transportation of pigment by wandering cells and the spread of infection in wounds. The spread of infection is due to a breakdown in one or more of the factors on which phagocytosis depends. Either the wandering phagocytes do not emigrate into the invaded area in sufficient numbers, or, having emigrated, they do not ingest the organisms, or the breakdown of the defensive mechanism may occur at a later stage. The phagocytes may emigrate and successfully intern the invading organisms, but they may fail to digest them, or things may go wrong during return-immigration. The organisms may be liberated by the leucocytes while still alive, just as the pigment granules may be liberated, and they may retain sufficient virulence to originate a recrudescence of infection in a new area. It is quite likely that some part of the auto-intoxication which attends the too free movement of infected limbs may depend on a liberation of ingested organisms brought about by injury to germ-containing leucocytes rather than to a mechanical pumping of the organisms or their toxins into the tissues.

Practical Conclusions.

If it be true that some part (how large a part is at present unknown) of the spread of infection in wounds is due to a breakdown in the defensive mechanism of phagocytosis, of which return-immigration is only one phase; if, in other words, a certain number of pus cells in infected wounds re-enter the circulation or the tissues carrying ingested but still living organisms with them, and if a certain proportion of the latent infection in sinus cases and the recrudescence of sepsis which breaks out in healed wounds is due to this same cause, then any method of wound treatment to be successful must deal with the problem of the return-immigration of germ-laden phagocytes. The subject is one, however, of great delicacy and difficulty. If antiseptic solutions when applied to wounds are sufficiently strong to kill the phagocytes and their cargoes, and so prevent their re-entrance into the tissues they will also inhibit emigration and phagocytosis. The irrigation of a wound with normal saline favours emigration and ingestion, but it does not prevent return-immigration. The application of hypertonic saline, on the other hand, may prevent return-immigration by killing the leucocytes, but it also checks emigration and phagocytosis.

A few experiments have been carried out to test the effect of various antiseptic agents on the capacity to ingest pigment or starch by the leucocytes and pus cells exposed to the action of such reagents when applied to wounds. The pus from a case of compound fracture of the tibia in which a cup-shaped granulating wound was dressed with the mixture of iodoform, bismuth subnitrate, and liquid paraffin known as "Bipp," to which also some sterilized rice starch had been added, showed that the percentage of leucocytes containing starch grains was reduced, while the proportion of starch grains free in the discharge was increased, although a considerable proportion of the pus cells from the same wound retained their vitality.

It is interesting to note that the starch grains in the pus from wounds which have been treated with the iodoform, bismuth, and paraffin paste are colourless. The grains in the interior of the phagocytes and the free grains only show the blue colour characteristic of the presence of free iodine after that reagent has been added. This bears on the question of the liberation of iodine in wounds dressed with iodoform paste.

Samples of pus from wounds previously dressed with gauze soaked in Dakin's solution (Dautresne's formula) with the addition of a little rice starch show that the inhibitory effect on the ingestive capacity of the leucocytes is less marked than in pus from wounds dressed with the iodoform paste. In one superficial granulating and healing wound, dressed twice daily with soaks of Dakin's fluid, nearly all the starch grains were ingested by the leucocytes in the fibres of the gauze dressing.

Certain facts stand out prominently in this world-wide and still unsolved problem of the routine conversion of infected into sterile wounds in war. An experiment in immunization is now being carried out in Europe on a colossal scale. The control of the virulent infection set up in the wounded tissues is far and away the biggest factor in the problem of the treatment of wounds in war. One is struck by the rapidity and efficiency of the immunizing process as carried out by Nature in some cases

and by the failure of it in others. The difficulty is that we cannot distinguish beforehand those cases in which it will succeed from those in which it will partly or completely fail.

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OBSERVATIONS UPON DYSENTERY CARRIERS.

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DURING the winter of 1915 several hundred typhoidal dysentery cases, in varying stages of convalescence, came under our observation for the purpose of eliminating, by bacteriological examination, those carrying infection. The methods we adopted in the routine examinations were the same in all cases no matter what was the original diagnosis accompanying the patient from overseas. We were led to adopt this uniformity of procedure as many of the cases had been diagnosed at the outset on clinical grounds only, and cases labelled "typhoid" or "paratyphoid" we found on examination to be dysentery.

