Report to the London School of Tropical Medicine on investigations on dysentery in Fiji during the year 1910 / by P.H. Bahr.

REPORT TO THE LONDON SCHOOL OF TROPICAL MEDICINE ON INVESTIGATIONS ON

DYSENTERY IN FIJI

DURING THE YEAR 1910.

BY

P. H. BAHR

M.A., M.B., B.C., D.T.M.&H., Cantab., M.R.C.S., Eng., L.R.C.P., Lond.

TOGETHER WITH AN ACCOUNT OF THE OCCURRENCE AND SPREAD OF DYSENTERY IN THE PACIFIC IN FORMER YEARS BY B. GLANVILL CORNEY, I.S.O., M.R.C.S., FOR TWENTY YEARS CHIEF MEDICAL OFFICER, FIJI.

WITH COLOURED AND MONOCHROME PLATES, AND MANY CHARTS.

LONDON:
WITHERBY & CO. 326 HIGH HOLBORN W.C.
1912.

Printed by WITHERBY & CO.
at their Printing Press in
Middle Row Place London

ARRANGEMENT OF PAPER.

A.—Epidemic Dysentery in Fiji.

B.—Amoebic Dysentery in Fiji.

LIST OF APPENDICES.

Bacillary Dysentery.

LIST OF ILLUSTRATIONS.

ERRATUM.—Sanduchean, nine lines from bottom of page 36, should read Gauducheau.

DYSENTERY IN FIJI

DURING THE YEAR 1910.

I. (a)—Introductory.

This paper embodies the results of thirteen months' study of dysentery in Fiji. For this purpose the author was appointed by the Committee of the London School of Tropical Medicine. A grant of £500 was placed at the disposal of the Committee by the generosity of Lord Sheffield. This was supplemented by a further sum of £300, provided by E. W. Blessig, Esq., of Bletchingley, Surrey, for the provision of skilled laboratory assistance. A report on researches on filariasis made concurrently has already appeared.

I do not propose to describe fully the local hygienic and sanitary conditions obtaining in these islands, as those have already been dealt with at some length in the report referred to.

Fiji consists of a group of 250 islands, many of which are uninhabited, and which in the aggregate approximate in area the size of Wales. The three larger islands only concern us here. These are mainly devoted to sugar, coconut and banana plantations. The population of the group comprises over 3,000 Europeans, 87,000 Fijians, 35,000 Indians and 3,000 Solomon Islanders. The two latter races are imported as labour coolies for work on the plantations, for which the native Fijian is unsuited It is amongst these immigrants that the scourge of dysentery is most prevalent.

Suva, the capital, is situated on Vitilevu, the largest island of the group. It has a population of over 1,000 Europeans, and a fluctuating population of natives and coloured immigrants of three or four times that number. My researches were carried out principally in the Colonial Hospital in Suva.

The climate is cool for the Tropics, and remarkably even throughout the year. The highest temperature recorded in 1910 was 96° F. in February, the lowest 61° F. in July. The rainfall is a large one, and is greatest in the hot months, November to March. In Suva it averages over 110 ins. per annum.

My opportunities for investigating the local dysentery were greatly enhanced by the Fijian Government, which not only contributed a monetary grant of £100 for this purpose, but also appointed me pathologist to the Hospital at Suva and a temporary medical officer of the Colony.

The necessary apparatus was brought out from England, and a laboratory was installed in the verandah of a house which I rented.

By the kindness of the Chief Medical Officer, the Hon. G. W. A. Lynch, I was permitted to conduct investigations on the patients under his care, and, further,

to try the effect of certain lines of treatment on an extended scale. To him, as also to other medical officers in the Government service, Drs. E. Prideaux, J. F. Smalley and F. N. Smartt, my best thanks are due for aiding me by every means in their power. I wish also to acknowledge my thanks to Dr. C. W. Daniels, late Director of the Tropical School, and formerly a medical officer of the Colony, for the great interest he has taken in my work, and to the present Director, Dr. H. B. Newham, for many kindnesses during the preparation of this report at the London School of Tropical Medicine. The Nursing Staff of the Colonial Hospital rendered me valuable service in many ways. I am also greatly indebted to Dr. B. Glanvill Corney, I.S.O., for contributing some valuable remarks on the earlier ravages of dysentery in the Colony, a subject on which he possesses unique knowledge.

Great credit is due to my laboratory assistant, Mr. W. J. Muggleton, and I am specially grateful to him for his painstaking services. A Fijian medical student, Jesse by name, who was devoted to this work, proved himself of great value, helping me in my dealings with the natives and the various immigrant races.

In the account of the investigations which follows, I propose, firstly, to state categorically the conclusions arrived at, giving in the text a brief statement of the facts and arguments on which these conclusions are based and referring the reader to the Appendix for statistical information and a variety of detail.

I. (b)—Dysentery in Fiji in former times. By B. Glanvill Corney, I.S.O., for twenty years Chief Medical Officer of that Colony.

It would be idle to affirm that we have an exact knowledge of how or when dysentery first occurred amongst the people of Fiji, or whether it has been endemic in these islands from remote ages. Native tradition does not reach much further back than three generations with the Fijians, even amongst those of high rank. As late as thirty years ago a story was current amongst them that dysentery was unknown to their ancestors until after the arrival of white men's ships. There exists one tradition in particular (1) that after and in consequence of the visit of a ship from the eastward an epidemic of great severity and extent occurred. The native name for dysentery is *coka dra*, which means exactly "bloody flux." That a genuine name for dysentery exists might be thought to point to its presence amongst the natives from remote ages, before any contact with Europeans or other stray voyagers. On the other hand, this name is no more than a descriptive or symptomatic one, and it may be argued that it is quite the most natural one for the Fijians to apply to an imported disease of foreign name of which they were ignorant, or which they found difficulty in pronouncing. We know of a certainty that whooping-cough is a disease of exotic origin in Polynesia, yet the Fijians have dubbed it with a purely native designation, *vu koli**—literally, "dog cough." A much more characteristically Fijian expression is *kalou ni wai*, which is applied by this people to the type of dysentery accompanied by great tenesmus and prolapse of the rectum, especially in children. This term is hardly susceptible of translation. Jarves (2) states that a devastating epidemic swept over Hawaii in 1802-1804, and occasioned a great mortality. It is now generally believed that this was

* The etymology of some of these Polynesian names is of great interest. Thus "koli," the Fijian for "dog," is their interpretation of "collie"; while in Rotumah, a dependency of Fiji, the dog is still known as a "kamia," the derivation of which is obviously the "Kemere" by which the first imported animal was addressed by its Australian owner (Basil Thomson).

either dysentery or influenza, or probably dysentery supervening on an epidemic of influenza, and that it was nearly synchronous with a great epidemic of undetermined origin and nature in Fiji.

