CHAPTER 2

AMOEBIASIS

AMOEBIC infections were not numerically of great signficance in the Australian armed forces during the war, but by reason of the diagnostic and therapeutic problems involved, and the degree of disability they caused, they were important. The number of days of treatment needed in hospital in each case ranged from 40 to 60 or more, and relapses increased the total time necessary for recovery. There were 783 cases of amoebiasis recorded in the A.M.F. during the war, excluding the 8th Division. In some periods the numbers rose to a proportion of 10 per cent of the figures for bacillary dysentery, though the latter were certainly greater than hospital figures indicate.

The figures and rates were as follows:

	Year	Number	Rate per 1,000
Middle East . . .	1940	8	0.562
	1941	135	1.77
	1942	2	0.248
Australia . . .	1943	207	0.514
	1944	161	0.436
	1945	79	0.308
	1946	45	0.30
New Guinea . .	1943	34	0.436
	1944	54	0.773
	1945	13	0.319
N.E.I. and S.W.P.A. other than N.G. .	Apr.-Dec. 1945	45	1.103

It must be understood that the places of notification are not necessarily those of origin of the infection, owing to the periods of latency and the occurrence of relapses.

Amoebiasis has occasionally appeared in localised limited outbreaks in Australia, and cyst carriers have been recognised in the few surveys that have been made, just as in most countries in the world. During the war it was necessary to be on the watch for amoebic infections because of the actual or potential risk to members of the armed forces and the civil community. Amoebiasis was known to be endemic in the action zones of the Middle East, and in New Guinea and other Pacific islands, and had to be looked for not only during active service, but also after the return of men to civil life. Prisoners of war, both in Australia and overseas, were also potential sufferers from, and carriers of the infection.

For the purposes of systematic description the lesions caused by the *Entamoeba histolytica* may be divided into colonic, hepatic and pulmonary types. In interpretation of the varied clinical signs awareness is necessary of the pathological features of the disease.

CLINICAL FEATURES

(1) DYSENTERIC

The clinical types of amoebiasis seen were chiefly those associated with subacute or chronic abdominal symptoms. Acute amoebic dysentery was rarely seen except in the Japanese prison and working camps, where terrible conditions often predisposed to severe and uncontrolled infections. In these the necrotic features of the pathological lesions were sometimes exceptionally severe, causing massive sloughing associated with haemorrhage. Under the controlled conditions even of active warfare on other fronts such acute manifestations of the destructive effect of *Entamoeba histolytica* were not seen. As a rule toxaemia was of moderate grade only in the usual subacute form of the infection. The characteristic stools, containing necrotic material, blood and pus cells, were sometimes found immediately, suggestive of destructive lesions of the colon; but such declared types were not very common. An attack of bacillary dysentery sometimes awakened a latent amoebiasis and this caused considerable confusion unless the latter was thought of; an amoebic infection could steal on under cover of a bacterial diarrhoea. In the Middle East in the later periods men were seen with an established amoebiasis who had been treated earlier for "clinical dysentery". The subacute type tended to merge into the chronic; indeed chronicity is a feature of amoebic infections. This fact, taken with the moderate grade of toxaemia usually observed, is evidence of the possession of some degree of immunity by the human subject. This will be referred to again later.

Temperature was usually raised at some stage. A polymorph leucocytosis sometimes reaching 15,000 or 20,000 cells or even more was a common feature, helpful in diagnosis. Eosinophile counts gave no useful information, for eosinophilia is not a feature of amoebiasis. When eosinophilia was observed another cause was practically always found. Abdominal disturbance of some kind was invariable. Even in the hepatic metastases some history of looseness of the bowels, dyspepsia or merely discomfort was usually obtained. In chronic states the presence of persistent discomfort was found suggestive. Pain was usually present to some degree all over the abdomen; in the types most commonly seen it was localised in the right lower quadrant, and often also in the right loin. Proctitis was occasionally a troublesome symptom: its existence was even suggestive of amoebic infection. In a few instances the pathological basis of anal fissure proved to be amoebic.

(2) HEPATIC

Amoebiasis cannot be sharply divided into colonic and hepatic types. Hepatitis was diagnosed in numbers of patients who never suffered from liver abscess, and yet abscess has occurred in other patients whose colonic signs and symptoms have been indeterminate, at least without careful enquiry.

