AMOEBIC DYSENTERY IN IRAQ.

THESIS

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by

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Introduction.

Dysentery, though a very ancient disease, is today still one of those conditions which must occupy our attention seriously, despite the greater knowledge which we have acquired as a result of the War.

During the War and since, it has become evident that further close study is necessary, if we are to differentiate still more between the various kinds of dysentery, and to prevent the occurrence of some forms of the condition in countries with a temperate climate. Whereas before the War, for example, amoebic dysentery was comparatively rare in England, it is reasonable to suppose that many more cases might result in the future, hence the great and growing importance of our keeping it in mind always, and not allowing ourselves to be aware of its great prevalence only in big campaigns.

It is not enough to be satisfied with the accepted term "dysentery" as meaning diarrhoea with the passage of blood and mucus from the bowels accompanied by pain and tenesmus. We must search for the real cause, and in this we are aided by our experiences during the War and the large volume of literature that has been published on the subject recently. The medical profession and the public generally are
greatly indebted to such careful investigators as Castellani and Chalmers, Dale and Dobell, Wenyon and O'Connor, and Rogers and Manson, not forgetting the scores of medical officers who served in the Near East and Mediterranean littoral during the War and after.

Dysentery was known in the time of Hippocrates, and now a considerable amount of interesting literature is available to show the undoubted prevalence and seriousness of the disease in most of the great campaigns of the past and in more modern times. Lyons in 1856 described the terrible ravages of the disease in the Crimean War.

Strangely enough, dysentery does not appear to have been a cause for great anxiety during the Russo-Japanese War. In the South African War many deaths resulted from dysentery, and there is no doubt that very arduous campaigns in countries where many hardships were experienced were responsible for the big increase of the disease and the numbers of deaths. Even in the Great War the severity of the disease and the death toll differed in the near East according to the theatre of operations. In Egypt it was always a source of anxiety, as also in Gallipoli and India, but in Mesopotamia, where to start with, the conditions under which our troops served were worse than in any other Eastern theatre of war, dysentery played havoc with our soldiers causing
many deaths, and rendering thousands useless and inefficient.

It must come forcibly to the minds of many who have had experience of dysentery in the tropics that in spite of preventive measures, cases of amoebic dysentery are liable to occur with greater frequency in this country owing to the many thousands of soldiers who returned from the East after the War and who are possible "carriers" today. Hence there is the necessity for regarding cases of dysentery in England as not necessarily being epidemic or bacillary in nature, but quite probably protozoal in origin. From what one has seen of the protozoal type of dysentery in Iraq since the War, it leads one to hold the above view, because a fair number of men of the Royal Air Force in that country have been invalided to the United Kingdom recently after having suffered recurrences of the disease, and many of these men are possibly "carriers".

Iraq is a country where, despite the great care in preventive measures taken during the last five or six years, dysentery is still rather a troublesome disease and one which renders the British soldier liable to attacks. The incidence of the disease is not high compared with the War period, but it is certainly a cause of inefficiency and non-effectiveness among the personnel of the Royal Air Force, and as such has to be viewed with
all seriousness.

The country is composed for the most part of a huge flat delta of sun-baked desert lying between the Euphrates river in the west and the river Tigris in the east. The Tigris and Euphrates join together to form the Shatt-el-Arab which flows south into the Persian Gulf. On the Shatt-el-Arab is Basra, the port of Iraq and the southermmost city, while Baghdad the capital and largest city is situated on the Tigris approximately in the middle of the country. Mosul and Kirkuk are big towns on the northern frontier of Iraq and it is in these northern latitudes that the only real mountainous country prevails. Palm belts stretch along the rivers on either side, and it is here that the population is mainly distributed. Among the native population dysentery is always prevalent, though in the northern parts of Kurdistan in the hill villages it seems to be definitely less. Among the Royal Air Force personnel, however, cases appear as frequently in Mosul and Kirkuk as in Basra, while in Baghdad which has by far the biggest number of troops situated in and near it, dysentery plays a big part in the sickness of the Royal Air Force and British Army.

The climate of Iraq is hot and dry. There is no heavy rainy season as in India, though wet days
in March and April are fairly numerous. The extremes of temperature are very great, and during the hot weather months of May, June, July, August and September, the temperature may reach as high as 120° Fahr. or higher, while in the winter months, especially December, January and February, it may even be below freezing point. No rain falls in the months of May, June, July and August, but hot dry winds are frequent, often bringing sandstorms in their train. The "Shamal" or northwest wind is dry but bearable, while the "date-ripener" of September is moist and hot and most trying to Europeans. In Basra where the temperature is higher, often 125° or over, the climate is more humid, but Baghdad and Mosul, though slightly cooler, experience their quota of hot, blistering days.

In this thesis it is proposed to give a brief resume of our present views of amoebic dysentery based on one's experience in Iraq in 1924 and 1925, when one had an opportunity of studying the condition both in the laboratory and in the wards of the Royal Air Force hospital in Hinaidi, and also on out-post stations where it was studied clinically from the onset of symptoms. It may be stated here that amoebic dysentery is much more prevalent in Iraq today than bacillary or epidemic dysentery. Very
few cases of bacillary dysentery are seen, so much so that it has become a habit in the service hospitals there to search first for the entamoeba histolytica before going on to further investigations for other causes.
Classification of the Various forms of Dysentery.

Until the last quarter of the 19th century no satisfactory differentiation was made between the different forms of dysentery. The commencement of our knowledge took place when Loesch discovered an Amoeba in a case of dysentery. This was in 1875 when he published his discovery. Then a few years later Shiga discovered the dysentery bacillus. This was a very great start.

Beside these two main varieties of dysentery, other forms have now been recognised, and probably as time goes on their number will be increased.

At the present time authorities are agreed in classifying the dysenteries into two main groups:—

A. Protozoal dysenteries.
B. Bacillary dysentery.

Under the protozoal group may be considered "Flagellate dysentery" said by some people to be caused by the flagellates Lamblia intestinalis, Trichomonas intestinalis and Tetramitus mesnili, while the dysentery attributed by others to the Balantidium coli may be mentioned here as well. But of all the protozoa we must at present place entamoeba histolytica as first in the causation of a definite and undoubted dysentery.

In addition, dysenteric symptoms have been
recorded in connection with Kala Azar (Leishmaniasis) and Malaria, while some observers have held a spirochaete responsible for a type of dysentery. Then too, Ankylostomiasis and Ascaris infections have been known to produce the symptoms, though we are not yet in a position to state that these symptoms are those of true dysentery. A classification may be tabulated in the following manner, when the main forms of dysentery with their known causes will be shown, with other sources which can only be said to be "possible".

1. Bacillary dysentery. Due to:-
   (a) B. dysenteriae Shiga.
   (b) B. dysenteriae Flexner.
   (c) B. Morgan No.1.
   (d) B. Eyre No.9.

2. Protozoal dysentery. Due to:-
   (a) Sarcodina - Entamoeba histolytica.
   (b) Flagellata - Lambia intestinalis.
       Trichomonas intestinalis.
       Tetramitus mesnili.
       Leishman-Donovan body.
   (c) Infusoria - Balantidium coli.

3. Other causes of dysenteric symptoms:-
   (a) Trematode worm - Schistosomum mansoni.
   (b) Nematoda - Ankylostoma duodenalis.
       Ascaris lumbricoides.
   (c) The malignant malarial parasite
       P. falciparum.
   (d) Spirochaete - Spironema eurygyratum Werner.

Amoebic Dysentery.
Iambi described the presence of amoebae in the intestines in 1859, and Loesch added another discovery in 1875 by demonstrating the presence of amoebae in the faeces of a patient who was suffering from dysentery. The next step was taken by Kartulis who at the instigation of Koch investigated the subject in Egypt, and produced evidence in 1891 on the causal relationship between Amoebae and dysentery by means of experiments on cats. But in 1887 he had shown the presence of Amoebae in a liver abscess. Not long after Kartulis had done this work, Osler discovered Amoebae in a liver abscess also, and Councilman and Lafleur, his pupils, carried on the investigation of the relationship between Amoebae and dysentery, thereby confirming Kartulis's discovery.

The next great discovery was in 1903 by Schaudinn who carried out a final differentiation between the Entamoeba coli and the Entamoeba histolytica. Vierack in 1907 described the Entamoeba tetragena, but recent observers such as Musgrave and Clegg, Walker and Sollard in the Philippines, Darling in the Panama, and Wenyon at the London School of Tropical Medicine have definitely established the pathogenicity of the Entamoeba histolytica.
or Entamoeba dysenterica, and have confirmed the view that the Entamoeba of Schaudinn and of Vierück are merely phases in the development of one parasite.

Description of the Parasite. The Vegetative E. histolytica.

It is necessary when searching the stools of a suspected case of amoebic dysentery to bear in mind that the presence of the non-pathogenic entamoeba coli is very apt to confuse our object. With practice, however, it is not difficult to distinguish between the two parasites and when the stool is fresh and the amoebae of dysentery active it simplifies the task considerably.

The E. histolytica varies in size, and according to Dobell measures from 18 μ up to 40 μ in diameter, but this is of small importance since it may vary widely in different stages of its development. Also its shape is of little consequence, because when it is quiescent it is round and when in motion it is very irregular in outline. This irregularity is due to the projections called pseudopodia which effect the amoeba's movement.

The nucleus in the unstained parasite is very hard to see distinctly and it is usually situated at the junction of the endoplasm and ectoplasm. In the E. coli it is more often centrally placed.
With the stained preparation the nucleus shows a delicate achromatic membrane studded with small chromatic granules. In the centre is a small spherical karyosome 0.5µ in diameter surrounded by an achromatic zone. Between the karyosome and the nuclear ring is a linin-network.

Vacuoles are occasionally seen and in the E. histolytica they are non-contractile.

The protoplasm is clearly differentiated into a granular endoplasm and a clear ectoplasm, and this difference is much more marked when the parasite is seen in motion. The pseudopodia show no granular appearance at any time. On occasion the endoplasm may show much coarser granules with perhaps the crystals of phagocytosed material. A most important feature is the frequent occurrence of red blood corpuscles which the parasite has ingested. This is of great diagnostic value since the E. coli rarely shows this. The protoplasm of E. coli is granular throughout though fine granules may exist in the ectoplasm. The pseudopodia are never so clear as in the case of E. histolytica.

The colour of E. histolytica is rather of a greenish tint, but the E. coli is greyish.

The pseudopodia of E. histolytica are clear,
refractile, and often active, and this is of diagnostic importance in distinguishing them from those of E. coli which are granular, small, blunt, and sluggish in motion. The former also are long finger-like transparent processes or large and blunt ones. A pseudopodium may be seen projecting forwards steadily and the whole endoplasm flows into it. This process is repeated until the amoeba may disappear out of the field of vision. The activity and the rate of motion of these pseudopodia depend on the freshness of the stool and its warmth. In cold weather the general activity of the whole amoeba is greatly decreased.

Reproduction and E. histolytica Cysts.

