, so that this hospital was more concerned with the relicts.

The diseases prevalent in Thailand were as follows:—

**Bacillary Dysentry** which was present at the commencement and which tended to burn itself out, very little being left after the first few months.

**Amoebic Dysentry** accounted for more deaths than any other disease, either alone or complicated. In addition to the ever-increasing case incidence of amoebic dysentery originating in Malaya and augmented by infection picked up from dirty water supply, dirty conditions, lack of sanitation, flies, native food purchased as extras, etc., etc., etc., there occurred a definite epidemic in at least one known locality, Konju, which spread throughout the entire prisoner population. It appears to have originated in this camp from an itinerant barge or barges run by Thais or Chinese, which sold food to the prisoners. The cases directly from this source were of a very virulent variety, out of all resemblance to generally accepted standards even of acute amoebic dysentery.

**Beriberi** appeared again, both the dry and wet types and also Shoshin, or acute cardiac beriberi.

**Nutritional Ðedema** was frequently confused with wet beriberi and the two of them with pellagra, all three being common.

**Pellagra** was characterized by typical glossitis and stomatitis, burning or aching feet, increased knee-jerks, œdema, diarrhœa and in some cases spastic paraplegia. Little dementia was seen.

**B, Deficiency** in the form of scrotal dermatitis was rife.

**Eye Diseases** were common, ulceration and night blindness from A deficiency, retrobulbar neuritis from B, deficiency and blurred vision from B, deficiency.

**Diphtheria**, both faucial, labial and cutaneous, was common.

**Tropical Ulcers** of the most appalling size were common, some 500 amputations being done. The aetiology of these is obscure but it is assumed that there must be a bacterial or virus causation aggravated by A deficiency.
Finally there was cholera—worst in June-September, 1943. Inoculation against this disease by the Japanese had been only half-heartedly carried out.

This somewhat lengthy preamble serves to show the epidemiology and aetiology of amoebic dysentery amongst P.O.W.s, together with the conditions complicating symptoms and treatment of the disease.

The infection would seem to have occurred in several ways and from several sources. Those who came into camp with the disease contracted in the campaign—a small number. Those who picked up the disease from the unhygienic conditions in Singapore at the capitulation and in the early days of Changi. Spread of the disease in P.O.W. life from those already with it. From infection in the dreadful conditions in Thailand, with the outbreak of a major mass infective epidemic at Konju. The presence of large numbers of Dutch Eurasian troops who are known to have a high carrier rate—about 25 per cent. Lastly one must not forget that a certain proportion of inhabitants of the U.K. and U.S.A. carry cysts. Yorks and Smith, working amongst Army recruits, give this figure from 5 to 19 per cent. Wenyon in Egypt found British soldiers had a carrier rate of 11 per cent. Pauson found New York food handlers to have a 4 to 5 per cent rate. Australia, however, has no cyst carriers per se.

Although most of these cyst carriers in European countries are reckoned to be harmless, Westphal considers that an infection of the bowel may light up these amoebae. With the bacillary dysentery epidemic in Changi this could certainly have happened. There is, however, still considerable doubt as to the varying degrees of pathogenicity of strains of Entamoeba histolytica.

Brumpt maintains there are virulent and non-virulent strains. Certainly later in this thesis will be shown that there are very wide responses in different patients to infection with Entamoeba histolytica.

Suffice it to say that the causes of this epidemic may be tabulated as:

2. Local infection due to bad sanitary conditions.
3. A mass food borne epidemic at Konju.
4. Possibly Westphal’s phenomenon.

