TABLE II.

Case No.	Treatment.			Consecutive Negative Examinations.		Date of Relapse (1916).
	Begun.	Ended.	Amount Given.	Dates (1916).	No.	
12	Aug. 5	Aug. 16	12 gr.	Aug. 5, 7, 9	3	Aug. 8
16	Aug. 5	Aug. 16	12 gr.	Aug. 12, 15, 23, 25, 28, 30	6	Sept. 1
21	Aug. 5	Aug. 16	12 gr.	Aug. 14, 16, 19	3	Aug. 8
23	Aug. 6	Aug. 17	12 gr.	Aug. 22, 26, 29, 31	4	Sept. 2
25	July 24	Aug. 2	10 gr.	July 31; Aug. 1, 5, 7, 9, 11, 13, 15, 19	9	Aug. 21
28	July 24	Aug. 2	10 gr.	July 31	1	Aug. 7
32	Aug. 5	Aug. 16	12 gr.	Aug. 7, 8, 14, 16, 18, 20, 24, 26, 29	9	Aug. 31
33	Aug. 5	Aug. 16	12 gr.	Aug. 14, 16, 19, 21, 23, 24, 25	7	Aug. 28
34	Aug. 5	Aug. 16	12 gr.	Aug. 8, 10, 12, 14, 16, 18, 20	7	Aug. 22
38	Aug. 10	Aug. 21	12 gr.	Aug. 19, 21, 23, 25; Sept. 1, 4	6	Sept. 6
39	Aug. 12	Aug. 23	12 gr.	Aug. 24, 26, 29	3	Aug. 31
40	Aug. 12	Aug. 23	12 gr.	Aug. 17, 18, 21, 23, 25, 28	6	Aug. 30

The other two cases were not given as much as 10 grains of emetine. No. 17 was discharged as cured after seven $\frac{1}{2}$ grain doses, and No. 30 relapsed after a course of 8 grains. Table III gives the records of these two cases.

TABLE III.

Case No.	Treatment.			Consecutive Negative Examinations.		Date of Relapse.
	Begun.	Ended.	Amount Given.	Dates (1916).	No.	
17	June 29	July 8	3 $\frac{1}{2}$ gr.	June 5, 17, 20, 27; July 3, 8, 14	7	
30	June 25	July 19	8 gr.	July 22, 24, 27, 31; Aug. 7, 8	6	Aug. 10

The authorities found it necessary to discharge No. 17 a week after the end of his treatment, so that his case must remain open. He had a very small infection, which, it is interesting to note, was not detected until the sixth examination; he had been negative again for some time before his treatment began.

After the injection of at least 10 gr. of emetine hydrochloride, then, 12 out of 21 cases—that is, 57 per cent.—were certainly not permanently freed from *E. histolytica*. In one case (No. 12) the drug had no visible effect on the infection, the three negative examinations appearing in Table II probably being one of the natural negative periods often occurring in this patient. In all the other cases the treatment was followed by a longer or shorter negative period, and subsequently a relapse.

TABLE IV.

Case No.	After Emetine.	Day when First Found Negative.	Date of Detection of Relapse after End of Treatment.
15	12 gr.	First after end of treatment	Twentieth day.
16	12 gr.	Eighth day of treatment	Sixteenth day.
21	12 gr.	Tenth day of treatment	Fourth day.
23	12 gr.	Fifth after end of treatment*	Sixteenth day.
25	10 gr.	Eighth day of treatment	Nineteenth day.
28	10 gr.	Eighth day of treatment	Fifth day.
32	12 gr.	Third day of treatment	Fifteenth day.
33	12 gr.	Tenth day of treatment	Twelfth day.
34	12 gr.	Fourth day of treatment	Sixth day.
38	12 gr.	Tenth day of treatment	Sixteenth day.
39	12 gr.	First after end of treatment	Eighth day.
40	12 gr.	Sixth day of treatment	Seventh day.

* Not examined between eleventh day of treatment and this date.

A similar distribution of first negative examinations holds among the cases that did not relapse within three weeks.