The above is a brief description of the active vegetative form of E. histolytica which throws out characteristic pseudopodia, absorbs nourishment and red corpuscles in the wall of the large bowel, and manufactures toxins. Outside the body it is non-resistant and dies quickly. We have yet to consider the reproduction of the amoebae and cyst formation. Reproduction takes place in two ways, on the one hand by binary fission and on the other by the formation of spore cysts which form resistant spores. This is no process of schizogony as in the case of the E. coli.
According to Dobell multiplication takes place by division in the tissues of the gut wall. The E. histolytica lives on the tissues and on red blood corpuscles. When conditions are adverse to the vegetative parasites they encyst, but before this they undergo a change in size and become smaller and are called precystic in type. The cysts when first formed contain one nucleus.

Cysts have a definite cyst wall and are much smaller than the vegetative form. Within they contain highly refractile masses called chromatoid bodies which look like blocks with rounded ends. A vacuole is present which contains glycogen and this stains a light brown with iodine. They multiply by binary fission so that finally there may be four small nuclei in a fully matured cyst. No further nuclear division takes place. Outside the body these cysts are resistant and can live for many days provided they can have warmth and moisture, and it is this which makes our task of eradicating the disease a very difficult one.

Other non-pathogenic intestinal amoebae exist and have been described recently. They are the Endolimax nana, Iodamoeba butschlii, and the Dientamoeba fragilis. If the main characteristics of the E. histolytica are kept in mind these can
readily be distinguished and no confusion need arise.

Practical Methods in Searching for the Parasites.

In the Royal Air Force hospitals in Iraq it was the custom to have a side-room fitted up in the dysentery block for the special examination of stools for *E. histolytica*. This saved time and delay was obviated through not having to send the specimens to the main pathological laboratory some distance away. Fresh stools could thus be examined at once, and in the cold weather, when exposure would diminish the activity of the amoebae, the specimens were kept close at hand and in a specially warmed room. This procedure was undoubtedly a good one here, since the two dysentery wards, where one was working, were both fairly well filled towards the end of the hot weather and early autumn, and it was considered very necessary to do prompt examinations of all stools.

The routine examination of all stools began at 8.30 a.m. in the summer months, and if vegetative *E. histolytica* could not readily be found, a search was made for cysts. For the parasite a drop of normal saline was put on a slide and a small piece of mucus was picked out of the bedpan, (which contained no urine,) and was mixed with the saline.
A cover glass was put over the drop and a 1/6 in. objective used. With practice one got readily used to all those things which at first might be mistaken for E. histolytica, such as E. coli and other non-pathogenic entamoebae like the Endolimax nana. At the beginning too, it was a simple matter to think that one had got on the track of a small rather inactive E. histolytica, when in reality it was only a large refractile macrophage cell derived from the submucosa and often containing red blood corpuscles.

The examination for cysts was carried out by employing Weigert’s iodine solution. This consists of iodine one part, potassium iodide two parts, and water one hundred parts. A drop of the fluid was placed on the slide and a piece of mucus picked out and mixed with it. The colouring of the glycogen mass of the cyst with the iodine usually produced a light brown colour and the nuclei were rendered more distinct.

Flagellates, particularly Lamblia intestinalis and its cysts, were often found in the stools which also contained E. histolytica. One can say with perfect confidence that of all the specimens one has examined, no definite causal role of dysentery could be ascribed to the flagellates, as far as could be
seen, because there have always been pathogenic
entamoebae there as well. I have not been fortu-
nate enough to find a case, where undoubtedly
flagellates alone were present, with no trace of
E. histolytica or cysts as well, after a very care-
ful search of successive stools several days on
end. Flagellates such as the above were common in
many stools examined, and though active E. histoly-
tica might not be found at the first examination,
they were found subsequently. It struck one that
any specimen containing flagellates was bound to
reveal in the end the presence of the pathogenic
amoeba as well. The Lamblia intestinalis and the
Tetramitus mesnili were the commoner ones met with
and any stool containing these made one confident
that the E. histolytica would be encountered also
some time in the search.

Several hours in the morning were spent on the
examination of specimens and one did not usually
finish until about 11 a.m. or a little later.
Twenty minutes to half an hour was given, at least,
to the examination of each stool and ample opportu-
nity existed for the careful search for the E.
histolytica.
Table of the Main Differences Between
the E. histolytica and E. Coli
in Unstained Specimens.

<table>
<thead>
<tr>
<th></th>
<th>E. histolytica.</th>
<th>E. Coli.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Size.</strong></td>
<td>18-40μ</td>
<td>18-40μ</td>
</tr>
<tr>
<td><strong>Protoplas.</strong></td>
<td>Endoplasm granular, ectoplasm clear and well differentiated from endoplasm.</td>
<td>Granular throughout. Distinction between endoplasm and ectoplasm.</td>
</tr>
<tr>
<td><strong>Nucleus.</strong></td>
<td>Not clearly seen and at junction of endoplasm and ectoplasm.</td>
<td>Clearly seen and in centre of protoplasm.</td>
</tr>
<tr>
<td><strong>Colour.</strong></td>
<td>Of a greenish tint.</td>
<td>Greyish in colour.</td>
</tr>
<tr>
<td><strong>Vacuoles.</strong></td>
<td>Few, non-contractile.</td>
<td>Rather less numerous, non-contractile.</td>
</tr>
<tr>
<td><strong>Pseudopodia.</strong></td>
<td>Large or finger-like, clear, and well differentiated from endoplasm.</td>
<td>Small, finely granular and not well differentiated from endoplasm.</td>
</tr>
<tr>
<td><strong>Movements.</strong></td>
<td>Very active.</td>
<td>Very sluggish.</td>
</tr>
<tr>
<td><strong>Contents.</strong></td>
<td>Often contains numerous red blood corpuscles.</td>
<td>Very rarely contains red blood corpuscles.</td>
</tr>
</tbody>
</table>
Naked eye appearances.

In the older literature, before the differentiation of the two main forms of dysentery came about, there were great discrepancies in the pathological accounts of the post-mortem changes in the large bowel. With the differentiation now so clearly put before us and with our greater knowledge and abundant recent material, the pathologic findings are more definite and all the previous confusion is cleared up.

With Amoebic Dysentery the great changes, and the earliest, are in the wall of the large intestine. The first lesions to appear are small elevations of the mucosa. These indicate a zone of commencing necrosis lying deeper in the mucous coat. When the zones increase in size flask-shaped ulcers are formed with their bases in the submucosa. These ulcers are confined to the large bowel only, but the appendix has been known to become involved similarly. The ulcers vary in size and become in many cases an inch or more in diameter. In advanced cases the edges are undermined, the margins are rounded, and the base may extend well into the muscular coat. The ulcers contain yellow, greenish, and dark sloughs. The intervening mucous membrane may be quite healthy.
Erosion into arterioles may produce haemorrhage of a serious nature, and the vessels at the base of the ulcers usually become thrombosed.

Perforation and gangrene of the gut have occurred and these, of course, may lead to peritonitis. It appears that the caecum is the site where this is most likely to occur and where the ulcers of Amoebic Dysentery are said to commence.

Cicatricial and pigmented scarring may be evident where the ulcers have been, and adhesions may form between adjacent coils of intestine.

In Enteric the post-mortem examination shows ulceration in the lymphoid tissue, (Peyer's patches), of the ileum and jejunum. Sometimes, however, in paratyphoid fever the large intestines may be involved.

In the caecum with Tuberculosis there is usually seen a hyperplastic form of intestinal tuberculosis. This is a diffuse infiltration of the submucous coat with tuberculous granulation tissue and the formation of fibro-fatty tissue in the subserous coat. The whole constitutes a tumour of considerable size.

When the entamoebae reach the liver through the portal vein and hepatic complications follow, the earliest change is a general congestion and enlargement of the liver. Later, greyish, irregular and anaemic patches may yield a reddish gummy pus when
they are expressed. Still later the centres of the patches may liquefy and abscess cavities are produced. The walls of the abscess are ragged in appearance. They are usually spherical on the whole, though they may have diverticula leading off from the central cavity. The pus is chocolate-coloured as a rule, and being (largely) free from pyogenic organisms, tends to be (rather) sterile. The cysts of E. histolytica are never found in liver abscesses, the active entamoebae alone being responsible for these pathological changes.

Microscopic pathology.

The opinion is now held by many that the E. histolytica enters the bowel wall by the crypts of Lieberkühn, and that in the wall they multiply. The tissues are disintegrated by means of a cytolysin and a gelatinous necrosis takes place. There is very little local inflammation of the surrounding tissues.

In advanced cases the amoebae may be found among the muscle bundles and in the peritoneal veins. From the veins they may reach the portal vein and then the liver, where Amoebic Hepatitis or an abscess may result.

Fortunately, fatal cases have been few in Baghdad among Royal Air Force personnel recently, and
recently, and in 1924 no opportunity was had of examining early pathological specimens of the changes in the large intestine or liver. Those pathological specimens examined were one or more years old and no record of the cases were available. But the typical lesions of Amoebic Dysentery as described by so many recent authorities were quite obvious in those museum samples which one had at hand to examine.

Judging by the cases in the wards, the parts of the large intestine mostly affected seemed to be the caecum and the lower part of the descending colon and sigmoid colon. Patients used mostly to complain of pain over those regions. The amount of blood in the samples of stools varied a great deal. In some it was very small, consisting of a few drops here and there, and in others it was large in quantity where two or more ounces of blood and mucus were passed. These latter cases usually meant there was a great deal of ulceration and haemorrhage in the bowel wall. As an example Case No. 7 in the Appendix shows this. Haemorrhage with that patient was very severe, extending over a long period, and loss of blood necessitated the transfusion of hyper-tonic saline.

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Etiology, Diagnosis, and Clinical Types.

Etiology.

Entamoeba histolytica cysts are the chief cause of the spread of the disease. Active entamoebae owing to their susceptibility to drying outside the body are probably never a direct cause of infection. Cysts are resistant and, if given a favourable opportunity of maintaining life for example within the housefly, or among moist green vegetables such as lettuces, can keep up sporadic outbursts of the disease, until climatic conditions like the winter in Iraq, are adverse to their existence. Dessication of the cysts is said to take place in sand and dust.

Water is a common source of infection and it may harbour cysts for days. All water is chlorinated in Royal Air Force units in Iraq, so that cysts do not find a favourable medium in tanks and other receptacles for holding drinking water. With detached units and Armoured Car convoys in the desert, however, there is every probability that the water is not chlorinated as carefully as it should be, hence dysentery is not uncommon with these men.

The climate in Iraq as stated earlier is very hot and dry. Even in winter there is very little rain despite the intense cold on occasion. One is
therefore not inclined to agree with some authorities who say that a hot temperature with great humidity is most favourable to entamoebae. It is during some of the hottest and driest months, June to October, that dysentery is at its worst. November is a dry month also and the incidence then is fairly high. On the whole the climate is too dry to suggest that moisture or humidity has any great influence on Amoebic Dysentery in Iraq.

