DYSENTERY—A CLINICAL STUDY.

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The following report is based upon the records of some 600 cases which were admitted into the dysentery wards of the General Hospital at Alexandria, between May, 1916, and February, 1917, and were under the care of the writers.

The cases were of very different character; some were admitted into hospital on their arrival from England acutely ill. Some, also acutely ill, came from local camps on the Western Front; while others were admitted from Salonica, India, Mesopotamia, or other hospitals in Egypt, more or less convalescent, suffering from acute relapses, or with chronic dysentery.

A certain number of patients were suffering from their first attack of dysentery. Others had suffered previously, and in a few the disease was of old standing, and must have been present for many months. The majority were otherwise healthy, but a minority were infected as well with malaria and paratyphoid fever; or had previously had attacks of these diseases, or sunstroke, sandfly fever, etc.; while in another group visceral disease of varying kind co-existed.

The cases were thus in no way comparable and we have in consequence laid little stress upon statistics. We may, however, state that entamoebae were detected in the stools in more than 58 cases, a dysentery bacillus in 132 cases, and lamblia in 50 cases.

One is apt to forget that dysentery—the bloody flux—is a symptom of many diseases rather than a disease in itself, although at times epidemics of dysentery of uniform origin may occur. The type of the infection may, however, vary somewhat suddenly even in the same place. At Cape Helles for instance, amoebic dysentery was rife during July, August, September and October; while at Suvla Bay the main cause was bacterial (Captain W. Campbell). The interchange which occurred among the troops on the Peninsula, however, led to cross infections, and before the evacuation the cases in either place might be bacterial or protozoal; or a double infection might be present.

The movement of troops in this world war is so extensive that diseases indigenous to one place may be readily imported into another. So that in medicine as with arms all ante bellum standards have been upset and war conditions necessitate an avoidance of a priori theories. Colonel Sandwith,
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for instance, tells us that it has been anticipated that the dysentery in Alexandria in the autumn would be chiefly protozoal whereas it has been mainly bacterial. Captain Lumb tells us that while the dysentery in East Africa prior to the war was wholly amoebic, the bacillary affection has been frequently seen there since the arrival of the Indian troops in the autumn of 1914. One of us had a patient under his care in the autumn of 1914 who came into hospital from the trenches on the Aisne, suffering from amoebic dysentery; and cases have been recorded where it has occurred in England in men who had never been away from home. The persistence and the latency of protozoal infections is responsible for a group of cases where an infection acquired in one country shows evidence of its existence when the carrier has reached the other side of the globe. The “Aisne” patient, for example, had had an attack of dysentery at Lahore eighteen months previously, and the infection had probably never been exterminated although all the symptoms remained in abeyance. Comparable histories are, of course, well known to occur in such diseases as malaria and syphilis.

This point cannot be too strongly emphasized as successful treatment of dysentery can only be ensured by an accurate diagnosis of its cause in the individual case. This can rarely be made in the wards. Laboratory findings are of great value, but pitfalls also exist in the laboratory, and its negative reports are of little value. The history of the illness, previous illnesses, and the itinerary of the patient are probably of as great practical importance; but all these factors, the history, the symptoms, and the laboratory reports, must be carefully correlated to ensure a correct solution. Even then one is often in doubt, and empirical treatment, with all its disadvantages, has to be instituted.

AMOEbic Dysentery.

The protean character of the symptoms of amoebiasis of the intestine is well known, but is not perhaps sufficiently recognized. A very large percentage of the population where amoebic infections are endemic are carriers, with presumably a small local lesion in some part of the bowel so trivial in its degree that no evidence of its presence exists during life. Captain Dunne tells us that he saw several such cases (from the Peninsula) on the post-mortem table during 1915, the patients having succumbed to gunshot wounds. Strong quotes some statistics, and others are available from the recent literature. Strong and Musgrove in the Philippines found amoeba in the stools of healthy individuals in four per cent. Musgrove found them in 101 of 300 prisoners in Manila, though only 61 were suffering from dysentery. Craig found them in 65 of 200 cases. Wenyon and O’Connor (Alexandria) found them in 5.3 per cent of the healthy troops, in 1.8 per cent of the prisoners in Gabbari Military Prison, and in 13.7 per cent of the Egyptian civil prisoners in Hadra. The incidence
of the infection is very considerable, too, among men who are convalescent from an attack of dysentery. Walker Hall, Adam, and Savage found them in 9°08 per cent, and Wenyon and O'Connor in 10°8 per cent of convalescent patients on their arrival in England. The incidence is not confined to cases of dysentery, but also obtains in enterica convalescents. Walker Hall's figures show 10°06 per cent amoebic carriers among this group.