The urine and faeces of all cases were examined and the procedure was as follows: The morning specimen of urine was obtained, the patient being instructed that while micturating he was to collect the latter portion in a sterile bottle. This was then centrifugized and the deposit planted out upon plates of MacConkey's bile salt neutral red lactose agar. For faeces a portion was emulsified in ordinary broth, and, after standing, the emulsion was plated upon MacConkey's medium. A small quantity of a second portion of the faeces was emulsified in double-strength Gram's iodine and then a drop of the emulsion was examined microscopically for protozoa. Sometimes an emulsion in saline was examined in addition. After forty-eight hours' incubation the plates were examined and likely colonies subcultured into broth, agar, and various sugar media. Finally, probable cultures were submitted to macroscopic agglutination tests with known serums.

All the cases were examined many times over so as to ensure that no carrier would slip through the net. This involved a large amount of work, as during the winter six months we made about 5,000 examinations altogether, but when the work is organized it can easily be overtaken.

BACILLARY CARRIERS.

We have only met with six cases carrying the *Bacillus dysenteriae*. Three were carriers of *B. dysenteriae* "Y" and three of *B. dysenteriae* Shiga. One of these latter was a combined infection with *Entamoeba histolytica*.

Of the three "Y" infections two of the patients contracted the disease in Gallipoli, and only one gave a history of typical dysenteric symptoms with blood and mucus in the faeces. His illness began in August, 1915, at Gallipoli; he had a relapse in the following October, and we recovered the "Y" bacillus from his faeces on January 28th, 1916.

The second case of "Y" infection contracted in Gallipoli had its onset about the beginning of October, 1915, with general malaise and fever; a week later the bowels became loose for four or five days only, during which period the patient was, at times, only partly conscious, but there was never any blood or mucus at any time, and the diarrhoea never recurred. On January 25th, 1916, we recovered the "Y" bacillus from the faeces, and on February 22nd and March 17th we again isolated the same organism from the faeces. Four examinations later, in April, gave negative results. The third case of "Y" infection is unusually interesting, for the man apparently contracted his infection in hospital. He was attached to a hospital in Lemnos as an orderly and was frequently on duty in the dysentery ward. He had not been anywhere else, and about the middle of October, 1915, after three months' duty in hospital, he was taken ill. For two to three weeks he was fevered and felt ill, but the symptoms were very indefinite and there was nothing to suggest that he was suffering from dysentery. The motions were normal and the bowels tended to be constipated, requiring enemata every second day during the first three weeks of his illness. Five weeks after the onset of his illness he was transferred home, and on January 25th, 1916, we

recovered the "Y" bacillus from his faeces. This patient has never had diarrhoea at any time, the bowels being constipated throughout his illness.

Of the three cases carrying *B. dysenteriae* Shiga, all contracted the disease in Gallipoli, and the symptoms in each were more or less typical of dysentery, diarrhoea with blood and mucus being present.

It is curious that the case with mildest symptoms was the one having the combined infection of the Shiga bacillus and the *Entamoeba histolytica*.

This patient also carried *Entamoeba coli*. His illness began about the end of August, 1915; mucus was present in the faeces, but only a little blood, and he never passed more than six motions per day, and frequently only two. The diarrhoea stopped about two or three weeks later while in hospital at Malta, and he was sent home. He never had any emetine. In October, while in hospital in this country, he had a recurrence of the diarrhoea for a few weeks. On January 22nd, 1916, we found the Shiga bacillus in the faeces, and also a large number of cysts of *Entamoeba coli*; later, in February, we found cysts of *Entamoeba histolytica*.

The second case of Shiga infection contracted his illness on August 10th, 1915, and it persisted with slight and varying improvement until his arrival in this country at the beginning of December. Slight jaundice developed a week before landing, but he made rapid progress in this country and soon became convalescent. On January 28th, 1916, we recovered the Shiga bacillus from the faeces.

In the third case carrying *B. dysenteriae* Shiga the illness began about the beginning of August, 1915, and the patient was invalided home to England on September 18th. The dysentery did not improve until after admission to hospital in this country. After dismissal from hospital at the beginning of October, the dysentery recurred, and he was readmitted on the 18th. He again improved, and left on December 6th for duty. He had another relapse, however, and was readmitted to hospital on January 10th, 1916. At the beginning of March we recovered the Shiga bacillus from the faeces; a few cysts of *Entamoeba coli* were also found.

With the one exception noted above, no *Entamoeba histolytica* cysts were found in the faeces in any of the cases, although frequent search was made, and no other protozoa except the *coli* cysts in the two cases mentioned.