Enlargement of the liver was suggestive of an amoebic lesion, but enlargement of the spleen, though stated to be a useful sign, was not found

helpful, since there were so many other causes. In fact, in service experience splenomegaly when associated with amoebic hepatitis was only secondary to hepatomegaly. The enlarged liver was commonly tender, and pain could be elicited by compression of the lower ribs. Jolting in transport over rough roads frequently excited pain, this information being sometimes volunteered by patients in New Guinea. Dyspeptic symptoms were common, although they sometimes drew attention away from the true focus. A combination of dyspepsia with symptoms involving the lower part of the abdomen was suspicious, especially if associated with tenderness of the liver. A long latent period between the presumed time of infection and the onset of relevant symptoms was often misleading, though sometimes actually suggestive. The picture of a febrile disease occurring in an endemic area and mainly characterised by upper abdominal discomfort and tenderness should always suggest the possibility of amoebic hepatitis, though the diagnosis may often be in doubt for a time. Where abscess had formed in the liver the diagnosis was usually readily made, though it might be complicated by the extension of the process to the thorax.

(3) PULMONARY

Involvement of a lung was not very uncommon; it was always found in association with hepatitis and particularly with liver abscess. It seems to have been more often seen in the Middle East than in New Guinea or base areas in Australia. The pulmonary signs were most frequently found at the base of the right lung; they were usually impaired resonance, crepitations, pleural friction, altered breathing or evidence of fluid in the pleural cavity. Occasionally an hepatic abscess became by extension a pulmonary abscess or discharged through the bronchial tree. Affection of a lung could be confusing if the patient was seen when this was the chief presenting symptom. Indeed the provisional diagnosis was frequently incorrect. M. L. Edwards reported a case of amoebic pericarditis successfully treated, though the diagnosis was presumptive on the grounds of physical signs which suggested involvement of the pericardium.

No detailed account of the pathology of amoebiasis need be given here: this information is readily accessible. But it should be stated that experience has shown that amoebiasis is essentially a disease whose successful diagnosis and treatment depend on a ready knowledge of its pathology.

DIAGNOSIS

Diagnostic errors were not rare; even experience could not always avoid them, but certainly minimised them. A thoughtful review of many amoebic infections evokes the reflection that in a great number an earlier diagnosis could have been made on the application of greater insight and persistence. Surgical explorations do not always reveal the true disability. Appendicectomy has been performed a number of times and a substantial lesion in the appendix has not always been found. Coates found that the frequency of appendicitis among 1,500 men in the dysentery block of Nakom Paton

hospital exceeded that of 6,500 other men. The usual finding at operation was a necrotic process without lymph or adhesions. Pulmonary consolidations have been thought to be primary, so too have pleural exudates and intrathoracic abscesses. Secondary infections confused the issue at times: for example *Haemophilus influenzae* was grown from an empyema which was found afterwards to be secondary to an amoebic liver abscess. A diagnosis of neurosis has even been made in cases in which persistent complaint was made without any cause being determined.

Special technical instructions were issued concerning the diagnosis of amoebiasis. Particular stress was laid on the frequent difficulty in recognising the hepatic form, though most of the difficulty disappeared with a thorough knowledge of the disease. The correct pathological outlook was well summarised as follows:

"The first stage in infection of the liver consists of so-called amoebic hepatitis, and here the amoebae have been carried up via the portal vein and distributed through the liver as emboli derived from thrombosed vessels draining submucous ulcers in the large bowel. Enzymes secreted by the amoebae lead to local necrosis of tissues, while the fusion of adjacent foci gradually produces an abscess. Abscess formation is the end result of a progressive infection commencing with intestinal amoebiasis which may or may not have given rise to clinical dysentery."

The importance of amoebic hepatitis as an obscure cause of fever was stressed. A constant awareness of this possibility was found to be one safeguard to error by omission. One frequent source of error lay in the misconception that amoebic hepatitis is a fulminating disorder, it was commonly less spectacular. This was specially necessary having regard to the variability of the intestinal history. Nevertheless, exact information was essential concerning the state of the mucous membrane of the colon and the nature of the cellular contents of the excreta. This could be gained only by the use of the sigmoidoscope and the frequent and repeated microscopic examination of the stools. As with bacillary dysentery, a great forward step was made when sigmoidoscopy became a routine diagnostic procedure. Usually the mucous membrane in amoebic infections was found to look healthy except for the distinctive lesions themselves. These consisted of areas each ringed by a zone of hyperaemia or petechiae, in the centre being a granular surface, yellowish nodules or oval ulcers. The base of the ulcer appeared only after swabbing or gently scraping away inflammatory material and debris; and from this base vegetative forms of *Entamoeba histolytica* could often be demonstrated under the miscroscope. The swab method used for diagnosis of bacillary dysentery was not satisfactory for the discovery of amoebae. The larger granulomatous or ulcerated lesions were not often seen, except in severe infections, for example, in debilitated patients. Such infections were seen in prison camp hospitals such as Chungkai where 20 per cent of the patients with amoebic dysentery died during 1943. Here specific drugs were not supplied by the Japanese, and the amount of emetine available was very small. The lesions in the bowel were frequently not circumscribed, but formed florid inflammatory swellings. Local gangrene ensued, often spreading to cause peritonitis with or without perforation. Severe haemorrhages occurred,

often following the passage of huge foul sloughs. The course and management of these desperate lesions, fortunately not seen under ordinary conditions, formed a special problem, which will be described later.

