

## A POLYVALENT VACCINE IN THE TREATMENT OF BACILLARY DYSENTERY IN EAST AFRICA<sup>1</sup>.

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THE research on which this paper is based, was carried out between September 1916, and April 1917, while the writer was in charge of the Carrier Depot Hospital, Nairobi, British East Africa.

Dysentery was the most serious disease met with amongst the porters of the Military Labour Bureau in the early days of the East African Campaign, and was the main cause of death and invaliding even as late as the latter six months of 1916, although stringent sanitary measures had done much to diminish its incidence. Apparently at first it was thought that the disease was amoebic in type, and treatment consisted almost entirely in the administration of emetine. Dr Pirie, Government Pathologist at Nairobi, was the first to point out that this idea was wrong, and in the first eight months of 1916, he conducted a series of bacteriological examinations of the stools of 56 cases of dysentery and showed that in only two cases were amoebae found while dysentery-like bacilli were actually isolated in 35·7 per cent., the main type isolated resembling *B. Shiga*, or frequently *B. Morgan*<sup>2</sup>.

It was just at the conclusion of Dr Pirie's work in June 1916, that the writer took over charge of the Carrier Hospital at Nairobi. From that time the stools of every patient admitted to hospital for dysentery or diarrhoea, were examined by the writer for protozoa, at least twice, and often thrice, as a routine practice, and it was soon apparent from the number of negative results that the conclusions of Dr Pirie were justified, and that we were dealing with an outbreak of dysentery of the bacillary type and not of the amoebic type. This was also borne out by the failure of emetine in the treatment of the disease. As a result of this work the routine administration of emetine ceased, and reliance was placed mainly on saline treatment, though all the usually recommended lines of treatment for bacillary dysentery were tried at one time or other. Still the death rate did not improve as it should have done, and a very large number of cases became more or less chronic. At last it occurred to the writer that a vaccine based on the results of Dr Pirie's research might improve matters, and Dr Ross, the Director of Laboratories,

<sup>1</sup> A preliminary note on this work has appeared in the *British Medical Journal*.

<sup>2</sup> *Journ. of Hygiene*, xv. No. 4.

B.E.A., and Dr Pirie were consulted on the subject. They thought that the idea was worth a trial and agreed to prepare the vaccine. The main difficulty lay in the highly toxic nature of dysentery vaccines prepared in the usual manner, but it was surmounted by Dr Ross's suggestion to sterilise the proposed vaccine by 0.4 per cent. carbolic acid and not by heat. It may be as well to state here, particularly as it is apparently the first occasion on which a dysentery vaccine sterilized in this way has been used on such an extensive scale, that the result was most satisfactory, for in no single instance, either in the prophylactic inoculation of 76,000 porters with the vaccine or in the therapeutic inoculations described in this paper, was an alarming general or even local reaction seen.

The earliest vaccine tried consisted of *B. Shiga* and *B. Flexner* in equal proportions, and as it was uncertain what was the minimum dose that produced effects and what was the maximum dose that could be given with safety, an initial dose of 5 million was tentatively tried. This was cautiously increased as the vaccine showed its value. The original vaccine was brilliantly successful in certain cases and just as distinct a failure in others, although these latter were also of the bacillary type. It was therefore concluded that probably the failures were due to other strains of dysentery bacilli causing the disease in those cases, and consequently the vaccine was made more polyvalent, and finally contained eight strains, namely *B. Shiga* three strains, *B. Flexner* two strains, *B. Morgan* three strains. This was the vaccine used in the cases recorded in the present paper.

Before discussing the results of vaccine therapy in bacillary dysentery, it seems best to give a short account of the disease as seen amongst Africans, in whom it appears to be slightly different in its manifestations and course to the disease amongst Europeans.

#### CAUSES.

In addition to the usually recognized predisposing cause of dysentery, the following factors seem to play an important part in the origination of the disease among Africans.

(1) *Change of Environment.* This factor was particularly noticeable in those recruits who arrived in Nairobi from the region around Lake Victoria Nyanza, involving a train journey of some 250 miles over a water-shed 9000 feet high. In many the disease showed itself during the actual train journey, in others it only appeared after a residence of two or three days in the Carrier Depot at Nairobi. Although in this depot there were recruits from the districts of Kikuyu and Akamba as well as these Lake people the incidence of the disease was distinctly heavier among the latter, and one was forced to conclude that the men were infected before arrival in Nairobi. The foregoing conclusion received support from the fact that this difference in incidence still obtained in Mombasa, to reach which place both Nairobi district and Lake porters had the same rail journey. This therefore raises the question as to whether Lake

tribes are dysentery "carriers," the change in environment leading to an exacerbation of the disease. The writer hopes to be able to throw further light on this suggestion in a later paper.

(2) *Change of Diet.* In the early stages of the campaign it was not sufficiently realized that a native is subject to the same laws of dietetics as an European, and that in his home an immense amount of labour is put into the preparation and thorough cooking of his meal by his wife or wives. When this was understood and a well cooked, well balanced diet provided for the porter, an immediate lessening of intestinal disorders resulted. The African, however, undoubtedly possesses intestines peculiarly liable to be attacked by inflammatory disease caused by errors in diet, resulting in diarrhoea or even dysentery.

(3) *Helminthiasis.* This is extraordinarily prevalent and, in the writer's opinion, is a most important predisposing cause of dysentery (possibly owing to the delicacy of the African's intestines as mentioned above) and in a paper to be published shortly he hopes to bring forward evidence to support this statement. It is sufficient here to give a summary of the helminth infections among the cases on record in this paper as obtained from the results of the examinations of stools.

Table I.

*Helminth Infections in Dysentery Patients.*

Class of case	Total cases examined	Number of species of Helminths observed										Total of infected cases			
		Negative		One		Two		Three		Four		Five		No.	%
		No.	%	No.	%	No.	%	No.	%	No.	%	No.	%		
Effectives ...	473	42	9	204	43	140	29.5	70	15	15	3	2	0.5	431	91
Invalids ...	272	28	10	100	37	98	36	39	14	7	3	—	—	244	90
Total ...	745	70	9.5	304	40	238	32.75	109	14.5	22	3	2	0.25	675	90.5

In contrast to this, take the following table compiled from the results of autopsies on cases not suffering from dysentery.

Table II.

*Helminth Infections in Non-Dysenteric Patients.*

Class of case	Total cases examined	Number of species of Helminths observed										Total of infected cases			
		Negative		One		Two		Three		Four		Five		No.	%
		No.	%	No.	%	No.	%	No.	%	No.	%	No.	%		
Effectives } Invalids )	260	80	31	124	48	49	19	7	2	—	—	—	—	180	69
Total ...	260	80	31	124	48	49	19	7	2	—	—	—	—	180	69

## PATHOLOGY.

One of the most noticeable features about Africans suffering from dysentery is the rapid emaciation which occurs in even moderately bad cases. Forty-eight hours seem sufficient to reduce a patient to a condition of skin and bone. It is a matter of regret that no actual weight statistics are available to illustrate this.

*Central Nervous System.* This usually shows no macroscopical change. In a few cases, however, congestion of the meninges is distinct and the cerebrospinal fluid is increased in amount. Possibly this may be associated with the acute toxæmia which is always present in these cases.

*Respiratory System.* The lungs, in almost all cases, show signs of congestion and oedema, and it is worthy of note that many cases of dysentery die as the result of a terminal pneumonia. In all the patients in which pneumonia developed in this series, the sputum invariably showed typical pneumococci only. It seems probable that this is a complication of dysentery due to the lowered resistance of the patient (especially as the African seems to be extraordinarily susceptible to pneumococcal infections), and not one of the manifestations of a generalized septicaemia.

*The Circulatory System* shows little change except in a degeneration of the heart muscle which starts as cloudy swelling and in some instances ends in actual fatty degeneration of the myocardium. The heart in most cases is somewhat dilated and often contains an antemortem clot on the right side.

*The Genito-Urinary System* is apparently unaffected, a few cadavers showed congestion of the kidney, which may have had no connection with the original disease.

*The Alimentary System* shows the majority of the lesions in this disease. In the first place the mouth, in very bad cases, may be the site of a certain amount of ulceration, and in an appreciable number of the very toxæmic patients, this developed into cancrum oris, a condition which usually led to the rapid appearance of the man in the mortuary. This lesion seemed in the majority of cases to have developed in the gum in close proximity to the teeth, and spread from there. The pus from these cases showed only the usual pyrogenic cocci and some bacilli, both Gram-positive and Gram-negative, but with the means at our disposal they could not be identified.

The stomach, as a rule, was normal in appearance, but in a few isolated cases, in which toxæmia was most distinct, ecchymoses were present in the mucous membrane and in the serous covering.

The small intestines presented a normal appearance, with a few exceptions in which ecchymoses were seen near the lower ends of the ileum, and occasionally slight superficial ulceration was present in the same area. Peyer's patches were not enlarged. The lymphatic glands in the mesentery were usually enlarged and sometimes showed small hæmorrhages in their substance, but no organisms could be isolated from them.

The large intestine was the site of the most extensive and characteristic lesions. As a rule these were confined to the sigmoid and descending colon, but were also found in seven cases in the ascending colon and caecum. No lesion of the appendix was ever observed.