Sand may not play any part in the dissemination of the disease, because of dessication of cysts, but it seems very likely that the ingestion of sand and dust along with food can be a predisposing cause. Inflammatory conditions of the mucosa caused by particles of sand may well be favourable to the lodgement of cysts or the resultant amoebae. The fine dust and sand accompanying sandstorms can penetrate to places where food is kept, no matter how carefully foodstuffs may be stored. This unpleasant dust seems to get everywhere in spite of precautions, whenever these storms are taking place.

One has seen cases where dysenteric symptoms, slight in nature, rather point to sand being the primary cause, and since entamoebae happened also to be present, the condition was of course labelled Amoebic Dysentery.
A case in point is No. 9 in the Appendix. This man passed semi-formed or formed stools nearly the whole time he was in hospital and only traces of blood were seen. The lesions were obviously not extensive enough to produce the typical dysenteric stool, and a long close search took place before the entamoeba was brought to light. It is admitted that this may have been amoebic from the beginning and that mechanical irritation played no part, but how can one explain the so-called "Clinical Dysentery" cases that one meets with in Iraq? With these, few in number, there could be found no definite cause, neither bacillary nor protozoal, and search was extended to beyond these two forms without result.

One entertains the opinion that these mild cases yielding dysenteric symptoms and called "Clinical Dysentery" are the result only of sand or other irritation in the bowel, and that, if cysts or entamoebae be present in addition, they may find the damaged mucosa more favourable to attack and Amoebic Dysentery may supervene. Enteritis of varying degrees of severity is common in the tropics, and this along with the other conditions which are conductive to the well-being and increase of cysts and entamoebae, may contribute something to the
indigenous nature of the disease in hot countries. This might certainly be the case in Iraq where sandstorms are very bad, diarrhoea is common enough, and sanitation, though most strictly supervised, is primitive in form.

Carriers of E. histolytica.

There are people who harbour active entamoebae in the large intestine and yet they do not suffer from the symptoms of Amoebic Dysentery. Also they may not have suffered from the clinical signs of the disease in the past. These persons are "contact carriers". Other persons may have had the disease and afterwards become "convalescent carriers". Any of the above who are thus liable to pass E. histolytica cysts in their motions are a constant source of danger not only in the tropics but in countries with a temperate climate. Given the conditions of the tropics the danger is far greater. The infection in carriers may persist for years and can be got rid of only by anti-amoebic treatment.

Yorke, Matthews, Melins Smith, and others in the years 1916 to 1919 have shown a percentage varying from 5 to 19 per cent of carriers in England among the personnel of the navy, in children, among the insane, and in army recruits. Many of these were not old dysentery convalescents and had never been out of England. The climate of England cannot be
ideal for the life of cysts outside the human body, but the above certainly proves the existence of "carriers" unassociated with the tropics at any time of their lives.

Hot weather is undoubtedly beneficial to E. histolytica. They are more active then and the examination for parasites is rendered so much easier. But, in cold weather, despite the fact that specimens might be examined immediately they are available, the parasites are most sluggish and greater difficulty arises as to certain diagnosis.

Cases of Amoebic Dysentery do arise in England though they are comparatively few. But there is no reason why more cases should not develop in the future. With the big number of carriers in this country one would expect more cases of Amoebic Dysentery, but, as will be shown later, sanitation and climatic conditions along with the even greater numbers of carriers in the East help considerably to make the incidence abroad higher than in England. I have seen only one case personally in England and that was before I went to the Near East and before I had had any experience of tropical medicine. This was in 1923 and the case was that of a young girl aged four years who contracted quite an acute attack of Amoebic Dysentery. Both her parents had
been in India for very short periods only during the War, though they had not had Amoebic Dysentery as far as they knew.

The father was free from E. histolytica and cysts, but the mother was found to be a "carrier" and entamoebae and cysts were discovered. Both mother and child had emetine treatment. It was summer when the child contracted the disease, but one did not ascertain whether there had been any history of enteritis some time previous to the onset of dysenteric symptoms. The child recovered well.

Incubation Period.

We owe it to Walker and Sellards that some idea of the incubation period has been worked out. When these two investigators administered gelatin capsules containing vegetative entamoebae to a certain number of persons, symptoms of dysentery showed in some cases twenty days after and in others anything from twenty to ninety-five days after.

In a country such as Iraq, where the disease is endemic and where so many sources of infection exist, it is a very difficult procedure, almost impossible in fact, to ascertain with any degree of accuracy correct data in relation to the incubation periods of many cases observed.
Diagnosis and Clinical Types.

The pathological laboratory and not the bedside is the only place where we can arrive at our correct diagnosis of Amoebic Dysentery. The discovery of active amoebae containing red blood corpuscles and exhibiting characteristic pseudopodia is quite sufficient. If there is reason to suspect a superimposed bacillary infection then further examination is necessary.

Sigmoidoscopic examination in cases where ulceration has extended into the rectum may be valuable in revealing the character of the ulcers. Care has to be exercised in the use of the sigmoidoscope, however, but if reasonable precautions are taken, this enables a useful examination to be made and no anaesthetic is required.

To judge a case by clinical data is fatal and in doing so one can rightly be accused of criminal negligence. The real cause must be found and this can come about only in the laboratory.

Amoebic Dysentery is more often insidious in onset, and it usually runs a chronic course. Relapses are frequent and latency is one of the most striking features of the disease. A patient may complain more of ordinary diarrhoea than of true dysenteric symptoms. It is often only when blood is definitely seen in the stool that the airman
becomes apprehensive and reports the matter at once. However, very acute attacks are seen too, where the general disturbance is serious and the loss of blood most alarming. These fulminating or gangrenous cases are fortunately comparatively few in these days. One has not heard of a case where an accident has occurred to a pilot through a fulminating attack having taken place during a flight. Such rapid onset with all its alarming acuteness might well be a source of danger to a pilot and perhaps be responsible for an accident taking place.

A clinical classification of Amoebic Dysentery according to the severity of the disease may be set down as below:

A. **Acute Intestinal Amoebiasis.**
   1. Simple acute attacks of moderate intensity.
   2. Gangrenous Dysentery.

B. **Chronic Intestinal Amoebiasis.**
   1. With symptoms.
   2. Latent.

Pain and tenderness on palpation varied a great deal with the types one saw in Iraq. Some men had only slight pain at intervals while others had it more or less constantly with more acute attacks at certain times. It was also not necessarily an index of the amount of blood and mucus that
could be found in the stools, though with those very acute and almost gangrenous cases pain and large quantities of blood in the stools often went together. The caecum and descending colon and sigmoid colon were the places over which most men complained of pain. Tenderness on gentle palpation was often useful to help in forming an idea of the extent the large bowel might be involved.

The average number of stools passed was about nine. One occasionally saw cases where the motions exceeded fifteen, and if the patient's condition had already been diagnosed definitely as amoebic in origin, then in addition examination was made for a superimposed bacillary infection. An acute exacerbation with ordinary cases meant an increase in the number of stools and usually far more blood and mucus in the specimens. In the mildly acute cases one often saw pultaceous stools, and sometimes even formed ones which would be streaked with blood and mucus.

In those cases where the diarrhoea is excessive and where perhaps fifteen to twenty motions are passed in the twenty four hours, the specimens consist almost entirely of blood and mucus. Such a stool is said by some people to be more typical of
Bacillary Dysentery, and if viewed from the clinical point of view only, this diagnosis might leap to the mind. But time and again with cases like this Bacillary Dysentery was not proved to be present and entamoebae were the only causal agents found. Hence no reliance can be placed with safety on the amount of diarrhoea during the twenty four hours and the appearance of the specimens to differentiate between the two main types. Only once did I see a motion which could truly be called "anchovy sauce" in appearance. In the ordinary course of events one never saw such stools or as a rule anything which in the least answered to that description.

With most patients there was little rise of temperature or pulse as a result of the disease. The thermometer sometimes registered over 100°F, and general disturbances and signs of toxaemia were few. However, cases in the wards could be seen where a slight rise of temperature persisted round about 99°F. and 100°F, and yet no evidence of hepatitis or other more serious reason could be found. These small rises could only be attributed to slight toxic absorption taking place in the bowel wall, but like Cases No. 5 and 7 in the Appendix, definite fever and toxaemia were present with no hepatic complications to serve as contributing factors.
The diarrhoea with case No.5 was never very excessive, but with case No.7 it was not uncommon for eighteen or twenty stools to be recorded in the twenty-four hours. They were for the most part all blood and mucus, with only traces of faecal matter here and there, and a few smallish sloughs.

The general disturbance of the more serious cases like No.7 was marked. Not only was the temperature raised to about 101°F. but the patient became rapidly very thin, he was pale through loss of blood, the tongue was coated though moist, he slept badly and had headaches, ced was complained of, and he became rapidly weaker and exhausted. He became obviously very ill. The sloughs that he passed indicated extensive and somewhat gangrenous changes in the bowel wall. He complained of pain mostly in the line of the descending colon though it was present also over the caecum.

Those patients with mildly acute attacks suffered no great discomfort, and ordinary good nursing with specific treatment soon improved them wonderfully. The same applied to the chronic cases which showed symptoms, and the majority of these were kept in bed only when undergoing emetine treatment with emetine-bismuth-iodide capsules.
Earlier in this section I have mentioned "Clinical Dysentery". I may add in further explanation that nearly all cases of dysentery which came into hospital from the various units were named in this manner by Medical Officers of units, unless an examination for the definite cause had been carried out at the Station Sick Quarters of the unit. Now and then these patients remained cases of "Clinical Dysentery" throughout the whole of their stay in hospital. When no amoebic, bacillary, or other reasonable specific infection could be blamed, the diagnosis had to remain. But it must be mentioned that the course pursued by this type of dysentery was mild and subacute with most patients. Diarrhoea was not severe, griping and tenesmus were slight, and blood and mucus small in amount. Also there was little constitutional disturbance.

The white blood count is a very useful indication of the severity of the disease. A leucocytosis of 11,000 per cm. or over is often seen with a simple acute attack. In patients where one is afraid of hepatic complications setting in, though no sign may definitely show, a high leucocytosis of over 20,000 may be very suggestive. Rogers believes that a very high white count can be almost diagnostic
of Amoebic Dysentery, since though Bacillary Dysentery also shows a leucocytosis it is generally not such a high one as with Amoebiasis.

**Differential Diagnosis.**

The differentiation of the dysenteries must be accomplished in the laboratory. When the two main kinds cannot be found other possible causes must be sought for. It was considered advisable in all cases where a high initial temperature was present to take a blood film to eliminate the malarial parasite. Case No. 4 in the Appendix suggested a "Malarial dysentery" at first until the entamoeba was discovered, and, though a blood film had been taken at the beginning, it was after this that the benign tertian parasite was found.

Not only Enteric, Cholera, and Syphilitic and Tuberculous infections of the gut have to be considered in differential diagnosis, but also Haemorrhoids and Carcinoma must be taken into account.

With Enteric there is not so much blood or mucus in the stools to the extent that there is with a typical attack of Amoebic Dysentery, nor is there quite so much diarrhoea as a rule. One cannot go by the stools alone. There is the patient's general condition and high temperature to consider in Enter-
ic, and such a patient looks very ill in addition. With Amoebic Dysentery the patient so often looks comparatively fit.