Ellis (3) gives an account of a kind of dysentery which afflicted Tahiti after the visit of Vancouver's ships to those islands in 1790. Lesson (4), who was a surgeon on the French corvette " La Coquille " (1823), confirms the statement that the natives attributed the occurrence of epidemic dysentery amongst them to this visit of Vancouver. Buzacott (5), a missionary pioneer in Raratonga (Cook Islands), was satisfied that there was no evidence that dysentery, or the *eke toto* (lit. : " bloody flux "), had ever raged amongst the people of that island before the arrival of foreigners. Its first manifestation (after the settlement of missionaries at Raratonga) took place soon after the arrival of a person from Tahiti, who was suffering from it.

John Williams (6), a missionary who had experience of all the Pacific Island group (excepting Tonga and Fiji) from Tahiti to Samoa, refers to the overwhelming nature of the epidemic just referred to ; he states that in the island of Rapa nearly half the population was swept away. In his experience most of the diseases which raged in these islands during his residence were introduced by ships, and this even if there was no apparent or recognized disease of a similar nature amongst the ships' crews ; he states that first intercourse between Europeans and natives was invariably attended with the introduction of virulent fever and dysentery.

Commodore Wilkes (7), writing in 1840, states that dysentery never had prevailed in Fiji as an epidemic. However, the missionary John Hunt died of dysentery in Fiji in 1845, a fact attested by his colleagues Calvert and Lyth.

Litton Forbes (8), a medical man, in 1875, remarked that dysentery was quite as fatal to Fijians as to whites, though, perhaps, owing to differences of food and other causes, somewhat more common among the latter.

The great measles epidemic of 1875 included dysentery as one, if not the most formidable and destructive, of its consequences.

The period since 1875 is within the memory of myself and of Sir William Macgregor. In 1876 dysentery was looked upon by Europeans in Fiji as the most dangerous and probable disease they had to fear. Cases of hepatic abscess in white men were noted in that year and afterwards, though I cannot call to mind having seen one in a Fijian native at any time. About the same year (1876 or 1877) a somewhat circumscribed epidemic of dysentery occurred amongst the Fijian labourers, natives of another island employed on a plantation on the Rewa river. It was popularly attributed to a surfeit of oranges ; the point is of interest because it indicates the season of the year, February, March and April, the advanced portion of the hot season and the rains, at which the epidemic took place, a coincidence noted in later epidemics. It is true that epidemics of dysentery have been noted as early as the latter part of November, especially in the Suva gaol, but they were of very limited extent. Amongst the Solomon Island immigrants, dysentery was seen in a very fatal form either during the sea passage in the labour schooners or soon after arrival in Fiji. In 1880 such an epidemic of great virulence was observed on board the schooner " Stanley " ; this continued in the shore depôt. In 1882 another similar epidemic occurred at Suva amongst the immigrants from another schooner, the " Surprise." In the same year another, but of lesser extent, occurred amongst the immigrants from the " Lord of the Isles " in the Suva depôt ; one or two cases had already occurred at sea. In 1884 a severe epidemic occurred amongst immigrants from the same group of islands on board the " Meg Merrilies," a report of which appeared in the Fiji Royal Gazette (9). Later, another epidemic broke out on the same vessel, which I, together with

Dr. C. W. Daniels, investigated. Dr. Daniels published an account of this in the *Practitioner* for 1890 (10). In the fatal cases the whole of the intestine, from duodenum to rectum, was acutely inflamed, thickened and disorganized. We used to term the dejecta "frog-spawn stools," from their striking resemblance to masses of frog's spawn, or to large grains of sago pudding. Dysentery of this particularly virulent or Melanesian type, so far as I am aware, did not and does not occur in the Fijian villages, or affect Fijians themselves brought into contact with patients in the depôts or the plantations; nor did Melanesian immigrants, who had been fortified by good living during previous indentured service in Fiji, Queensland, New Caledonia or Samoa, fall victims to it, even when associated with the affected batches; but no information is available to show whether their immunity was due to stronger physique—though this condition was obvious at a glance—or whether it was an immunity conferred by a previous attack.

The year 1882 was marked by a disastrous prevalence of dysentery amongst immigrants working on plantations in Fiji, especially in the alluvial district of the Rewa river. The occurrence was attributed chiefly to the underfeeding, exposure, and unaccustomed and continuous labour which the unseasoned Solomon Islanders had to face when employed in opening up new land for sugar plantations. The mortality amongst the new immigrants was very high, even in 1884 it was still 133·67 per *mille* over all indentured inmigrants in the Rewa district (from all causes, but chiefly from dysentery) and in the Navua district 322·17 (9).

With the shrinkage of Melanesian immigration into Fiji after the principal sugar plantations were established, and the gradual substitution of East Indian coolies for these natives, this particularly severe type of dysentery noticed amongst the Solomon Islanders notably diminished, and has now (as far as I know) practically ceased.

The Indians, however, appear to have brought with them a new form, of less severity and much more amenable to treatment. It was, furthermore, often associated with intestinal worms, especially *Ascaris lumbricoides* and *Ankylostoma duodenale*. Practically all the post-mortems on Indians in Fiji revealed the latter parasite; whereas in Melanesians, Fijians and whites, ankylostomes were rarely met with. About 1885, I think, the Indians began to be severely reduced by ankylostomiasis, associated with an abnormal prevalence of dysenteric symptoms, and it was almost ten years before a radical improvement in this respect took place.