At one end of the chain lies the carrier with no symptoms of intestinal disturbance; at the other end the patient with acute dysentery. The intervening links are represented by cases presenting an infinite variety of symptoms. The difference depends upon several factors, the site and the degree of the lesions, and the presence of associated bacterial infection.

Tenetsus, for instance, depends upon the involvement of the lower part of the large bowel, and is absent when this is not involved. Diarrhoea is mainly due to intestinal catarrh of very varied bacterial origin. The presence of mucus in the stools predicates inflammation of intact, i.e., undestroyed mucous membrane. Blood is present if ulceration is progressive, or catarrh severe. An uncomplicated ulceration of the cecum and ascending colon presents very different symptoms from those of widespread involvement of the colon generally, with some associated catarrh.

The whole series may be seen in the post-mortem room: the small uninflamed ulcer; the ulceration surrounded by acute catarrh; and the gut almost wholly devoid of mucous membrane, and necessarily incapable of secreting any appreciable amount of catarrhal products.

We are becoming somewhat sceptical as to the existence of pure acute amoebic dysentery, i.e., amoebiasis unassociated with bacterial infection. We have seen several such cases where the dysenteric symptoms subsided under general treatment before the entamoebae were detected in the stools, and appropriate treatment instituted. And everyone knows that amoebiasis may exist without any evidence of the gastro-intestinal disturbance, and may not be recognized until an hepatic abscess or the rupture of an ulcer occasions notable distress. In one of our cases dysenteric symptoms were absent though death was due to the rupture of an amoebic ulcer. In this case the patient was admitted from Mesopotamia with a diagnosis of malaria, and after ten days' residence was sent to a convalescent hospital. A fortnight later fever recurred and he was admitted again to the wards on September 9, 1916. He was then seriously ill with a high temperature which frequently reached 105° F. Bacillus paratyphosus A was isolated from the blood, and the clinical symptoms were those of enterica. There was little or no diarrhea until the end of September, when he became dull, lethargic, and semi-comatose, and incontinence of feces ensued. A little mucus was now occasionally present in the stools, but these never suggested the presence of dysentery, and entamoebae were not detected at the single examination that was practised. On October 13, 1916, signs of peritonitis made their appearance, but his general condition negated operative
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measures. He died on October 17, 1916. Post-mortem examination revealed widespread amoebic ulceration of the large bowel.

The post-mortem findings in this case were unexpected as the existence of amoebic dysentery was not suspected during life in view of the character of the general symptoms and the positive blood culture. There was never any blood in the stools, and mucus was only present during the terminal stages of the illness, and never in large amount.

In many of our cases the history was one of diarrhoea, or alternating diarrhoea and constipation following dysentery, rather than of chronic dysentery. A medical man, for instance, had a mild attack of dysentery at Cape Helles, in November, 1915, which did not necessitate hospital treatment; diarrhoea for three days in December, a recurrence in March, and frequent repetitions subsequently which only ceased after the stools had been examined in May, the Entamœba histolytica recognized, and appropriate treatment instituted. Another patient gave a history of an attack of dysentery in June in Mesopotamia, which lasted for some weeks, and was succeeded by chronic diarrhoea which sometimes ceased for many days at a time. !His stools however, were never formed, and in November contained E. histolytica. Less commonly the symptoms are those of diarrhoea from beginning to end. One patient had diarrhoea on his way to Egypt in April, 1915, at Chatby in May, and on four subsequent occasions, none of which necessitated treatment in hospital. In October, 1916, it recurred so severely that he had to go to hospital, where, however, he was only detained for six days, and then sent to Montazah Convalescent Hospital. The diarrhoea recurred at once, and examination of the stool revealed E. histolytica. The attacks of diarrhoea were never severe, but might last for a fortnight at a time. He had no tenesmus, and never to his knowledge passed either blood or mucus with the stools.