Microscopic examination of the dejecta is of great value, provided that rash conclusions are not drawn from the failure to find amoebae of the vegetative or cystic type in one or two specimens. Major A. T. H. Marsden, who acquired an immense experience in the recognition of the entamoeba under these conditions, has pointed out that cysts are passed intermittently, and that their detection will depend upon the degree of this intermittency and the actual numbers excreted. Where less than 10,000 cysts a day are passed the detection of chronic dysentery by this means is not practicable except in a small minority. In 1,100 cases he found that six consecutive stools examinations disclosed one or more cysts in about half the infections. His own estimate was that in 20 per cent of cases cysts were found on one examination, 30 per cent on two, and 37 per cent on three. These figures confirm the necessity for the examination of material obtained by sigmoidoscopy. Bruce Hall from an analysis of some hundreds of cases considered that sigmoidoscopy yielded positive diagnostic information in 40 per cent, results confirmatory of history and clinical examination in 20 per cent and no positive help in 40 per cent. Under ordinary conditions the essential feature of amoebic lesions was seen to be a focus, especially in the subacute infections. The microscopic features of the stool, or rather of the cells and other material found in an appropriately selected flake of mucus, were not always distinctive. The exudate was not typically a "bacterial" one, but rather of the "indefinite" type. Red cells were found with considerable constancy and some degenerate tissue cells were commonly seen. Charcot-Leyden crystals have proved to be of some diagnostic value in arousing suspicion. It is unnecessary to repeat here descriptions of the entamoeba in its active trophozoite form or its vegetative cyst. Great care was taken that those upon whom devolved the duty of identifying this protozoon should have sufficient experience, and intensive training was given not only to pathologists but also to clinicians handling the disease. Errors in the recognition of this organism have occurred before by mistaking other protozoa or amoeboid macrophages for amoebae. The rapid movement of the blunt pseudopods, the delicate tracing of the nuclei and the inclusion of red cells should be distinctive. In the microscopic examination of suitably fresh material it was not found essential to use a warmed stage under the conditions usually prevailing in the tropical areas where most of the work was done. The finding of other protozoa or of worms or their ova in the stool gives rise in itself to a suspicion that the entamoeba may also be there. The presence of *Iodamoeba butschlii*, though itself usually considered non-pathogenic, may be a pointer. *Strongyloides* infestation and entamoebic infection have also been known to co-exist in some tropical areas. Despite all endeavours, the microscopic diagnosis could not always be made, and there remained the method of therapeutic trial, of which more will be said later.

DIAGNOSIS OF LIVER ABSCESS

Once the end result of liver abscess had been reached, special regional methods were of value in diagnosis, which depended on demonstration of structural changes rather than of the causal organism. The local signs were all the more convicing when they occurred after a period of remittent fever and sweating with no other obvious causes. Abscess in the right lobe usually gave rise to pain referred to the right shoulder, and to signs of diminished air entry at the base of the right lung. Where the abscess involved the lower portion of the lobe localised tenderness, particularly on movement or pressure, was more common. Owing to this the patient often preferred to lie on the right side. Abscess of the left lobe often caused epigastric signs, which could be confusing. Radiological methods were of value in diagnosis. The signs most commonly found were diminished movement or "splinting" of the right or occasionally the left cupola of the diaphragm, or distortion of a cupola, or increased density of the liver shadow. Signs of consolidation or collapse of a lung or of pleural effusion were also suggestive.

EPIDEMIOLOGY

The investigation and treatment of individual patients was mainly a matter of penetrating investigation and deduction, but the epidemiological problems were often even more obscure. In certain areas the higher incidences were associated with poor hygiene; the experiences of the 8th Division in captivity bore witness to this. An extreme example may be quoted from Thailand: during one year out of 11,347 men admitted to hospital in Chungkai, 1,309 had amoebic dysentery. It was of interest here that the figures for bacillary dysentery were only half those for amoebic. The conditions were of course exceptional. On other fronts no important outbreaks of amoebic dysentery occurred, though rates of incidence varied. Spread of amoebiasis was by the means familiar in other intestinal infections. Where there was a risk of bacillary dysentery amoebic infection might be a concomitant hazard. Water has been recognised as an important carrier of amoebae. Chlorination of water within potable limits would not kill the vegetative forms of *Entamoeba histolytica,* but filtration effectively removed them. The army mollinite filter was estimated to be as adequate for this purpose as some 6 feet of soil. The trophozoites were much more vulnerable, but safe methods of sanitation were essential, though cysts could not survive drying. In parts of New Guinea the sanitary problem of disposal was complicated by the proximity of the water table to the surface of the ground. In the Moresby area at one time some concern was felt about the complete safety of chlorination of the water of the Laloki River, which ran some risk of contamination by the presence of a temporary dysentery hospital on its banks some miles upstream. Insects such as flies could act as vectors, and contaminated food was also a real risk. It was unlikely that food soiled by carriers could maintain an epidemic, but it could initiate an outbreak. The site of origin of an infection was not always easy to determine. A man seen in New Guinea with amoebiasis might have been infected in Syria. Of 50 patients seen in