The lesions were divisible into three main types:

Type (1). This was evidently an early form and was seen in cases which succumbed to toxæmia in the very early days of the disease, or in cases which were suffering from some other ailment and in which dysentery had developed in the later stages of that illness. The sigmoid colon and descending colon showed a dark red velvety surface, the mucous membrane being slightly thickened, and here and there small petechial hæmorrhages. The surface of the mucous membrane was covered with a layer of mucus, more or less faecal-stained. The blood vessels running to the affected portion of the large intestine were engorged as a rule and the veins in the actual walls of the intestine were in a condition of venous congestion. No signs of ulceration were present, nor were there any raised yellow nodules on the surface of the intestine suggestive of an amoebic origin of the disease. Sections of the intestine showed engorgement of the mucous cells of the glands of the large intestine, and a diapedesis of polymorpho-nuclear leucocytes into the submucous layer. In three cases a similar appearance was seen in the caecum and ascending colon, either as a concomitant of the disease in the descending colon, or as a solitary lesion.

Type (2). The second stage was evidently only an extension of the first. The lesions were found in the same portions of the intestines, but as a rule occupying a more extended area. The surface of the intestine was covered with a yellowish or dark brown slough, either continuously in the very worst cases, or in patches of varying size. On removal of this slough, an ulcer was left beneath, which, as a rule, presented an appearance of only affecting the mucous membrane and the submucous coat. The edges were irregular in outline, and perpendicular more or less to the floor, which was sometimes rough and sloughy, but usually smooth and formed of the muscular tissue of the intestinal wall. In bad cases the ulceration occasionally extended down to the surface of the serous membrane, but in none of the cases which died, amongst the ones recorded in this paper, was perforation seen. A section of the intestine showed, in the ulcerated area, the usual appearance of coagulation necrosis at the edge of the ulcers, and outside this area the appearance seen in the first stage of the disease described above.

Type (3). The third stage was only seen in chronic cases which had responded to treatment at first, inasmuch as the acute signs of dysentery ceased, but which had developed into a condition of chronic diarrhoea with emaciation. The appearances presented varied enormously. The areas of the intestine affected corresponded to those in the first and second stages. In these cases the large intestine was full of a light-coloured yellowish fluid containing nodules of solid faecal material. Very little mucus was noticeable

on the surface of the intestine. In some cases ulceration of the intestine was still present, but the ulcers were clean with puckered, thickened edges, and apparently in a state where an attempt at healing was taking place. The ulcerated areas were, as a rule, small compared to those in stage 2. The intervening mucous membrane showed scarred areas of fibrous tissue, evidently the site of former ulceration, and much thickening of the remaining unaffected surface. This latter portion showed a velvety appearance with marked rugae. Sections through the ulcers showed regeneration changes in the edges, but, as a rule, little or none in the floor of the ulcer which was practically always formed of the muscular coat.

In other cases no actual ulceration was present, but large areas of the large intestine showed a smooth glazed surface which were the sites of former ulcers, and between these, thickened rugose mucous membrane with markedly velvety appearance. In these cases death was apparently due to loss of fluid owing to a large portion of the large intestine having been thrown out of operation as an absorbing area.

The liver in acute cases was enlarged and engorged. No abscess was seen in it, except in one case which was suffering from *Schistosomum mansoni*, and a scraping of the walls of the abscess showed the eggs of this helminth, but no amoebae.

The spleen in acute cases was enlarged and soft. In chronic cases it was also usually enlarged, but whether this was due to dysentery or to chronic malaria, it would be difficult to say.

The other organs in the alimentary system were normal.

#### SYMPTOMS.

In an African native the symptoms usually associated with dysentery are not so distinct as in the case of Europeans, either from the native dislike of hospital treatment, or from the undoubted fact that, taken as a whole, he is less sensible to pain. The writer personally has seen cases which have gone out to duty involving hard manual labour while suffering with severe dysentery of several days standing, and which, on being called out as suspected cases, have indignantly denied being ill. The consequence of this is that, in a large proportion of cases, men have not come for treatment till the disease is well advanced, and a toxæmic condition already manifest. Dysentery is not usually a disease simulated by the malingerer, owing to the native's dislike of light diet. As a rule the patient only complains of passing blood in the stools, and diarrhoea. On further questioning he may confess to having a certain amount of pain in the stomach and straining at stool. Headache and fever are also complained of in a small proportion of cases.

## PHYSICAL SIGNS.

As has been noted above, the patient often shows signs of the onset of toxæmia in wasting, lethargy, and general weakness. The rapidity with which a native wastes as the result of disease, particularly abdominal disease, is remarkable, and is only equalled by the rapidity with which he puts on flesh once a cure is effected and good diet instituted.

The temperature varies considerably. Some cases never show any rise of temperature from the beginning to the close of the illness, others show an intermittent temperature running up to 101° F. throughout the whole course of the disease, the majority show an initial rise of temperature from 100° F. to 102° F., which, with remissions, lasts for two to three days. A subnormal temperature is usually noticeable before death.

The pulse and respiration rates closely follow the temperature, but as a rule are slightly more rapid than normal during apyrexia, rising to about 80 and 30 respectively.

The tongue is covered with a brownish white fur, but in cases showing distinct toxæmia, it becomes shrunken, covered with brown fur, and is moved with difficulty. The teeth and lips in this toxæmic stage are covered with sordes.

The abdomen as a rule presents no abnormal appearance, though if the disease has been in existence some days, it may be sunken. On palpation, a tender area in the left iliac fossa is discernible. The area of tenderness varies in extent with the severity of the disease, but is usually most marked in the region of the sigmoid colon, and diminishes as one palpates along the length of the descending colon to the splenic flexure. In very bad cases, the colon may be tender and thickened along its whole length.

The stool varies greatly in appearance. The amount of blood may be almost infinitesimal, and in some cases requires a microscope for its detection, or it may constitute the major portion of the evacuation. Mucus is always found if searched for, and usually in fairly large quantities. It is not mixed intimately with the blood, the latter forming streaks on its surface. The actual faecal content of the stool in the early stages of the disease is small, but increases with the administration of purgatives. It is rarely normal in colour, being either paler than usual and even in some cases quite white, or else green. In either case the smell indicates an increase in intestinal fermentation gases. In the very early stages of the disease the stool may be quite formed, the mucus and blood forming a coating for more or less scybalous masses, but this rapidly disappears, and diarrhoea becomes a constant feature. It is very noticeable that the number of stools rarely exceeds 20 a day and is usually not more than 10 to 15.

The liver nearly always shows an increase in size, to about a finger's breadth below the costal margin, and may be slightly tender to the touch. The spleen is rarely affected.

The respiratory system shows no physical sign, unless death is supervening, when indications of hypostatic congestion are found, and in certain cases a terminal lobar pneumonia may end the scene.

The genito-urinary system is not as a rule affected. The urine is usually highly acid, may be small in quantity and consequently highly coloured. Although it was never actually determined, the writer is inclined to believe that the colouring matter is actually increased and not relatively so as the result of concentration.

The blood shows a slight decrease in the content of red blood corpuscles, and a slight polymorphonuclear leucocytosis.

In cases in which toxaemia is marked, the face becomes sunken, the lips and teeth covered with sordes, the tongue covered with brown fur, the abdomen boat-shaped, the patient restless and semi-conscious or delirious. Hiccough then starts, and is nearly always a sign that death is near. In only a few cases when hiccough once sets in, has recovery occurred. In certain patients, hiccough was the first symptom of the onset of toxaemia.

The circulatory system does not, as a rule, show any change. In a few cases when the disease had lasted some considerable time, or toxaemia was well marked, haemic murmurs appeared, but very little dilatation of the heart was observable antemortem.

#### COMPLICATIONS.

(1) *Cancrum Oris*. This occurred in a few cases in which the dysentery had lasted for some time, and the patient was in a state of chronic toxaemia.

(2) *Perforation*. No case of perforation was ever seen.

(3) *Peritonitis*. No case with peritonitis occurred among these cases.

(4) *Toxaemia*. This is a very frequent complication of bacillary dysentery. Owing to the pressure of routine work, it was impossible to estimate the acidity of the blood in these cases, but it seems probable that an acidosis was responsible for these symptoms. It is hoped in a later paper to elucidate this point. At least it can be recorded here that alkaline treatment gave promise of better results than any other in combating this complication.

(5) *Tissue Desiccation*. This is a frequent cause of death in bacillary dysentery, the constant loss of fluid by the bowel causing a corresponding loss of water by the body cells. Although everything was done to increase the quantity of fluid taken by the patient, it was difficult to overcome the African's rooted idea that the drinking of water leads to increased diarrhoea. Objections were even raised by the natives to fluid diet on this score. Recourse to subcutaneous saline infusion was consequently often necessary.

(6) *Post-Dysenteric Diarrhoea*. Frequently after all signs of dysentery have ceased, an intractable diarrhoea persists, often accompanied by marked toxaemia and wasting. The most probable explanation seems to be that a secondary infection of the ulcers in the large intestine with *B. coli communis* or other intestinal organisms takes place.