This study was commenced first in Changi purely from the microscopic angle and mainly as a differential diagnosis from bacillary dysentery. In the Thailand Base Hospital at Kanchanburi (or Kanburi) the writer had charge both of the laboratory and the dysentery wards. Here the study of the disease at its worst was undertaken and treatment mainly without emetine attempted. Later, from June, 1944, onwards, the writer was in charge of the dysentery wing of the Allied Prisoner of War Hospital, Nakom-Paton, some 1,200 beds in the dysentery wing alone. Here the residue of the amoebic dysentery epidemic were housed, i.e. those who had not died or been cured and who had in the main passed into the chronic or carrier states. They were kept here more or less in sanctuary—but unfortunately near the end of the war the Japanese started cutting down the hospital beds and sending out carriers and chronics, making the final round up of amoebics at the end of the war extremely difficult.

Most of the cases were Europeans, Americans and Australians but some were
Eurasian Dutch in whom amebic dysentery is a common and not much feared disease and in whom the aetiology was somewhat different.

One hundred and forty cases treated with no emetine or with under 6 grains were studied in Kanchanburi as well as considerable numbers before the emetine gave out or when some appeared. In addition the writer had 300 cases at Nakom-Paton as well as the administrative charge of the block of 1,200.

Nothing in this thesis suggests any alterations to the preconceived ideas of the causal organism or its aetiology—the particular factors in this epidemic fitting in with what is already generally known. It is considered, however, highly possible that there is a difference in virulence between certain *Entamoeba histolytica*—a classification as yet not accepted.

**SYMPTOMATOLOGY.**

Several distinctive types are noted:—

1. The acute fulminating type.
2. Severe infections which in most cases died if emetine was not given.
3. Cases pushed over the summit to a chronic stage by various drugs, and which remained in this stage or ultimately reached the carrier state or in a few cases were cured.
4. Mild cases. Some of these could be cured without emetine.

**The Acute Fulminating Case.**—Although most authorities speak of acute and chronic forms, the existence of fulminating cases is rarely given prominence. Osler in the 13th Edition of his “Practice of Medicine” refers to “acute cases.” Manson-Bahr in “Tropical Diseases” states that acute cases are rare—he himself only having seen three which he would call acute. Some of the cases seen in Thailand would have needed immediate and full courses of emetine and its compounds if a cure was to have been effected and life saved and even then the issue might well have been in doubt.

The incubation period would appear to be short—ten days to a fortnight. The disease comes on briskly and in a few days the patient is passing many stools a day. The patient then goes downhill with considerable rapidity and dies within seven to ten days of onset. The last days are truly terrible as nothing touches the dysentery and in a few cases where a grain or so of emetine was obtained to alleviate suffering, no effect was noted at all. Stools reached 40 a day in numerous cases and the diagnostic point that amebic dysentery can be distinguished from bacillary by the frequency of stools *per diem* here fails completely. These facts, the violence of the symptoms and frequency of stools were noted by many others including Street and Vardy at Tarsao, Thailand, and are at variance with established ideas.

Great pain and tenesmus accompanied the motions and excoriation from acid stools added greatly to the patients misery. Intolerable pain was complained of from distension especially over the cæcum: In the last few days nausea followed by incessant vomiting occurred and the cases resembled cholera in many respects, except that they were conscious and very violent, often hurling themselves around the hut spurt orang facés and vomit in their agony—such
a description being in no way an exaggeration. The writer has never seen it described elsewhere.

Stools were typical, starting with faecal matter mixed with blood and passing on to the so-called “anchovy sauce” stool. These progressed to sloughs from the bowel and pure blood, the faecal matter naturally getting less and less. There was no resemblance to the odourless blood and clear mucus of bacillary dysentery even where the stools were 30 to 40 a day. The odour was typical and highly unpleasant. Finally only a very short time before death the patient became comatose and presented all the signs of acute dehydration—wrinkled fingers, dry tongue—shriveled, shrunken, mummified appearance, with well-marked hippocratic facies before expiring.