In Cholera there is the profuse diarrhoea, which shows the typical "rice-water" stools. These generally contain no blood or mucus. There are also cramping pains in the limbs and the signs of great prostration quickly supervene. "Rice-water" fluid may also be vomited, and the symptoms soon become very grave.

In Syphilitic and Tuberculous infections of the bowel the diarrhoea is not usually so frequent nor so persistent. Blood and mucus will more often be less in the stools. The history and general condition of the patient must help in the diagnosis.

The rectum may be examined for Haemorrhoids and Carcinoma, but a cancer high up will require a bismuth meal to help us to reveal the cause. The laboratory, in any case, is essential for aiding us to arrive at our correct diagnosis.

Complications.

The immediate complication most to be feared with a fulminating attack is perforation of the bowel with its resultant peritonitis.

The next most important complications to
think of are Amoebic Hepatitis and Abscess of the Liver. The E. histolytica is the exciting cause of such conditions, but it seems that predisposing factors appear to be necessary before the serious associations of amoebic infection make their presence felt in the liver.
Prophylaxis and Treatment.

The problem of the prevention of Amoebiasis is a big one and it has to be faced not only in the tropics but in countries of the temperate zones. Those factors which go to make the prevalence of the disease greater and a source of constant anxiety during war must not be lost sight of in times of peace. Imperfect sanitation, trying conditions of service, doubtful and unwholesome food, and perhaps lack of proper and adequate hospital treatment, all tended to increase our difficulties. Not only were there these conditions to contend with then, but in addition and almost as important, there was the bewildering problem of knowing how to deal with the chronic "carriers". This was so in the Great War and now, though we can deal more successfully with the majority of relapsing cases, there remains the additional question of what to do with the "contact carriers" who in England today probably exist in thousands and who have had no symptoms of the disease.

Malins Smith in the British Medical Journal of November 15th, 1924, gave a very good resumé of the work on the spread and incidence of the protozoal infections in the population in England.
These investigations were carried out by several people at the Liverpool School of Tropical Medicine from 1916 to 1919. In this article he stated the position in 1916, when it was thought that the Entamoeba histolytica was a parasite of man only in the tropics and subtropics. The almost complete absence of the disease in this country was thought to be an indication that the causal parasite was absent. As a result of the above work, however, those ideas were soon modified, and it was finally proved that not only were there numbers of people who harboured the parasite and had never had the disease, but that the Entamoeba histolytica had probably long established itself in England and did not depend on a foreign source of spread. With regard to the spread it was found too that many of the children who showed the parasite in the stools revealed at the same time a family history of heavy infection.

Thus from the above it follows that this entamoeba normally can be found in England as well as in the tropics. Food and water contaminated by infected faeces, or rendered harmful through the agency of flies, are the chief cause of the spread.
Hence the subject of sanitation becomes more prominent, and it is a matter of vital importance wherever the Entamoeba histolytica is likely to exist that pollution of food and drink be rendered impossible.

From all this work it appears that the parasite is not the only requisite for the onset of the disease, but that some factors which may be more prevalent in the tropics are necessary in addition. It is likely that in England the destruction of tissue caused by the entamoebae in the intestines of healthy carriers is easily made good and a balance is maintained between the host and the parasite - as Malins Smith holds. There is no doubt that the Entamoeba histolytica is widely prevalent in this country and yet Amoebic Dysentery is not often seen. Cases probably arise which are not diagnosed or are labelled "ulcerative colitis".

It seems to me that Malins Smith in his article is rather influenced by the high incidence of healthy carriers in England into minimising the extent of the disease abroad. I do not agree with him when he says that "even in tropical countries only a small percentage of dysentery is amoebic". With the Royal Air Force abroad in 1923 and 1924 the prevailing type of dysentery was amoebic in Iraq, India, and the Mediterranean littoral, as the tables in the
Reports of the Health of the Royal Air Force will show. The percentage of Bacillary Dysentery was small. Of a total of 118 cases in Iraq in 1924, 101 were amoebic and none were bacillary. The Report for 1924 also says "only 8 cases of dysentery were reported from India, these cases being scattered amongst five stations. The prevailing type in this command was also protozoal, only one case being bacillary in origin".

One holds the opinion strongly then that Amoebic Dysentery is still a disease mostly of the tropics. Also it seems that acute cases are more frequently met with than in England, and that some conditions, as yet not fully explained, are favourable and responsible for this.

However, the problem to be dealt with is the prevention of the spread of infection whether in England or the tropics. This must be done in two ways, (1) by freeing the convalescent and known contact "carriers" from their infection, and (2) by making our sanitation as perfect as possible.

Carriers of all kinds are a danger to the public and must be treated whenever occasion demands. Old convalescents should be examined from time to time, and if necessary emetine treatment should be given. It is obviously too big an undertaking to
examine the population for the possible thousands
who are healthy contact carriers, but in those
families where a case of Amoebic Dysentery is found
the rest of the family should be examined at once
and treated where positive. Close contacts of all
definite cases should be dealt with in the same way.

Dale in 1916 was the first person in England
to make remarkable progress in the treatment of
chronic carriers. He found that only a temporary
freedom from cysts was present in many of his
cases after the usual course of emetine injections
of one grain daily for ten or twelve days had been
given. He considered that the entamoebae were
localised in a partly-healed pocket or sinus in the
bowel wall and that they were more or less shut off
completely from the circulation and consequently
from the action of the hypodermically injected
emetine. Dale utilised the double iodide of emetine
and bismuth. This given orally passed the gastric
juice practically unaltered and in the alkaline
juice of the duodenum became dissolved and further
on in the intestines liberated emetine and precipi-
tated bismuth sulphide.

This oral treatment had greater action on the
entamoebae, and it bore out the good results which had
been primarily obtained by the older ipecacuanha treatment by mouth. Cysts also were destroyed much more effectively, and on the whole the results were most encouraging.

Three grains of the emetine-bismuth-iodide were calculated to be equivalent to the one grain of emetine hydrochloride injected hypodermically. Such beneficial results took place with this treatment that today with the chronic cases of dysentery, and even with simple acute attacks sometimes, it has not been superseded.

We now come to the urgent question of sanitation. If we are to keep down the numbers of healthy and convalescent carriers we must not only treat them but prevent them from infecting others. If such people live in insanitary surroundings the risk of infection to others in the same dwellings is great. Many of those healthy carriers who were discovered in England had lived in places where sanitation was imperfect. The urgency of cleanly habits is fairly obvious. Among unclean persons it is an easy matter for food to become contaminated by infected faeces. These possibilities must be eliminated whether in England or the tropics. If the chances of infection are greater, there must be
greater liability to acquiring the disease.

Sanitation in this country is on the whole excellent. In some places it may not be perfect, but generally it is far superior to any system which can be found abroad.

Sanitation and Prophylaxis in Iraq.

In Iraq the sanitary system is a great problem. Among the native population there is none worth mentioning, though the Iraq Health Service today is doing a tremendous amount of work in improving things. With the Royal Air Force and Army units we still find primitive methods in use, though these are the only possible ones that could be utilised at present.

There is no sewage-disposal system laid down on the lines we are accustomed to in England. All water and other liquid waste has to be disposed of into soakage pits and grease traps. When these become too full new ones have to be constructed.

There are no flush-water latrines in the military cantonments and bucket latrines are universal. The excreta are incinerated after getting rid of the urine into urine-soakage pits. All possible rubbish and material is burnt.

Despite these comparatively primitive methods
the results of cleanliness and efficiency from most Royal Air Force and Army stations in Iraq are amazing. The strictest supervision by the Medical Branch of the Royal Air Force is exercised over sanitary matters and the distribution and care of food, and this is rewarded by the absence of cholera and plague outbreaks and the minimum of enteric infections. Sandfly fever, malaria, and dysentery are the three diseases most common in Iraq today and of these dysentery probably gives the most trouble taken all round.

Therefore it can be seen that great responsibility rests upon the Station Medical Officer, when it is reflected that he has to deal with a system of sanitation which is poor, though the only one available under the circumstances of our temporary occupation of the country. In Egypt, Palestine, and India the conditions are much better, and Amoebic Dysentery does not show such a high incidence as in Iraq.

It will be readily understood that such latrines as those of the bucket type, even though they may be carefully encased all round to make them flyproof, are sometimes left open and flies get ready access to the contents. Then apart from the infection of food by cyst-bearing flies there are native cooks, waiters,
oolies and other native employees who may be carriers and so help in the progress of the disease. Whenever possible cooks and mess waiters are always carefully examined for E. histolytica, but it is an impossible undertaking to do the complete and thorough examination of a shifting and ever-changing native personnel so employed.

The chief prophylactic measures employed in Iraq among Royal Air Force personnel and native followers are the examination of all cooks and mess waiters for E. histolytica, and "pinking" with potassium permanganate solution of all vegetables and fruit, the prompt and frequent emptying of latrine buckets, and protection of food in flyproof foodsafes and the suppression of flies. A rule very strongly enforced among the cooks and kitchen helpers was the thorough cleansing of hands at intervals and the soaking of them in potassium permanganate solution.

On those stations where medical efficiency is undoubted and where the sanitation, such as it is, cannot be improved, the smallest numbers of Amoebic Dysentery cases are to be found. The violation of these rules of strict sanitation has only one result, the increase of dysentery and enteric.
Treatment.

Ipecacuanha was the first specific drug as far as we know used for the treatment of Amoebic Dysentery. Its special destructive power only on the E. histolytica was first demonstrated by Vedder, and he also proved that if the drug was used without one of its alkaloids, emetine, it had no deleterious effect upon the parasite of Amoebic Dysentery.

Manson also successfully carried out extensive experiments in the treatment of cases with ipecacuanha and the value of the drug became still further established.

Rogers, however, followed up the work of Vedder and recommended the use of the alkaloid emetine alone. He thought that if the alkaloid was responsible for the specific action on the E. histolytica, it could be better utilised if given by itself and if it was injected into the circulation. This procedure has now largely superseded the use of ipecacuanha, and no doubt gives better results. Emetine is easier to give and there is not so much trouble with nausea and vomiting.

The usual routine with ipecacuanha was to give doses of 20 to 30 grains once a day after an initial purgation with castor oil. It was given on
an empty stomach and all food was stopped for three hours. Just before administering the drug it was advisable to give 15 to 20 drops of laudanum in a little water in order to minimise the desire to vomit. It could be given in pill form, bolus, or in capsules. The above dose was given once or twice a day for a week. Vomiting was fairly frequent. Strict watch over the patient for signs of overdosage was necessary and a good plan was to reduce the dose by 5 grains every days.

Whatever drug is used, ipecacuanha or emetine, care must be taken in the quantities administered. Emetine is a poisonous alkaloid in large doses, and it can have an irritative effect on the mucosa of the intestines producing an intractable diarrhoea. Its toxic effect is also seen in the production of cardiac irregularities, and on some occasions great asthenia and collapse. A skin condition has been shown recently to be associated with overdosage. This is exhibited as a fine branny desquamation, and even if other signs are absent this is a sure indication of a toxic condition.

