OBSERVATIONS ON EAST AFRICAN BACILLARY DYSENTERY.

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Introductory.

East African dysentery has ceased to be of merely local importance. With troops here from nearly all parts of the Empire carriers are almost certain to take infections acquired here to other places. I am emboldened therefore to put these observations on record, because, with one exception, so far as I am aware no observations have been published on bacillary dysentery in Eastern Africa. I am the more encouraged to do so because I have to record the common occurrence of an organism which has not hitherto been recognised to be a frequent cause of dysentery, viz. Morgan's bacillus.

Manteufel, writing upon an outbreak of dysentery at Dar-es-Salaam, in German East Africa, states that he was the first to demonstrate the occurrence of *B. dysenteriae* in that colony. In 161 cases investigated he found 45 to be bacillary, 9 amoebic, and 107 negative. Of the 45 bacillary cases 15 were positive both by agglutination and by culture from the stools; 21 positive by agglutination, negative by culture; and 9 positive by culture, negative by agglutination.

Of 16 strains isolated and tested 2 appeared to be identical with *B. Flexner* and 7 with *Bac. Y.* No strains corresponded with *B. Shiga* but several curious anomalous varieties were found.

In the *Nairobi Laboratory Report* for Jan.–June, 1914 (vol. v, Part 1), I published a preliminary note on 5 cases. These five and one other are included in this paper although their investigation was incomplete.

In the *Annual Medical Report of the Protectorate* for 1912 it is stated that in this country dysentery invariably depends upon the *Amoeba*. This statement, I fancy, was based upon insufficient evidence. Be that as it may the general impression here now is that amœbic dysentery during the past few years has become less common while dysentery presumably of bacillary origin (the opinion being based mainly on negative evidence—absence of amoebae from the stools) has become increasingly frequent.

During the campaign it has only been possible to have a very small proportion of the dysentery cases examined bacteriologically or even microscopically, but of those examined comparatively few proved to be amoebic. Tentative emetine treatment in undifferentiated cases has also failed to be of benefit in most instances. Other protozoa (*e.g. Lamblia*) occur occasionally, but they may be ruled out as comparatively negligible factors in the causation of dysentery. Fine suspended particles of mica in the water of certain districts (especially the Tsavo river) may account for some cases of a chronic nature but I think it may be taken as reasonably certain that most of the dysentery here at present is bacillary in origin.

Source of Strains.

56 cases in all (A to F and 1 to 50) are included in this paper. Six of these (A to F) date back to June, 1914. The study of these was unfinished when I was called out for Field Service and as the cultures had all died out before my return to the Laboratory they must remain incomplete. The remaining 50 cases were obtained between January and August, 1916.

All 56 cases with the exception of case C, a European lady suspected of being a typhoid carrier, were clinically dysentery, the stools containing blood, pus, and mucus. They were of varying severity, some slight but most of them severe. In all cases save two the presence of amoebae was excluded by microscopic examination, but it is possible that in some instances amoebae may have been missed or that the bacillary infection may have been secondary to an amoebic one. In two cases amoebae were found. One of these proved negative for dysentery bacilli. In the other (Case 44) no amoebae were found at first, but they were present on a later examination.

Incidentally I might note here that the macroscopic appearance of the stools seems to be of no value whatsoever in determining whether

the disease is amoebic or otherwise in origin. Microscopically however if the stools are examined within the first day or two of the disease, a marked paucity of bacteria suggests the presence of amoebae even although they may not actually be found, and in such circumstances a trial of emetine is recommended. In later stages of the disease this difference between amoebic and bacillary cases does not hold good.

Most of the cultures were made from stools, a few from the scrapings of ulcers in the large intestine *post-mortem*.

Of these 56 cases 39 yielded non-lactose fermenters on MacConkey plates, and from these 39 dysentery-like bacilli were obtained in 20. These 20 cases were as follows. Two other cases (21 and 24) are also included in the list.