In five of the cases we examined the blood for the presence of specific agglutinins. Of the three Shiga cases, only one gave a positive result; this was the third case described above, and on March 10th his serum gave complete agglutination at a 1 in 15 dilution only, and partial down to 1 in 40 dilution. The two "Y" cases, tested at the end of February, gave a curious result. Both failed to agglutinate their own organisms in dilutions 1 in 20, but with a known strain of the "Y" bacillus they gave positive reactions, in one case complete down to 1 in 500, and in the other complete only down to 1 in 80.

The length of time these cases carried the bacilli varied from four to seven months.

HISTOLYTICA CARRIERS.

Twenty-four cases infected with *Entamoeba histolytica* have come under our observation, including one combined with *B. dysenteriae* Shiga, and two combined with *Lambliia intestinalis*; several also were found to be carrying *Entamoeba coli*. Fourteen cases contracted their infection in Gallipoli and four in Egypt, one in the Cameroons, one in India, one in South Africa or India, and another in Central Africa; one man contracted his infection in Lemnos, and another in England.

All the cases except three gave histories of more or less typical attacks of dysentery—diarrhoea and tenesmus, associated with the passage of blood and mucus.

Of the three atypical cases, one was only taken ill two weeks after arrival in this country from Egypt. He had been quite well in Egypt, and was invalided home for defective vision; after being two weeks in hospital at Netley he developed frontal headache and constipation, and the condition was thought to be enteric fever; he was kept in bed for three weeks. On examining the faeces three weeks later we found cysts of *Entamoeba histolytica*. It is possible that he may have acquired his infection at Netley, but it is more likely he became infected while in Egypt, and, although free from symptoms, carried the infection home. He never at any time had typical dysenteric symptoms. The second atypical case was that of a man who acquired the infection in Gallipoli at the beginning of November, 1915. He suffered from feverishness, headaches, pains in the back, and some vomiting, but he never had diarrhoea at any time, the bowels, in fact, being very constipated. Seven months after the onset of infection we found him to be carrying *histolytica* cysts. The third atypical case was one in which we found some difficulty in eliciting reliable information. The man had been in Central West Africa about eight years previously, but denied

having had dysentery there, although he had suffered from indigestion and colicky pains. He had been in France during the latter half of 1915, and was quite well, but had to be sent home about the end of December with an injury to his leg. He was at his own home in the South of England when he was taken ill with vomiting, continuous diarrhoea, associated with the passage of mucus, but no blood. He was admitted to hospital on January 31st, 1916, and rapidly improved under a course of emetine hypodermically. On February 3rd we found *histolytica* cysts, but only in small numbers.

Of the typical cases several showed some features worthy of further mention. One was that of a man who had never been out of England, and who contracted an acute attack of amoebic dysentery while living in contact with a number of soldiers who had all been abroad at some previous time, and several of whom had had dysentery. This case has already been recorded in full elsewhere,¹ and it suffices for us to say that we found active *Entamoebae histolyticae* in the faeces; he recovered completely under a course of emetine hypodermically, and *histolytica* cysts did not persist in the faeces. There can be little doubt that he became infected from some carrier among the soldiers with him.

A somewhat similar mode of infection is illustrated in the case of a camp orderly in Lemnos.

He had been in Egypt for three months before being transferred to Lemnos, but enjoyed good health there. He was orderly corporal in the camp, where there was a large number of dysentery cases, and part of his duty was to supervise the latrines. Three or four weeks after arrival in Lemnos he was seized with a typical attack of dysentery. His condition, however, was said to be enteric, and, slowly improving, he arrived in England at the end of August, 1915. He was in an enteric hospital for some time before being sent on furlough, and never at any time received any emetine. Returning to his dépôt he felt rather weak, with dizziness and palpitation, but there was no return of diarrhoea. In April, 1916, fully ten months after the onset of the dysentery, we found *histolytica* cysts in his faeces, but his serum failed to show any agglutinins to *B. typhosus*, *B. paratyphosus* A, *B. paratyphosus* B, or any of the dysentery bacilli.

The patient who contracted his infection in the Cameroons has an interesting history.