Meanwhile, dysentery proper continued to occur in varying degrees of extent and severity in different seasons and years. Virulent dysentery among the Fijian natives was usually observed in local, circumscribed outbreaks.

The best place for observing circumscribed epidemics of dysentery was, unfortunately, the principal gaol of the Colony. For some years it showed a marked tendency to recur there. It usually began about December or January, and affected all races and classes of prisoners. Some years were notably more fatal than others. In some years, too, dysentery was more prevalent throughout the Colony than in others; and when this was the case all races were more or less affected by it in an approximately similar degree.

There is no doubt that nowadays the Eu opean, after his first year in Fiji, is on the whole much less liable to dysentery than in the early days of the settlement This comparative immunity is probably the result of better living, i.e. better houses, better food, better protection in the field against hot sunshine and cold rain, more regular working hours, less exhausting work, greater care as to drinking water, diminution of insobriety, the better quality of alcoholic drinks consumed, more appropriate clothing—in fact all the conditions

incidental to the increased knowledge, experience, caution and hygienic care which enable men to resist the action of specific micro-organisms.

The diminution and gradual disappearance of pioneering dangers may to some, if not to a large extent, be attributable to the prolonged annual cultivation of the soil in the localities where white men and immigrants are now employed. We used always to find that the turning up of previously untilled soil over considerable areas was coincident with the occurrence of much dysentery amongst the labourers, and even amongst the native Fijians when thus employed, and we were inclined to regard that as an element of some importance in its causation.

Liver abscess has not, I think, *pro rata* of the population, diminished, but rather increased; especially is this the case in the sugar district of Navua,* where many unseasoned white mechanics and office employees have of late years been introduced. Although many more persons of this class are now working in other sugar districts, they appear to be less liable to liver abscess. On this point, however, as I write without the figures, I can only state my impression.

I. (c)—Dysentery in Fiji at the present time.

From the above account it will be gathered that the mortality from epidemic dysentery used to be much greater than it is at the present day. In 1886 C. T. Hirsch placed the case mortality amongst Solomon Islanders at 40 per cent. In the epidemic on the "Meg Merrilies," quoted above, it was 48 per cent. (10). The type was of great virulence. According to Dr. Daniels, the small intestine was inflamed throughout its whole extent, and the last 2·18 ins. of the ileum, as well as the whole of the large intestine, was covered with a blackish-green substance. Death took place within four to ten days from the appearance of the first symptoms. Stomatitis and ulceration of the gums were common. In two cases the prepuce and glans penis were covered with a dirty green membrane similar to that seen in the intestines. Five convalescent patients developed arthritis, and recovery took several months to complete.

Apart from the severity of its effects and its virulence, there is no reason to suppose that the actual infective agent was specifically different from that in operation at the present day, yet pathological lesions of so extensive and serious a nature as those described by Daniels are not met with at the present day, nor is the mortality so high.

In the statistical returns of the Colonial Hospital at Suva, no distinction is made between amoebic and bacillary dysentery; nevertheless the case mortality from these causes is nowadays not a very large one, as may be gathered from the statistics given in Appendix II., which show the mortality from dysentery for the last six years. It varies from 5·2 per cent. to 12·8 per cent.

In the provincial plantation hospitals devoted to the Indian coolies the case mortality from dysentery is considerably lower than in similar hospitals for Fijians. This is a well-recognized fact in Fiji. The explanation generally accepted is to the effect that the Indian coolies, being under European supervision, are taken to hospital immediately they show signs of this disease; whereas a Fijian in his native village never seeks medical aid till the disease is well advanced.

In 1908 there was an epidemic of dysentery amongst the Fijians, with a mortality of 25·7 per cent.

* It is interesting and instructive to note in this connection that all the cases of amoebic dysentery and liver abscess, with one exception, encountered during the course of this investigation came from the sugar districts, especially Navua.

The case mortality statistics of epidemic dysentery are given by Vaillard and Dopter for the following countries :—

Japan, 24 per cent. (Shiga) ; Moscow, 12-17 per cent. (Rosenthal) ; Westphalia, 11 per cent. (Kruse) ; Toulon, 6-9 per cent. ; Brittany, 20-50 per cent. ; Finistèrre, 50-60 per cent. ; the difference in the severity of the disease being due, according to these authors, to differences in the virulence of the bacilli causing the several epidemics.

Owing to the absence of endemic malaria, so frequent a complication of dysentery in other countries, Fiji offers an exceptionally favourable opportunity for the study of this disease. It was for this reason that Fiji was selected as the field for my investigations.

The epidemic of dysentery in and about Suva during the year 1910 was one of the most extensive of recent years. Over 300 cases were admitted to the Colonial Hospital. A great increase in the average number of the dysentery cases admitted occurred after the devastating hurricane which swept over Suva on March 24th, 1910.

In the town of Suva dysentery is mainly an institutional disease, the majority of cases occurring in the two gaols, one of which accommodates the Indian, the other the Fijian prisoners. Thus, out of a total of 159 cases studied, over one-half, or 57·8 per cent., were prisoners from these gaols.

The annual epidemic of dysentery occurs during the hottest weather, November to April, which is also the period of greatest rainfall. Comparatively few cases occur during the months of June to October, which is the coolest time of the year and has the smallest rainfall (Appendix I.).

In making these investigations the distinction between the amoebic and bacillary dysentery was based on the discovery of amoebae in the dysenteric discharges in the former, and the isolation of the dysentery bacillus from the stools in the latter. Owing to the labour entailed, this latter procedure was not always feasible. In all cases dealt with in the course of this inquiry and classified as epidemic, or bacillary dysentery, no amoebae were ever found in the stools after repeated search. In a number of instances the dysentery bacillus was isolated from the stools and the sera of the majority gave agglutination reactions with the dysentery bacillus. Only cases passing typical stools containing blood and mucus, directly on admission or during their stay in hospital, were investigated. Some selection of this sort was necessary ; otherwise many cases reported as having passed blood and mucus in their stools before admission would have been included.