a hospital in New Guinea 14 were believed to have contracted the infection in the Middle East, 22 in New Guinea, and the remainder had infection of doubtful origin. A definite crop of infections from Syria, for example, was seen after the return of troops to Australia.

In 1943-1944 the incidence of amoebic infections was watched closely in New Guinea. By this time most relapses from infections originally acquired in the Middle East would have declared themselves, and since diagnostic accuracy was then of higher degree, clinical infections seen among troops in New Guinea would presumably have arisen there. Though it was not expected that the numbers would be great, the troops strengths were considerable, ranging from 60,000 to 100,000. As shown in the figures quoted above the rate was not unduly high. The average of amoebic infections among all forms of dysentery in New Guinea over this period was recorded as 3.5 per cent, but at a general hospital in Lae out of 1,400 admissions to the dysentery ward over a period of twelve months the proportion of amoebic infections was 8.7 per cent. Subsequent experience has shown that the figure of 3.5 per cent is unduly low: the higher estimate is probably nearer the truth, even allowing for the known association of bacillary and amoebic dysentery.

The problem of the carriers of amoebic cysts caused earnest thought on several occasions. Its importance depends on the possibility of an outbreak of amoebic dysentery in a community. A survey made by Lancaster (quoted by Andrew) of 1,000 Australian troops over a period of five months is of interest. Of these men 5.5 per cent had cysts of *Entamoeba histolytica* demonstrable in the stools. This figure is within the apparent norm found by surveys in most parts of the world not specially subject to amoebic infections. Dissection of the results showed that the percentages were highest among those who had been exposed to infection in the Middle East, reaching 10 per cent; the figure for those exposed to possible infection in New Guinea alone was 3.7 per cent and in Australia alone 3.2 per cent. Other earlier Australian figures as presented by Heydon suggest a corrected average rate of 8 per cent in Townsville, and another later series in soldiers never out of Australia gave 2 per cent.

A. J. Bearup, J. J. Lawrence and G. A. M. Heydon also surveyed soldiers in the General Details Depot in Sydney from December 1941 to November 1943, to determine what percentage showed evidence of being cyst carriers or of active infection. Multiple examinations were not always possible; the best results were obtained by flotation methods, with the use of zinc sulphate. They only found *Entamoeba histolytica* in 1.3 per cent of 2,365 men who had never been out of Australia. Their series included 210 men who had served outside Australia, but no histolytic amoebae were found among these. The results of these surveys, though variable in their figures, suggest that the cyst carrier rate in Australia does not exceed the usual figures for other parts of the world.

The internment of Italian prisoners of war in Australia brought up the cyst carrier as a problem of public health. In 1941 prisoners were brought to Australia in whom there was good reason for suspecting amoebic

infection. Numbers of such prisoners had suffered from amoebic dysentery in the Middle East, their previous hygiene had been poor and the conditions of the compounds in which they had been confined immediately after surrender had been very bad. Surveys were made and a considerable number were found to be harbouring cysts. At Tatura camp a single examination showed a cyst rate of 6 per cent, a figure consistent with civilian average. At Murchison 161 suspects showed a rate of 11 per cent, whereas others showed only a rate of 5 per cent. At Cowra, in New South Wales, a special sewered compound was established for carriers of amoebic cysts, and here 350 prisoners were kept under very good conditions. After several months re-examination revealed only 122 as persistent carriers, but no clinical affections had occurred among them. At Loveday and other camps a certain number of clinical attacks occurred, but yielded to treatment. At the end of 1941 and early in 1942 examinations were made of Japanese internees at an army mobile bacteriological laboratory, and the Walter and Eliza Hall Research Institute in Melbourne. Only 3 per cent of 228 were found to harbour cysts. It is obvious that such surveys can yield only approximate results, but the figures are sufficiently consistent to be of some value.