## TREATMENT.

Practically every recommended method of treatment was tried during the time the writer was in charge of the hospital. Before his advent, emetine was given to all cases with any symptoms of dysentery, and as far as could be seen from clinical records, without success of any sort. The routine dose was one grain hypodermically once daily for seven to ten doses. Inasmuch as the clinical benefits were negative, and no evidence of amoebic infection was found microscopically or postmortem, the administration of emetine was stopped, except in cases in which amoebae were found by microscopic examination.

Small doses of calomel namely half a grain every hour for eight hours daily for three days, was a form of drug treatment largely used in the earlier cases of this series. Theoretically the idea of stimulating the secretion of bile seems to be an excellent one, inasmuch, as has been mentioned above, the biliary function of the liver seems disorganized in bacillary dysentery, as shown by the green or light coloured stools so frequently seen. Practically it was found that this treatment was too drastic for the majority of the patients suffering from this disease and eventually it was abandoned.

The sulphates of magnesium and sodium were next tried, and it was found that better results were secured by the former salt. Although it is said to exercise a specific effect in bacillary dysentery, this was found not to be the case, as alone without vaccine treatment, it had hardly any perceptible effect on the course of the disease. Combined with vaccine treatment, it was however excellent, and consequently it remained the main drug in the oral treatment of the disease. It may be remarked here that it seems strange that sodium sulphate, which is believed to act more strongly on the biliary secretion than magnesium sulphate, should have a weaker therapeutic action than the latter, but such is the practical experience in this series of cases. Other drugs tried for oral treatment were *mistura chlorinata* consisting of potassium chlorate  $2\frac{1}{2}$  grains, acid hydrochloric pure  $3\frac{1}{2}$  minims, quinine hydrochloride 3 grains, syrup of lemon 40 minims, water to 1 ounce, given every four hours, this being without any appreciable effect on the disease, and castor oil in 4 drachm doses thrice daily also with results distinctly inferior to those of magnesium sulphate.

In addition to these medicines, the object of which was either to wash out the toxins from the bowel or to inhibit bacterial development in the intestines, other drugs were used. Thus, as a result of the discovery of the vast number of dysentery patients suffering from worms (one patient passed a *Taenia saginata* which was rolled into a ball the size of a football, took 20 minutes to pass, and when laid out was 250 feet long and only one head was found in the whole mass; another passed 80 *Ascaris lumbricoides*, 30 on the first day and 50 on the second), anti-helminthics were always administered directly a patient entered hospital, and repeated at intervals till microscopic examination showed that the stools were negative. Of the anti-helminthics, thymol was found to

be the best, given in three doses of 30 grains at intervals of 2 hours on an empty stomach, followed by a dose of magnesium sulphate. Of the others, beta-naphthol was found the second best, while santonin proved the most effective in the treatment of ascariasis. Thymol was equally effective with tapeworms as with ankylostomes, surpassing *Felix mas* in the elimination of the former infection.

At a late stage of this series, the writer's attention was drawn to a note in the *British Medical Journal* relating to the use of *Tinctura Rhei Composita* in post-dysenteric diarrhoea. This drug was tried in three cases, and found so efficient in 4 drachm doses repeated at intervals of four days when necessary, that it became a routine treatment, and in at least two cases of children suffering from dysentery, it was found to effect a cure quite apart from other treatment.

As mechanical methods for cleaning the inflamed bowel and diminishing toxin absorption, rectal irrigations of sodium carbonate, potassium permanganate, tannic acid, silver nitrate, and protargol were all tried in succession, but it must be confessed without any improvement in the case. It seems probable that the injection, inasmuch as it could only be given by native orderlies, failed to reach the inflamed portion of the bowel, and was so disliked by the African that the mental depression it produced probably more than counteracted any good done by the actual irrigation.

As regards complications tissue desiccation was met by enforcing the taking of fluid by mouth, and when this was not enough, by subcutaneous saline infusions. In the later cases of the series, when the condition of the patients was extremely critical even when admitted, the latter treatment was called for from the first, and in many cases undoubtedly saved the patient's life. In the light of later knowledge, however, the writer would use an isotonic solution of sodium bicarbonate, and would be tempted to try Professor Bayliss's 6 per cent. gum arabic in this solution. The infusion acts partly by replacing lost tissue fluid, partly by raising the blood pressure and partly by diluting the toxins in the blood.

Toxaemia was combated by the administration of potassium bicarbonate in 30 gr. doses four times daily, and when the potassium salt was not available, that of sodium. This was based on a supposition yet unproved that the toxaemia was due to acidosis. Certainly in some cases it did good, and was one of the most effective drugs in relieving hiccough, which again was probably only a result of acidosis. As a further attempt to diminish the absorption of toxins from the bowel, caecostomy was performed in two cases and continuous saline irrigation instituted. Unfortunately though both cases survived the actual operation, it was apparently performed at too late a stage of the disease, and both succumbed to toxaemia. The main difficulty in this treatment seems to be to select cases late enough to justify the performance of the operation, yet early enough to give the patient a reasonable hope of recovery. Still it seems better to operate too early than too late.

For hiccough, potassium bromide in 10 gr. doses thrice daily, and a mustard plaster on the epigastrium seemed to be the most effective measures of treatment. Tinctura iodi in one minim doses in water every hour for six hours was very useful in certain obstinate cases.

Cancrum Oris was always fatal until it was treated by a potassium chlorate gargle combined with the internal administration of 15 gr. of potassium chlorate thrice daily. Under this treatment about 50 per cent. of the cases recovered.

Post-dysenteric diarrhoea was treated with considerable success by giving a vaccine of *B. coli communis* (2000 million per c.c.) in doses of  $\frac{1}{2}$  c.c., 1 c.c., and 2 c.c. at intervals of three days, Tinctura Rhei Composita in 4 drachm doses at intervals of four days was also useful in certain cases.

DIET.

Copies of two of the latest hospital dietaries are attached. In these dietaries Diet B combines the Fluid Diet and Semi-solid Diet of the dietary in force when the cases referred to in this paper were in hospital.

DIET I.

DIET A			DIET B		
(Green Ticket)		Hour	(Yellow Ticket)		Cooking instructions
Milk	8 oz.	2 a.m.	Milk	10 oz.	<i>Milk.</i> All milk must be boiled and served to the patients warm.
Brandy	$\frac{1}{2}$ "	4 a.m.	Sugar	1 "	
Sugar	$\frac{1}{2}$ "	5 a.m.	Milk	10 oz.	
			Sugar	1 "	<i>Arrowroot.</i> This must be boiled with milk and sugar for half an hour.
Tea	1 pint	6 a.m.	Tea	1 pint	
Sugar	1 oz.		Sugar	1 oz.	
Milk	4 "		Milk	2 "	<i>Matama Uji.</i> This must be boiled for two hours, forming a thin gruel.
Arrowroot	4 oz.	8 a.m.	Matama uji	6 oz.	
Milk	2 "				
Sugar	$\frac{1}{2}$ "				<i>Rice Jelly.</i> The rice must be cooked for at least three hours, or longer if the rice has not reached the stage of jellification.
Milk	4 oz.	10 a.m.	Milk	4 oz.	
Brandy	$\frac{1}{2}$ "		Sugar	$\frac{1}{2}$ "	
Soup	1 pint	12 noon	Rice jelly	8 oz.	
Bread	4 oz.		Milk	4 "	
Milk	4 oz.	2 p.m.			
Brandy	$\frac{1}{2}$ "				
Tea	1 pint	4 p.m.	Tea	1 pint	
Sugar	1 oz.		Sugar	1 oz.	
Milk	4 "		Milk	2 "	
Arrowroot	4 oz.	5 p.m.	Matama uji	6 oz.	
Milk	2 "				
Sugar	$\frac{1}{2}$ "				
		6 p.m.			
Milk	8 oz.	8 p.m.			
Brandy	$\frac{1}{2}$ "	10 p.m.	Milk	10 oz.	
Sugar	$\frac{1}{2}$ "		Sugar	1 "	
		12 midnight			

Uji is a thin gruel.  
Ugali is a very thick porridge.

*Bacillary Dysentery*

## DIET I (cont.).

## DIET C. Full (White Ticket).

Monday, Wednesday, Friday, and Sunday.