B. European infant. Severe attack but recovered. Stool.
C. European lady. Suspected typhoid carrier. Stool.
D. European male. Mild case. Stool.
1. Kavirondo. Carrier Corps porter. Post-mortem.
2. Kavirondo. Carrier Corps porter. Post-mortem.
5. European. Sergeant. L.N. Lancs. Stool.
8. Kavirondo. Carrier Corps porter. Stool. Case died later.
12. Indian baby. Stool. Case died later.
15. Kikuyu. Carrier Corps porter. Stool. Case died later.
17. European. Private. L.N. Lancs. Stool.
19. European. Private. L.N. Lancs. Stool.
21. Kavirondo. Stool.
23. Indian child. Stool.
24. European lady. Stool. Chronic case. Acute attack about a year previously. Passing pus and mucus ever since.
28. Swahili houseboy. Stool. Ran away from hospital. Probably recovered.
33. European child. Stool. Recovered.
34. Kavirondo. Carrier Corps porter. Stool. Case died later.
38. M'Kamba. Carrier Corps porter. Stool. Recovered.
39. Kavirondo. Carrier Corps porter. Stool. Recovered.
44. Baganda. Stool. Case still severe after three months in hospital.
45. Baganda. Stool. Case died later.
50. Kavirondo. Carrier Corps porter. Stool. Recovered.

That only 20 of the 56 cases (35·7 %) yielded dysentery-like bacilli is probably partly accounted for by shortage of laboratory materials severely limiting the number of colonies that could be examined from each plate. Intermittent occurrence or actual absence of dysentery bacilli may account for others. Various observers report similar findings, *e.g.* Manteufel, quoted above; Denier, from 329 cases found dysentery bacilli in 30·48 % of dysenteric stools and in 17·72 % of

TABLE I.

Culture No.	Lactose	Gelatin	Mannite	Glucose	Saccharose	Dulcite	Maltose	Salicin	Litmus milk: 1 day	Litmus milk: 3 days	Litmus milk: 15 days	Indol	Motility	Laevulose	Dextrin	Inulin	Adonite	Agglutinations: Flexner	Agglutinations: Shiga	Agglutinations: Y	Agglutinations: Polyvalent "D 25"	Agglutinations: Patient's Serum	Approximate Grouping
B	–		–	A	–	–							–				–						Shiga
C	–		A	A	–	–							–				–						Flexner-Y
D	–	–	A	A	–	A slight			A	Alk		+	–				–						Flexner-Y
1 A	–	–	–	A	–	–	–	–	0	A	A	–	–	A	–	–	–	–	–	–			Shiga
1 B	–	–	A	A	–	–	–	–	0	0	A clot	–	+	A	–	–	A	–	–	–			?
3 A	–	–	–	AG	–	–	–	–	0	0	A clot	–	+	AG	–	–	–	–	–	–			Morgan
3 B	–	–	–	AG	–	–	–	–	0	0	A clot	–	+	AG	–	–	–	–	+ trace	–			Morgan
5 B	–	–	–	AG	–	–	–	–	0	0	Alk	+	+	AG	–	–	–	+ slight	–	+ trace			Morgan
8 A	–	–	–	AG	–	–	–	–	0	0	Alk	+	+	AG	–	–	–	–	–	–			Morgan
8 C.	–	–	–	AG	–	–	–	–	0	0	Alk	+	+	AG	–	–	–	–	–	–			Morgan
10	–	–	–	A	–	–	A	–	0	0	A clot	+	–	AG	–	–	–	–	–	–			?
11	–	–	–	AG	A	–	AG	–	0	0	0	+	+	Alk (?)	A (?)	–	–	+	–	+ trace			?
12	–	–	–	A	–	–	–	–	A	Alk	Alk	–	±	A slight	–	–	–	+ slight	+ +	–			Shiga
15 A	–	–	A	A	–	A	A	–	A	Alk	Alk	+	–	A	A	–	–	–	–	–			Flexner-Y
15 B	–	–	A	A	–	A	A	–	A	Alk	Alk	+	–	A	A	–	–	–	–	–			Flexner-Y
17	–	–	A	A slight	A late	–	–	–	0	0	A clot	+ slight	–	A	A slight	–	–	–	–	–			Flexner-Y