A double infection is by no means uncommon, and occurred in twelve of our cases, entamœba and a dysentery bacillus being detected in the stools. But in amoebiasis, associated with dysenteric symptoms, the majority of patients show no dysentery bacilli. Other bacteria, however (B. Morgan, B. fecalis alkaligenes, B. paracolon, B. C.L.A. 1, B. C.L.A. 2, streptococci, etc.), are commonly present, sometimes in enormous numbers, and, whether capable or not of originating dysenteric symptoms in normal intestines, may quite conceivably produce them in a bowel already invaded by entamœba. And this, we think, is the explanation of the variation in the symptoms met with in amoebiasis, variations which are dependent upon difference in the site and the degree of the amoebic invasion; and in the kind and the mass of the bacterial infection.

1 C.L.A. type of bacilli. A number of organisms of the Morgan group of intestinal bacilli have been isolated at the Central Laboratory, Alexandria, and have been designated C.L.A., No. 1, No. 2, etc. These organisms are feebly pathogenic to animals, and are probably diarrhoea producers in man.
The failure to recognize the double character of the cause in cases where it exists may have a tragical ending. In the majority of such cases the subsidence of the febrile and toxic symptoms unaccompanied by the usual associated improvement in the character of the stools suggests the presence of the protozoal infection, or this may be recognized on repeated examinations of the stools, and appropriate treatment secures the desired result. In one case we failed to appreciate this point.

The patient, a seaman on a trawler, dined in town on October 10, 1916, and acute dysentery succeeded next day, the abdominal pain being severe and the tenesmus constant. When he was admitted into hospital on October 10, 1916, he was much exhausted and looked toxic and ill. The tongue was thickly coated, the abdomen flat and mobile but tender in the iliac fossae, and the stools were numerous and composed of bloody mucus. The pulse was febrile and numbered 101. The temperature ran between 100° and 102°F. In the days succeeding his admission his condition improved to some extent. The fever persisted, but the stools became less frequent and faecal and the blood lessened in amount; on October 14, 1916, some small superficial sloughs were recognized in the stools. On October 19, 1916, he seemed better, the stools being less frequent and less fluid, with no blood or mucus, though some exudate was present, while his general strength was well maintained though he was appreciably thinner. The abdomen, too, was slightly distended and occasionally he had some pain. During the ensuing week, however, he continued to lose ground. The fever persisted, assuming a hectic character and being accompanied by severe sweating. The diarrhoea continued, the stools still retaining the same character. Towards the end of the month vomiting ensued at intervals, the diarrhoea continued, and the abdominal pain recurred. The abdomen became slightly more distended, but no evidence of general peritonitis was manifest. He died of cardiac failure on the morning of November 1, 1916. Post-mortem examination showed a widespread amoebic ulceration of the colon with an acute bacterial infection of the unaffected mucous membrane.

The lower three feet of the ileum were also acutely inflamed. The whole course of the illness suggested a bacterial cause—the sudden acute onset, the evident toxemia and the continued pyrexia; and this seemed confirmed by the result of the examination of the stools which failed to reveal the presence of any protozoa, while a dysentery bacillus—not fermenting mannite—was isolated on culture from the stools on October 12, 1916. On subsequent examination dysentery organisms were absent, but B. Morgan was isolated on several occasions, and an intestinal infection of non-specific character engrafted upon a dysenteric lesion was assumed to be the cause of his illness, the enterica group having been excluded by examination of the urine, the stools, and the blood. The protozoal infection was not suspected and was not treated. The patient was definite that his illness only commenced upon October 7, 1916, though the condi-
tion found post mortem must have existed for some considerable time prior to this date, the amoebiasis being thus latent. Death, of course, ensued as a direct result of a bacterial infection, but this might perhaps have been resisted if emetine treatment had been instituted on admission, and the protozoal infection overcome.