Diet per man	Cooking pots per 500 men	Hour	Cooking instructions
	3 of 25 gals.	2 a.m.	Boil water.
		3 a.m.	Put in crushed mealies 12 oz. per man and 1 lb. salt per 25 gallon pot. Boil fiercely till 10 a.m.
	2 of 25 gals.	4 a.m.	Put 4 oz. beans per man and $\frac{3}{4}$ lb. salt per 25 gallon pot into cold water, bring to boil and boil hard till 11 a.m.
Tea	1 of 100 gals.	5.30 a.m.	Boil water, make tea 1 pint per man with 2 oz. sugar per man. Serve at 6 a.m.
Sugar	2 of 25 gals. (matama)	6 a.m.	Put 6 oz. matama flour per man into some boiling water without salt and boil till 8 a.m. making uji.
	5 of 5 gals. (rice)		Put 2 oz. per man of rice into cold water with 1 oz. salt per 5 gallon dixie and boil till 7.30 a.m., then evaporate off water over slow fire.
Matama	6 oz.	8 a.m.	Serve matama uji with rice.
Rice	2 "		
	1 of 25 gals. (meat)	9 a.m.	Put meat $\frac{1}{2}$ lb. per man with 1 lb. salt per 25 gallon pot into some boiling water.
	1 of 25 gals. (bones)		Put bones from this meat into cold water and boil up.
			Boil meat and bones till 12 noon, mix soup from each, and give 1 pint per man.
		10 a.m.	Pour off water from crushed mealies, transfer latter to small dixies, evaporate off remaining water over slow fire.
	2 of 25 gals.	10.30 a.m.	Put peeled bananas $\frac{1}{2}$ lb. per man with $\frac{1}{2}$ lb. salt per 25 gallon pot into boiling water, boil till 11.30 a.m., pour off water and beat bananas into a mash.
Bananas	$\frac{1}{2}$ lb.	11.30 a.m.	Pour off water from beans, put latter in small dixies over a slow fire to evaporate off rest of water.
Mealies	$\frac{3}{4}$ "		
Beans	$\frac{1}{4}$ "		
Meat	$\frac{1}{2}$ "		
Soup	1 pint	12 noon	Mix beans, crushed mealies and banana mash into a thick paste and serve with meat and soup.
		3 p.m.	Put washed unpeeled sweet potatoes 3 lb. per man without salt into cold water, boil till 4.30 p.m., then pour off water and steam over a slow fire.
Sweet potatoes	3 lb.	5 p.m.	Serve sweet potatoes.

*Alternatives.*

1. Matama flour, mwele flour, wimbe flour and mohogo meal are all interchangeable when made into uji.

2. Crushed mealies can be replaced by mealie meal ugali. For this mealie meal need only be boiled for five hours but should be treated exactly the same as crushed mealies otherwise.

3. Sweet potatoes may be replaced by ordinary potatoes 2 lb.

DIET I (cont.).

DIET C. Full (White Ticket).

Tuesday, Thursday and Saturday.

Diet per man	Cooking pots per 500 men	Hour	Cooking instructions
Tea 1 pint } Sugar 2 oz. }	1 of 100 gals.	5.30 a.m.	Boil water, make tea 1 pint per man with 2 oz. sugar per man. Serve at 6 a.m.
	2 of 25 gals. (matama)		Put 6 oz. matama flour per man in some boiling water without salt and boil till 8 a.m. making uji.
	5 of 5 gals. (rice)		Put 2 oz. of rice per man in cold water with 1 oz. of salt per 5 gallon dixie and boil till 7.30 a.m., then evaporate off water over slow fire.
Matama uji 6 oz. } Rice 2 ,, }	2 of 25 gals.	8 a.m.	Serve matama uji with the rice.
		„	Put crushed mealies 6 oz. per man in boiling water with 1 lb. salt per 25 gallon pot. Boil hard till 3 p.m.
	1 of 25 gals.	8.30 a.m.	Put 2 oz. beans per man and $\frac{3}{4}$ lb. salt per 25 gallon pot into cold water, bring to boil and boil hard till 4 p.m.
	1 of 25 gals. (meat)	9 a.m.	Put meat $\frac{1}{2}$ lb. per man with 1 lb. salt per 25 gallon pot into boiling water.
	1 of 25 gals. (bones)		Put bones from this meat into cold water and boil up.
			Boil meat and bones till 12 noon, mix soup from each and give 1 pint per man.
	22 of 5 gals. (dixies)	10 a.m.	Put rice 12 oz. per man into boiling water in dixies. Boil and stir well till 11.30 a.m. Add 1 oz. salt per dixie and $\frac{1}{2}$ oz. ghee per man at 10.30 a.m.
		11.30 a.m.	Put rice over small fire to evaporate off water.
Rice 12 oz. } Ghee $\frac{1}{2}$ ,, } Meat $\frac{1}{2}$ lb. } Soup 1 pint }		12 noon	Serve rice, meat and soup.
		3 p.m.	Pour off water from crushed mealies, transfer latter to small dixies and evaporate off remaining water over slow fire.
	1 of 25 gals.	3.30 p.m.	Put peeled bananas $\frac{1}{4}$ lb. per man with $\frac{1}{2}$ lb. salt per 25 gallon pot into boiling water, boil till 4.30 p.m., pour off water and beat into mash.
		4 p.m.	Pour off water from beans, put latter into dixies over a slow fire to evaporate off remaining water.
		4.30 p.m.	Mix beans, crushed mealies and banana mash into a thick paste.
Bananas $\frac{1}{4}$ lb. } Mealies 6 oz. } Beans 2 oz. }		5 p.m.	Serve the mixed beans and mealies.

Alternatives.

1. Matama flour, mwele flour, wimbe flour and mohogo meal are all interchangeable when made into uji.

2. Crushed mealies can be replaced by mealie meal ugali. For this mealie meal need only be boiled for five hours but should be treated exactly the same as crushed mealies otherwise.

*Bacillary Dysentery*

## DIET II.

DIET A			DIET B		
(Green Ticket)		Hour	(Yellow Ticket)		Cooking instructions
Milk	8 oz.	2 a.m.	Milk	10 oz.	All milk must be boiled and served to the patient warm.
Brandy	$\frac{1}{2}$ "		Sugar	1 "	
Sugar	$\frac{1}{2}$ "				
Milk	8 oz.	4 a.m.			<i>Arrowroot.</i> This must be boiled with milk and sugar for half an hour.
Brandy	$\frac{1}{2}$ "				
Sugar	$\frac{1}{2}$ "				
Milk	8 oz.	5 a.m.	Milk	10 oz.	4 ounces making 2 pints.
Brandy	$\frac{1}{2}$ "		Sugar	1 "	
Sugar	$\frac{1}{2}$ "				
Tea	1 pint	6 a.m.	Tea	1 pint	<i>Mealie meal uji.</i> This must be boiled for two hours forming a thin gruel.
Sugar	1 oz.		Sugar	1 oz.	
Milk	4 "		Milk	2 "	
Arrowroot	4 oz.	8 a.m.	Mealie meal uji	6 oz.	
Milk	2 "		Sugar	1 "	
Sugar	$\frac{1}{2}$ "				
Milk	4 oz.	10 a.m.	Milk	4 oz.	<i>Rice Jelly.</i> The rice must be cooked for at least three hours or longer if the rice has not reached the stage of jellification.
Brandy	$\frac{1}{2}$ "		Sugar	$\frac{1}{2}$ "	
Soup	1 pint	12 noon	Milk	4 oz.	
Bread	4 oz.		Rice jelly	8 "	
Milk	4 oz.	2 p.m.			
Brandy	$\frac{1}{2}$ "				
Tea	1 pint	4 p.m.	Tea	1 pint	
Sugar	1 oz.		Sugar	1 oz.	
Milk	4 "		Milk	2 "	
Arrowroot	4 oz.	5 p.m.	Mealie meal uji	6 oz.	
Milk	2 "		Sugar	1 "	
Milk	8 oz.	6 p.m.			
Brandy	$\frac{1}{2}$ "				
Sugar	$\frac{1}{2}$ "				
Milk	8 oz.	8 p.m.			
Brandy	$\frac{1}{2}$ "				
Sugar	$\frac{1}{2}$ "				
Milk	8 oz.	10 p.m.	Milk	10 oz.	
Brandy	$\frac{1}{2}$ "		Sugar	1 "	
Sugar	$\frac{1}{2}$ "				
Milk	8 oz.	12 midnight			
Brandy	$\frac{1}{2}$ "				
Sugar	$\frac{1}{2}$ "				

Uji is a thin gruel.

Ugali is a very thick porridge.

## DIET C. (White Ticket.)

Diet per man	Cooking pots per 500 men	Hour	Cooking instructions
Tea	1 of 100 gals.	5.30 a.m.	Boil water, make tea 1 pint per man with 2 oz. sugar per man. Serve at 6 a.m. with 1 oz. milk.
Sugar	2 of 25 gals.	6 a.m.	Put 6 oz. mealie meal flour per man in boiling water and $\frac{1}{2}$ lb. salt per 25 gallon pot and boil till 8 a.m. forming a thin gruel.
Milk	(mealie meal uji)		
	5 dixies rice (5 gals.)	6 a.m.	Put 2 oz. rice per man in cold water with 1 oz. salt per 5 gallon dixie and boil till 7.30 a.m. Then evaporate off water over a slow fire.

DIET II (*cont.*).

Diet per man	Cooking pots per 500 men	Hour	Cooking instructions
Mealie uji 6 oz. } Rice 2 ,, }		8 a.m.	Serve mealie meal uji with rice.
	2 of 25 gals. 1 of 25 gals.	8.30 a.m.	Put 6 oz. beans per man into cold water and boil hard till 11.30 a.m. Pour off water and serve at 12 noon.
		10 a.m.	Put rice 12 oz. per man into boiling water in dixies, boil and stir well till 11.30 a.m. Add salt 1 oz. per dixie and $\frac{1}{2}$ oz. ghee per man at 10.30 a.m.
		11.30 a.m.	Put rice over a small fire to evaporate off water.
Rice 12 oz. } Ghee $\frac{1}{2}$ ,, } Beans 6 ,, }		12 noon	Serve rice and beans.
	2 of 25 gals. 1 of 25 gals. (meat) 1 of 25 gals. (bones)	2 p.m.	Put meat $\frac{1}{2}$ lb. per man with 1 lb. salt per 25 gallon pot into boiling water. Put bones from this meat into cold water and boil. Boil meat and bones till 5 p.m. Mix soup from each and give 1 pint per man.
		3 p.m.	Put 6 oz. mealie meal flour per man in boiling water and $\frac{1}{2}$ lb. per 25 gallon pot and boil till 5 p.m. forming a thin gruel.
Mealie meal uji 6 oz. } Meat $\frac{1}{2}$ lb. } Soup 1 pint }		5 p.m.	Serve mealie meal uji and meat and soup.