During the period of this inquiry there were eleven deaths from bacillary dysentery at the Colonial Hospital. Two of these fatal cases were admitted in a moribund state, and died before receiving any treatment.

A separate ward is set apart at the Colonial Hospital for the treatment of dysentery. As a further precaution against infection the dysenterics are not permitted to mix with the other patients till fully convalescent.

Out of 159 cases studied, only six occurred in females (i.e. two Fijians, two Indians and two half-castes). There are certain reasons for this apparent disproportionate liability of the male sex to infection. Firstly, there are very few female prisoners and the majority of the dysentery cases came from the gaols. Secondly, there is great disproportion between the numbers of male and female Indians in the Colony, only 40 women being imported from India to every 100 men. No Solomon Island women are imported. The cases amongst the Fijians were mostly in constables from the police barracks, or labourers employed in gangs on the wharves. Bacillary dysentery occurs in all the various races found in these islands. The same remark applies to amoebic dysentery, to which a special part of this report is devoted.

References Quoted Above.

1. Commissioners' Report on the Decrease of the Native Population (91-95) 1893 (quoted in Appendix I. of that report).
2. Jarves: History of the Hawaiian or Sandwich Islands (pp. 16 and 174), London, 1843.
3. Ellis: Polynesian Researches, London, 1829, Vol. 2, p. 269.
4. Lesson: Voyage Médical autour du Monde, Paris, 1829, p. 58.
5. Buzacott: Mission Life in the Islands of the Pacific, London, 1866, p. 100.
6. Williams: Narrative of Missionary Enterprises in the South Sea Islands, London, 1837, pp. 280-284.
7. Commodore Wilkes: Narrative of the United States' Exploring Expedition, Philadelphia, 1845, Vol. 3, p. 329.
8. Litton Forbes, M.D., L.R.C.P.: Two Years in Fiji, London, 1875, p. 169.
9. Fiji Royal Gazette, 1886. Annual Report on Polynesian Immigration.
10. Daniels: Practitioner, Vol. 45, 1890, pp. 343-346.

As a result of detailed study, the following conclusions have been arrived at. These, for convenience, are briefly indicated here, to be discussed subsequently in greater detail.

Bacteriological.

1. Epidemic dysentery in Fiji is of bacillary origin. Bacilli morphologically and culturally identical with Shiga's and Flexner's bacilli have been isolated from the stools and from post-mortems. Other bacilli, morphologically similar but giving an atypical reaction with various sugars, were also obtained. No special strains of the dysentery bacillus were found to be connected with special clinical types of the disease, or peculiar to any special race.
2. After long subculture of the bacilli the sugar reactions they gave were found to be variable and inconstant, but the reactions as regards mannite and dextrose remained constant.
3. Agglutination tests with dysentery bacilli of various types and with sera of patients in all stages of the disease proved neither constant nor reliable.

Clinical.

1. Amoebae were never found in the stools from which the dysentery bacilli were isolated.
2. Cases of all degrees of severity were encountered. These could be classified clinically into three main types, namely, the mild or catarrhal, the acute or ulcerative, and the toxic or fulminant.
3. An attack of bacillary dysentery was often a terminal affection in such chronic wasting diseases as pulmonary tuberculosis.

Pathological.

Macroscopic. The large intestine was affected throughout its whole extent in every case. The small intestine appeared normal in all but two cases. Necrosis and gangrene of the mucous membrane were the most constant features. In cases of long duration sloughing of the necrosed mucous membrane over considerable areas had taken place. The rectum in many instances was the part most severely affected.

Microscopic. The changes in the bowel wall were of the most intense inflammatory nature. Destruction of the epithelial cells of the mucous membrane and of the nutrient blood-vessels in the submucosa was the most constant feature.

Epidemiological.

An epidemic of dysentery is of annual occurrence in Suva, the season corresponding with the period of greatest heat and greatest aggregate rainfall. There is evidence that the house-fly is the principal agent in the spread of the disease. Bacilli of the Shiga and Flexner type, on recovery from the intestinal tract of these flies, showed variable but inconstant reactions with the sugar tests.

Treatment.

Of a series of cases treated in different ways, those injected intravenously with polyvalent anti-dysenteric serum gave the best results, as far as could be ascertained.

Prophylaxis.

The abolition of epidemic dysentery in a town of the size and importance of Suva depends upon an efficient sanitation, mainly directed against the house-fly.

Amoebic Dysentery.

Amoebae were found in the stools in a limited number of instances. The incidence of these cases bore no relation to the season of the year.

The clinical history and course of all amoebic cases were quite distinct from those of the epidemic variety. The pathological lesions were also dissimilar.

No differential diagnosis could be made by the simple inspection of the stools.

Although *Amoeba coli* is a common parasite in the stools of normal Fijians, amoebic dysentery appears to be a rare disease among them.

The amoebae found in the dysenteric discharges approximated the type of Schaudinn's *Entamoeba histolytica*.

II. Bacteriology.

The bacteriology of acute or epidemic dysentery is a very complex subject, owing mainly to the fact that bacilli, morphologically and culturally similar, give different fermentative reactions with the glucosides. Many such have been isolated by different observers from cases of acute dysentery.

The study of these has been complicated further by Kruse, who proposed to term all types, not conforming with the type as originally described by him and Shiga, as para- or pseudo-dysentery.

There is a tendency in the later writings on the subject to regard the bacillus as very variable in its reactions, and to include under one group the different sub-varieties described.

Shiga, in 1897, isolated from the stools and post-mortem of cases of epidemic dysentery in Japan a rod-shaped Gram-negative organism which, from its morphological and cultural characteristics, he classified as belonging to the coli group. There was considerable doubt at first as to its motility; it is now generally conceded to be non-motile, but possessed of peculiarly active Brownian movement. It is admitted that flagella are present; these have been described by Shiga and Duval. It produces acid, but no gas in dextrose broth; it gives a feebly acid reaction with milk, which on further incubation becomes alkaline. No indol is said to be formed by this type under any circumstances.