19 A	–	–	AG	AG	–	–	AG	–	0	0	A clot	+	–	AG	A slight	–	–						?
19 B	–	–	–	AG	–	–	–	–	0	Alk	Alk	+	–	AG	–	–	–						Morgan
21	–	–	AG	AG	–	AG	AG	A	0	0	A slight	+	–	AG	–	–	–						Bac. enteritidis
22 A	–	–	–	–	–	–	–	–	0	Alk	Alk	–	–	–	–	–	–						?
22 B	–	–	–	A	–	A (?)	A late	–	0	Alk	A slight	–	–	A	–	–	–	+ trace	+ +	+ slight			?
23	–	–	–	A	–	–	–	–	Alk	A	A clot	–	–	A	–	–	–	+ +	+ +	+ +			Shiga
24 A	–	–	AG	A	–	AG	AG	–	A	A	0	+	–		A	–	–						Bac. enteritidis
24 B	–	–	AG	AG	–	AG	AG	–	A	0	0	+	–	AG	A	–	–						Bac. enteritidis
28 B	–	–	–	AG	–	–	AG	–	0	Alk	Alk	+	+ slight	AG	–	–	–				–		Morgan
28 C	–	–	–	AG	–	–	–	–	Alk	Alk	Alk	+	–	AG	–	–	–				–		Morgan
33 B	–	–	–	AG	–	–	–	–	Alk	Alk	Alk	+ trace	–	AG	–	–	–				+ slight		Morgan
34 A	–	–	–	AG	–	–	–	–	0	Alk	Alk	+	+	AG	–	–	–				–		Morgan
38 A	–	–	–	AG	–	–	–	–	Alk	Alk	Alk	+ trace	+ slight	AG	–	–	–				+ slight	+	Morgan
38 B	–	–	–	AG	–	–	–	–	Alk	Alk	Alk	+	+ slight	AG	–	–	–				+ slight	+	Morgan
39 A	–	–	–	A	–	–	–	–	0	Alk	Alk	–	–	A	–	–	–		+ +		+	+	Shiga
44 A	–	–	–	AG	–	–	AG slight	–	Alk	Alk	Clot	+	+ slight	AG	–	–	–				–	+	Morgan
44 B	–	–	–	A trace G	–	–	–	–	Alk	Alk	Alk	+	+ slight	AG	–	–	–				–	–	Morgan
45 A	–	–	–	AG	–	–	–	–	Alk	Alk	Alk	+	+ slight	AG	–	–	–				–	+	Morgan
45 B	–	–	–	A	–	–	AG	–	Alk	Alk	A clot	+ trace	+ slight	AG	–	–	–				–	+	Morgan
50 B	–	–	–	A	–	–	–	–	0	Alk	Alk	–	–	A	–	–	–		+ +		+ +	+	Shiga

diarrhoeic stools; in 197 of Heffernan's cases dysentery bacilli were found in 50 %, amoebae in only 5 cases. Tribondeau and Fichet in 217 cases from the Dardanelles found amoebae in 10 cases and dysentery bacilli in 38 (17 %), but in this instance it must be noted that their cases were all past the acute stage and in process of recovery.

Technique.

The method of procedure employed was based on those recommended by Henderson Smith and by Ledingham and Penfold. A loopful of mucus from the stool or bowel was shaken up in a tube of broth. After standing for about an hour a loopful of broth taken from the top layers of the tube was plated on MacConkey's bile-salt neutral-red lactose agar and incubated at 37° C. for at least 18 hours. Colourless colonies were then picked off, inoculated individually into broth, incubated again for a few hours or overnight, and then inoculated again into mannite-peptone water and on to agar. Examination for motility in the young broth culture and the behaviour towards mannite as a rule decided whether we were to proceed further with the examination or not. Further inoculations into gelatin and the various sugars etc. available were made either direct from the agar slope or from a broth sub-cultured from it.

Table I gives the characters of 36 organisms isolated.