**Severe Type.**—This type may start with a severe bout of dysentery with slight fever, 20 to 30 stools a day, tenesmus and pain and may be thought to be of bacillary nature until stools are examined both naked-eye and microscopically. Far more commonly the case starts with slight diarrhoea or bouts of diarrhoea alternating with normal or constipated periods. The condition gradually gets worse or the attacks more frequent until the patient seeks medical advice or, alternatively, seeks medical advice as he has “seen blood.” Generally the stool picture was found to be that of soft diarrhoeic motions with blood and brown faecal fluid and semi-solid material intermixed. Sometimes the stools were solid or semi-solid but streaked with blood. In other cases the stools were just watery diarrhoea. In all cases the motions were offensive and the great majority acid causing excoriation and pain when being voided. Without emetine these cases with a few exceptions, which will be described later under treatment, proceeded on a downward path slowly but surely, some took months to finally die and experienced temporary amelioration during this downward path from time to time—a particularly distressing aspect from a psychological point of view, both for patient and clinician. Each attack, however, proved more severe, the stools increasing and the frank “anchovy sauce” type being seen. Later, as in the fulminating type, sloughs appeared. Considerable blood loss occurred in these cases, the patients becoming dehydrated, anaemic and emaciated. Irritating foods such as sauces, peanuts, vegetables and beans provoked severe bouts and pain was almost constant. This pain was increased by great distension of the bowel, with extreme discomfort and in many cases masses appeared in the cæcum due to intense thickening of the bowel from the amœbiasis.

With shortage of equipment and staff it was not possible to carry out blood-counts or blood-pressures on these patients except where definitely indicated.

Gastric symptoms were severe and distressing, vomiting, heart-burn, and anorexia being prominent.

Intense dry mouth and frequently hæmorrhoids added to the patients general misery.

Anuria and oliguria caused great pain and discomfort. A frantic desire to pass water led to nothing. Catheterization not only failed to find urine in the bladder but increased the pain and discomfort.

Hiccoughing was a late phase and always heralded death. The writer has
never known a case developing hiccoughs in the late stages recover. “Blackouts” and periods of unconsciousness supervened on these symptoms and the patients gradually sank into states of extreme pain, misery and wretchedness before finally dying.

The violent convulsions, copious vomiting and maniacal symptoms noted in the first type were, however, entirely absent.

In the cases observed at Kanchanburi, no equipment or facilities for post-mortems were available. No buildings could be used, the hospital being too crowded and cramped. Occasionally the surgeon performed a partial post-mortem on the operating table. This was out of the question for dysentery patients. However, at Nakom-Paton, such facilities were available and for a considerable time were used until the Japanese unaccountably suddenly forbade all autopsies.

The post-mortem appearances in these acute and severe cases were typical. The whole lower bowel could be a mass of undermined ulcers with only small areas of healthy tissue remaining, the appearance being black and necrotic with large sloughs. More often the condition was confined to three areas—caecum, splenic flexure and sigmoid-rectum with considerable healthy areas between. Around these affected areas great thickening of the bowel occurred, but the plum-coloured swelling distended bowel of bacillary dysentery was quite distinct and could never be confused. The writer saw many of the latter at Changi. The greenish colour and the pseudo-membrane of bacillary dysentery with the characteristic hollows and ridges crowned with exudate makes a distinct picture from these amoebic cases.

Many of the amoebic ulcers seen were deep, penetrating far into the musculature. The bowel, however, was not thinned out as in bacillary dysentery, rather it thickened in step with the development of ulceration.

Perforation of amoebic cases was extremely rare—the writer only seeing one clinical case.

Cases are quoted of spread of amoebic infection to the lower small intestine. This is very rare and the line of demarkation seen at autopsy is distinct and striking.

Both sigmoidoscopy and proctoscopy were impossible at Kanchanburi but the latter was carried out at Nakom-Paton. Severe ulceration could often be seen right down to the external sphincter. The ulceration was characteristic, the undermined edges of the craters being demonstrable. Many of the ulcers were pin-point and quite distinct from bacillary. Surrounding inflammation was often absent and exudate non-existent. Often the ulcers involved the internal sphincter. The painlessness of the ulcers to the touch per rectum was noteworthy; the pain of amoebic dysentery would appear rather to be due (1) to distension, (2) to the acidity of the stool. In bacillary dysentery this is reversed—apart from soreness due to repeated evacuations, the condition is relatively painless—the ulcers are, however, sore to the touch per rectum.