An organism having the same morphological and cultural characteristics was discovered by Kruse in cases of epidemic dysentery occurring at Laar, in Germany, about the same time.

In 1900 an organism morphologically similar, but differing from the organism of Shiga and Kruse in producing acid from mannite, was isolated from cases of dysentery occurring in Manila, by Flexner, Strong and Musgrave.

Flexner, in 1901, made a comparative study of the strains then known, and found that the serum of dysentery patients under his care would agglutinate them all; he found no morphological differences in any.

In 1902, Vedder and Duval isolated bacilli, similar to those obtained by Flexner, from cases of asylum dysentery in the United States.

Martini and Lentz made an exhaustive study of the strains of dysentery bacilli then known, and still further subdivided them by their reactions with various glucosides. They support Kruse's classification into true and

pseudo-dysentery bacilli, according as these organisms do not, or do, ferment alcohol mannite.

The serum of a patient, suffering from an infection with the one variety, was found to agglutinate a bacillus giving totally different fermentation reactions, an observation confirmed by de Blasi. The only satisfactory differentiation they could obtain was by agglutination tests with sera of artificially immunized animals.

Lentz attempted a still further differentiation by absorption tests, using rabbit serum immunized against a certain strain of the bacillus.

In 1904 Hiss gave a very able résumé of the subject up to date, and tried to differentiate between the different groups by further cultural tests. He found that all the different strains of dysentery bacilli, while agglutinating in lower dilutions with the serum of an animal immunized against one particular strain, could yet be differentiated in higher dilutions. The so-called Y bacillus of Hiss and Russel, which fermented dextrose and mannite alone, was described, while one of Flexner's original strains was found to ferment saccharose and maltose in addition to these.

In 1906 Shiga, working in conjunction with Ohno, made an exhaustive inquiry into the different types of dysentery bacillus, and while finding the fermentation properties of a single group constant towards dextrose and mannite, the reaction towards maltose, saccharose and lactose varied both quantitatively and qualitatively. The production of indol he also found to be variable, and any classification based on that property artificial. The true Shiga-Kruse type formed no indol under any circumstances. The types are referred to as acid and non-acid, as first proposed by Lentz, according to their reactions with alcohol mannite. Basing his classification on these fermentation reactions, he has added another group to the four already proposed by Hiss. This type is intermediate between the classical type of Shiga-Kruse and Flexner's bacillus, in that, after twenty-four hours' cultivation, it produces acid from mannite, the reaction, on further incubation, once more becoming alkaline. The agglutinative characters of these five groups, as regards immune sera, on the whole agreed with their fermentative reactions; there were, however, notable exceptions.

Ohno, in his elaborate investigations on 74 strains of dysentery bacilli derived from different sources, concluded that none could be distinguished either by morphological or cultural qualities, but that the acid types caused a severer clinical form of dysentery than the non-acid types; the exact reverse of this has been stated by other observers. The grouping of different strains of organisms, according to differences in their powers of fermentation, did not correspond to the results obtained by agglutinative and bacteriolytic reactions with specific immune sera. As a result of the inquiry, he considered that there is no valid reason for separating the dysentery bacilli into distinct groups—the acid and non-acid—as proposed by Lentz, nor into the true and pseudo-dysentery bacilli as proposed by Kruse.

In 1907 Twort, in investigating the fermentative reactions of certain members of the coli group, utilized a number of rare glucosides, but obtained no assistance from them in his endeavour to separate the members of the dysentery group into yet other types. In his last paper (1908) Shiga submits that there are still more varieties than Ohno has discovered. He now uses bacilli, conforming in their reactions to five groups, in the production of anti-dysenteric serum in Japan. These are as follows:—

(1) Fermenting dextrose alone (Shiga, Kruse, Flexner (Newhaven)).

(2) Fermenting dextrose and mannite (Hiss and Russel's Y bacillus, Ferran, Seal Harbour bacillus).

(3) Fermenting dextrose, mannite and saccharose (Flexner, Strong (Manila).
(4) Fermenting dextrose, mannite, maltose and saccharose (Harris, Gay, Baltimore and Woolstein's bacillus).
(5) Fermenting dextrose and maltose, and giving only a feebly acid reaction with mannite (Shiga).

[The acid reaction with mannite only appears after 24 hours' incubation, and after two to three days this becomes alkaline again. Shiga considers this group as occupying a place half-way between the acid and non-acid groups of Lentz.]

Morgan (1911) investigated 25 strains obtained from British sources, but all belonging to the acid type, and found great variability in their reactions; he concludes that the monosaccharides, galactose, arabinose, raffinose, adonite, glycerin, and amygdalin, were of little use in determining any differentiation.

Observers, other than those already mentioned, have found that not only are bacilli of different types found in the same epidemic, but even in the same stool. Thus Gay and Duval isolated acid and non-acid types of bacilli from the same stool. Duval and Shorer, in an investigation of summer diarrhœa occurring amongst infants in the United States, found dysentery bacilli in 94 per cent. of the stools investigated, and again acid and non-acid types were found in the same stool; the latter type, also, they succeeded in isolating from stools of children who were in perfect health. Again in this same summer-diarrhœa, Martha Woolstein and Grace Dewey isolated only acid types of bacilli 48 times out of 62 cases investigated. Duval and Basset isolated the true Shiga's bacillus from the intestine, liver and mesenteric glands, post-mortem, in this same affection, whilst Duval and Torrey have described types of lactose-fermenting dysentery bacilli; and Fisher isolated an acid type of bacillus (called bacillus F) forming permanent acidity in milk. In asylum dysentery, in Germany, Ritterhaus, Kemp and Metz have isolated several varieties from the same stool.