Agglutination was tested by the microscopic method, four hours being allowed before finally deciding whether it was positive or negative. The anti-Shiga, anti-Flexner, and anti-Y sera were kindly supplied me by the Lister Institute, also the "D 25" serum, this being a polyvalent anti-dysenteric serum prepared for therapeutic purposes. Further details regarding degree of agglutinability will be found in the subsequent discussion of results.

Blanks in the table mean "not tested."

Grouping of these 36 strains.

Cultures 1 B, 10, 11, 19 A, 22 A, and 22 B may I think be dismissed from further consideration as not corresponding even approximately with any recognised pathogenic organism.

ORGANISMS RESEMBLING *B. SHIGA*.

Six cases. Cultures Nos. B, 1 A, 12, 23, 39, and 50 B.

B was insufficiently tested. 1 A and 23 agree in their cultural reactions with the typical *B. Shiga* except in their behaviour towards milk. 1 A is not agglutinated by anti-Shiga, Flexner, or Y sera in a dilution of 1 in 200 while 23 is agglutinated to practically an equal extent by all three in that dilution. Remlinger and Dumas (1915) have recently recorded from the Argonne an organism closely related to *B. Shiga* but not agglutinated by Shiga serum. Generally speaking however the Shiga bacillus has been found to occur in many parts of the world very true to type and to remain so even after repeated sub-cultivation (although variations do then occur; see, for instance, Barber, also Hallenberger). My observations are only sufficient to enable me to put these organisms on record as resembling but not identical with *B. Shiga*.

No. 12 agrees with the typical Shiga organism very closely. It is agglutinated by Shiga serum very markedly, slightly by Flexner serum, and not at all by Y (all in dilutions of 1 in 200). Its character as regards motility however is curious. Bacilli of this class frequently show oscillatory and turning movements which make one doubtful at times whether one is dealing with a feebly motile organism or not, but the first two generations in broth (off the original MacConkey plate) of culture No. 12 left one in no doubt. It was an actively motile organism. Subsequent generations (young broth cultures) taken from an agar slope, however, appear to be non-motile, or, at most, to show nothing more than ordinary oscillatory and turning movements. Whether this organism therefore should or should not be accepted as a *B. Shiga* is also doubtful, although anyone working with it as it is now would, I think, accept it as such, unless possibly further tests which I am not in a position to apply, showed further discrepancies.

Nos. 39 and 50 B correspond with the typical *B. Shiga* both by fermentation and agglutination tests. As regards agglutination 39 reacts slightly with "D 25" 1 in 30, well with anti-Shiga 1 in 100 and slightly 1 in 500, also well with the patient's own serum diluted 1 in 100.

No. 50 agglutinates well with the polyvalent serum 1 in 30, very well with anti-Shiga 1 in 100 but not 1 in 500, also well with the patient's serum 1 in 50.

Organisms resembling the *Flexner-Y* Group.

Four cases. Cultures C, D, 15 A and B, and 17.

C and D were insufficiently tested but it might be noted that in case C (a suspected typhoid carrier) the organism agglutinated with the patient's own serum markedly in a 1 in 90 dilution, slightly with 1 in 180. The patient's serum also agglutinated *B. typhosus* markedly with a 1 in 180 dilution.

Nos. 15 A and B correspond with the typical *B. Flexner* but ferment dulcite additionally. They do not agglutinate with either Shiga, Flexner, or Y sera 1 in 200.

No. 17 is less typical. It resembles the *Bac. Y* except in fermenting saccharose and clotting milk. In these two respects it resembles the *B. Strong* but differs from that organism in fermenting dextrin and in not fermenting dulcite and maltose. It also does not agglutinate with Shiga, Flexner, or Y sera 1 in 200.

Great variability has been recorded amongst this group both as regards fermentation and agglutinability (see especially Morgan, also Gettings and Barber), but I think that at least the strains from case No. 15 have sufficient claim to be regarded as belonging to the mannite-fermenting group of dysentery bacilli. Whether such organisms should be labelled "Para" or "Pseudo" dysentery bacilli and be regarded as distinct species, or whether they are to be looked upon as merely varieties of one common type is part of a very large question which is beyond the scope of this paper.