The questions raised here were the indications for treatment, if any, and the possibility of risk to the civil community. Excepting where clinical symptoms appeared no treatment was adopted: in any case all anti-amoebic drugs were then so scarce in Australia that it was difficult to secure enough to treat men with overt disease. Under conditions of good nutrition and strict hygiene the proportion of carriers diminished. Later the Cabinet decided to allow numbers of these men to work in rural districts and the question of risk was considered. Though medical opinion was not completely unanimous, the D.G.M.S. of the Army thought that any danger was unsubstantial, and the men worked well and remained in good health and without any known hazard to others. After the end of the war a check of prisoners of war from prison camps in Italy showed that 60 to 70 per cent were passing cysts, but none had any symptoms. Yet the 2/14th Australian General Hospital, sent to Malaya to receive 8th Division prisoners of war, surveyed 1,297 men, including those with clinical diarrhoea, and found only 3 per cent to have cysts. Seventy-three cases of clinical infection, most of them severe, were confirmed at the same time. It must be admitted that unless comparable conditions of investigation exist in such surveys, no statistical conclusions can be drawn.

Under static military conditions instances occurred of infection apparently arising from within. Thus Kay and Johnston reported a series of seven cases of amoebiasis in the Northern Territory: of these men five had never been out of Australia. They presented symptoms of persistent diarrhoea with vague abdominal pain and three came from one unit. A series of fourteen cases was reported from the 2/2nd A.G.H. at Atherton in North Queensland. Most of these were thought to have arisen in the Middle East, but some were probably New Guinea infections. In a few the source was thought to be North Queensland. On the other hand Aus-

tralian experience in Syria indicated that while amoebiasis was a well recognised risk there, it did not appear to be common as local reports suggested. In New Caledonia also it was stated to be common, yet Captain Patrick, with an Independent Company in 1942, reported that it had not been encountered in Australian troops, nor was it found by a special American board of investigation. In Borneo too amoebic dysentery was said to be common, even more common in places than bacillary dysentery. This was contrary to Australian experience at the time, though evidence of the disease was found and confirmed in later observations of men in these campaigns. Here the military conditions, except for brief periods, were relatively favourable and the special measures then possible against fly infestation probably lowered the risk.

It is difficult to reconcile the conflicting aspects of this disease, but the overall experience of the Australian medical services has confirmed that it is a condition apparently widely, though not densely, spread in all communities. It does not readily assume acute epidemic form, but may do so under conditions of privation, and poor nutrition and hygiene. Small outbreaks may occur from time to time, even in families. The answer at present lies in adequate hygiene, thorough treatment of patients, and a high standard of cure.

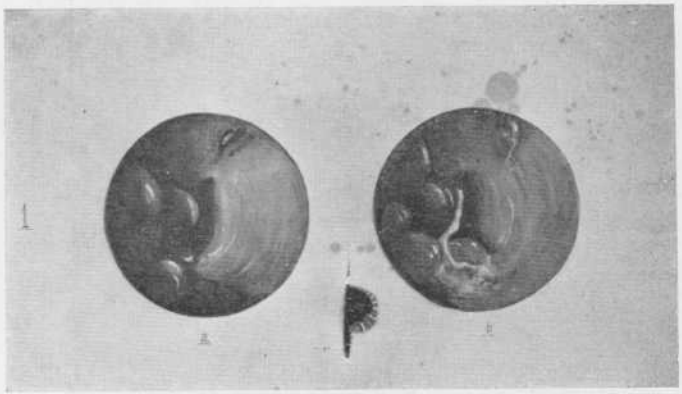
IMMUNITY

Consideration of the epidemiology of the disease must also take into account the degree of immunity established by clinical attacks, as well as such natural resistance as most people seem to possess under ordinary conditions. The apparent harmlessness of cysts in the intestine of healthy people is witness to the existence of some natural barrier to infection, which can be broken down by malnutrition or intercurrent disease, particularly other dysenteric disease. When clinical infection occurs the amoebae may live for periods of years within the body, yet even with specific treatment the disease is notorious for relapsing. In a vast experience in Malaya, Burma and Thailand Australian medical officers were able to demonstrate the gradual acquirement of some degree of immunity once their patients, even though malnourished, had surmounted the initial perils of severe infection. In Nakom Paton 1,346 men out of 1,500 men were found to be carriers of active amoebiasis. A few men suffered from liver abscess; in others massive and florid infections of the colon were present, some requiring surgical drainage. In spite of the lack of specific drugs, numbers of these men eventually recovered, some of them after being desperately ill. At Changi also spontaneous improvement was noted in a number of patients. No fewer than 120 new cases were diagnosed in one period of six months, and though few facilities for treatment existed, some men improved in spite of the handicap of substandard nutrition.