Dysentery bacilli of the true type and various sub-varieties have been found all over the world: in Japan by Shiga and Amako; in the United States by the observers already mentioned; by Castellani in Ceylon; by Rogers in the gaol dysentery of India; by Loghem and Schüffner in Java; by Deycke in Constantinople; by Rosenthal in Moscow; in Germany, in addition to the observers mentioned, by Drigalski (Doberitz), Lüdke (Barmen), Müller (Styria), and in asylum dysentery by Liefmann, Kühn and others.

Shiga's bacillus was first isolated in England by Eyre in asylum dysentery from the stools and also from the bile post-mortem, and in Ireland by McWeeney. Aveline, Boycott, Macdonald and Macalister also have isolated bacilli of the acid type from cases of asylum dysentery. Cases of acute dysentery occurring in this country in which these bacilli have been isolated have been quoted by Saundby and Marshall.

Technique adopted in the Isolation of Dysentery Bacilli from Stools.

It will readily be understood that in dealing with natives on a large scale, in consequence of the utter impossibility of supervising a number of men with the means at my disposal, the collection of dysenteric discharges for the purpose of bacteriological examination, under strict aseptic conditions, was almost an impossibility. During the first two months of my work in Fiji, it was found that stools which had stood for a few hours in the tropical climate became so overgrown with moulds as to be useless. The insanitary habits of the natives, especially the Indians, completely frustrated the collection of stools in sterilized receptacles. The universal presence of house-flies, which obtain access to the pails, considerably added to the contamination. The following method of

collecting stools for examination was adopted and was the only one which proved successful. The pail to be used by the patient was washed out with boiling water, and a Fijian medical student, who was specially detailed for this work, watched the patient till the stool was passed. Immediately a platinum loopful of the blood and mucus was diluted in broth and plated out on Conradi-Drigalski plates; eight to ten drops of the emulsion were found sufficient for one plate. When pure mucus, without contamination with faecal matter, was passed by the patient, the dysentery bacilli were on several occasions obtained in almost pure culture. Dilutions and cultivations in MacConkey bile broth proved unsatisfactory, as the dysentery bacillus appeared to become rapidly overgrown by other organisms of the coli group.

With the equipment at my command, not more than one, or at the most two, plates could be made from any one stool; but, the technique once established, I had no difficulty in isolating dysentery bacilli from every freshly-passed stool containing blood and mucus.

The table given in Appendix III. shows the number of times dysentery bacilli were isolated from stools, and also the types encountered.

On Conradi-Drigalski medium colonies of the dysentery bacillus were recognized by their peculiar transparent blue colour. Often the plate smelt strongly of spermin, a fact noted by Shiga, Eyre, and Amako. Some freshly-passed stools consisting of mucus and blood very often had a similar smell. On subculture in broth, they formed but a faint turbidity. They were further distinguished by their morphological and Gram-negative characters, and lastly by their sugar reactions. Six sugars were used, namely, those which have generally proved the most serviceable in differentiation of types—mannite, lactose, saccharose, maltose, dulcite, and dextrose, in 1 per cent. solution in peptone water. The reaction with litmus milk, indol production, and their agglutination with Lister Institute anti-serum complete the list of tests employed. Seven types (according to their fermentative reactions) of dysentery bacilli were isolated (Appendix III.). They were derived from every clinical variety of case, from the mildest to the most severe. No single variety was found peculiar to one class of case or any particular race. Thus the true Shiga's bacillus was isolated from three of the most severe cases encountered, as well as from two of the mildest; the so-called acid type (Flexner's bacillus) was also isolated from every variety of case. Lactose-fermenting forms were met with on four occasions. A non-acid bacillus fermenting maltose and saccharose (type "C," Appendix III.) was isolated from the same stool as the pure Shiga type; it was agglutinated by the patient's serum in a dilution of 1 : 200, and produced permanent acidity in milk, together with a slight clot. It was not agglutinated by the Lister Institute anti-serum. It is doubtful whether it can be classified as a true dysentery bacillus, as it was non-toxic when injected into guinea-pigs intraperitoneally, whereas all other types were intensely toxic to these animals.

Isolation from Post-mortems.

Post-mortems were secured on every available case. Unfortunately for the success of this investigation, the first six deaths from this disease occurred shortly after my arrival in the Colony, and before the laboratory was satisfactorily established. Shiga's bacillus was, however, isolated from the intestine post-mortem in two cases, once in almost pure culture.

The difficulty of obtaining a post-mortem directly after the death of the patient was considerable. Putrefaction commences almost immediately in a tropical climate. There was no refrigerating apparatus available, and as the relations had to be summoned before a post-mortem could be performed there was often considerable delay.

In making cultures from the intestines post-mortem, a loop of the large intestine was first tied off and removed, then opened in the laboratory and the faecal contents washed out with sterilized water. The necrosed surface was then seared with a metal rod and a platinum loop plunged through the surface into the underlying tissue. Dilutions in broth of material so obtained were plated out on Conradi-Drigalski medium, and the bacillus recognized by the tests already given.

The reactions given in the table, Appendix III., by which the bacilli are classified, were obtained in a 1 percent. dilution of the sugars in peptone solution after 24 hours' incubation. According to Ohno, these reactions change considerably after further incubation. One bacillus of each type was selected and incubated for a period of ten days on three subsequent occasions. Variation in the reactions obtained was noted in two instances (Appendix IV.). Thus a bacillus of the type B failed to ferment saccharose and maltose after nine months' subculture, while a lactose-fermenting type, G, after four months' subculture no longer fermented lactose, and had apparently temporarily lost the power of fermenting saccharose. These later tests were made in England with cultures brought home from Fiji.

The indol reaction was found to be very variable. No indol was ever produced by the true Shiga's bacillus, type A or B, whereas a marked reaction was obtained with types E, F and G. The reaction in type D was variable after 24 hours' incubation, but constant after ten days.

In their reaction with litmus milk, acid was produced after 24 hours' incubation in every case. In types C and E the reaction remained acid throughout. In the other types the reaction again became alkaline after three or four days' further incubation.