The table shows that there may be a good deal of variation in the time elapsing before the disappearance and re-appearance of the cysts in the stools. The interval before the detection of the latter must be kept in mind when fixing the criterion for discharge as cured. In only one case (No. 18) were there any troublesome sequelae to the injections. This patient had a fairly severe local swelling and a rash, and complained of feeling sick and giddy. Although rather more than half of the cases were certainly not cured, these results would seem to be good compared with other records of emetine treatment under similar conditions—*cf.* Dobell, where two-thirds of the cases were certainly not cured, and 5 out of the 14 "showed no improvement whatsoever" under emetine treatment.

Permanent clearance of *E. coli* and *Lamblia* was not observed in any case. The latter seems quite unaffected by emetine given hypodermically, and no special treatment was undertaken for its elimination in the absence of any conclusive evidence for its pathogenicity. *E. coli* sometimes disappears during treatment.

Acknowledgements.

In conclusion, I would express my thanks to Captain J. R. Dickson, R.A.M.C., pathologist of the Central Laboratory, for his constant interest in this work, and for the trouble he has taken in making arrangements for the examination and treatment of these cases, finally undertaking the latter himself. I am indebted to Mr. A. G. Thacker for all records since I left Brighton on August 18th. My thanks are due to Dr. C. M. Wenyon (now Lieutenant-Colonel, R.A.M.C.), and to Dr. A. C. Stevenson, of the Wellcome Bureau of Scientific Research, for very kindly allowing me to use the resources of their laboratory, where I have always received the kindest assistance. I cannot sufficiently thank Mr. C. Dobell for the very kind way in which he has always been ready to give invaluable instruction and advice throughout the year, for which I have never hesitated to ask, and without which the shortcomings of this work would have been multiplied exceedingly. My thanks are also due to Lieutenant-Colonel Coutts, R.A.M.C., Officer Commanding the Kitchener Hospital, for his permission to publish this account of the cases in his hospital.

A SEARCH FOR DYSENTERY CARRIERS AMONG SOLDIERS COMING FROM GALLIPOLI AND EGYPT.

BY

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THIS investigation was undertaken, at the instance of Colonel Sir William Leishman, with the object of attempting to determine the extent to which men coming from countries in which dysentery is endemic are likely to be dysentery carriers. It is well known that a small percentage of those who have suffered from dysentery act, after convalescence, as carriers, and the possibility of those who have not suffered from an attack of dysentery, but have been in a district in which dysentery is endemic, serving as carriers must also be borne in mind.¹

The faeces of fifty soldiers who had reached France after service in Gallipoli and Egypt were examined for entamoebae and dysentery bacilli (Table I). The soldiers were chosen at random in the medical wards of the hospital, no attempt at selection being made. Seventeen out of the 50 had not had a previous attack of dysentery; the remaining 33 stated that they had suffered from previous attacks and had been admitted to hospital, passing blood and slime. Nine cases were admitted with a diagnosis of dysentery, and in one of these (No. 33, Table I) it was stated that *B. dysenteriae* (Flexner) had been isolated from the stools. All the patients examined, with a single exception (No. 17, Table I), had been in districts (Gallipoli and Egypt) in which dysentery was known to be endemic.

The faeces obtained for examination were in all except three cases either firm and well formed or semi-liquid and dark brown or yellowish in colour, with or without scybala, the latter resulting from the administration of a purgative—in other words, the faeces presented no abnormal change of pathological import. The exceptions were as follows: Case 31 (diagnosis dysentery), in which

TABLE I.—Examination of Faeces of Fifty Soldiers in Hospital who had served in Districts in which Dysentery is Endemic.