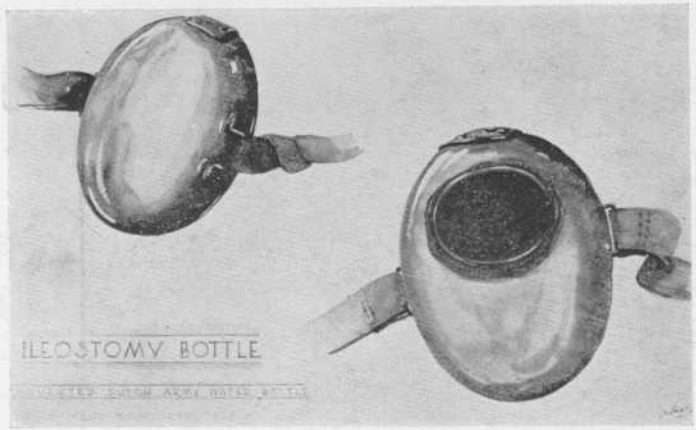
TREATMENT

Treatment has had its disappointments, as well as its dramatic successes. Throughout the war on all fronts the Australian medical services

had to exert the strictest economy of drugs. Emetine and in particular emetine-bismuth-iodide were scarce; the latter was sometimes and in some places unobtainable. The standard laid down originally for the A.I.F in the Middle East was followed as far as possible. A total of 8 to 10 grains of emetine hydrochloride was given, preferably by intramuscular injection, in daily doses of 1 grain in 1 c.cm. of water. A light diet was usually allowed. In acute and subacute infections the immediate effect was most satisfactory. Chronic infections were the least satisfactory to treat. The advice given in the official instructions was usually followed that the patient should be kept in bed during the course and for some days after on account of the possible toxic effects of emetine on the myocardium. No evidence of cardiac disturbance was ever seen, even when such strict precautions were relaxed, and the Australian experience suggests that the importance of this caution has been exaggerated. Emetine-bismuth-iodide was officially advised for use in chronic or relapsing infections and for symptomless carriers who were passing cysts, but it was not used for the latter purpose, owing to its scarcity and the dubious need for it in the absence of symptoms. As a routine the best method was found to be a course of emetine, followed by a course of up to 30 grains of emetine-bismuth-iodide. The usual dosage was 1 grain for 3 successive days and then 3 grains daily for 7 days. An interval of three weeks was considered advisable to allow time for the elimination of the accumulated emetine, but no harm was found on shortening this interval. During convalescence carbarsone 0.25 gramme twice a day for ten days was advised. Later stovarsol was used in place of carbarsone, and no objection was found to giving this drug during the course of emetine-bismuth-iodide or of emetine. Occasional trouble was found with nausea or vomiting following the administration of the latter drug, but by always giving it at night after a sedative, little difficulty was experienced. In general it was found better not to excite any apprehension in the patient and to let him take the dose without special comment. Yatren (Quinoxyl) 2 to 2½ per cent solution was found useful as a retention enema of about 6 ounces, when obtainable, but supplies were restricted. Double iodine compounds such as "Diodoquin" were not readily available, and experience with them in the services was insufficient for opinion as to their value. Where circumstances allowed thorough treatment to be given the results were very good, and relapses were few and much less frequent than after a partial treatment. Emetine alone was sometimes apparently sufficient, but remembering the pathology of the disease the value of emetine-bismuth-iodide with its local action was evident. The value of early and adequate treatment was borne out in Australian experience. There was no advantage in giving repeated courses of emetine where the patient was passing cysts and had symptoms of relapsing or chronic disease. However, when the patient presented signs of acute recrudescence, especially with diarrhoea, a brief series of emetine injections could be used with good results to control acute symptoms, followed later by the other drugs. Amoebic involvement of the liver and lung required the normal full course even if no colonic symptoms