Organisms resembling *Morgan's Bacillus.*

Ten cases. Cultures Nos. 3 A and B, 5 B, 8 A and C, 19 B, 28 B and C, 33, 34, 38 A and B, 44 A and B, and 45 A and B.

Nos. 5 B, 8 A and C, 34 A, 38 A and B, 44 B, and 45 A agree completely in their cultural and fermentation reactions with Morgan's No. 1 bacillus.

Nos. 38 A and 44 B are, however, only feebly motile, not actively motile as the typical Morgan's No. 1 is stated to be.

No. 5 agglutinated slightly with Flexner serum but not with Shiga or Y (dilution 1 in 200).

Nos. 8 A and 8 C did not agglutinate with these sera in 1 in 200 dilution.

No. 34 A did not agglutinate with polyvalent serum 1 in 30.

Nos. 38 A and 38 B agglutinated slightly with polyvalent serum 1 in 30, with the patient's serum well 1 in 50, and slightly 1 in 100, not with Shiga serum 1 in 100.

No. 44 B did not agglutinate with polyvalent serum 1 in 30 nor with the patient's serum 1 in 50.

No. 45 A did not agglutinate with polyvalent serum 1 in 30 but gave a slight reaction with the patient's serum 1 in 50 although not 1 in 100.

Nos. 19 B, 28 C, and 33 B correspond in their characters with Morgan's No. 1 bacillus except that they are non-motile. In this connection it might be noted that Tribondeau and Fichet in their 13 cases found 7 actively motile, 4 feebly, and 2 non-motile. 28 C did not agglutinate with polyvalent serum 1 in 30; 33 B did slightly. No other agglutinations were tried with this lot.

Nos. 3 A and B, 28 B, 44 A, and 45 B present characters intermediate between Morgan's bacillus and Bowman's "Bac. S," although varying amongst themselves. Bowman's bacillus, said to be a common cause of dysentery, especially amongst children in the Philippines, is described in the abstract of Musgrave and Sison's paper in the *Bull. de l'Inst. Pasteur* as being "très voisin du bacille Morgan I"; in the abstract of Bowman's original paper there are at least three characters mentioned which distinguish it from that organism, viz. fermentation of maltose, non-production of indol, and clotting of milk. The following table (Table II) shows the variations in this series of strains.

TABLE II.

	Maltose	Milk	Indol	Motility
"Typical Morgan I" ...	–	Alk.	+	active
"Typical Bac. S" ...	AG	Clot	–	active
3 A and 3 B	–	Clot	–	active
28 B	AG	Alk.	+	slight
44 A and 45 B ...	AG	Clot	+	slight

45 B also differed from Morgan's and Bowman's organisms in forming acid only without gas from glucose.

As regards agglutinations 3 B gave a very slight reaction with anti-Shiga serum 1 in 200, not with Flexner or Y in that dilution. 3 A gave no reaction with those three.

No. 28 B gave no reaction with polyvalent serum 1 in 30.

Nos. 44 A and 45 B agglutinated slightly with patient's serum 1 in 50 but not 1 in 100, nor with polyvalent serum 1 in 30.

Morgan's bacillus was originally found and described from cases of summer diarrhoea amongst children in England. I am unable to

refer to the original descriptions of the various strains by Morgan and by Morgan and Ledingham, but have taken the characters of Morgan's No. I Bacillus as summarised by Muir and Ritchie and Henderson Smith to be correct. Possibly some of my atypical strains correspond with some of Morgan's varieties other than No. I.

The occurrence of Morgan's Bacillus in cases of what clinically appears to be dysentery has been recorded by Gettings and by Orr in asylum dysentery in England. In India, Morison and Chitre from 95 cases of diarrhoea and dysentery studied some 1500 organisms. *Morgan's No. I* bacillus was found in 16 cases, *B. Shiga* in 16, and *B. Flexner* in 10. Ten other cases also showed organisms having fermentation reactions approaching those of recognised dysentery bacilli.