This drug, if used with discretion, is the only means of effecting a radical cure, and if the patient is watched carefully there is no reason why compli-
complications should arise. No drug has displaced it and the results obtained in the treatment of both acute and chronic cases fully justify its use.

When Rogers advocated the use of the alkaloid emetine alone he and Vedder were of the opinion that when the drug was injected hypodermically it had a greater effect on the entamoebae in the tissues of the body than if given by the mouth alone. This opinion is still largely held by many people, but the question is not entirely settled yet. Dale and Dobell in 1916 in careful experiments with the alkaloid failed to confirm fully the original observations made by Vedder and Rogers that emetine injected hypodermically had a particularly destructive power on E. histolytica actually in the tissues of the bowel wall. All are agreed, however, that the drug is specific, and it seems reasonable to suppose that no matter how it is given, if it does actually reach the parasites, whether in the tissues of the bowel wall or when they are practically free in the lumen, it must have a deleterious effect on them.

Dale also in 1916 as stated previously certainly showed that when E. histolytica were lying in partly-healed pockets in the intestinal wall emetine-bismuthous-iodide given by mouth had excellent results, and encysted E. histolytica and cysts were easily
destroyed.

Today it can be said that injections of emetine and the oral method of administering it are both extensively used. In acute cases the usual procedure is to start with the hypodermic injections of one grain daily of emetine hydrochloride for 10 or 12 days and, after an interval, if the stools are still positive to give a course of emetine-bismuthous-iodide (E.B.I.) by mouth as well. To start with E.B.I. in acute cases sometimes brings on increased irritation of the mucosa and this aggravates the disease.

**Treatment of Acute Cases.**

The treatment carried out in Royal Air Force hospitals in the tropics at present is briefly detailed below.

The patient is confined to bed on a starvation diet and put on a Sodium Sulphate mixture of Sodii Sulphas. drachms one, Acid. Sulph. dil. minims fifteen, Tinc. Zingiberis minims five, and Aqua menth. pip. to one ounce. If the case is not one where collapse is present Calomel, grains three, precedes the mixture. The Sodium Sulphate mixture is given every four hours for the first two days of the course of emetine. This flushes the bowel and
removes organisms and their toxins and clears the mucous surface so that the specific drug can act better.

Emetine hydrochloride, one grain in one cubic centimetre of distilled water, is injected hypodermically at night every day for 10 or 12 days with no break in the course, but not for longer as a general rule. A close watch is kept on the heart. In very severe cases it is given intravenously dissolved in ten cubic centimetres of normal saline. If there is a tendency to vomit tincture of iodine three minims, or ten to fifteen minims of Chlorodyne half an hour previous to the emetine usually prevents it.

Enemas of a thin starch solution with laudanum, thirty minims, are found useful in bad tenesmus.

The diet to start with is a minimum and consists of barley-water, imperial drink, and tea. This is added to gradually throughout the course by Brand's essence, arrow-root and cornflour, Benger's food and chicken-tea, jellies and milk puddings, beef-tea, and a flip of egg and milk. When the course is finished a little fish and minced chicken is added.

Colon lavage is carefully carried out once
daily in ordinary acute cases after the Sodium Sulphate is stopped. This is of normal saline, one pint, or a lavage containing quinine bichloride ten grains to one pint of normal saline. The quinine colon lavage seems to help materially if carefully given. Alcohol is strictly forbidden.

If the patient was doing well and his illness was only a mildly acute one, he was allowed up for a little on the second day after the course had finished.

Treatment of Chronic Cases.

Although sometimes mildly acute cases were treated with E.B.I., the usual procedure was to adopt hypodermic emetine until at least the excessive diarrhoea had subsided. With the chronic cases, or where further courses of emetine were required after the initial one, E.B.I. was always used. It was given once a day at night in capsules containing three grains and occasionally in bigger doses of two capsules a day, each of two grains, one at night and one in the morning. This was also given for 10 or 12 days depending on the case and its response to treatment. The patients were kept in bed while undergoing treatment. Colon lavage was kept up also but not daily. It was given every other day.
A drug which has been largely used in the Royal Air Force in Palestine and Egypt recently with chronic cases of amoebic dysentery is Thymol. In Iraq this treatment was not extensively employed. It is thought that owing to its antiseptic and especially anthelmintic properties it can free the bowel of many organisms and give the E.B.I. a much better opportunity of effecting a cure. Sodium bicarbonate, ten grains thrice daily, is given for three days previously to the administration of Thymol. On the fourth day on an empty stomach and after good purgation with Calomel and Magnesium Sulphate, three doses of Thymol of thirty grains each (in two cachets of fifteen grains each) are given at intervals at 9 a.m., 10 a.m., and 11 a.m. Two drachms of Magnesium Sulphate are given and, when the bowels have moved, a cup of tea without milk and a dry piece of toast are allowed. At night the E.B.I. course is started but with only one grain for that dose and for the dose next day. On the third day the usual doses of E.B.I. are given. No fat or alcohol must be given with the Thymol, hence castor oil is never used along with it.

This appears to give satisfactory results, but the sheet-anchor in the whole treatment of chronic
cases of amoebic dysentery is emetine. Great care must be taken to watch the heart and the pulse rates after giving E.B.I. especially if more than one course is ordered. Cases 8 and 12 in the Appendix are typical examples one meets with occasionally in Iraq, but fortunately, if the signs of toxic effects are noted early the patients soon recover. It was very rarely necessary to give more than two courses of E.B.I.

The rule was that before chronic cases were discharged, eight negative stools were found in the course of three weeks after treatment. But it nearly always meant that many more were examined than eight specimens for each convalescent, and patients were not discharged until they were free of E. histolytica.

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Summary.

It is the desire in this summary to discuss the situation today with regard to Amoebiasis in Iraq with the help of the foregoing sections; and to endeavour to point out some of the possible reasons why it is still largely prevalent there, and so at the same time explain its comparative absence in England.

There is a diversity of opinion in the literature of the War period and immediately after as to which of the two main types of dysentery, Amoebic and Bacillary, actually provided the greatest number of cases in the Eastern War areas taken as a whole. Some authorities held that, because Amoebiasis was undoubtedly a disease of the tropics and subtropics, there must at least have been as much Amoebic Dysentery present as Bacillary, or even more of the former. Others were equally certain that Bacillary Dysentery was more prevalent, and according to their arguments and the figures they could produce, this certainly appeared to be a reasonable conclusion. Undoubtedly in Gallipoli, Egypt, Salonica and Palestine there was a great deal of Bacillary Dysentery, probably more than the protozoal type, but it might be argued too that in India and Mesopotamia
there may have been more cases of Amoebic Dysentery.

One point of view which strikes one forcibly is that for some periods of the war in Gallipoli, Palestine, and especially Mesopotamia, there were not the opportunities to study the type of dysentery with the degree of accuracy required, and that a good deal depended on clinical diagnoses alone. This lack of laboratory confirmation accounted for cases being called Bacillary Dysentery, when in reality they were Amoebic, and vice versa.

From one's own experiences in the War (not in the Medical Services) one knows that thousands of troops in Gallipoli had dysenteric symptoms and no specimens of stools were ever submitted to examiners, because facilities for proper microscopic examination and other laboratory means happened not to be available at the particular time or place. Also, scores of soldiers in Gallipoli had dysenteric symptoms of a mildly acute nature, and, since no very great constitutional disturbance was present and because the rather urgent diarrhoea soon subsided, they never reached the Field Ambulances or hospital ships. One believes that this was also the case in Mesopotamia.

Those men who went to hospital through the urgent nature of the symptoms were probably diagnosed
mostly as suffering from Bacillary Dysentery, but the others who did not go and ran no raised temperatures and did not feel really ill, though they numbered hundreds, were very possibly cases of mild Amoebic Dysentery. Later they may have become relapsing cases of Amoebiasis or escaped notice altogether and are now healthy carriers. In Mesopotamia, I believe, in the early stages of the campaign hundreds of troops had dysentery of a mildly acute type, who never reached hospital and were thus never diagnosed. These also may have been amoebic in type.

Therefore my own opinion is that more Amoebic Dysentery existed during the War in the tropics and subtropics than Bacillary Dysentery, and that the Bacillary type was rendered more noticeable in some countries owing to the necessity of greater numbers of these cases being compelled to go to hospital through showing more severe constitutional signs of disturbance, than may be seen with Amoebiasis, especially when the latter is only a mild attack.

If there is doubt as to which of the two forms created the higher incidence in the War, it is clear from the Reports of the Health of the Royal Air Force for 1923 and 1924 that Amoebic Dysentery
today, at any rate, shows higher total figures than the other kinds for all countries in the East where Royal Air Force personnel are stationed. Iraq easily shows the highest case incidence of dysentery as being due to Amoebiasis for both years, while Egypt, Palestine, and India are not quite so constant but nevertheless show the majority of cases to be protozoal. One also knows that, though there are no figures available at the moment, the year 1925 in Iraq shows very little difference from the previous year.

Many investigators, as we know, have shown that carriers are fairly plentiful in England. They why, we might ask, is there not more Amoebic Dysentery? There cannot be a great deal because it would not altogether escape being detected.

For this answer we might look to Iraq because, if we can find good reasons why it is particularly prevalent there, it is justifiable to assume that the lack of those reasons as applied to England may account for the higher incidence in the warmer country.

One gives the following reasons why Amoebiasis might be said to exist to a much greater extent in Iraq than in England.
1. The climate is more suitable for *E. histolytica* cysts outside the body and they live longer. Consequently more carriers arise from this greater chance of infection.

2. If there are more carriers there is greater possibility of infection spreading and manifesting itself.

3. Sanitation is not as perfect as in England, India, or Egypt, therefore flies can help in the spread of cysts contained in faeces.

4. Flies are particularly very bad just before the hot weather, April and May, and at the end, September and October. This helps to explain the high monthly incidence in June and November.

5. Sandstorms are very frequent in the hot and dry weather, and sand and dust is ingested with food to a great extent. This may bring on an inflammation which may cause damage to the intestinal mucosa.

6. The mucosa may be sufficiently injured to allow the *E. histolytica*, in carriers, to thrive on this damaged tissue and so become more fully active and pathogenic.

7. If a heavy infection takes place, where cysts are more numerous in the intestinal canal,
this combined with some of the above factors may produce more likelihood of pathological changes commencing.

The frequency of diarrhoea of varying degrees of acuteness, whether brought on by ingested sand particles or by other irritant causes such as unwholesome and tinned food, is noted in Iraq. The mucosa in the bowel in these cases must be rendered more susceptible to the onslaught of many organisms, and if the E. histolytica be present in very large numbers, which might well be the case in such a country, there seems no reason why further pathological changes should not be produced upon the intestinal mucosa. Once inflammatory changes have commenced the conditions for E. histolytica may be so improved by greater proximity of broken down tissue and red blood corpuscles to live on, that their full activity is evinced and multiplication takes place readily. Even other organisms present may, by their irritant effect, help to encourage the E. histolytica to settle in the inflamed bowel.