The Chronic Type.—Many cases of the preceding type as a result of medication with drugs available other than emetine managed to survive long enough to obtain a degree of resistance enabling them to pass into the chronic stage.
In this condition patients remained, some ultimately becoming symptom free of their own accord, others carrying on until emetine became available and others remaining stationary.

Of the last, two chief variants were noticed: (1) Those with a consistent diarrhoea from 3 to 5 motions a day—sometimes passing Entamoeba histolytica or cysts—more often not of considerable interest in these cases was the part played by secondary infection and by a pellagrous condition. When such drugs were available this could generally be easily decided by the effects of sulphaguanidine or nicotinic acid. Nevertheless a distinct number could be said to be entirely of amœbic origin. (2) The more typical chronic amœbic—days of comparative health with increase of bodily condition, amounting almost to obesity, broken at intervals with acute relapses with all the typical signs of pain, tenemus, “anchovy sauce” stools, the finding of active Entamoeba histolytica and/or cysts. These relapses as a whole tended in time to become more infrequent. The care of these cases was one of the greatest difficulty under the existing conditions. To keep them in hospital was difficult in view of their apparently robust appearance in the quiescent periods; it was almost impossible to convince the Japanese of their ailments; if one did manage to retain them because of “infectivity” hospital working parties believed of our captors invariably claimed them—furthermore, these working parties were liable overnight to be considered outside working parties and discharged forthwith. It was a noteworthy feature of the condition that the patient in a relapse lost all semblance of health and well-being almost within forty-eight hours.

The Mild Case.—A considerable number of these cases commenced and remained mild. There were cases in this series who were cured without emetine although in the majority these alternative drugs merely had an alleviating effect, and possibly prevented the disease becoming more serious. Furthermore, large numbers of these cases without any treatment at all appeared to remain in the same stage or quietly cleared up to reach the carrier condition.

They were a constant problem—to keep them in hospital was difficult, both by their looks and the apparent injustice to other patients needing the bed space or being needed for working parties; to send them out risked aggravating the disease and spreading the infection in the insanitary P.o.W. Camps.

The protection of these cases proved a long and weary struggle, by no means always successful.

The mild case ranged from occasional sharp bouts of typical amœbic dysentery to slight abdominal and gastric discomfort with occasional looseness of stools. Very often the diarrhoea was so slight and infrequent that the patient did not trouble to report it and the condition was found in a routine examination. Occasionally the only reason for reporting sick was “seeing blood.” Not infrequently the patient reported because of piles and the condition was only discovered then. Completely symptomless passers of Entamoeba histolytica vegetative forms was sometimes seen and when one descended to this level the differentiation between a “carrier” and a “case” became a matter of difficulty.
The following is an illustrative case:

Pte. E.—Argyle and Sutherland Highlanders—had several attacks of amoebic dysentery in 1940, 1941 in Malaya and again in Changi P.O.W. Camp, Spring, 1942. On 20.5.43 he was admitted to Kanchanburi Base Hospital. No emetine was available and various substitutes were tried. He had no diarrhoea and no symptoms after 7.6.43 yet active *Entamoeba histolytica* in large quantities were found in his stools on 5.7.43 and were still present on his transfer to another camp in November, 1943—still symptom free.

During the last year of captivity the cases in Nakom-Paton Hospital consisted to a very great extent of chronics and mild cases which had survived up to date with improvised treatment. Emetine was available in small quantities sufficient to preserve life. New cases of course occurred but sufficient emetine was available to keep them alive or in mild cases to cure, consequently the types observed were almost entirely 3 and 4.

*To be continued.*