Variation in the reactions of a given strain of dysentery bacillus after subculture has been found by many observers. Thus Hiss found the fermentative characters of a number of dysentery organisms isolated by him were modified by subculture. Torrey found a bacillus which at first fermented dextrose only, but after several subcultures fermented mannite as well; he also found that the power of fermenting maltose was augmented after anaerobic culture. Kruse, in investigating a strain obtained from Strong (Manila), found that on subculture it fermented the disaccharides, which it at first failed to do. Later, in conjunction with Ritterhaus, Kemp and Metz, the same bacillus fermented lactose and clotted milk; Morgan, four years later, found that the descendants of this Strong's bacillus, although they clotted milk, now no longer fermented lactose. Twort found that dysentery bacilli at first unable to ferment saccharose were able to do so after being grown for some time on a medium containing this ingredient, and by similar means the true Shiga-Kruse bacillus was induced to ferment lactose. Shiga, in his last paper, gave instances in which the sugar reactions of the same strain had become altered on subculture. Lentz describes an acid strain which he had isolated, and which after nine years' subculture completely lost its power of fermenting maltose.

Penfold has lately induced intestinal bacteria, notably the typhoid bacillus, to alter their classical reactions, thereby confirming Twort's statement that the sugar tests constitute an impracticable means of grouping these bacilli.

As regards the morphology of the different types isolated in Fiji, they all exhibited the same characteristics on first isolation; no difference in shape or motility was noted They were extremely small, short organisms, with round ends, about 1 μ in length, and only assumed the typical rod shape on subculture in broth. On Conradi-Drigalski medium they formed dense cobalt-blue colonies, round in shape, with a slightly irregular margin. The Brownian movement is extremely active when first isolated.

The bacilli grew well anaerobically. The fermentation reactions of types A and E were re-tested after subculture; they gave the same result as when first isolated. A limited number of guinea-pigs were injected intraperitoneally with broth cultures of the different types.

After six months' subculture in Fiji, Shiga's and Flexner's bacilli brought out from England and kindly provided by Professor R. T. Hewlett and Dr. F. W. Twort were non-toxic to guinea-pigs. These Flexner bacilli belonged to Firth's "type 4," and fermented maltose as well as mannite and dextrose.

According to de Blasi and Castellani, dysentery bacilli injected intraperitoneally kill guinea-pigs in 14 to 30 hours I injected two cc. of a 24-hour-old broth culture of types A, B, E and G, immediately after isolation from dysenteric stools. The guinea-pigs all died from eight-and a-half to twenty-four hours after; the bacilli were recovered in three cases from the heart blood. In every instance there were signs of peritonitis and excess of peritoneal fluid: in the guinea-pigs thus injected bacilli were recovered from this fluid and from smears from the liver and spleen. These bacilli, after passage through the guinea-pig, gave the same sugar reactions as when first isolated, and were agglutinated by anti-serum in a dilution of 1: 200.

Shiga states that the dysentery bacilli rapidly lose their virulence on subculture, but retain it if kept in the refrigerator.

This apparent decline in the toxicity was seen in three strains tested. Injections of broth cultures (2 cc.) of the bacilli of type A, isolated post-mortem from the intestines, killed guinea-pigs within twenty-four hours; however, this same bacillus, injected after two or three months' subculture on agar and injected in the same manner, produced no symptoms at all in these animals, and could not be recovered from the tissues post-mortem. A bacillus of type E gave similar results after only six weeks' subculture.

Agglutination.

The agglutination test with anti-serum prepared by the Lister Institute was used for identifying the dysentery bacilli. This is a polyvalent horse serum derived from inoculation with Shiga's, Kruse's and Flexner's original strains, in addition to a Shiga's bacillus isolated by Eyre in asylum dysentery and also strains isolated from infantile diarrhœa in America.

I found that in broth cultures of bacilli recently isolated this serum agglutinated bacilli of type A up to 1 : 10,000, and type E up to 1 : 1,000. Aveline, Boycott and Macdonald state that it agglutinates the strains of bacilli used in its preparation up to a dilution of 1 : 10,000; Shiga states that two kinds of agglutination occur; in one the bacilli unite in chains, in the other in clumps; these phenomena were often noted in my Fiji work.

The dysentery bacilli, especially after long subculture, show a great tendency to auto-agglutination; to obviate wrong conclusions my broth cultures were always examined for this phenomenon before the agglutination tests were applied. Various authors have stated that the agglutination of the dysentery bacilli is not a satisfactory test in this disease. The agglutination reaction in my hands proved neither constant nor reliable.

Vedder and Duval have found that the serum of patients from whose stools dysentery bacilli had been isolated did not agglutinate those bacilli. In several instances I found that with bacilli isolated from the stools the reaction still remained negative after repeated trials with the patient's own serum (Appendix V.). Castellani states that in lower dilutions (1 : 40) the sera of many normal people agglutinate this bacillus.

I made a number of control tests with the sera, in a dilution of 1 : 50, of normal individuals belonging to different races—36 East Indians, 36 Fijians and 8 Solomon Islanders—many of them suffering from diseases other than dysentery. Broth cultures of bacilli, types A and E, recovered from different sources, were used. Fifty cases were tested with Shiga's bacillus (type A) and thirty with Flexner's (type E). Five gave a positive reaction; two of them were suffering from severe tertiary yaws, one from elephantiasis of the scrotum, and two were normal individuals.

The sera of 112 cases under treatment for bacillary dysentery were tested. A positive agglutination with Shiga's bacillus (type A) was noted in 74·1 per cent. of the cases, in dilutions from 1 : 100 to 1 : 200; in some instances positive reactions in dilutions of 1 : 500 were noted. The agglutination reaction was, on the whole, more marked with cultures of Shiga's bacillus (type A), though other types were also tested. Although a considerable number of the mildest cases of dysentery gave a positive result, yet the sera of those that were the most severe clinically gave the most marked agglutination reaction. Only broth cultures after twenty-four hours' incubation were used.

As Shiga has stated that after long subculture these bacilli are unsuitable for testing sera, only recently isolated cultures were used for this purpose, when once the dysentery bacillus had been successfully isolated from the stools in Fiji, the time limit assigned for each test being fixed at one hour.