One other patient in this series died, but not as the result of dysentery. He was admitted into hospital from Mesopotamia and could give no account of his illness, as he was then mildly delirious. He died two days later. Post-mortem examination revealed a widespread pulmonary tuberculosis and one or two small healing ulcers (probably of amoebic origin) in the colon. A malarial infection was also present.

The diagnosis of amoebic dysentery is beset by many difficulties. In a general way the onset of symptoms is subacute or insidious, the course is essentially chronic, fever is absent or slight in degree (100° to 101° F.), and there are no toxic symptoms such as headache, malaise anorexia, prostration, etc., the picture contrasting strongly with acute cases of the bacillary type. But the onset may be acute, fever high, and general symptoms severe in cases where a bacillary infection is superadded, and all these may be absent in the milder bacillary infections.

An accurate diagnosis can only be made by microscopic examination of the stools. Macroscopic examination is insufficient, for in amebiasis any type of stool may be present, and the only real test is the discovery of the E. histolytica.

A single negative examination is of little value, and in our cases the parasite was only found at the first examination in sixty per cent. In several instances it was only detected at the third or fourth, and in one case at the eighth examination. This is by no means surprising when we consider the relative bulks of the stool and of the morsel examined. The stools, too, must be fresh and clean, for cold and prolonged exposure to the atmosphere rapidly destroy the amebae, a result which also follows the admixture of stools and urine, and treatment by emetine. Microscopic search should always be made, too, in evident cases of bacillary dysentery, for the infection is often mixed. A laxative should be administered in every case and mucus, exudate, or sloughs of the mucus membrane examined in particular.

It seemed possible that the failure to recognize entamoebae in the stools might be due in part at any rate to the co-existence of a bacterial infection destroying the parasites. In twenty-one cases in which entamoebae were found the same stool was examined bacteriologically. In five cases a heavy bacterial infection was present (B. dysenteriae mannite fermenter, B. dysenteriae non-mannite fermenter, B. faecalis alkaligenes, B. para colon A., B. C.L.A. 1). In sixteen cases abnormal bacteria were absent.

In 23 of our cases (E. histolytica 18, E. coli 5), a protozoal infection was not recognized on the first examination of the stools. The bacteriological examination of these stools showed negative results in 13 cases,
but positive results in only 10 (B. dysenteriae mannite fermenter 5, B. dysenteriae non-mannite fermenter 1, B. paracolon A 1, B. C.L.A. 6, B. faecalis alkaligenes 1, and streptococci 1). The other causes which have been suggested as reasons for the failure are thus more important than a coincident bacterial infection.

Another suggestion indicative of a protozoal cause of active dysentery is the presence of E. coli in the stools. We have been led to this conclusion by the satisfactory result of the administration of emetine in such cases, but we are unfortunately unable to give statistics upon this point. Mr. Savage, however, has kindly permitted us to publish his figures, which show that a double infection with histolytica and coli is nearly three times as common as a pure histolytica infection. A pure coli infection is, however, more than twice as common as the double infection, so that the indication is merely suggestive. In acute dysentery, however, a pure coli infection is not quite twice as common as the double infection, and E. histolytica was present in forty-three per cent of the cases in which entamoebae were found.

In cases where microscopic examination is impossible, and in fact in every case, due attention should be paid to the other data. The probable source of the infection and the nature of the dysentery there prevalent are particularly important. Cases which originated on the Peninsula, especially at Cape Helles, and in Mesopotamia, particularly if the early symptoms were slight and did not necessitate treatment in hospital, are most frequently amoebic. Recurring attacks of dysentery, chronic dysentery, and dysentery which fails to react to general treatment, and dysentery associated with enlargement of the liver, hepatitis and perihepatitis, are most generally amoebic in origin. Repeated examination of the stools, too, may reveal the cause even if the symptoms have subsided, and in one of our cases E. histolytica cysts were found for the first time in the first normal semi-solid stool passed after the commencement of the illness.