The patient was treated with a fluid diet until his stools had been solid for two days and without any suspicion of blood or mucus. He was then put on to the semi-solid diet for two days, and if no diarrhoea showed itself in this time, he went on to full diet.

## GENERAL TREATMENT.

All dysentery cases were kept in bed as far as possible during the acute stage of the disease. Their mouths were cleaned twice daily with potassium permanganate lotion, and, if necessary, also in the intervals between these routine washings. They were also given a pint of water six times daily in addition to the fluid matter of the diet.

Most of these methods of treatment of bacillary dysentery by means of drugs were tried before vaccine treatment was commenced, and although a certain amount of improvement was attained in the reduction of the death and invaliding rate, yet as will be shown later from the results of treatment in 138 cases without anti-dysenteric vaccine, the results were far from encouraging.

## TREATMENT WITH ANTI-DYSENTERIC VACCINE.

The vaccine as used in this series of cases consisted of:

- (1) *B. Shiga* (3 strains) 500 millions.
- (2) *B. Flexner* (2 strains) 250 millions.
- (3) *B. Morgan* (3 strains) 750 millions.

Each strain was inoculated separately into a flask containing peptone bouillon, incubated for 72 hours, and then killed by the addition of 0.5 per

cent. carbolic acid. Each flask was then allowed to stand for 48 hours, and at the end of this time was tested for sterility. If sterile, the dead cultures were standardized, mixed in the above proportion, and bottled.

In order to compare the results of vaccine treatment, it was found necessary to record the cases under different groups, each group of cases being treated with a different dose of vaccine. As some of these groups contained only a very small number of cases, too inadequate to form any conclusions on, the groups were re-collected into two main divisions, namely *Division X* (Tables III–VI) into which all the early cases fell, and in which the dosage in the light of later experience was small, and *Division Y* (Tables VII–XIII), into which all the later cases fell, and in which the dosage was much larger.

It was further found that cases had to be divided into two main classes, namely “Effectives,” and “Invalids,” to admit of adequate comparison, “Effectives” being porters who were actually doing duty at the time of the onset of dysentery, or who had been passed as recruits for work: “Invalids” being porters who had been returned from posts nearer the actual fighting line, for disease contracted in the field which rendered them unfit for further duty. Thus under each group will be found two main classes, namely Effectives and Invalids.

Again it was obvious that it was unfair to take each case of dysentery as the same in severity, for some men reported sick as soon as the disease showed itself, others not for ten days afterwards, when toxæmia had set in and the patient had been reduced to a very weak state. The disease itself was also not always equal in severity. Consequently it was decided that some sort of subdivision of cases according to the severity of the disease and to the condition of the patient was essential.

The severity of the attack was adjudged on four main points, namely the symptoms, the amount of toxæmia, the amount of blood and mucus in the stools, and the number of evacuations daily.

The following classification was adopted:

(a) *Slight cases* in which the patient's physical condition was good, and the disease was mild and unaccompanied by toxæmia.

(b) *Medium cases* in which the patient's physical condition was good, but the disease was more severe, and in which a certain degree of toxæmia was seen.

(c) *Bad cases* in which the patient's physical condition was fair, but in which the disease was severe, and toxæmia marked.

(d) *Very bad cases* in which the patient's physical condition was poor, and in which the disease was severe and marked toxæmia was present.

(e) *Hopeless cases* in which the patient was admitted either in a comatose condition, or in which the patient was so emaciated and toxæmia so marked that no hope of the patient's recovery could be entertained.

It must here be remarked that the standard of physical condition was of necessity made much lower for invalids than for effectives, and that even then



possibly a considerably larger proportion of them should have been included amongst the "hopeless" class, but in every case the most optimistic view possible was taken.

A classification based on the characters of the case and the condition of the patient necessarily depends largely on the judgment of the observer, consequently in each group, a total is made of all cases included in that group, so that conclusions based on these totals are absolutely independent of any personal bias. It is unfortunate that whereas the vaccine cases were consecutive admissions to hospital, no case being excluded, the only non-vaccine cases available for comparison of which a record exists, were more or less selected men, in that all were "Effectives" and "Hopeless" cases were definitely excluded, this series having been recorded before vaccine treatment was started or even thought of. Consequently in order to compare total cases, it has been found essential to show in Table XV not only totals showing all cases, but also totals including all cases but "hopeless" ones.

As a further note it may be stated that even a comatose "hopeless" case sometimes recovered consciousness, and with the aid of stimulants and intravenous salines lived sometimes for a considerable number of days after admission, but in not a single case was the classification of "hopeless," which was always made on the day of admission, found unjustified, for no case in this class ever survived to be cited as a recovery.

A further division of porters was also necessary for statistical purposes. A certain number of porters received a dose of 1 c.c. (1500 million) of anti-dysenteric vaccine, others received a dose of 4 c.c. of anti-dysenteric vaccine, in both cases as a prophylactic measure when recruited. A large proportion of the cases received no prophylactic inoculation at all.

We have therefore the following outline classification under which all cases are brought.

- (i) *Division.* X or Y, i.e. small or large therapeutic doses vaccine administered.
- (ii) *Group.* I—X depending on therapeutic dose of vaccine administered.
- (iii) *Class.* I. Effectives, II. Invalids.
- (iv) *Sub-Class.*
  - A. Receiving no prophylactic inoculation.
  - B. Receiving 1 c.c. A.D. vaccine as a prophylactic.
  - C. Receiving 4 c.c. A.D. vaccine as a prophylactic.
- (v) *Character of Case.* As described above.

Table III.

GROUP A. Not treated with anti-dysenteric vaccine.

Class	Character of case	Recoveries						Deaths			Average number of days in hospital			
		Total cases	Total No.	%	To duty No.	%	Invalided No.	%	No.	%	Duty	Invalided	Died	All cases
<i>Effectives</i> Prophylactic inoculation Nil	Slight	34	32	94.12	10	29.41	22	64.71	2	5.88	11.40	19.18	14.50	16.62
	Medium	43	34	79.07	6	13.95	28	65.12	9	20.93	10.00	20.68	18.33	18.69
	Bad	46	24	52.17	1	2.17	23	50.00	22	47.83	9.00	16.61	15.41	15.87
	Very bad	15	4	26.67	—	—	4	26.67	11	73.33	—	21.25	16.64	17.87
	Hopeless	—	—	—	—	—	—	—	—	—	—	—	—	—
Total ...		138	94	68.12	17	12.32	77	55.80	44	31.88	10.76	19.06	16.27	17.15

Table IV.

GROUP B I. Initial dose. Variable, 5 million to 200 million.

Class	Character of case	Recoveries						Deaths			Average number of days in hospital			
		Total cases	Total No.	%	To duty No.	%	Invalided No.	%	No.	%	Duty	Invalided	Died	All cases
<i>Effectives</i> Prophylactic inoculation Nil	Slight	—	7	100.00	4	57.14	3	42.86	—	—	16.25	17.66	—	16.86
	Medium	6	5	83.33	3	50.00	2	33.33	1	16.67	12.00	24.00	5.00	14.83
	Bad	3	1	33.33	1	33.33	—	—	2	66.67	11.00	—	11.00	11.00
	Very bad	—	—	—	—	—	—	—	1	100.00	—	—	6.00	6.00
	Hopeless	1	—	—	—	—	—	—	—	—	—	—	—	—
Total ...		17	13	76.47	8	47.06	5	29.41	4	23.53	14.00	20.20	8.25	14.47
<i>Invalids</i> Prophylactic inoculation Nil	Slight	—	—	—	—	—	—	—	—	—	—	—	—	—
	Medium	—	—	—	—	—	—	—	—	—	—	—	—	—
	Bad	1	—	—	—	—	—	—	1	100.00	—	—	7.00	7.00
	Very bad	—	—	—	—	—	—	—	—	—	—	—	—	—
	Hopeless	3	—	—	—	—	—	—	3	100.00	—	—	14.33	14.33
Total ...		4	—	—	—	—	—	4	100.00	—	—	12.50	12.50	

Table V.  
GROUP B II. Initial dose. 90 million. (Doses every second day increased by half as much again.)