"Clinical Dysentery", as stated earlier, comprises those cases where no satisfactory explanation can be given for their origin after careful search. One is convinced that many, if not most, of these cases in Iraq are the result of the ingestion of
sand and dust only.

One entertains the opinion that the earliest pathological changes in the intestine typical of Amoebiasis are of a nature secondary to primary disturbances of the mucosa. The pathological changes first seen by the naked eye appear to be small sites of commencing necrosis with healthy mucous membrane between. These, however, may be sites where the entamoebae, being already present in the host, find a more favourable opportunity to gain maturity quickly thereby gaining increased activity and vitality. Normally it is thought that when conditions are unsuitable for entamoebae they encyst, but the above opportunity may be just what the parasites require to make them flourish.

There are carriers enough in Iraq today in all probability to produce the hospital patients, provided the above predisposing factors make themselves felt.

One considers that a patient may have pursued a course such as follows. He may have been a healthy contact carrier, we shall say, in March or April. From that time on his latent infection may be added to tremendously by further cysts owing to the great increase of flies up till June. Then in
May and June, when sandstorms and dry dusty weather commence, irritation of the mucous membrane may give the required impetus to the heavy infection, and a primary dysentery results. The same may happen in September and October when flies begin to be bad again after the hottest months have passed, but when the weather is still dry and dusty. This weather and the renewed activity of flies accounts for the big number of cases in November. In July, August and part of September, flies are not numerous, because of the great heat which may be 120° to 125°F. in the shade during the hottest part of the day.

The high incidence during the fly seasons, or just after, certainly indicates that food is probably very quickly infected with cysts by flies and that the dessication of cysts by dust is a negligible matter in the spread of the disease.

India, Egypt, and Palestine, as far as the Royal Air Force are concerned, do not show so much Amoebic Dysentery as Iraq. This is due to three things:—firstly, general conditions and sanitation especially are better in those countries, second, there is less dry and dusty weather, and lastly, there is probably less abdominal trouble and diarrhoea than in Iraq.
There is no doubt that flies and primitive sanitation, combined with the fact that carriers among the natives are most numerous, are responsible for heavy infection of personnel in Iraq. The number of cases of dysentery from a unit is an index to the sanitary efficiency displayed by that unit. The nearer conditions become to a state of war, such as with forces on the frontier or armoured car units away in the desert for weeks, the greater is the liability to get the disease. People become more careless with their food and water, latrines are not carefully constructed, and food is not clean and so often has sand and dust mixed with it.

Microscopic Findings.

One was never satisfied in all the specimens of stools examined that flagellates such as Lamblia intestinalis and Tetramitus mesnili which one frequently saw were a cause of dysentery alone. No case was seen where these existed by themselves, for E. histolytica were always eventually found. But it may be that these flagellates are the primary cause of a dysentery or are responsible for inflammatory changes in the mucosa, and E. histolytica in small numbers might be present as well. Often it was easy to see many flagellates may thus be a
predisposing cause to an Amoebic Dysentery.

Clinical Features.

Happily in Iraq there are very few serious cases, and deaths due to Amoebiasis are rare. In the years 1924 and 1925 there were no deaths due to Amoebic Dysentery alone. Paratyphoid and other diseases of the enteric group, combined with Amoebic Dysentery or Amoebic hepatitis, have caused a few deaths in the last three years.

Malaria of the benign tertian type and occasionally malignant malaria was seen to run concurrently with Amoebic Dysentery now and then. An example is given in the Appendix of Cases of a case of paratyphoid associated with Amoebic hepatitis. These cases in the Appendix are more or less typical of what one might expect to see in Iraq now. The case of Amoebic hepatitis quoted was that of a patient who had done much armoured car convoy work on the frontier and it was likely that he had at some time or other had one of those mild attacks of dysentery which soon subsided and which he did not report. When he contracted paratyphoid he was compelled to come to hospital and the Amoebic hepatitis was discovered.

Very acute cases with great diarrhoea and
obviously extensive ulceration and somewhat gangrenous changes in the large bowel were few. The majority of cases were mildly acute with no high temperature nor marked signs of toxaemia.

**Prophylaxis and Prompt Treatment.**

Despite the primitive sanitation in Iraq most units are carefully looked after and all means are taken to keep down flies and get rid of native carriers. This has good results in the numbers of dysentery cases seen in hospital. If more extensive active service operations were to take place no doubt there would be more patients suffering from Amoebic Dysentery. In this country most of the conditions which exist in Iraq are absent and to this I attribute the fact that one hears of comparatively few cases in England.

Prompt laboratory examination at once and specific treatment quickly started, when cases were diagnosed, made a tremendous difference to results. All doubtful cases of diarrhoea were sent to hospital as soon as possible for the examination of stools. To these measures one can attribute the success in the Royal Air Force hospitals in Iraq in combating the disease in a country where one would expect much more dysentery. Also it is undoubtedly the
reason why so few cases of Amoebic hepatitis and Liver Abscess are encountered. These are noticeably few, and early treatment by emetine no doubt accounts for this.

Recurrent Amoebic Dysentery patients are still met with, but not a big number. Usually a recurrent case is invalided to the United Kingdom, when he is free of E. histolytica, since it is thought that he has less likelihood of getting another attack there, and also it gives him a better chance to regain his normal health.

Emetine-bismuth-iodide has remained the only reliable drug available for use in chronic carriers. Colon lavage of quinine bihydrochloride carefully combined with emetine in E.B.I. form has helped considerably to bring down the number of relapsing cases to a great extent during the last three years.

The problem of Amoebic Dysentery, it seems to me, must ever face military units abroad in hot, very trying countries like Iraq. One holds the view that infection is definitely more liable in those countries and that everything points to favouring its occurrence. If precautionary measures slacken the disease increases by leaps and bounds.
With our climatic conditions as they are in England, with our more or less perfect sanitation, and the comparative absence of carriers (in contrast to their numbers in the East,) Amoebiasis, while ever kept in mind, should not provide us with that anxiety which confronts the European population and especially military personnel in hot countries.
Table 1.
Nosological Table of Dysentery
by Geographical Areas, 1924.

<table>
<thead>
<tr>
<th></th>
<th>Mediterranean Littoral.</th>
<th>Iraq.</th>
<th>India.</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Average Strength 3,491.</td>
<td>Average Strength 3,660.</td>
<td>Average Strength 1,912.</td>
<td></td>
</tr>
<tr>
<td>Dysentery.</td>
<td>Ratio per 1,000 of strength.</td>
<td>Ratio per 1,000 of strength.</td>
<td>Ratio per 1,000 of strength.</td>
<td></td>
</tr>
<tr>
<td>Clinical, primary.</td>
<td>1.1</td>
<td>-</td>
<td>4.1</td>
<td>-</td>
</tr>
<tr>
<td>&quot; recurrent.</td>
<td>0.6</td>
<td>-</td>
<td>0.5</td>
<td>-</td>
</tr>
<tr>
<td>Bacillary, Primary.</td>
<td>0.6</td>
<td>-</td>
<td>-</td>
<td>0.5</td>
</tr>
<tr>
<td>&quot; recurrent.</td>
<td>0.3</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Amoebic, primary.</td>
<td>3.7</td>
<td>-</td>
<td>22.4</td>
<td>-</td>
</tr>
<tr>
<td>&quot; recurrent.</td>
<td>1.4</td>
<td>-</td>
<td>5.2</td>
<td>-</td>
</tr>
<tr>
<td>Total Dysentery.</td>
<td>7.7</td>
<td>32.2</td>
<td>-</td>
<td>4.2</td>
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</table>
Table 2.
Case Incidence of Dysentery at Stations Abroad for years 1923 and 1924.

<table>
<thead>
<tr>
<th>Station</th>
<th>Average Strength</th>
<th>Clinical</th>
<th></th>
<th>Bacillary</th>
<th></th>
<th>Protozoal</th>
<th></th>
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</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Primary</td>
<td>All Cases</td>
<td>Primary</td>
<td>All Cases</td>
<td>Primary</td>
<td>All Cases</td>
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<tr>
<td>Mediterranean Littoral</td>
<td>3,491</td>
<td>4</td>
<td>6</td>
<td>2</td>
<td>3</td>
<td>13</td>
<td>18</td>
</tr>
<tr>
<td>(Egypt, Malta, Palestine)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Iraq.</td>
<td>3,660</td>
<td>15</td>
<td>17</td>
<td>-</td>
<td>-</td>
<td>82</td>
<td>101</td>
</tr>
<tr>
<td>India</td>
<td>1,912</td>
<td>-</td>
<td>-</td>
<td>1</td>
<td>1</td>
<td>4</td>
<td>7</td>
</tr>
<tr>
<td>Mediterranean</td>
<td>4,112</td>
<td>26</td>
<td>35</td>
<td>12</td>
<td>15</td>
<td>9</td>
<td>13</td>
</tr>
<tr>
<td>For year 1923</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Iraq.</td>
<td>3,524</td>
<td>22</td>
<td>26</td>
<td>2</td>
<td>3</td>
<td>63</td>
<td>79</td>
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<td>India</td>
<td>1,896</td>
<td>3</td>
<td>5</td>
<td>1</td>
<td>1</td>
<td>12</td>
<td>28</td>
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</tbody>
</table>
Table 3.
Monthly Case Incidence of Dysentery
Abroad 1924.

<table>
<thead>
<tr>
<th>Command</th>
<th>Mediterranean Littoral</th>
<th></th>
<th>Iraq</th>
<th></th>
<th>India</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Month</td>
<td>Primary Cases. No.</td>
<td>All Cases. No.</td>
<td>Primary Cases. No.</td>
<td>All Cases. No.</td>
<td>Primary Cases. No.</td>
<td>All Cases. No.</td>
</tr>
<tr>
<td>January</td>
<td>1</td>
<td>1</td>
<td>8</td>
<td>8</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>February</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>March</td>
<td>-</td>
<td>1</td>
<td>3</td>
<td>3</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>April</td>
<td>1</td>
<td>1</td>
<td>9</td>
<td>12</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>May</td>
<td>1</td>
<td>2</td>
<td>12</td>
<td>16</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>June</td>
<td>1</td>
<td>1</td>
<td>19</td>
<td>21</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>July</td>
<td>2</td>
<td>2</td>
<td>7</td>
<td>9</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>August</td>
<td>5</td>
<td>7</td>
<td>10</td>
<td>11</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>September</td>
<td>2</td>
<td>2</td>
<td>7</td>
<td>7</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>October</td>
<td>4</td>
<td>5</td>
<td>8</td>
<td>9</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>November</td>
<td>2</td>
<td>4</td>
<td>10</td>
<td>12</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>December</td>
<td>-</td>
<td>1</td>
<td>4</td>
<td>8</td>
<td>-</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>19</td>
<td>27</td>
<td>97</td>
<td>118</td>
<td>5</td>
<td>8</td>
</tr>
</tbody>
</table>
Case 1.

Admitted to hospital on 22.11.24 from Kurdistan where he had been on armoured can convoy work for several months.