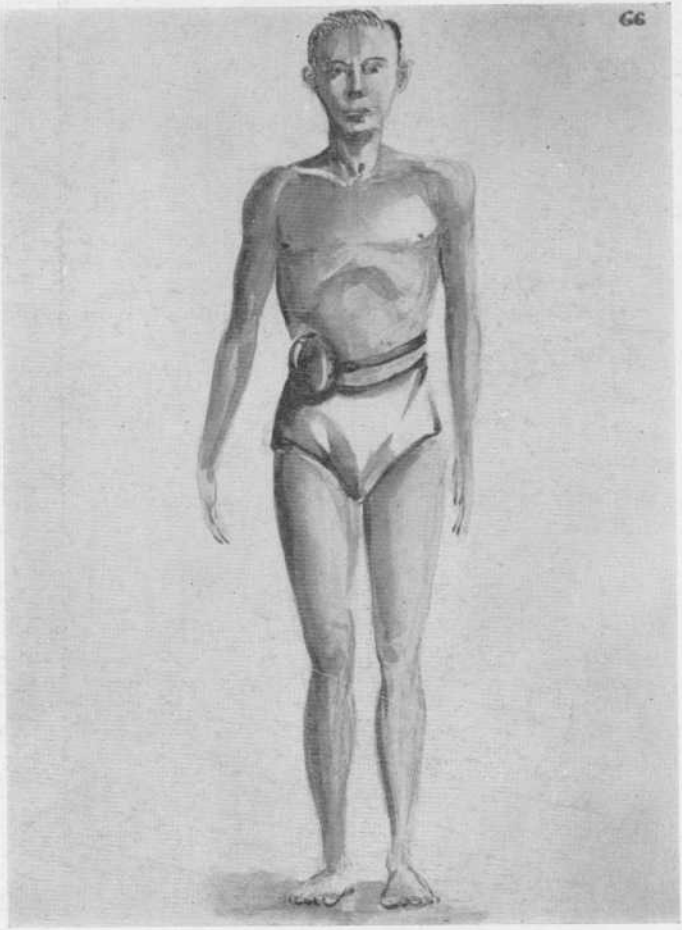


Proctoscopy of amoebic ulcers—Nakom Paton.



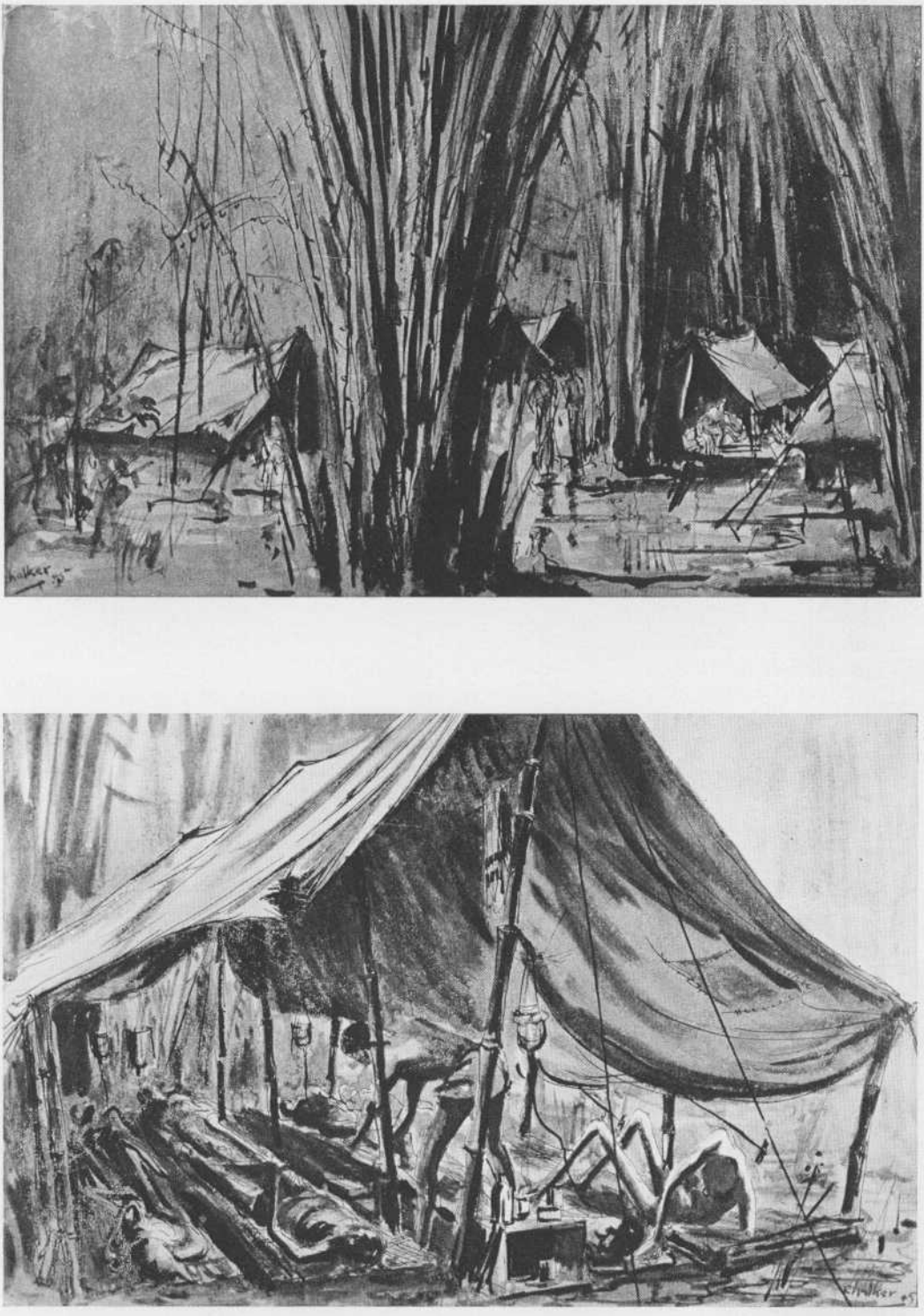
(Drawn by J. Chalker)

Ileostomy bottle made from Dutch water bottle.