Going to the Cameroons at the outbreak of war he was quite well till June, 1915, when he was taken ill with a typical attack of dysentery. The symptoms came on a short time after drinking from a small brook, and other sixty men who drank from that stream went off sick with dysentery at the same time. The patient stated that the bodies of dead natives were found later buried further up the stream. Receiving only two injections of emetine in Africa he was invalided to England, where he remained in hospital from July to October, 1915, before his condition improved. In October he went to France, and at Christmas the dysentery recurred, but he did not report sick with it till March, 1916. He was then admitted to hospital and sent back to England. In April, May, and June we found him to be carrying cysts of *Entamoeba histolytica* and *Lambliia intestinalis* in the faeces.

Two other of the typical cases are worthy of mention.

The first of these two patients contracted a typical attack of dysentery in Gallipoli in June, 1915, and five days after the onset he received a shrapnel wound in the leg, for which he was sent to Malta. The diarrhoea seemed to stop immediately after wounding, and did not recur except for three or four days in July, when it was associated with vomiting. Abdominal pains, however, persisted from the start, and they were of a colicky nature, chiefly over the colon. On August 17th his appendix was removed, and he was afterwards sent home to this country as a convalescent appendix case. In the middle of December he had a relapse of dysentery, associated with similar abdominal pains, and diarrhoea, alternating with constipation, persisted till February, 1916, when he was admitted into hospital here. Finding cysts of *Entamoeba histolytica* in the faeces, we put him on a course of emetine hypodermically, and he made an uninterrupted recovery. He had never had emetine before.

The second patient also contracted his infection in Gallipoli, and typical symptoms began in July, 1915, whereupon he was sent to Egypt, and he improved under emetine. Returning to duty at a base camp, he had a relapse, and was admitted to another hospital in Egypt. The symptoms were typical of dysentery, and were associated with a good deal of abdominal pain.

He was given a prolonged course of emetine, but the first few doses only appeared to do good, and eventually abdominal section was done in the middle line above the umbilicus. He was thereafter sent home, and the dysentery got worse on the ship. He improved in hospital in this country, but in March, 1916, after rejoining his dépôt, he had another relapse. At this time we found active *Entamoebae histolyticae* in his faeces, and put him on a course of emetine hypodermically, 24 grains in all. We noted improvement after the first few doses only, and for about a fortnight the amoebae disappeared from the faeces. Active forms of the amoebae reappeared, and were associated

with an exaggeration of the dysentery. He was then put upon pulv. ipecac. and pulv. ipecac. co. administered by the mouth, and he made undoubted progress under this form of treatment. For a month after this treatment we failed to find any amoebae or cysts in the faeces. We understand, however, that cysts have since appeared, and it seems likely that the infecting *Entamoeba* are of an emetine-fast strain.

In connexion with these two cases we may mention that the patient previously noted above who contracted his illness in this country had colicky pains in the abdomen for five or six days before the onset of the typical diarrhoea, and a surgical specialist who saw him at this time advised operation for duodenal ulcer; but the onset of diarrhoea and the discovery of *Entamoeba histolytica* in the faeces gave the indication for the successful medicinal treatment with emetine.

The next two cases illustrate the long persistence of the infection and its eventual spread to the liver.

The first patient had been in the South African war from 1900 to 1902, and suffered from "dysentery" there. He was transferred to India for the next five years, during which period he had three separate attacks of dysentery, and in 1907 he was invalided home with "dysentery and a sluggish liver." In 1908 he returned to India, and being in fairly good health he remained till 1914, when he was transferred to France. In February, 1915, he had to be sent home because of "rheumatics" and haemorrhoids. The haemorrhoids were removed, and he remained well until April, 1916, when he began to suffer from pains in the right side over the liver. The liver was found to be enlarged below the costal margin, a tender smooth swelling was obvious in front, and a liver abscess was diagnosed. There was intermittent fever and the bowels were loose, but there was no real "dysentery." We found *Entamoeba histolytica* cysts in the faeces, and the patient was given a course of emetine hypodermically. The hepatic swelling disappeared, the liver diminished in size, and the patient got quite well, although he was still passing *histolytica* cysts three weeks later.

The second case of liver abscess had a less fortunate termination. This patient had had dysentery in India in 1905, and while in France he had a recurrence for three weeks in November, 1914, which was associated with an attack of "pleurisy." In September, 1915, he was admitted to a hospital in England for pain in the right side of the chest; he lost flesh considerably, and in the following month was transferred to the Royal Herbert Hospital, Woolwich. Dullness was found in the right subaxillary region with little or no breath sounds and some pleural friction. Some fever was present, but there was no diarrhoea except for five days at the end of October. The liver dullness increased, and on January 18th, 1916, liver puncture was done in four places with negative result. A subsequent puncture on January 26th was successful and pus was obtained which was found to contain active *Entamoeba histolyticae*. We searched the faeces on three separate occasions previous to this and failed to find any protozoa, and we only found *histolytica* cysts in the faeces after demonstrating the active forms in the liver pus. The liver abscess was drained, but the patient did not improve and gradually became weaker and died.