The usual diarrhoea had been complained of before admission and the passage of "slime", though he stated that he saw very little blood at any time. On admission there was very little abdominal discomfort and no real diarrhoea, such as had been present eight days before he was sent to hospital.

He was put on a mixture of sodium sulphate every four hours, the mixture being made up of Sodii sulph. drachms 1, acid sulph. dil. mins 15, Tinc. Zingiberis mins. 5, and aq. menth. pip. ad 1 oz. The diet was light and consisted of tea, light biscuits, arrowroot, cornflour and milk puddings.

The stools varied from the almost fluid variety to semi-formed and were examined every morning. Mucus was always present but only slight traces of blood, and microscopically flagellates were numerous more especially Lamblia intestinalis and cysts and Tetramitus mesnili. For ten days the stools were searched daily but no sign of Entamoeba histolytica.
or cysts could be seen. The patient was comfortable during this time and having only two or three motions a day.

On the eleventh day active Entamoeba histolytica were found and he was put to bed on a strictly light diet of tea, a little milk, and light milk puddings and started on a course of capsules of emetine-bismuth-iodide, grains 2 twice daily for ten days, totalling 40 grains. He vomited a little on the third day but the powder was not returned. The stools kept to an average of two to three a day. After finishing the course he was put on a fish diet. Only very slight discomfort was evident throughout the course of emetine, and no cardiac irregularities showed.

One stool was examined during the first week after the course, but no Entamoeba histolytica or cysts were found. The second examination, eleven days after the finish of the course, revealed the presence of free Entamoeba histolytica and the capsules of E.B.I. were started again. A nine days' course totalling 36 grains was given, and thereafter six stools were examined, one the first week, two the second, and three the third. All six stools were negative to E.H. The stools after the second course were practically normal in consistency with
no blood, and only now and then showed traces of mucus. After being given general tonics he was discharged feeling quite fit and comfortable.

Case 2.

A.C.L. H--.  Amoebic Dysentery (recurrent)

This patient had been in hospital five months before with a primary attack, and that time was in eight weeks. After discharge he had had vague abdominal pains now and then, but no distinct diarrhoea.

On admission on 3.11.24 he had considerable abdominal pain and diarrhoea, the stools showing a fair amount of blood and mucus. The temperature was normal, the pulse 84, and he looked pale and anaemic and was somewhat exhausted. The heart and lungs were normal and there was no liver enlargement. Eight motions were passed on the first day. He was started on Sodium sulphate four hourly.

On first examination the stools macroscopically were semi-formed to fluid with a good deal of mucus and a fair amount of blood. Microscopically red blood corpuscles were in plenty as were Lamblia intestinalis and cysts, but no entamoebae histolytica could be seen after a very close search. After four days of close searching active entamoebae
histolytica were found and also cysts, but in this stool there was no obvious blood.

A course of E.B.I. was started consisting of grains two twice daily for ten days. During the course there was only slight discomfort with the drug and the diarrhoea lessened considerably towards the tenth day. After the E.B.I. was finished the average number of stools was about three a day with only a trace of blood and mucus in them. His diet was increased, he was allowed up after two days, and was put on a tonic of Mist. ferri. arsenical oz. thrice daily. Colon lavage of Quinine Bihydrochloride, ten grains to the pint of saline was used on alternate days with the emetine.

There was no liver enlargement and a blood count revealed no marked leucocytosis. Temperature and pulse remained more or less constant throughout, and there was no sign of discomfort in the liver region.

Entamoebae cysts were found twelve days later, though the diarrhoea had stopped and there was no obvious blood or mucus in the stools. Slight pain was more or less constant particularly over the region of the splenic flexure, descending colon and sigmoid colon indicating extensive lesions in the large bowel. This airman was eventually invalided to the U.K. early in 1925.
Case No. 3.

A.C.E. B--- R.A.F. Amoebic Dysentery. (primary)

Admitted on 19.12.24 with abdominal pain and diarrhoea and that day he had six motions.

His previous history and medical records showed he had been in hospital in Palestine a year before with Bacillary Dysentery. He had had it moderately severely, and on the first day of admission then had passed about twenty stools containing much blood and mucus. No entamoeba histolytica or cysts were found on that occasion. It was a Shiga infection and the patient responded well to the anti-dysenteric serum treatment and recovery was good.

On this second occasion in Iraq there was blood and mucus in the stools, and he was sent from his unit to hospital with the usual diagnosis of "Clinical Dysentery". His temperature and pulse were normal.

The stools were searched closely daily and eight days after entamoebae histolytica and cysts were found. He vomited the emetine-bismuth-iodide capsules several times in spite of precautions being taken, so he was put on injections of emetine hydrochloride one grain, daily for five days then started on E.B.I. capsules again. The capsules were then retained quite comfortable and he finished his
course without much discomfort, totalling five grains of emetine by injection and thirty-six grains of E.B.I. by capsule. The diarrhoea responded well to the injections of emetine, and at the end of the whole course it had stopped.

It was not necessary to give him another course, since his progress was steady and all stools for eight subsequent examinations were negative to entamoeba histolytica and cysts. His blood showed a white count of 9,000 per cm. and at no time did he have any symptoms of hepatitis.

The examination of blood and mucus on a MacConkey agar plate showed no sign of a bacillary infection.

Case 4.
Private O---, Beds and Herts Regiment.

1. Amoebic Dysentery (primary).

The patient had not had Dysentery before, but had had a primary attack of benign tertian malaria in India nine months before.

He had arrived from India on the 14th December and next day was admitted to hospital suffering from severe abdominal pain, feverishness, headache and diarrhoea. For two days previously he had had diarrhoea.
On admission to a medical ward he had abdominal pain and a temperature of 101.4°F and a pulse of 124. He vomited several times and his tongue was dirty. There was tenderness in the epigastric region and towards the left costal margin, but no definite rigidity of the abdominal wall. There was also no obvious enlargement of the liver or spleen on palpation. Later in the day he passed several stools which contained a good deal of blood and mucus. On examination these were found to contain free Entamoeba histolytica, and he was transferred to the Dysentery ward. Blood films were taken on the same day, but these revealed no malarial parasites. He was started on hypodermic injections of emetine, one grain daily for four days. Later in the evening his temperature fell to normal.

Next day he was much more comfortable, with no abdominal pain, no vomiting, and his bowels did not open. On the third day he vomited again several times and the abdominal pain returned. The temperature also rose to 99.8°F. There was no cold stage, and blood films were negative to malarial parasites. The bowels remained closed until the evening, when a severe diarrhoea started again and
the stools were typically dysenteric.

On the fourth day the temperature was a little higher but his bowels again did not move. Injections of emetine were stopped on the fifth day, and he was started on E.B.I. but the powder was vomited. Parasites of benign tertian malaria were found in a blood film, however, and he was started on quinine bihydrochloride, grains 10 thrice daily. The bowels still remained closed.

The sixth day found the patient much more comfortable, but two stools were passed each of which contained blood and mucus. Later in the day the temperature rose to 104°F and he did not feel so well. He also vomited the E.B.I. powder again. Next day he was given an injection of emetine.

After this his progress was steady and his temperature came down to normal in two days. The diarrhoea gradually lessened and there was no further rise of temperature. Emetine was continued daily for ten days. Thereafter his stools remained negative for seven days, but entamoeba histolytica cysts were found on the next examination, and he was given a course of E.B.I., grains two, twice a day for nine days, combined with colon lavage with quinine in normal saline on alternate days. On discharge he
was quite fit and the stools were negative to entamoebae histolytica and cysts.

Case 5.

L.A.C. F---. R.A.F. Amoebic Dysentery, (primary)

Admitted as an ordinary typical case of Amoebic Dysentery in November, 1924, but it was not a severe case. The usual symptoms were present on admission, but there was no raised temperature or pulse, and the heart and lungs were normal. The diarrhoea was not very severe, and the bowels opened only six times the first day and the stools contained only a moderate amount of blood and mucus. His general appearance and health appeared to be very good.

He was put on a nourishing but light diet, and had Sodium Sulphate mixture, one ounce four-hourly while his stools were being searched. On the ninth day entamoeba histolytica cysts were found, but no entamoebae. He was put to bed and a course of E.B.I. was started when he was given three grains a day for twelve days. Colon lavage was also given.

With the course started the diarrhoea lessened a great deal and he had only two to three motions of the bowels a day. But his temperature rose on the second day of the course to 100°F in the evening.
with a pulse of 84, and he had vague abdominal pains. A blood film was examined for malarial parasites but none were found. There was no discomfort in the hepatic region. On the third day of the course the temperature was down to about 99°, and the pulse varied from normal to 80. The fourth day the temperature and pulse were normal and he felt quite comfortable. The fifth and seventh days he had further rises and on the latter occasion it went up to 102° with a pulse of 100. There was slight general abdominal pain only, but his diarrhoea was just a little worse, he having had six stools on the seventh day. No malarial parasites were found.

After this and until the end of the course of E.B.I. the temperature remained practically normal, and his general improvement was good. No further sign of entamoeba histolytica or cysts was found, the stools improved rapidly, and on discharge, about one month after finishing the course, he was quite fit and had no diarrhoea.

Case 6.
S/Leader C---. Royal Air Force.

1. Paratyphoid B.
2. Amoebic hepatitis.

This case was admitted to a special medical
ward in January 1925 suffering from general abdominal discomfort, a temperature of 104°, headache, and signs of toxaemia. The patient looked obviously very ill. There was no diarrhoea the day he came in, but the following three days stools were passed which strongly suggested the enteric group, and on one occasion a fair amount of blood was passed.

The patient in the past had refused to be protected from the enteric group by T.A.B. inoculation, and there was no history of Amoebiasis, though he had been a lot on out-post duty on the frontier.

The spleen was somewhat enlarged and tender, but the liver was found to be considerably enlarged. Also there was discomfort in the hepatic region. The liver was three to four inches below the costal margin. Serological examination confirmed a diagnosis of enteric fever, and paratyphoid B. was finally found to be the cause.

The stools continued to be rather loose and blood was present on several occasions. One week after his admission definite cysts of entamoeba histolytica were found, though no entamoebae. Along with his other treatment he was given emetine, one grain daily for ten days. The liver was also explored on six different occasions for hepatic
abscess, but these attempts were all unsuccessful in yielding pus. A month after his admission the liver had subsided a great deal, and was almost normal in size, though some discomfort was still felt in that region.

During the whole of this time his condition was bad, however, and he ran a very high temperature, he was very apathetic, his toxaemia became worse and resistance also, and he died just on two months after his admission.

Case 7.

A.G.I. H---. R.A.F. Amoebic Dysentery. (Primary)

This patient was admitted in November, 1924 and was nearly four months in hospital at the end of which time he was invalided to the United Kingdom. He stated that he had had Dysentery during the War six years earlier in Mesopotamia, but that he did not know what type, and no medical records were available to find out.

When admitted he had only moderate diarrhoea, passing five stools the first day, and these contained blood and mucus in fair amount. He was not a robust looking patient and he was rather anaemic looking. Pain was only slight, the temperature and pulse were normal, and there was no sign of hepatitis. Numerous active entamoebae
histolytica containing red blood corpuscles were found next day in the stools, and a few flagellates of Lamblia intestinalis.