The treatment of amœbiasis of the intestines consists in the administration of ipecacuanha or emetine in sufficient doses over a sufficient length of time. The well-known discomforts produced by the administration of large doses of ipecacuanha given by the mouth have led to the almost universal use of emetine administered hypodermically. The current routine is to give twelve grains of emetine hydrochloride in one-grain doses at night. In a large number of acute cases this treatment is

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successful, the symptoms lessening in severity after four to five days and rapidly disappearing, while the entamœbes disappear from the stools. But in a considerable number of cases the entamœbes disappear from the stools, though the symptoms may be absent or trivial. In one of our cases twelve grains of emetine were given between June 8 and 19, amœbe reappeared in the stools on August 1. In another case they were detected for the first time on August 12, though 12½ grains had been given between July 21 and August 6. (The earlier examination had been made after emetine treatment had been commenced.) Our experience in this coincides with that of Wenyon and O'Connor.

In another group the dysenteric symptoms persist. This is most usually in cases where the illness is chronic or relapsing, and where presumably ulceration of considerable severity and extent still obtains. It seems reasonable in such cases to give ipecacuana or emetine by the mouth in the hope of reaching organisms in the crevices of chronic ulcers which are out with the reach of the blood-stream. We therefore follow up the hypodermic course of emetine by a course of ipecacuana given by the mouth in five or ten-grain doses for a fortnight. So far as we know our cases thus treated have not relapsed, but we are, of course, unfortunately unable to secure reliable data of the history of our patients after their discharge from hospital.

It seems necessary still to emphasize the fact that a "ten-day course" will not cure amœbic dysentery. It is well known that such protozoal infections as malaria and syphilis require prolonged treatment to ensure a cure, and amoebiasis also requires prolonged treatment, even after the dysenteric symptoms have completely disappeared. The presence of dysenteric symptoms in amoebiasis indicates active ulceration of the bowel, and, like gastric and duodenal ulcer, amœbic ulceration requires time to heal. The statistics of Wenyon and O'Connor with regard to emetine treatment, and those of Savage and Young with regard to the treatment by the double iodide, afford confirmatory evidence of the correctness of our reasoning.

The rapid disappearance of dysenteric symptoms in amoebiasis has in our opinion led to an unwise shortening of the period of treatment in hospital with the result that relapses have been frequent, and the period of absence from duty unnecessarily prolonged. To put it in another way, "a stitch in time saves nine." Recent infections respond rapidly to treatment, but recurring cases with deep and large ulcers have as a rule to be invalided home, and are in consequence absent from the field for many months. We are unable to give statistics of the average duration of treatment in hospital of our acute cases, but it must have been between five and six weeks. An earlier dismissal was rarely possible on account of either local or general symptoms, and we invariably kept them in hospital until they had been for a week on ordinary diet without discomfort. They
were then sent to a convalescent home for a fortnight before their return to duty.

We have been unable to obtain membroids or such like protection for the ipecacuanha, but we have found that it rarely occasions sickness if given with thirty grains of bismuthi carb., and five grains of sodii bicarb. about 9 o'clock at night and three hours after the last meal. The patient was in bed for at least thirty minutes before the dose was administered.

We have used salol coated tabloids of emetine with apparently good results, but we dislike using tabloids on account of the impossibility of ensuring their disintegration within the bowel. Our supply of the double iodide of bismuth and emetine was limited and only allowed its use in three cases; in two the result was satisfactory. The tabloids are keratin coated, and given immediately after meals sometimes excited a little nausea, but only rarely actual vomiting.

We have invariably kept our patients in bed during the emetine course, though they were subsequently allowed up for a few hours each day during the ipecacuanha course, if their general condition permitted. We have had no serious symptoms in any case, but our patients, if in poor health, have not infrequently complained of nausea, giddiness, weakness, etc., on exertion, and in consequence we have refrained from permitting any exercise. We have tried Wenyon's plan of giving emetine simultaneously by the bowel and hypodermically, but these general symptoms seemed intensified by this measure and we in consequence adopted the sequence detailed above, with satisfactory results.