No.	Duration of Service.		Locality in which Previous Attack of Dysentery Occurred.	Diagnosis at Time of Examination.	Pathogenic Organisms Isolated from Faeces.	No.	Duration of Service.		Locality in which Previous Attack of Dysentery Occurred.	Diagnosis at Time of Examination.	Pathogenic Organisms Isolated from Faeces.
	In Gallipoli.	In Egypt.					In Gallipoli.	In Egypt.			
1	4½ mths.	—	No previous attack	Malaria	—	25	3 mths.	5 mths	Gallipoli.	Shell shock	—
2	3 "	7 mths.	" "	Bronchitis	—	26	6 "	2 "	"	Malaria	—
3	3½ "	8 "	" "	Acute parenchymatous nephritis	—	27	5 "	8 "	"	Pyrexia	—
4	8 "	3 "	" "	Bronchitis	—	28	3 "	5 "	"	Shell shock	—
5	1 "	7½ "	" "	Gunshot wounds	Inagglutinable <i>B. dysenteriae</i> (Shiga).	29	3½ "	22 "	"	Pneumonia	—
6	4 "	6 "	" "	Cardiac	—	30	4 "	7 "	"	Pyrexia	<i>B. dysenteriae</i> (Strong).
7	5 "	5 "	" "	Dysentery (two days' duration)	—	31	3 "	5 "	"	Dysentery	—
8	—	12 days	" "	Dysentery	<i>B. paratyphosus</i> B.	32	4 "	5 "	"	"	—
9	—	1 mth.	" "	Diarrhoea	—	33	5 "	4 "	"	Dysentery (convalescent)	—
10	—	3 weeks	" "	Bronchitis	—	34	—	4 "	Egypt	Gastritis and influenza	—
11	—	2 mths.	" "	"	—	35	—	5 "	"	Bronchitis	—
12	—	4 "	" "	Influenza	—	36	—	6 "	"	Influenza	—
13	—	3 "	" "	"	—	37	—	7 "	"	"	—
14	—	3 "	" "	"	—	38	—	7 "	"	Myalgia	—
15	—	7 days	" "	Diarrhoea	<i>B. typhosus</i> .	39	—	1 "	"	Influenza	—
16	—	4½ mths.	" "	Malaria	—	40	—	7 "	"	Dyspepsia	—
17	—	—	" "	Dysentery	Inagglutinable <i>B. dysenteriae</i> (Shiga).	41	—	7 "	"	Enteritis	—
18	2 mths.	6 mths.	Gallipoli	Shell shock	—	42	—	3 weeks	"	? Colitis	—
19	1 "	9 "	"	Influenza	—	43	—	5 days	"	Dysentery	—
20	4 "	10 "	"	Tumour of lung	—	44	—	1 mth.	"	Gall stones	—
21	1 "	3 "	"	Erythema nodosum	—	45	—	10 days	"	Influenza	—
22	7 "	2 "	"	Malaria	—	46	—	6 mths.	Mediterranean	Dysentery	—
23	3 "	6 "	"	Bronchitis	—	47	—	3 days	"	"	—
24	4 "	6 "	"	Mumps (convalescent)	—	48	8 mths.	4 mths.	New Zealand	Tonsillitis	—
						49	—	5 days	Queensland	Malaria	—
						50	2 weeks	7 mths.	Lemnos	Gastritis	—

Remarks.

No. 19: Lambliae present in faeces.
 No. 21: Faeces examined on three occasions.
 No. 28: Streptococci present in faeces.
 No. 31: The discharge from bowel consisted of pus, blood, and slime in faeces; three specimens were examined.
 No. 33: *B. dysenteriae* (Flexner) reported isolated from faeces.
 No. 34: Streptococci present in faeces.

No. 41: Discharge from bowel consisted of blood-stained slime; three specimens of faeces were examined.
 No. 42: Lambliae present in faeces.
 No. 46: Streptococci present in faeces. In this case and in Case 47 a previous attack of dysentery occurred in the Mediterranean, two days after leaving Alexandria.
 No. 50: Lambliae present in faeces.

the discharge from the bowel consisted of pus; Cases 32 (diagnosis dysentery) and 41 (diagnosis enteritis), in which blood and slime were being passed, and the disease was clinically dysentery at the time of examination; in all these cases there was a history of previous attacks of dysentery. It will be noted that in seven other cases, which had been admitted with a diagnosis of dysentery, no blood or slime was present in the faeces examined; in three of these cases (Nos. 7, 8, and 17) there was no history of a previous attack of dysentery; in four cases (Nos. 33, 43, 46, and 47) a history of previous attacks of dysentery was given.

In none of the cases could entamoebae, motile or encysted, be recognized in the faeces. Encysted lambliae, it may be observed, were found in three cases (Nos. 16, 42, and 46).