The diet was kept very light but nourishing, he was put to bed, and was given injections of emetine grain one once a day for twelve days. On the fifth day the temperature was up a little, 99.5° and the diarrhoea and abdominal pain were worse. On the ninth day the diarrhoea was very severe and he passed fifteen stools in the twenty-four hours. All these stools consisted almost entirely of blood and mucus, and had a red currant jelly appearance. There was a little bile as well. They had the appearance suggestive of Bacillary Dysentery stools, and specimens were sent to the laboratory for plating. The patient was kept warm with hot bottles and his diet consisted mainly of albumen water, Benger's food, and arrowroot. Bismuth salicylate one dram was given thrice daily in addition to his emetine treatment.

For over a fortnight the diarrhoea kept severe, a lot of blood was passed, the temperature kept up to over 100°, and the patient's condition was rather serious. He became rapidly thinner and more anaemic looking. Several stools which had been carefully examined in the laboratory failed to show
any Bacillary infection. However, at last it was necessary to give an intravenous transfusion of warm hypertonic saline, three pints, to which was added forty cubic centimetres of anti-dysenteric serum. Almost immediately after this treatment he had a short attack of serum sickness, but brandy appeared to remedy this very quickly. Next day he was given more anti-dysenteric serum, twenty cubic centimetres, intramuscularly in the thigh, and the following day twenty more intravenously by syringe. Tincture of chloroform and morphia twenty to thirty minims, last thing at night added a lot to the patient's comfort.

About one month after admission the patient began to mend slowly but steadily. The diarrhoea became less and the stools became more formed and more faecal in colour. No hepatic complications came on, and he began to put on weight slowly. Microscopically the stools showed no entamoeba histolytica or cysts, but macrophages, pus cells, red blood corpuscles were still fairly abundant with now and then a few entamoeba coli. There were no flagellates. Towards the end of his term in hospital, however, entamoeba histolytica cysts were found, and he was given a course of E.B.I. capsules.
Colon lavage of quinine solution was also given. On discharge he was very fit and was a good deal fatter than he had been for years. The stools were negative to E. histolytica and cysts, but he was nevertheless invalided to the United Kingdom.

Case 8.

L.A.C. P---. R.A.F.

1. Amoebic Dysentery (P)
2. Cardiac irregularities.

Admitted in November, 1924, with diarrhoea, slight abdominal pain and passing blood and mucus. He had three motions only on the day he came in. There was no previous history of dysentery. The heart and lungs were normal on examination, he had a temperature of 99° and his pulse was 68. He stated he had had pleurisy in 1918 with effusion and that he was rather subject to sore throats. The tonsils were slightly enlarged. The liver appeared to be normal and he had no discomfort there.

The specimens showed a little bile pigment besides blood and mucus. There were no flagellates and active E. histolytica were not found until eight days had passed. The first day of his course of E.B.I. he vomited the powders in the morning and evening, so next day he was started on injections of
emetine. He took the course fairly well, though attacks of nausea affected him several times. His pulse rate too was a bit irregular, but there was no discomfort otherwise.

About two weeks after he had finished the emetine treatment and after he had been up for nearly the whole of that time, he began to complain of cardiac pain and palpitation. This was more marked at night. He was put to bed. His pulse was irregular in time and varied from normal to over 100, and this was not accounted for by any appreciable rise of temperature. There was no history of acute rheumatism or rheumatic fever.

At the apex area could be elicited a faint systolic murmur, but the heart appeared to be normal in size. No evidence of valvular impairment could be made out at the other areas.

He showed no sign of heart failure. He was given Tincture of digitalis minims x thrice daily for four days after which, with the rest in bed, he improved steadily. Before discharge he felt fit and no bruit could be heard at any area.

Case 9.

Pte. E.---. Inniskilling Fusiliers.

Amoebic Dysentery (P).
Admitted from the frontier town of Kirkuk in October, 1924. He was in hospital nearly three months. He gave a history of having had severe diarrhoea for two days, when he noticed blood and "slime" in his stools.

On arrival at hospital he had very slight diarrhoea only and the stools were semi-formed. Just a trace of blood could be seen, with a little mucus. He felt quite fit and looked a healthy man. There was no raised temperature or pulse, and no real pain.

The specimen first examined contained nothing interesting at all, no flagellates or cysts, only a few red blood corpuscles and other normal contents. It was not till the seventh day after close examination that active E. histolytica were found. Meanwhile the stools averaged three a day and he felt quite comfortable. The entamoebae were not numerous. A white blood count revealed a leucocytosis of 14,000 per cmm.

Treatment.

He was started on E.B.I. in capsules for ten days taking a capsule of two grains twice a day. Along with this he was given colon lavage with quinine bihydrochloride solution (ten grains of quinine to
one pint of saline) on alternate days. There was no vomiting or discomfort and the emetine was taken well. He was allowed up the second day the course had stopped, and lavage was stopped with the emetine.

The stools were free of blood macroscopically and microscopically nothing of interest showed for ten days. Then cysts were found and it was necessary to give more E.B.I. Lavage was left out this time and he was given a longer trial with E.B.I. being put on two grains twice a day for fourteen days. His heart and pulse rates were watched closely during this time and other signs of overdosage were watched for also. He stood the fifty-six grains of E.B.I. very well indeed. The pulse rates remained normal and no cardiac or other complications ensued. He was allowed to get up one week after the second course of emetine had finished.

This case showed remarkable tolerance to emetine. One month after the emetine treatment he was discharged perfectly free of E. histolytica or cysts after fifteen stools had been found negative.

Case 10.

A.C.2. C---.

Clinical Dysentery.
This patient came in November from the northern frontier. He had had fairly severe diarrhoea before admittance, when he was reported as having passed a good deal of blood and mucus. When seen in hospital the diarrhoea had practically stopped, the stools were semi-formed, and the first specimen showed only a mere trace of blood and mucus. He had only three motions the first day.

He was comfortable and complained of no pain whatsoever. The heart and lungs were normal.

Treatment.

A Sodium Sulphate mixture, one ounce, was given every four hours. The specimen examined that day showed no *E. histolytica* or cysts. Macrophages and a few red blood corpuscles were the only things of note.

He was kept on the Sodium Sulphate for two days, then this was stopped. The motions after this fell off until he was having only one a day which was free of blood and contained just a trace of mucus now and then. There was no pain or discomfort and he appeared quite well and fit. For a fortnight specimens were carefully examined once a day but no pathogenic entameobaee or other causes could be found. Flagellates or flagellate cysts
were never found, and after a week no blood or mucus could be seen.

He was in hospital nearly a month and was discharged with the diarrhoea quite gone.

Case 11.

L.C.A. L---. Amoebic Dysentery. (P)

When this patient was admitted in September 1924 he had profuse diarrhoea, a good deal of pain over the caecum and sigmoid colon on gentle palpation, and a slightly raised temperature of 99.5°F. Diarrhoea was frequent (he had nine motions the day admitted) and there was a great deal of blood and mucus in the specimens. His heart and lungs were normal, and he had a pulse of 80.

This patient had been on armoured car convoy work in Kurdistan just previously for several months.

The first specimen showed microscopically numerous flagellates of Lamblia intestinalis, macrophage cells, red blood corpuscles, and active E. histolytica in good numbers. He had already been put on Sodium Sulphate four-hourly and was in bed.

Treatment.

Emetine hydrochloride was started hypodermically one grain daily being given for twelve days. After the emetine, which was equivalent to 36 grains of E.B.I., his diarrhoea had subsided a great deal and
he passed on an average of three motions in the twenty-four hours. In consistency they had also improved and the amount of blood and mucus was less as well. The patient felt tolerably comfortable and pain was only slight.

Two days later he got up and his diet was increased. One week after the course had finished he complained of slight discomfort, but no pain, in the cardiac region. There was no tachycardia. The pulse was 65, but irregularities in the form of extra-systoles were present. There was no sign of valvular complications and the heart was not enlarged. There was no history of heart trouble. He was put to bed and given Tincture of Digitalis minima ten thrice daily for three days. After three days the condition was not noticeable. He was kept in bed, however, for another week.

When he got up the irregularity did not return, and no further trouble was experienced. He was given no more emetine, however, and when he was discharged he was free from E. histolytica and cysts after ten negative stools had been found.

Case No. 12.

A.C. 2. B---. Amoebic Dysentery (P)

Admitted July, 1924, with severe diarrhoea and passing blood and mucus. The usual preliminary
treatment was given. Active E. histolytica were found very quickly on first examination.

He had no raised temperature and the pulse was steady. Heart and lungs were normal. His physique was good, and he took the injections of emetine quite well.

A fortnight after the first course of emetine had finished more active E. histolytica were found. He was then started on E.B.I. three grains a day for twelve days. There was very little discomfort with this total of thirty-six grains, and by the time he had finished he was feeling quite fit and the diarrhoea had stopped. Pain was away and generally he felt much better. The stools were semi-formed to formed and no blood or mucus could be seen.

Four days after the E.B.I. treatment, however, he developed a slight temperature of 100.5° and had a headache. He was put to bed. The pulse was up to 90 and, though he did not feel as fit as usual there was nothing in particular that he complained of. Next day he had a fine branny rash all over his body with a suggestion of a circum-oral pallor. The pulse was 104, but the temperature was normal. The rash looked very like that of Scarlet Fever.
He felt much more comfortable however, and no sore throat or other signs of Scarlet Fever were present.

The third day the rash had gone and he felt quite fit and well. The heart had behaved in a normal manner except that the pulse rate was raised on the first two days. He was given Tincture of Digitalis for three days M.XV. twice daily, and kept in bed. After a week more he was considered to have recovered from the mild attack of emetine poisoning, and he was allowed up.

He was finally discharged free from E. histolytica.
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Rogers - Dysenteries, Their Differentiation and Treatment.

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Walker, Adams and Savage - Convalescent Paratyphoidal and Dysenteric cases considered from the preventive standpoint. B.M.J. August, 5th, 1916.
CASE 4. AMOEBAIC DYSENTERY (P)  
MALARIA B.T. (R).

TEMPERATURE CHART.

DECEMBER, 1924.

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Case 8. Amoebic Dysentery (P).

Temperatue Chart.

**November 1914 - January 1915**

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**CASE 7**
A.C.I.H.

**Amoebic Dysentery, (P).**

**Temperature Chart.**

**November 1924.**

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**Pulse Rate:**
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**Motions per 24 Hrs.:**
5 9 10 14 16 13 15 11 15 15 20 15 18 12 10
Case No. 1 in Appendix (A.C.R.)

A. E. Histolytica (with R.B.Cs.).
B. " " "
C. Cyst of Lamblia Intestinalis.
D. Spiroforma Eugyratum.
E. Red Corpuscles.
CASE NO. 11 IN APPENDIX. (L).

1. ACTIVE E. HISTOLYTICA WITH R.B.CS.
2. '    '    '
3. FLAGELLATE OF LAMBLIA INT.
4. MACROPHAGE CELL.