Class	Character of case	Recoveries						Deaths			Average number of days in hospital		
		Total cases	Total %	To duty	To duty %	Invalided	Invalided %	No.	%	Duty	Invalided	Died	All cases
<i>Effectives</i>	Slight	5	100-00	4	80-00	1	20-00	—	—	8-00	9-00	—	8-20
	Medium	3	100-00	2	66-67	1	33-33	—	—	8-50	13-00	—	10-00
	Propylactic	10	90-00	6	60-00	3	30-00	1	10-00	19-50	26-33	19-00	21-50
	Very bad	8	62-50	3	37-50	2	25-00	3	37-50	19-67	39-50	27-00	27-37
	Hopeless	—	—	—	—	—	—	—	—	—	—	—	—
Total ...	26	84-61	15	57-69	7	26-92	4	15-39	15-00	25-71	25-00	19-42	
<i>Invidids</i>	Slight	9	88-89	2	22-22	6	66-67	1	11-11	12-50	14-50	1-00	12-55
	Medium	2	50-00	1	50-00	—	—	1	50-00	10-00	—	18-00	14-00
	Bad	8	50-00	2	25-00	2	25-00	4	50-00	34-00	8-50	24-75	23-00
	Propylactic	10	40-00	—	—	4	40-00	6	60-00	—	78-25	19-83	43-20
	Hopeless	3	—	—	—	—	—	3	100-00	—	—	24-00	24-00
Total ...	32	53-12	5	15-62	12	37-50	15	46-88	20-60	34-75	20-60	25-91	

Table VI.  
GROUP B III. Initial dose. 90 million. (Doses repeated weekly increased by half as much again.)

Class	Character of case	Recoveries						Deaths			Average number of days in hospital		
		Total cases	Total %	To duty	To duty %	Invalided	Invalided %	No.	%	Duty	Invalided	Died	All cases
<i>Effectives</i>	Slight	2	100-00	2	100-00	—	—	—	—	9-00	—	—	9-00
	Medium	—	—	—	—	—	—	—	—	—	—	—	—
	Bad	—	—	—	—	—	—	—	—	—	—	—	—
	Propylactic	2	—	—	—	—	—	2	100-00	—	—	52-50	52-50
	Hopeless	1	—	—	—	—	—	1	100-00	—	—	29-00	29-00
Total ...	5	40-00	2	40-00	—	—	3	60-00	9-00	—	44-66	30-40	
<i>Invidids</i>	Slight	—	—	—	—	—	—	—	—	—	—	—	—
	Medium	1	100-00	—	—	1	100-00	—	—	—	27-00	—	27-00
	Bad	—	—	—	—	—	—	—	—	—	—	—	—
	Propylactic	—	—	—	—	—	—	—	—	—	—	—	—
	Hopeless	—	—	—	—	—	—	—	—	—	—	—	—
Total ...	1	100-00	—	—	1	100-00	—	—	—	27-00	—	27-00	

Table VII. GROUP B IV. Initial dose. 180 million. (Doses repeated weekly increased by half as much again.)

Class	Character of case	Recoveries						Average number of days in hospital				
		Total		To duty		Invalided		Deaths	Duty	Invalided	Died	All cases
No.	%	No.	%	No.	%	No.	%					
<i>Effectives</i>	Slight	5	100-00	4	80-00	1	20-00	—	16-00	10-00	—	14-80
	Medium	5	100-00	4	80-00	1	20-00	—	9-25	11-00	—	9-60
	Propylactic inoculation	4	100-00	3	75-00	1	25-00	—	14-67	35-00	—	19-75
	Very bad	4	50-00	2	50-00	—	—	2	50-00	49-00	—	25-50
Nil	—	—	—	—	—	—	—	—	—	—	—	—
Total ...	18	88-89	13	72-22	3	16-67	2	11-11	18-69	18-67	25-50	19-44
<i>Invalids</i>	Slight	1	100-00	1	100-00	—	—	—	8-00	—	—	8-00
	Medium	—	—	—	—	—	—	—	—	—	—	—
	Propylactic inoculation	2	100-00	—	—	2	100-00	—	—	—	29-00	29-00
	Very bad	—	—	—	—	—	—	—	—	—	—	—
Nil	—	—	—	—	—	—	—	—	—	—	—	
Total ...	3	100-00	1	33-33	2	66-67	—	—	8-00	29-00	—	22-00

Table VIII. GROUP B v. Initial dose. 750 million. (Doses doubled at weekly intervals.)

Class	Character of case	Recoveries						Average number of days in hospital				
		Total		To duty		Invalided		Deaths	Duty	Invalided	Died	All cases
No.	%	No.	%	No.	%	No.	%					
<i>Effectives</i>	Slight	4	100-00	4	100-00	—	—	—	7-50	—	—	7-50
	Medium	3	100-00	3	100-00	—	—	—	12-33	—	—	12-33
	Propylactic inoculation	4	100-00	4	100-00	—	—	—	11-25	—	—	11-25
	Very bad	2	50-00	1	50-00	—	—	1	50-00	52-00	—	33-00
Nil	—	—	—	—	—	—	—	—	—	—	—	—
Total ...	13	92-30	12	92-30	—	—	1	7-70	13-66	—	33-00	15-15
<i>Effectives</i>	Slight	1	100-00	1	100-00	—	—	—	13-00	—	—	13-00
	Medium	1	—	—	—	—	—	1	100-00	—	—	18-00
	Propylactic inoculation	2	100-00	2	100-00	—	—	—	—	14-50	—	14-50
	Very bad	—	—	—	—	—	—	—	—	—	—	—
Hopeless	—	—	—	—	—	—	—	—	—	—	—	
Total ...	4	75-00	3	75-00	—	—	1	25-00	14-00	—	18-00	15-00
<i>Invalids</i>	Slight	3	33-33	—	—	1	33-33	2	66-67	—	9-00	9-33
	Medium	1	100-00	1	100-00	—	—	—	—	33-00	—	33-00
	Propylactic inoculation	7	85-71	5	71-43	1	14-28	1	14-28	43-20	65-00	41-57
	Very bad	1	—	—	—	—	—	1	100-00	—	—	12-00
Nil	2	—	—	—	—	—	2	100-00	—	—	49-00	
Total ...	14	57-14	6	42-86	2	14-28	6	42-86	41-50	37-00	23-17	33-00

Table IX.  
GROUP B VI. Initial dose. 1500 million. (Doses doubled at weekly intervals.)

Class	Character of case	Recoveries						Deaths			Average number of days in hospital		
		Total cases	Total %	To duty No.	To duty %	Invalided No.	Invalided %	No.	%	Duty	Invalided	Died	All cases
<i>Effectives</i> Prophylactic inoculation Nil	Slight	7	100-00	7	100-00	—	—	—	—	—	—	—	16-71
	Medium	2	100-00	2	100-00	—	—	—	—	11-00	—	—	11-00
	Bad	3	100-00	3	100-00	—	—	—	—	24-67	—	—	24-67
	Hopeless	3	—	—	—	—	—	3	100-00	—	—	60-33	60-33
Total ...	15	80-00	12	80-00	—	—	3	20-00	17-75	—	60-33	26-26	
<i>Effectives</i> Prophylactic inoculation I c.c.	Slight	1	100-00	1	100-00	—	—	—	—	15-00	—	—	15-00
	Medium	13	100-00	13	100-00	—	—	—	—	12-46	—	—	12-46
	Bad	6	83-33	5	83-33	—	—	1	16-67	25-60	—	9-00	22-83
	Very bad	7	71-43	5	71-43	—	—	2	28-57	41-40	—	31-50	38-56
Hopeless	3	—	—	—	—	—	3	100-00	—	—	12-00	12-00	
Total ...	30	80-00	24	80-00	—	—	6	20-00	21-33	—	36-00	20-67	
<i>Invalids</i> Prophylactic inoculation Nil	Slight	2	100-00	—	—	2	100-00	—	—	—	9-50	—	9-50
	Medium	14	100-00	9	64-29	5	35-71	—	—	16-44	21-40	—	18-21
	Bad	11	81-82	6	54-54	3	27-28	2	18-18	38-67	31-33	51-00	38-91
	Very bad	12	4	33-33	3	25-00	1	8-33	8	66-67	31-33	13-00	28-75
Hopeless	6	—	—	—	—	—	6	100-00	—	—	21-83	21-83	
Total ...	45	64-44	18	40-00	11	24-44	16	35-56	26-33	21-18	28-94	26-00	





Table XII.

GROUP B IX. Initial dose. 1500 million. (Dose doubled every three days.)