The length of time these cases carried *histolytica* cysts varied from two and a half months to thirteen years. Less than half of them received emetine in the early stages of their illnesses, and some of these only had a few doses. The case occurring in England, which came under our notice from its commencement, received thorough emetine treatment and apparently became completely free from the infection. Thorough emetine treatment from the beginning of the illness, controlled by repeated microscopic examination of the faeces, seems to us the most hopeful method of preventing the chronic carriers.

While the patient is carrying the cysts there is always the liability to recrudescence of the acute disease and, more serious still, the development of liver abscess. Again, there is the danger of transmitting the infection to other people, which, although slight in this country, is possible, as shown by the case of the man who had never left England.

In only ten of these *histolytica* cases did we have time to test the agglutination reaction of the patient's serum with *B. dysenteriae* "Flexner," "Shiga," and "Y." All gave negative results except two. These showed positive reactions with *B. dysenteriae* "Y" only, one in dilution 1 in 80, the other in 1 in 40. These dilutions are probably too high to be indicative of infection.

LAMBLLIA CARRIERS.

In a previous paper² we recorded in detail twelve cases of infection by *Lambllia intestinalis*. Since then we have met with another eleven cases in which *Lambllia* was the sole organism isolated. It seems to us there can be little doubt that *Lambllia* may of itself give rise to considerable

intestinal irritation and consequent diarrhoea. In each of these cases we have examined the excreta many times without finding any other parasite. In the majority of the cases encysted forms only were found, but in a few, when diarrhoea was present, active free-swimming forms were found. The infection is a very persistent one lasting several months, and slight intestinal symptoms may be present all the time, or there may be exacerbations, and at these times active forms of the flagellate appear in the faeces. In some cases the patient states he is quite well, but on questioning him it will be found that an excitable condition of the intestinal tract is present. He has to be careful with his food; if he takes anything hot—for example, hot fluid—then active peristalsis is set up, and he passes a loose motion shortly thereafter.

The onset of the illness in all cases resembled an ordinary attack of acute dysentery—diarrhoea with, as a rule, passage of blood and mucus, and frequently tenesmus. The acute symptoms persisted for a varying length of time—one to six months—and relapses were common. It is, of course, quite possible that at the outset a double infection with *histolytica* was present and that the amoebae had disappeared under treatment with emetine, which had been given in most of our cases, and that the persisting *Lambllia* kept up the intestinal irritation. But the evidence in our series of cases is all in favour of the conclusion that *Lambllia* of itself can give rise to dysenteric symptoms. The following case is sufficiently illustrative:

A young officer who had previously been a medical student was taken ill in Gallipoli in the early part of June, 1915. He had had diarrhoea with, at first, no blood or mucus, and he controlled it by taking lead and opium pills, with which he had supplied himself before proceeding to the peninsula.

He continued to take the pills until he was wounded about three months later, when he was sent on hospital ship to Malta. As he stopped taking the pills the diarrhoea got worse, and blood and mucus appeared in the faeces. He was a month in hospital in Malta, where he was treated with milk diet and bismuth but no emetine. There was some improvement in the dysentery before he left Malta, but on the ship coming home to this country it recurred as bad as ever, and abdominal pains were severe. After a short time in a London hospital he was sent home on leave. Loose motions persisted all the time, but on only two or three occasions did blood or mucus appear. Early in February he was sent into the Royal Herbert Hospital, Woolwich, by a medical board on account of abdominal pains and diarrhoea associated with the passage of mucus but no blood. On February 12th a specimen of faeces was examined by us, and we found large numbers of *Lambllia intestinalis* present, both active and cystic forms. No other protozoa were found, and no organisms of the typhoid-dysentery group were isolated. The patient was put upon a course of emetine hypodermically, but, although this alleviated the abdominal pains, it had not the slightest effect upon the diarrhoea or the *Lambllia*. An interesting point, by the way, is that the emetine during its administration caused a temporary loss of memory for recent events—for example, the patient forgot his own home address.