Morgan's bacillus would also appear, according to Musgrave and Sison, to be a common cause of dysentery, especially amongst children, in the Philippines, where also occurs *Bowman's Bac. S.*

More recently Tribondeau and Fichet record 13 cases of Morgan's bacillus dysentery from the Dardanelles and refer to a small epidemic of 5 cases amongst troops at Toulon. They produce good evidence for their belief that the presence of Morgan's bacillus is not fortuitous in those cases but that it is the active pathogenic agent and must be added to the growing list of dysentery bacilli. Of their 13 cases there were three types; 7 corresponding with Morgan's No. I, 1 with Morgan's No. 3, and 5 with Morgan's No. 29.

They found agglutination and deviation of complement, as tested with the patient's serum, entirely negative. In this connection it might be noted that Ten Broeck and Norbury state that agglutinins are not found in the blood of cases from which the bacillus is isolated, but Ledingham in reviewing their paper states that this is not correct and refers to his work published in a communication by Morgan and himself (*Proc. Roy. Soc. of Med.*, Epidemiol. Sect., March, 1909). In the six of my cases in which agglutination with the patient's own serum was tested it will be noted that evidence of slight agglutinating qualities was found in five.

Organisms related to the *Gaertner-Paratyphoid* Group.

Nos. 21, 24 A, and 24 B show affinities with this group so far as their fermentation reactions go but they are all three non-motile and produce indol abundantly. Their non-motility would apparently make them more closely allied to the *Bac. enteritidis B.*, an organism which however I only know from the table given by Castellani and Chalmers.

RELATIVE PATHOGENICITY OF THE DYSENTERY BACILLI.

The cases are too few in number to generalise upon the question whether the Shiga bacillus produces a more severe type of the disease than other varieties, but it may be noted that of the seven fatal cases in my series two yielded the Shiga bacillus, one a Flexner-like organism, and four Morgan's bacillus.

Kuenen states that pseudo-dysentery bacilli (meaning types other than Shiga) as a rule set up a less severe form of the disease in Sumatra, although they may nevertheless cause a high percentage of deaths.

Hallenberger writes that in the tropics it does not always hold good that the *B. Shiga* produces a more serious illness than the mannite-fermenting varieties of *B. dysenteriae*. The opposite may occur.

SERUM TREATMENT.

Through the kindness of the Lister Institute I received a large quantity of polyvalent anti-dysenteric serum for trial treatment. Three strains (D 23, D 24, and D 25) were supplied me. Capt. Thompson, E.A.M.S., gave me an opportunity of testing these sera on cases in the African Base Military Hospital, Nairobi. A large number of cases were available and the treatment was given a thorough trial on cases which microscopic examination showed to be non-amoebic. Only severe cases were chosen as so many of the milder cases were recovering under ordinary medicinal treatment. Some severe cases were at the same time not given serum so as to serve as controls. All cases received similar medicinal and dietetic treatment.

The serum was employed in doses of from 20 c.c. to 100 c.c., in most cases the amount given being nearer the upper limit than the lower. The dose was injected into the subcutaneous tissues of the flanks and was as a rule repeated daily.

I regret to have to report that in no single case did the serum appear to have the slightest beneficial effect. The great majority of the cases in which it was employed died. A few recovered but not a larger proportion than amongst those who did not receive serum.

Dr R. W. Burkitt allows me to state that he gave a polyvalent serum a considerable trial on Europeans in his private practice in Nairobi but that he had given it up as he had failed to get any good results. He injected subcutaneously 100 c.c. as a general rule in adults, at the earliest possible stage of the attack.

The failure to get benefit by polyvalent serum treatment here is very disappointing seeing that it has yielded good results in various other places.

Willmore and Savage quote Vaillard and Dopter as still insisting on the value of a monovalent Shiga serum as efficient in the treatment of all bacillary dysenteries, but state that these writers appear to stand practically alone in this contention. Their own experience at El Tor (where Shiga cases are rare) was all against this view, but with polyvalent serum they obtained very good results. Their case mortality rate in 1911–1912 when no serum was available being 70 %, falling to 12 % in 1912–1913 when polyvalent serum treatment was employed. The rate for the earlier period was probably abnormally high but they adduce good evidence to show that the fall coincident with the introduction of serum treatment was not entirely fortuitous.