Captain Lumb informs us that he obtained good results in East Africa prior to the War by continued treatment with emetine hypodermically. He gave it daily until the symptoms disappeared, and then twice weekly for a month, and once a week for another month. We have had no experience of this method of treatment.

**Bacillary Dysentery.**

The symptoms of bacillary dysentery are quite as varied and confusing as those of the amoebic variety.

In the typical acute case the onset is abrupt, the patient being suddenly seized with severe diarrhoea, accompanied by griping pains in the abdomen; and in the course of a few hours the stools lose their copious watery faecal character, and become small in bulk, though even more numerous than before, and composed of blood, or blood and mucus. Tenesmus is extreme, and the patient refuses to relinquish the bedpan or to leave the latrine, while the abdominal pain continues, intensified from time to time with the passage of a stool. The abdomen, too, is exquisitely tender to the touch. And with these local symptoms there is fever, the temperature reaching 101° to 102° F., and all the usual signs of toxemia, headache, anorexia, nausea, furred tongue, hot skin, and frequent bounding pulse.
On the second or third day his appearance is fairly characteristic. The face is flushed, and his eyes are languid, the patient being wholly absorbed in his own discomforts, and indifferent to his surroundings. But at the same time the brain is unclouded and the intelligence acute, and the dazed lethargic features of enterica are absent—though he will doze indefinitely unless aroused by a call to stool. The fever is usually remittent or intermittent, and rarely touches 103°F. Exhaustion is extreme from the pain, sleeplessness, and starvation; but the pulse is usually not unduly frequent, running about 90, and is of fair quality. An increasing pulse-rate, or one over 100, is of more serious significance than the height of the fever or the number of the stools.

The whole picture—the facies, the toxæmia, the fever, etc.—are those of an acute bacterial infection, in which gastro-intestinal symptoms are prominent.

The subsequent course of events varies greatly in different cases. In some the attack is short, the fever disappearing within forty-eight hours, while the diarrhæa rapidly ceases, and the blood disappears from the stools. In others the fever persists, and may last for a week, or rarely for a fortnight, while blood and pus and mucus continue present in the stools. There is no difficulty in these cases in recognizing the bacterial origin of the illness.

But in another group the symptoms can hardly be separated from those of the amœbic type. Two men were admitted into hospital on the same day from the same camp. One had arrived in Egypt from England on December 27, and on December 30 dysentery ensued, accompanied by considerable abdominal pain. His temperature on December 31 was 100°F., and the stools consisted of bloody mucus. On January 1 the fever and abdominal discomfort had gone, while the stools consisted of bloody muco-pus. On January 3 the blood had gone, and the stools were faecal, with a little cellular exudate, which in its turn had disappeared on January 5. The recent arrival of the patient in Egypt, and the acuteness of the symptoms at the onset, made the diagnosis clear even before a dysentery bacillus was isolated on culture, and the absence of entamoebæ determined by the microscope.

The second patient had been taken ill on December 25 with similar symptoms, had gone sick on December 27, and was admitted on December 31. He had then no fever and no abdominal discomfort save in a slight degree when at stool; while the stools at first faecal only showed the presence of blood and mucus on January 2, forty-eight hours after admission, and seven days after the onset of the symptoms. He had been in Egypt for a year, and had always been liable to diarrhœa though it was never severe, and had never necessitated his admission to hospital. A history of chronic diarrhœa, a subacute onset, an afebrile illness, but again due to a dysentery bacillus.

Diarrhœa, too, may be due to a dysentery bacillus. A man was admitted
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to hospital on December 21 on account of diarrhoea, which had ensued two
days previously. He said that he had passed some blood and slime on the
morning of the day of admission, but the stools after admission were always
faecal, without addition, and after December 26 formed. The number of
stools passed per diem after admission was 3, 1, 1, ——, 1, 1, 1, 1, but on
December 23 a dysentery bacillus was isolated. Another man whose stools
were examined in the course of an investigation into the flora of the stools
in Egypt, and found to contain a dysentery bacillus, had had diarrhoea for a
couple of days immediately before the stool—a normal one—was investiga-
gated. Dysentery carriers are by no means uncommon. The majority of
the cases discovered had at some time suffered from diarrhoea or dysentery;
but one man who had been on the Peninsula from September 15 until the
evacuation, and subsequently in Egypt, and whose stools contained a
dysentery bacillus on September 20, 1916, and on October 11, 1916, would
not admit having ever had any diarrhoea or dysentery since he came East,
and had never been off duty on account of illness save for a couple of weeks
in June, 1916, when he had “inflammation of the lungs.”