Class	Character of case	Recoveries										Average number of days in hospital						
		Total		To duty		Invalided		Deaths		Duty	Invalided	Died	All cases					
		No.	%	No.	%	No.	%	No.	%									
<i>Effectives</i>	Slight	7	100-00	7	100-00	—	—	—	—	—	—	—	—	18-14	—	—	18-14	
	Medium	31	100-00	30	96-77	1	3-23	—	—	—	—	—	—	14-73	31-00	—	15-26	
	Bad	67	61-04	52	77-61	9	13-43	6	8-96	—	—	—	—	22-62	37-56	21-50	24-52	
	Very bad	24	14-58-33	6	25-00	8	33-33	10	41-67	—	—	—	—	26-33	30-37	14-80	22-87	
	Hopeless	2	—	—	—	—	—	—	2	100-00	—	—	—	—	—	6-50	6-50	
Total ...	131	113	86-26	95	72-52	18	13-74	18	13-74	—	—	—	20-03	34-00	16-11	—	21-41	
<i>Effectives</i>	Slight	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	Medium	3	100-00	2	66-67	1	33-33	—	—	—	—	—	—	21-50	44-00	—	—	29-00
	Bad	5	100-00	5	100-00	—	—	—	—	—	—	—	—	23-80	—	—	—	23-80
	Very bad	1	100-00	1	100-00	—	—	—	—	—	—	—	—	41-00	—	—	—	41-00
	Hopeless	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Total ...	9	9	100-00	8	88-89	1	11-11	—	—	—	—	—	25-37	44-00	—	—	—	27-44
<i>Invalids</i>	Slight	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	Medium	3	100-00	—	—	3	100-00	—	—	—	—	—	—	—	23-00	—	—	23-00
	Bad	10	90-00	1	10-00	8	80-00	1	10-00	—	—	—	—	24-00	36-87	47-00	—	29-20
	Very bad	11	8-72-73	—	—	8	72-73	3	27-27	—	—	—	—	—	25-50	16-00	—	22-91
	Hopeless	2	—	—	—	—	—	—	2	100-00	—	—	—	—	—	11-00	—	11-00
Total ...	26	20	76-92	1	3-85	19	73-07	6	23-08	—	—	—	24-00	26-00	19-50	—	—	24-42
<i>Invalids</i>	Slight	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	Medium	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	Bad	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	Very bad	1	100-00	—	—	1	100-00	—	—	—	—	—	—	—	15-00	—	—	15-00
	Hopeless	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Total ...	1	1	100-00	—	—	1	100-00	—	—	—	—	—	—	15-00	—	—	—	15-00
<i>Invalids</i>	Slight	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	Medium	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	Bad	7	100-00	—	—	7	100-00	—	—	—	—	—	—	—	24-57	—	—	24-57
	Very bad	9	4-44-44	—	—	4	44-44	5	55-56	—	—	—	—	—	38-75	14-20	—	25-11
	Hopeless	2	—	—	—	—	—	2	100-00	—	—	—	—	—	—	7-50	—	7-50
Total ...	18	11	61-11	—	—	11	61-11	7	38-89	—	—	—	29-73	29-73	12-29	—	—	22-94





Table XIV—contd.  
DIVISION Y. Summary of Tables VII to XIII.—contd.

Class	Character of case	Recoveries										Average number of days in hospital			
		Total		To duty		Invalided		Deaths		Duty		Invalided	Died	All cases	
		No.	%	No.	%	No.	%	No.	%	No.	%				
<i>Effectives</i>	Slight	3	100-00	3	100-00	—	—	—	—	—	—	10-67	—	10-67	
	Medium	27	88-89	23	85-19	1	3-70	3	11-11	18-00	44-00	39-00	—	21-29	
	Bad	56	49	87-50	47	83-93	2	3-57	7	12-50	22-74	86-00	—	24-73	
	Very bad	56	39	69-64	37	66-07	2	3-57	17	30-36	29-05	83-00	—	33-52	
	Hopeless	8	—	—	—	—	—	—	8	100-00	—	—	—	21-87	
Total ...	150	115	76-67	110	73-33	5	3-34	35	23-33	23-55	82-40	29-77	—	26-96	
<i>Invalids</i>	Slight	10	8	80-00	5	50-00	3	30-00	2	20-00	11-40	9-33	—	10-40	
	Medium	24	24	100-00	14	58-33	10	41-67	—	—	17-21	21-90	—	19-17	
	Bad	37	28	75-68	16	43-24	12	32-44	9	24-32	34-00	37-83	—	39-78	
	Very bad	47	19	40-43	6	12-77	13	27-66	28	59-57	30-00	26-08	—	27-74	
	Hopeless	19	—	—	—	—	—	—	19	100-00	—	—	—	16-89	
Total ...	137	79	57-66	41	29-93	38	27-73	58	42-34	24-93	27-37	27-57	—	26-72	
<i>Invalids</i>	Slight	—	—	—	—	—	—	—	—	—	—	—	—	—	
	Medium	7	5	71-43	3	42-86	2	28-57	2	28-57	22-00	51-50	—	28-57	
	Bad	12	7	58-33	6	50-00	1	8-33	5	41-67	29-33	78-00	—	43-00	
	Very bad	31	10	32-26	8	25-81	2	6-45	21	67-74	45-50	44-00	—	24-55	
	Hopeless	10	—	—	—	—	—	—	10	100-00	—	—	—	10-00	
Total ...	60	22	36-67	17	28-33	5	8-34	38	63-33	35-65	53-80	18-47	—	26-28	
<i>Invalids</i>	Slight	—	—	—	—	—	—	—	—	—	—	—	—	—	
	Medium	3	3	100-00	3	100-00	—	—	—	—	35-33	—	—	—	35-33
	Bad	17	12	70-59	3	17-65	9	52-94	5	29-41	16-67	30-78	—	26-47	
	Very bad	20	7	35-00	3	15-00	4	20-00	13	65-00	49-00	38-75	—	19-38	
	Hopeless	11	—	—	—	—	—	—	11	100-00	—	—	—	87-28	
Total ...	51	22	43-14	9	17-65	13	25-49	29	56-86	33-67	33-23	16-24	—	23-65	

Table XV.  
Summary of all Vaccine cases in Divisions X and Y.

Class	Character of case	Recoveries										Average number of days in hospital		
		Total cases		To duty		Invalided		Deaths		Duty	Invalided	Died	All cases	
		No.	%	No.	%	No.	%	No.	%					
Division X <i>Effectives</i>	Slight	7	100-00	6	85-71	1	14-29	—	—	8-33	9-00	—	8-43	
	Medium	10	100-00	6	60-00	4	40-00	—	—	13-67	16-50	—	14-80	
	Bad	16	14	87-50	9	56-25	5	30-25	2	12-50	17-00	12-00	19-00	
	Very bad	13	6	46-15	4	30-77	2	15-38	7	53-85	17-50	29-71	27-46	
	Hopeless	2	—	—	—	—	—	2	100-00	—	—	17-50	17-50	
Total all cases		48	37	77-08	25	52-08	12	25-00	11	22-92	14-20	23-42	24-27	
Total excluding "Hopeless" cases		46	37	80-43	25	54-35	12	26-08	9	19-57	14-20	23-42	25-78	
Division X <i>Invalids</i>	Slight	9	8	88-89	2	22-22	6	66-67	1	11-11	12-50	14-50	1-00	
	Medium	3	2	66-67	1	33-33	1	33-33	1	33-33	10-00	27-00	18-33	
	Bad	9	4	44-44	2	22-22	2	22-22	5	55-56	34-00	8-50	21-22	
	Very bad	10	4	40-00	—	—	4	40-00	6	60-00	—	78-25	19-83	
	Hopeless	6	—	—	—	—	—	6	100-00	—	—	19-17	19-17	
Total all cases		37	18	48-65	5	13-51	13	35-14	19	51-35	20-60	34-15	18-89	
Total excluding "Hopeless" cases		31	18	58-06	5	16-13	13	41-93	13	41-93	20-60	34-15	18-77	
Division Y <i>Effectives</i>	Slight	25	25	100-00	24	96-00	1	4-00	—	—	13-29	10-00	—	
	Medium	111	107	96-39	104	93-69	3	2-70	4	3-61	15-34	28-67	33-75	
	Bad	175	157	89-71	145	82-80	12	6-91	18	10-29	21-54	45-42	23-94	
	Very bad	142	95	66-90	81	57-04	14	8-86	47	33-10	29-29	48-36	30-47	
	Hopeless	22	—	—	—	—	—	22	100-00	—	—	26-59	26-59	
Total all cases		475	384	80-84	354	74-53	30	6-31	91	19-16	20-93	43-93	28-38	
Total excluding "Hopeless" cases		453	384	84-77	354	78-15	30	6-62	69	15-23	20-93	43-93	28-96	
Division Y <i>Invalids</i>	Slight	10	8	80-00	5	50-00	3	30-00	2	20-00	11-40	9-33	9-50	
	Medium	34	32	94-12	20	58-82	12	35-30	2	5-88	20-65	26-83	15-50	
	Bad	66	47	71-21	25	37-88	22	33-33	19	28-79	30-80	36-77	45-21	
	Very bad	98	36	36-73	17	17-35	19	19-38	62	63-27	40-65	30-63	21-71	
	Hopeless	40	—	—	—	—	—	40	100-00	—	—	12-93	12-93	
Total all cases		248	123	49-60	67	27-02	56	22-58	125	50-40	28-82	31-09	22-18	
Total excluding "Hopeless" cases		208	123	59-13	67	32-21	56	26-92	85	40-87	28-82	31-09	26-53	

Having therefore explained the method of classification used, we can turn to the other point, namely what conclusions can be drawn from the recorded results of these cases?

(1) In the first place we may note that, as far as the death rate is concerned, there is practically no difference in the results of vaccine treatment between cases which received prophylactic inoculation and those which did not, indeed if anything the cases which received a prophylactic inoculation did worse and had a higher death rate than those which received no such inoculation. The real difference between cases which received a prophylactic inoculation and those which did not, lies in the percentage of those returning to duty after the disease. Here the cases which received a prophylactic inoculation show a considerably higher proportion of men returning to duty than cases which received no prophylactic inoculation, among effective porters. The opposite is the case amongst invalids, but here a fallacy may possibly be introduced as it is still uncertain how long any immunity conferred by prophylactic inoculation lasts. It seems probable from statistics at present available, that the immunity, if any, only lasts for a comparatively short time, in which case invalids coming under the head of prophylactically inoculated, should really be classed among the non-inoculated.