Beta-naphthol gr. xv and bismuth salicylate gr. xx were given thrice daily in several seven-day courses, but this had practically no effect. The *Lambllia* cysts disappeared for a few days after each course, but reappeared later when the temporary constipation passed off, and on one occasion active forms of the flagellate came back. A dose of calomel would produce violent diarrhoea with blood and mucus.

We have examined the faeces of this patient on numerous occasions up to the beginning of June, 1916, and practically every time have found encysted forms of *Lambllia* present. No other protozoa were seen, and at no time did we isolate any typhoid, paratyphoid, or dysentery bacilli. The patient, who has never felt fit since his illness began, does not look well, and, twelve months after onset, is still passing two or three loose motions daily.

The majority of our cases contracted their infection in Gallipoli, a few in Egypt, one apparently in France, and another in India ten to fifteen years previously, when he had an attack of true dysentery. This last patient was admitted to hospital for an indefinite illness, with fullness and pains in the abdomen. The motions were loose, and contained mucus, and active forms of *Lambllia* were present in large numbers. A commencing liver abscess was suspected, and the patient improved under emetine, but encysted forms of *Lambllia* persisted in the faeces.

We have tried several drugs in the treatment of the chronic *Lambllia* carriers, but we have not yet found a specific. Emetine hypodermically has not the slightest effect, and neither has the internal administration of beta-naphthol combined with bis. salicylate; pulv. ipecac. with pulv. ipecac. co. given by the mouth also produced no effect on the cysts. - Latterly we have tried naphthalene

by the mouth in keratin capsules, but this also has failed. We have found most promise of success in the internal administration of small doses of calomel over a prolonged period associated with a mild morning saline.

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A CASE OF AMOEBIIC ABSCESS OF THE LIVER OCCURRING TWENTY YEARS AFTER THE ORIGINAL ATTACK OF DYSENTERY.

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CLINICIANS have often noticed that abscess of the liver may follow many years after an attack of dysentery, or even appear with no history of dysentery at all. Such occurrences have, up to the present, been very puzzling, but recent work carried out on cyst carriers helps largely to explain them.

It has been shown that a person may become a contact carrier and present *Entamoeba histolytica* cysts in his stools without ever having had dysentery or even diarrhoea, and, further, that a person who has had dysentery may become a relapsing carrier and go on discharging cysts regularly or irregularly for many years. Either of this class of case may develop an abscess in the liver at any time, and there is no reason why they should not, as the presence of cysts in the stools indicates that they are still the subjects of amoebiasis. What is still difficult, however, to explain is why some cases develop abscesses and others do not. There would even seem to be a geographical predilection, because in India and other places, for example, they are common sequelae of amoebic dysentery, while in other parts of the world, though there is plenty of dysentery, they are distinctly rare. Further, why should a man the subject of amoebiasis live for years and then suddenly get liver symptoms and develop an abscess? These are points that still require solution.

Recently I have had the opportunity of fully studying a very interesting case of amoebic liver abscess appearing years after a dysenteric attack, and as it raises many points of interest no apology is necessary for publishing it.

A ship's chief engineer, aged 54, was admitted into the Seamen's Hospital, London School of Tropical Medicine, under my care, complaining of pain over the liver and stomach, with vomiting and hiccough.

History.

He was born in England. He went to sea at the age of 22, and has followed the sea ever since with the exception of three years, when he lived ashore in England. He has travelled all over the world—to India, Australia, South America, but not to Africa. In his time he had drunk a fair amount of alcohol, but lately had been more temperate. He had had gonorrhoea in his youth, and, at the age of 25, a chancre, for which he was never treated; apparently he had no secondaries. He had had malaria several times, slight attacks. He had dysentery twenty years ago at Port Said, with typical clinical symptoms—blood and mucus, tenesmus, straining, etc. He was treated in hospital there, and got better in a week or two. After that he had slight touches of diarrhoea for two or three years; thereafter all signs of bowel irritation disappeared. He is perfectly certain that he never had dysentery again—that is, a fresh infection. In September, 1915, he passed blood. This was found to be due to piles; he was operated on and cured.

Present Illness.

Nine weeks previous to admission to hospital he began to get feverish attacks, with general aching all over the body, diagnosed as influenza and treated as such. He had also slight vomiting. A week later he developed ague attacks, shivering followed by sweating, with headaches; this lasted four weeks. He was treated by his own doctor with quinine. A week later hiccough developed, with pain in the epigastrium and under the right costal margin. As his condition did not improve, and as increasing weakness and loss of weight also became apparent, he was sent into hospital, arriving there on July 5th, 1916.