The same series, then, obtains in bacillary dysentery as in amoebic
dysentery, and while the severe cases in the two groups present distinctive
pictures, the milder forms are practically indistinguishable; in both
diarrhoea rather than dysentery may prevail; and in both gastro-intestinal
symptoms may be in complete abeyance.

Like others we have at times imagined that the character of the stools
conveyed a definite indication of the cause of the illness. And arguing in
some measure from comparison with the secretions in acute coryza, we have
thought that mucus or muco-pus, streaked with blood, and fluid-purulent
stools were characteristic of bacillary cases; while a copious crimson stool
and a faecal stool, studded with sago-like masses, were characteristic of the
amoebic variety. But increasing experience has taught us like others that
no reliance can be placed on such criteria, and that every type of normal
and abnormal stool may be present with amoebic and with bacillary
infections of the bowel. After all, mucus comes from inflamed mucous
membrane, pus comes from the same source or from an ulcer, blood flows
from a ruptured or congested vessel; and inflammation of the mucous
membrane of varying degree, and ulceration, may and do obtain in both
forms of the disease.

The general features of the illness, an epidemic character, batches of
patients coming at the same time from the camp, an acute onset, high fever
and severe toxæmic symptoms, are the most characteristic symptoms of
bacillary dysentery, but the only sure test is the recognition of the specific
cause in the stools. Extreme exhaustion, for instance, may closely simulate
toxæmia, and a rough passage on a hospital ship from the Western Front
so exhausted one of our patients that we had little doubt on his admission
that his illness was bacillary, though it turned out to be a pure amoebic
infection.
A point of considerable practical importance may be considered here. In more than half (58.8 per cent) of the cases with blood and mucus in the stools examined microscopically and bacteriologically, no dysentery bacilli or Entamoeba were detected, and the cause of the illness remained undetermined.1

It has been suggested that the absence of dysenteric bacilli from the stools is merely a question of the stage of the disease; that in dysentery the specific bacteria cannot be recovered from the stools after the first few days, as they either die out or become submerged in the general flora of the inflamed bowel. As Major Ferguson puts it, the ground may still be wet, though the shower has ceased.

It is quite true that mucus and blood and pus may persist after dysentery bacilli have ceased to be recovered. In one of our cases no dysenteric bacilli were isolated after the fourth day, though blood and mucus were present in the stools as late as the ninth day. In another case blood and pus were present on the eleventh day, though bacilli were absent after the ninth. But in other cases the bacilli may persist after blood and mucus and pus have gone, and when the stools are normal. In 15 out of 28 cases in which the stools were examined repeatedly, bacilli were present in the second week, and in 3 in the third week. In one case they persisted until the eighteenth day, and were found subsequently on the twenty-fifth. In three cases they had disappeared after the fourth day.

Their presence, too, or at any rate their recognition, may be intermittent. We have found them present on the fourth and fifth days, absent on the sixth, and present on the eighth and ninth; present on the sixth and seventh, absent on the eighth, and present again on the ninth day. Isolated observations are of little value, and repeated examinations are required if the bacillary cause is to be fairly excluded. But the number of cases in which they are not detected seems too large to be explained in this way, and their absence must be accounted for by other circumstances.

(To be continued.)

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1 Central Laboratory, Alexandria, April to December, 1916: 888 cases with blood and mucus in the stools were examined; 78 cases showed E. histolytica, and 278 B. dysenteriae = 41.2 per cent; 173 cases showed atypical, i.e., inagglutinable B. dysenteriae.