(2) The only available records of cases not treated therapeutically with vaccine are of effective porters from which "hopeless" cases were excluded. On comparison of the death rate amongst these men, which was 31·38 per cent., with the cases treated with vaccine, we see that amongst effective porters treated with small doses of vaccine, the death rate was lowered to 22·92 per cent. whilst amongst a similar class of men treated with large doses of vaccine, it fell to 19·16 per cent. Here there is no room for any individual bias to enter, for all the vaccine cases were consecutive admissions to hospital whilst the control cases were to a large extent selected cases. If we exclude "hopeless" cases from the vaccine series, the death rate falls to 19·57 per cent. and 15·25 per cent. respectively. Therefore from these figures it may be concluded that the therapeutic use of the vaccine definitely influences the course of the disease for good and markedly reduces the death rate from bacillary dysentery when administered in sufficiently large doses.

In the case of invalids there are unfortunately no records of cases untreated with vaccine for comparison, and it is only possible from the recollection of the writer of the feeling of utter hopelessness in dealing with these cases in pre-vaccine days, to give an assurance that, poor as are the results shown for invalids under vaccine treatment, matters were far worse in the days when no vaccine was available. If the figures are thought to show an appallingly high death rate, it must be remembered that these invalids had been returned as useless to the forces in the field, that many of them were frightfully wasted from disease, and with no resistance left to protect them from the invasion of fresh hostile bacteria, and one may even look upon it as a triumph of medicine that, of their number, we were able to send back to the front even as small a

proportion as 13.51 per cent. in one set of cases and 27.02 per cent. in the other.

It will be noted that the statistics show improvement in the death rate with vaccine, not only amongst the milder cases, but also amongst the "very bad" or toxæmic class.

Besides comparison of the death rate, it is interesting to compare the statistics showing the percentage of cases returned to duty, a matter of great importance in a campaign when every effective porter was an asset to our forces. In pre-vaccine days it was considered that, if a man went into hospital suffering from dysentery, it was almost a certainty that, if he escaped death, he would be sent home as an invalid, only 21.32 per cent. being returned to duty. Compare with this the figures of effective porters treated with vaccine. Even with small doses, inadequate as they seemed in the light of later experience, over 50 per cent. of cases of dysentery were returned for further service at a time when the standard of fitness for duty was very high, and with the larger doses almost 75 per cent. of treated cases were sent back to duty. As has been mentioned above, a small proportion from 13.51 per cent. up to 27.03 per cent. of men returned as invalids and admitted to hospital with dysentery, were saved for a further period of usefulness in the field, a rather larger proportion than were returned to duty from the pre-vaccine series of selected effectives. From the point of view therefore of military efficiency, the vaccine was an important asset in the medical armamentarium.

In the statistics of treated cases, the average number of days spent in hospital has been included. Apparently the vaccine has very little effect on the number of days cases remained under treatment, though the average time in the hospital of vaccine cases was undoubtedly increased by the longer time cases, which eventually died, remained in hospital. In the pre-vaccine series of porters, similar cases died in a much shorter time.

(3) As regards the dosage of vaccine advisable, full statistics of each different dosage tried are included in the Tables. Certain of these were used with too small a number of cases to be useful for any statistical purpose, but on consideration of the various groups, when a sufficient number of cases are included to make conclusions reliable, it would seem that the results obtained with a dosage of 2 c.c. (3000 million), 4 c.c. and 8 c.c. vaccine on the first, fourth and eighth days were the most satisfactory and this is the dosage which is now recommended for general use.

(4) A small series of cases was tried with autogenous dysentery vaccines, but the numbers were too few to admit of statistical use. The general impression gathered, however, was that there was no advantage in their use over the polyvalent dysentery vaccine.

From the above facts it is recommended that the treatment of dysentery cases should be carried out on the following lines:

(1) Eliminate amoebic, bilharzial, and malarial dysentery by microscopical examination.

(2) Put the patient to bed.

(3) Give an aperient, and keep the bowels open by the use of magnesium sulphate in small doses three or more times a day.

(4) Administer *on the first day* or at the first possible moment, an initial dose of 2 c.c. (3000 million) of the polyvalent anti-dysenteric vaccine, followed by 4 c.c. on the fourth day and 8 c.c. on the eighth day, if necessary.

(5) Keep the patient on a light diet till the stools are normal in appearance and semi-solid, then gradually change to normal diet, being guided by the appearance of the stools.

(6) In the event of intractable diarrhoea setting in, give *B. coli communis* vaccine in  $\frac{1}{2}$  c.c., 1 c.c., and 2 c.c. doses at intervals of 3 days subcutaneously and by the mouth Pulvis Rhei and sodii bicarbonas one drachm of each thrice daily till the stools become normal in colour and tenesmus disappears.

It may be mentioned that, as post dysenteric diarrhoea was so common, and prolonged treatment to such an extent, an attempt was made to combine the treatments with anti-dysenteric vaccine and *B. coli* vaccine by giving them simultaneously, but no advantage was gained, the *B. coli* failing to produce the same effect as it does when it follows anti-dysenteric vaccine.

It must be noted that certain cases clinically and microscopically of bacillary type failed to respond at all to the anti-dysenteric vaccine. An explanation of this class of case may possibly be that heterologous dysentery bacilli were the causal organisms, or again the case may have been due to *Entamoeba histolytica* originally and treated as such, but secondary organisms, such as *B. coli communis*, may have got implanted on the healing amoebic ulceration, and have kept up the dysenteric condition. Certainly some of these cases responded markedly to a *B. coli* vaccine.

It is also worth recording that the vaccine in certain patients produced an immediate effect, the stools diminishing rapidly in number, their character improving, and the general condition of the patient becoming much better. It has happened too often to be merely coincidence, and it must be concluded therefore that one of its effects may be the immediate stimulation of phagocytosis. Independent testimony to this and also to the value of the vaccine as a therapeutic agent has been received from other observers from whose reports the following statements are quoted.

Capt. Shircore, E.A.M.S., Native Civil Hospital, Mombasa: "In my experience the best results obtained with anti-dysenteric vaccine are in chronic dysenteries. Its effects are immediate, *i.e.* within 18 to 24 hours, and whatever may be the explanation of such rapid action, nevertheless this result can be observed clinically. I regard it as a valuable therapeutic agent and would use it without fail in the type of cases mentioned in preference to any other line of treatment that I know of; and with confidence that if it was going to do good at all, it would do so promptly."

Capt. Mackinnon, E.A.M.S., K.A.R. Hospital, M'bagathi: "Although the preparation is a vaccine, I have usually found that there is an immediate

marked improvement after the first dose in nearly all cases in which a cure is obtained. There is usually a marked reaction and the temperature may run up to 103° F. or even more. I have noticed in several cases that, on the day following the inoculation, patients who have been passing 15 to 20 motions a day are suddenly reduced to one, or even to none, and show no signs of relapse thereafter."

Other observers have also noted a similar result with anti-dysenteric vaccine. See Skalski and Sterling (1917, *Deutsche med. Wochenschr.* XLIII. 713, abstract in *Trop. Dis. Bull.* x. 140), and Margolis (1917, *Deutsche med. Wochenschr.* XLIII. 783, abstract in *Bull. Inst. Pasteur*, xv. 557).

A testimony to the efficacy of the vaccine in treatment was given to the writer by two of his hospital dressers, who contracted dysentery while on duty, and insisted on being treated with the vaccine, although as a rule a native is distinctly averse to having needles thrust into him.

As a fact which requires further investigation, the writer wishes to record a general constitutional effect of the anti-dysenteric vaccine. A patient who had been in hospital for some considerable time with a bad ulcer of the foot, and a chronic inflammatory condition of the calf of the leg probably resulting therefrom, contracted dysentery. He was treated with anti-dysenteric vaccine in the usual way, and not only rapidly recovered from the intestinal disease, but the ulcer and the inflamed leg also became cured although they had resisted all other forms of treatment for months. So marked was the effect, that a case with a similar condition of ulcer and chronic cellulitis of the leg was tentatively given a dose of anti-dysenteric vaccine, and immediately cleared up. Unfortunately no other cases with exactly similar lesions have occurred, and no smear was taken from the ulcers of these two patients. The results with the vaccine are suggestive, however, and it has yet to be investigated whether dysentery bacilli do or do not occur in similar ulcers of the skin.

In conclusion the writer's thanks are due to Dr P. H. Ross, Director of Laboratories, E.A.P., whose suggestion to sterilise the vaccine with carbolic acid instead of heat made it possible to use large doses in treatment, to Dr J. Harvey Pirie, Institute of Medical Research, Johannesburg, from whose work on the dysentery bacilli of E. Africa, the idea of vaccine treatment was first formulated by the writer, and to both these officials for the way in which, under great difficulties and stress of other work, they so maintained the supply of the vaccine as to meet even excessive calls on it. The writer has also to thank Capt. J. A. M. Clarke, R.A.M.C., who supplied the records of cases detailed in Group B IX, and to Colonel Clemesha, D.D.M.S., E.A.E.F., for permission to publish this paper.