The bacteriological examination of the faeces was carried out according to the scheme adopted by Henderson Smith,² the cultural characters on nine different media (gelatine, lactose, mannite, glucose, saccharose, peptone, inulin, arabinose, and litmus milk), together with the motility and agglutinative reaction—to seven antisera: *B. dysenteriae* (Shiga), *B. dysenteriae* (Flexner), *B. dysenteriae* Y, *B. typhosus*, *B. paratyphosus* A, *B. paratyphosus* B, and *B. enteritidis* (Gaertner)—being noted.

In no case could an agglutinable *B. dysenteriae* (Shiga) or *B. dysenteriae* (Flexner) be obtained; if these bacilli were present in the faeces examined they must have been in such small numbers, relatively, that they escaped recognition throughout the series. In one case (No. 30) a

bacillus probably identical with *B. dysenteriae* (Strong) was isolated.* In two cases (Nos. 5 and 17) an inagglutinable bacillus corresponding in culturable characters to *B. dysenteriae* (Shiga) was obtained; in the first case the patient's own serum agglutinated in a dilution of 1 in 200; in the second no agglutination occurred with the patient's own serum, even in a dilution of 1 in 50.

Other pathogenic organisms were discovered incidentally: in Case 8, *B. paratyphosus* B was isolated; in Case 15, *B. typhosus*; in Cases 14 and 2 bacilli allied to Morgan's bacillus No. 1 and *B. paratyphosus* A respectively were found. The cultural and agglutinative characters of these bacilli are given in Table II, which contains also those of four other allied types, not known to be pathogenic, which were isolated, three (Nos. 9, 21, and 32) being late lactose fermenters.† Table II, it will be observed, exhibits in Nos. 15, 2, and 9 the co-agglutinative power of the serum employed.³ The serums were used in half the maximum dilution with which agglutination could be obtained.

Streptococci were abundant in the faeces of Cases 28, 34, and 46; the pathological significance of these bacteria is as yet undetermined.

Cases 5, 17, and 30 may be regarded as carriers. No other carriers could be recognized among the fifty men examined (excluding Cases 15 and 8, which, though carriers at the time of examination, do not belong to the

* Agglutination of this bacillus with Shiga, Flexner, and Y serums was obtained in 1 in 50 dilution, but not in 1 in 150 dilution.
 † Two of the strains isolated produced gas in inulin, but no acid.

TABLE II.—Cultural and Agglutinative Characters of Bacilli Isolated from Faeces.

No.	Bacillus Isolated from Faeces.	Lactose.	Gelatin.	Mannite.	Glucose.	Saccharose.	Litmus Milk.	Indol.	Motility.	Inulin.	Arabinose.	Agglutination Tests.							
												Control.	Shiga 1:2000.	Flexner 1:3000.	Y. 1:1000.	Typhoid 1:4000.	Paratyphoid A, 1:8000.	Paratyphoid B, 1:8000.	Gaertner 1:4000.
42	—	0	0	0	0	0	A	0	0	0	0	0	0	0	0	0	0	0	0
14	—	0	0	0	0	0	0, then alk.	0	+	0	0	0	0	0	0	0	0	0	0
5	Inagglutinable Shiga	0	0	0	A	0	A, then alk.	0	0	—	A	0	0	0	0	0	0	0	0
17	Inagglutinable Shiga	0	0	0	A	0	A, then alk.	0	0	—	A	0	0	0	0	0	0	0	0
15	<i>B. typhosus</i>	0	0	A	A	0	A	0	+	0	A	0	0	slight	slight	+	0	0	0
30	<i>B. dysenteriae</i> (Strong)	0	0	A	A	A	slight A	0	0	0	A	0	0	0	0	0	0	0	0
8	<i>B. paratyphosus</i> B	0	0	A G	A G	0	A, then alk.	0	+	0	A G	0	0	0	0	0	0	+	0
2	—	0	0	A G	A G	0	A	0	0	0	A G	0	0	slight	+	0	0	0	0
9	—	A G	0	A G	A G	0	A, late C	0	0	G	A G	0	0	0	0	0	+	0	+
21	—	A G	0	A G	A G	A G	A	0	+	0	A G	0	0	0	0	0	0	0	+
32	—	A G	0	A G	A G	A G	A	0	0	G	A G	0	0	0	0	0	0	0	+

A = Acid; G = gas; C = clot. The concentration of serum employed is of one-half the maximum dilution capable of giving macroscopic agglutination in two to four hours at 37° C.

dysentery group). Neither entamoebae nor dysentery bacilli of the Shiga or Flexner type could be found in the stools of patients 32 and 41, who were undoubtedly suffering from subacute attacks of true dysentery, the dejecta containing abundance of slime and blood during the period of examination.