Kuenen reports good results with Shiga-Kruse serum in epidemic (Shiga) dysentery and states that the serum appears to act equally well in cases of pseudo-dysentery.

Bahr in Fiji, using the Lister Institute polyvalent serum, had only two deaths in 106 cases (1·8 %), whereas in 53 cases treated without serum the case mortality rate was 13·2 %.

The same observer in abstracting the article by Musgrave and Sison, who found polyvalent serum fail, states that their experience "is entirely opposed to that of other investigators of the disease." Whether this be so or not I am not sufficiently in touch with recent literature to say, but Remlinger and Dumas working in the Argonne region, with cases due mainly to a Y bacillus, report the inefficacy of both anti-Shiga serum and polyvalent anti-Shiga-Flexner-Y serum.

Musgrave and Sison consider that for practical purposes, in places where the means of identifying the infecting micro-organism are not available, treatment by polyvalent serum fails. They ascribe this to the important part played by other organisms such as staphylococci and colon bacilli in producing the inflammation of the intestinal mucosa.

What the reasons for the failure of serum treatment here may be is as yet uncertain. One probable factor is the frequency of Morgan's bacillus as the cause of the dysentery, as I fancy this organism is not employed in the preparation of the polyvalent serum. It may be also that the other strains of dysentery bacilli found here are too remotely allied to those from which the sera were prepared.

Once let the dysentery bacilli get a good start I am of opinion that much may be said for the view that common intestinal organisms play

an important secondary *rôle*. Clinically it is frequently noted here that cases apparently get over the acute attack, but that they do not pull round and sooner or later die, although their motions have ceased for some time to have a dysenteric character. Seven cases of this sort were amongst those I investigated. Post-mortem examination of four showed extensive ulceration of the large intestine, in two instances there being very little mucosa left throughout its whole length. Cultures in four of the seven cases failed to yield any dysentery bacilli; in the other three they were found either ante- or post-mortem.

Such cases would suggest that the other bowel organisms may be responsible in the later stages of the disease for the destruction of the mucosa; death following on absorption of faecal or bacterial poisons through the extensive raw surface.

The recent view advanced by Remlinger and Dumas that death is in many instances due to a supra-renal insufficiency and the results recorded by Parhon with treatment by adrenalin are suggestive. The treatment by adrenalin is being given a trial here.

Vaccine treatment with mixed Shiga-Morgan vaccines is also being tried both therapeutically and as a prophylactic measure.

Specimens of these various cultures have been sent to the Lister Institute, London.

SUMMARY.

Fifty-six cases examined for Dysentery bacilli. All save one were clinically dysentery, and all save two were negative for amoebae and other protozoa.

Twenty cases (35·7 %) yielded dysentery-like bacilli.

From these twenty cases thirty-six organisms were isolated and studied. Of these—

Six corresponded with no familiar pathogenic organism.

Six resembled *B. Shiga*. Two agreed completely both by fermentation and agglutination. One also gave typical fermentation and agglutination results but was at first a motile organism, although in later generations it became non-motile. The other three are less definitely Shiga bacilli.

Five were mannite-fermenting organisms but only one agreed very closely in its fermentation reactions with *B. Flexner* and it was not agglutinated by Flexner serum.

Sixteen cultures from ten separate cases corresponded closely with Morgan's bacillus.

Eight of these agreed completely in their characters with *Morgan's No.* 1 bacillus.

Three differed only from *Morgan's No.* 1 in being non-motile.

Five presented characters intermediate between those of *Morgan's No.* 1 and Bowman's "*Bac. S.*"

Of six cases yielding Morgan's bacillus, agglutination with the patient's own serum was positive in five.

Three cultures showed affinities with the *Bac. enteritidis.*

Polyvalent anti-dysenteric serum treatment tried on a considerable number of cases was a complete failure.

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