State on Examination.

The patient's mouth was in a very septic condition, an extreme degree of pyorrhoea alveolaris existing. The tongue

was dry and coated, and there were numerous small ulcers in the mouth. The temperature was 101°. There was nothing abnormal in the lungs, and no dullness at the right base. The heart was a little enlarged, the sounds were very faint (? fatty). The pulse was irregular, missing beats regularly, rate 90 to 100. Abdomen: Pain on pressing epigastrium; liver enlarged, upper border fifth and sixth ribs, lower border three inches below costal margin; spleen 9 to 11, not enlarged. No tenderness over colon, nothing in groins. Urine: Specific gravity 1020, no albumin, blood, or sugar. Faeces: No amoebae, cysts, or flagellates; no ova.

Blood examination:

Red cells	...	...	...	...	4,800,000
White cells	...	...	...	...	14,000
Haemoglobin	...	...	...	...	100 per cent.
Differential count:					
Polymorphonuclears	...	...	...	...	60 per cent.
Large mononuclears	...	...	...	...	11 ..
Lymphocytes	...	...	...	...	27 ..
Eosinophile	...	...	...	...	1 ..
Mast cells	...	...	...	...	1 ..

No malarial parasites or pigmented leucocytes.

Diagnosis.

Hepatitis and gastritis, probably septic in nature.

Treatment.

All his teeth were removed at once; Mr. Vaughan, who did this, agreeing that the trouble might all be coming from the mouth condition.

Progress.

July 6th. A little better. July 10th. Temperature keeping up, two or three small rigors. Feels better, however. Faeces: No cysts or amoebae. July 13th. Much better; no more rigors, no sweating; temperature down a little. Leucocytes 14,000. Faeces: Again negative.

July 17th. Worse, rigors again increasing in severity; pain over liver on shaking him. Tender on deep pressure over lower axilla (? a liver abscess starting). Leucocytes, 15,000; no malarial parasites. Faeces: Again negative. Give him emetine, beginning to-night, one grain; mouth quite clean now. There does not seem to be any source of infection there.

July 18th. A dead amoeba and two *E. histolytica* cysts found in faeces to-day. This clears up the diagnosis and verifies the suspicion of an abscess forming in the liver. No rigor to-day.

July 20th. Temperature still raised; no further rigors, however, and no more sweating. The patient feels more comfortable, and says he has lost the liver pain. Sick in the afternoon. Decided that surgeon should see him and explore his liver for an abscess the following morning. No further sickness after afternoon, but pulse became very irregular in evening.

July 21st. Seized with a sudden heart attack at 2 a.m. and died at 2.45. Owing to the irregularity of his heart, and also because of its weakness, the patient had been on digitalis since July 14th.

Table giving Details of the Emetine Treatment and Results of the Examinations of the Faeces and the Leucocyte Counts.

Date.	Dose and Drug Employed.	<i>E. histolytica</i> Free.	<i>E. histolytica</i> Cysts.	Leucocytes.
1916.				
July 5	..	—	—	14,000
" 10	..	—	—	
" 13	..	—	—	14,000
" 17	Emetine hydrochloride subcutaneously	—	—	15,000
" 18	Ditto ..	+	+	
" 19	Ditto ..	—	—	
" 20	Ditto ..	—	—	16,500

The temperature is shown in the chart.

Autopsy.

The patient died at 2.45 a.m., July 21st, 1916. Post-mortem examination 4 p.m. same day.

External Appearances.—Body well nourished; good layer of fat; old scars on legs look like specific; no scars on penis; otherwise nothing to note.

Head, neck, and spine not examined.

Thorax.—Pleurae: Right, no fluid, adhesions between lung at apex; left, no fluid, no adhesions. Right lung, 1 lb. 9 oz., adhesions at apex; no signs of tubercle, however; body of lung congested, and lower lobe oedematous; no pneumonia. Left lung, 1 lb. 6 oz., similar to right.

Heart.—Pericardium: No fluid, adherent to wall of heart by old fibrous adhesions. In addition to this, a recent exacerbation of acute pericarditis with fresh lymph. Muscle substance very soft and flabby, advanced fatty degeneration. Valves: Nothing abnormal on right side. On left, aortic nil; mitral, old sclerosing endocarditis on both cusps. Aorta: Early atheroma.