It would seem that when soldiers who have had dysentery are admitted into a base hospital the condition of their intestinal tract is ordinarily normal or rapidly becomes so, any dysenteric lesions quickly subsiding. The results of the above investigation emphasizes the danger of troops who are dysenteric subjects bringing dysentery into this country by the agency of carriers. The risk would presumably be greater among men leading a

more arduous existence, conducing to a recrudescence of their malady, such as obtains in camp life at the front, and it appears desirable that further examinations should be made under the latter conditions.

In conclusion, I desire to express my indebtedness to Major Sidney Rowland for the constant advice and assistance which I have received from him throughout the course of this investigation.

REFERENCES.

¹ Ledingham and Arkwright, *The Carrier Problem in Infectious Disease*, London, 1912. ² The Identification of the Pathogenic Members of the Typhoid-Colon Group of Bacilli, *BRITISH MEDICAL JOURNAL*, July 3rd, 1915, p. 1. ³ Cf. H. Chick, The Preparation and Use of Certain Agglutinating Sera, *Lancet*, 1916.

NOTE UPON A CASE OF JAUNDICE FROM TRINITROTOLUOL POISONING.

BY

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BEFORE the war began the attention of the Home Office had already been drawn to the occurrence of poisoning among the men employed in the manufacture of high explosives belonging to the group of the nitro-derivatives of benzene and toluene, and in 1909 the Home Office issued regulations which were to be observed in the manufacture of these compounds. It was not, however, till the extended use of these bodies for war purposes began that it was realized that the handling of the finished product was also dangerous. The poison affects the persons employed in different manners, but it seems to be probable that no one is completely immune. The commonest symptoms are drowsiness, headache, and nausea, and to these are commonly added some degree of cyanosis, due probably to the conversion of the oxyhaemoglobin of the erythrocytes into methaemoglobin. Most of the workers show some of these symptoms in a greater or less degree, but fortunately the majority recover quickly and even gain some tolerance to the poison. Among the severer cases high grades of anaemia, methaemoglobinaemia, and jaundice occur, and of the cases of jaundice a proportion prove fatal. Another group of symptoms, which may be termed focal rather than constitutional, is the occurrence on the exposed surfaces of a dermatitis, which has been accurately described by Dr. Prosser White.¹

The woman whose case is here recorded was aged 24, and was strong, well-developed, and muscular. She had been engaged in munition work for a period of six

months, and four months before her fatal illness became an examiner in a munition factory. Her especial duty lay in examining the bags which were filled with the yellow powder, and this involved moving in an atmosphere filled with the dust, "enough," she said, "to make one sneeze." She also complained of the bitter taste which was always present in the mouth, and that all the food which she ate tasted of it. She wore a plate in the upper jaw, and, though she washed this regularly after her meals, she did not do so before eating. One month before her illness she noticed that she was passing dark-coloured urine, but it was not till a fortnight later that she began to feel ill, with loss of appetite and headache. She also had some vomiting after food with much retching. Four days later she felt worse and noticed that she was slightly yellow. This condition persisted, although she was sent to bed, and ten days later she was admitted to St. Bartholomew's Hospital.

She was deeply jaundiced, and the urine was stained almost black with bile pigments. There were one or two small petechiae on the body, but these were faded and did not appear to be of recent origin. The liver dullness began at the fifth rib, the organ could be felt just below the margin of the ribs, and there appeared to be some tenderness in the region of the gall bladder. The spleen could not be felt, nor on percussion did it seem to be enlarged. Her temperature was normal, and, apart from the jaundice and the vomiting, she did not appear to be ill.

On October 4th she was quite free from headache and nausea, but on October 6th she became very drowsy and vomited everything which she took. On October 7th she became very restless, and in the evening delirious, shouting and moaning. On the morning of October 8th she was quite unconscious and wildly delirious, and gradually sank and died in the evening.

At the *post-mortem* examination the chief features were