(Drawn by J. Chalker)

Patient wearing ileostomy bottle at Nakom Paton.



(*Drawn by J. Chalker*)

Cholera Camp, Hintok.

were present. The specific action of emetine was especially remarked in the more acute and the hepatic forms of the disease. It was gratifying to see temperature fall rapidly, and pain, tenderness, and enlargement of the liver subside after a few injections. Therapeutic trial has been mentioned. The greater the experience of a physician with amoebiasis, the sooner will he try the effect of emetine in obscure illnesses, realising the uncertainty of microscopic diagnosis. Five grains of emetine or less will soon prove whether the diagnostic nail has been hit on the head. Therapeutic response alone is the touchstone, for the drug does not increase the ease of finding amoebae; it is a resolving and not a provocative agent.

Special technical instructions were issued for aspiration of liver abscesses. For the most usual site the upper part of the right lobe, the eighth or ninth intercostal space was advised, in the anterior axillary line, the needle being directed inwards, backwards and a little upwards. For abscesses nearer the lower edge of the liver a combination of laparotomy and needling was safer. Drainage was advised only if secondary infection had occurred. Though amoebic hepatitis was occasionally seen, frank abscess was very uncommon in service experience.

The advanced forms of disease seen under conditions of captivity under the Japanese have been mentioned. Lieut-Colonel Coates had difficulty in persuading the Japanese that amoebic dysentery existed in the camps of Burma and Thailand, but the evidence of deep penetrating ulcers seen *post mortem* eventually produced some effect, and a microscope was obtained to confirm the diagnosis. In Kanchanburi Hospital also the demonstration of vegetative amoebae to a Japanese medical officer helped to get supplies of emetine and Yatren. Emetine was made by Captain Van Boxtel from extract of ipecacuanha, and used with immediate relief in the massive colonic infections, with their devastating haemorrhage and pain. But the appalling prevalence and severity of the disease in these camps made it necessary to seek more radical means of relief, and surgery was undertaken. The chronic infections had lasted in some cases for several years, in spite of drugs, and such patients were greatly relieved by ileostomy which allowed their bowels to rest. Dutch water bottles were used to make improvised ileostomy bottles. Great relief was given by this procedure. Even early infections profited by surgical drainage, where rapid deterioration was taking place, and here appendicostomy or caecostomy enabled irrigation to be used without discomfort. Colostomy carried the risk of causing invasion of the abdominal wall. Further details are given in the section on abdominal surgery. These surgical measures, used under unusual conditions proved life saving in numbers of cases. They also illustrated how, given an opportunity, the body was still able to build up some immunity to the disease. Even some of the extensive and severe infections yielded to later treatment, and were apparently cured. Repatriation medical officers have advised that in all cases of amoebic infection periodic reviews should be made in which radiological, pathological and sigmoidoscopic examination are carried out. Where indications of residual infection are found a further course of treatment will then be given.

STANDARD OF CURE

As a rule patients required some care in hospital and convalescent depots after treatment was finished, chiefly to make good a loss of weight and vigour which was usually considerable. During this period abdominal symptoms should be absent; macroscopic and microscopic examination of the faeces was always carried out. Finally sigmoidoscopy was performed as a final check. Where doubt existed and opportunity offered check examinations were carried out after an interval of some months.

IODAMOEBIASIS

One instance was seen of what appeared to be general infection with *Iodamoeba butschlii*. Dr E. H. Derrick found in a Japanese prisoner of war ulcerated lesions in the colon resembling those of the *Entamoeba histolytica*. In the stomach colon, lungs and brain were also found amoebae which have been considered identical with *Iodamoeba* by Dr C. M. Wenyon. No report on the stool was available, but this evidence indicates that this protozoon can be pathogenic. It is well known as an occasional inhabitant of the colon. The possibilities of *Iodamoeba butschlii* as a pathogen have perhaps been overlooked. Major H. O. Lancaster found that nineteen of 849 soldiers who had served in the Middle East or New Guinea harboured cysts of this amoeba, though more were found in 155 men who had not left Australia. It is known to be an occasional inhabitant of the colon in apparently healthy people. Andrew found *Iodamoeba* in 4 persons who had abdominal symptoms. The *Iodamoeba* has been found to be very susceptible to emetine, but clinical experience is too small to decide the importance of this protozoan as a cause of dysentery. Evidence suggests that where such infection is suspected emetine-bismuth-iodide will be of value.

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