A detailed account of the number of tests performed and the results obtained is given in Appendix VI.

Vaillard and Dopter state that the agglutination reaction only appears after the seventh to twelfth day from the date of infection. In several instances I obtained no reaction till after the first week of the illness.

Opportunities occurred for testing the sera of patients who had been under treatment at various periods (from two to eight months) after recovery; these were mostly Indian prisoners in the gaol. Out of twenty-five so tested, twenty-one still gave a positive reaction with cultures of Shiga's bacillus (type A) and Flexner's (type E) in a dilution of 1 : 50; all of these cases had given a positive reaction previously.

Agglutination tests were performed with other bacilli in seven cases which gave a positive reaction with the dysentery organisms. For this purpose a coli organism, obtained from the stools, was used, and also *Bacillus pyocyaneus* isolated post-mortem from the bile in a case of bacillary dysentery. No agglutination took place.

Several observers, notably Escherich, maintain that the serum of dysentery cases agglutinates the true coli bacillus. Kühn and Gildmeister have recently written on this paragglutination of the coli bacillus in cases of dysentery.

Attempts were made to isolate *Bacillus pyocyaneus* from the stools, especially those containing light green faecal matter, but were never successful.

III. Clinical.

Clinical observations were made on 159 consecutive cases. Seventy-nine of them were of Indian nationality, of whom 70·8 per cent. were prisoners from the gaol. Fifty-four were of Fijian nationality; seventeen prisoners and twelve constables, or 53·8 per cent., came from the gaol; there were also seven boys from the Government High School. Of the twenty Solomon Islanders 35 per cent. were prisoners. There were four Europeans and two female Fijian-European half-castes.

The Indians form the bulk of the criminal classes, hence the Indian prisoners constituted the majority of my patients.

There are certain characteristics peculiar to each of these different races—characteristics which not only influence their tolerance of the disease, but also have a distinct bearing on their behaviour while under treatment.

The Indian prisoners in Fiji were all adept malingerers ; any sign of dysentery was hailed by them as intimating a rest in hospital and a welcome release from irksome prison labour. They endeavour, therefore, not only to simulate the disease, but actually to acquire it. It was often extremely difficult in mild cases to detect malingering, and a Fijian student had to be detailed to watch their behaviour. By this means different methods of malingering were discovered and guarded against. A favourite method consisted in spitting blood and mucus, obtained by pricking the gums, into a diarrhoeic stool. Very often sago and arrowroot were added to simulate mucus. One prisoner in particular was seen to add blood and mucus, obtained from another patient's stool, to his own. A favourite method of prolonging convalescence was by faking diarrhoea, either by adding water to the motions, or by swallowing large doses of sodium sulphate, obtained surreptitiously.

The Fijians, on the other hand, are in many ways model patients, and do not attempt to malinger ; but they are in other ways impracticable. When suffering from a high temperature they are apt to discard all clothing and lie naked on the floor, or even to escape from the ward and spend the night naked under a tree in the rain. It is owing to this habit that the mortality from dysentery amongst the Fijians, when left uncontrolled, is so high.

Of all the races the Solomon Islanders appear to be the most susceptible to the severe forms of dysentery, and they exhibit but a feeble resistance. They are extremely liable to tuberculosis also, and in them an attack of dysentery is almost always a terminal event in tubercular disease.

Europeans living in Suva itself are not especially liable to bacillary dysentery, and, unless alcoholic, resist the infection well.

Nearly all my cases were young adults. There were, however, eleven children under the age of fourteen. The youngest patient was an Indian child of one-and-a-half years ; Shiga's bacillus was isolated from its stools.

A thorough physical examination was made of every patient immediately on admission to hospital. Notes were made of daily progress and of the appearance and number of the stools. Special attention was paid to any symptoms or appearance of an unusual nature. Of the mass of information gathered in this way I must confine myself to the briefest summary.

In a disease exhibiting so many and varied clinical forms, it becomes necessary for the purpose of observation and treatment to adopt some classification of the various clinical types encountered. A classification of this sort, however essential, must necessarily be a rough one. No hard and fast line can be drawn in classifying the cases of a disease of such varying degrees of severity. Every clinical variety of case was observed from cases in which, with hardly any constitutional disturbance, motions coated with blood and mucus were passed for a few days only, to others with stools of the foulest description and who died in a short time, prostrated by the severity of the infection and exhausted by the constant straining and passing of stools.

For convenience I arrange my cases, according to their severity, into three clinical types :—

(1) *Mild or catarrhal* : that type of case in which the constitutional symptoms were either absent or not severe. The temperature was often not raised during the whole attack. In many instances the motions were faecal from the commencement, coated with blood and mucus, and not exceeding twelve in number per diem (Appendix VII., Charts 1 and 2).

In many instances the tongue remained perfectly clean throughout the attack,

and the pulse-rate remained normal. The majority of patients complained of tenesmus on passing stools, and the abdomen was tender, especially in the left iliac fossa. In about a quarter of the cases the thickened and contracted coils of the sigmoid flexure could be felt in that region. From the stools of nine such cases, dysentery bacilli were isolated. Cases of this type constituted the majority of those which came under observation. A concise analysis of these is given in Appendix VII.

(2) *Acute or ulcerative :* In these the constitutional symptoms were pronounced, the temperature generally being raised for a considerable period. The motions consisted, for the most part, of blood and mucus, and exceeded twelve in number in the twenty-four hours. In such cases, necessarily a longer time elapsed before the establishment of convalescence. There were some instances in which, although constitutional symptoms were severe, the temperature was not raised above normal (Appendix VII. and Charts 3 and 4). Dysentery bacilli were isolated from the stools of such cases twelve times.

The clinical picture in this type was of a different character from that in the mild catarrhal cases, and indicated a more serious disturbance of the intestinal tract. Although the tongue was furred in almost every case, and although many of the patients, especially Fijians, were actually ill and suffering from a high temperature, yet the pulse-rate was accelerated above normal in only about half the number of cases.* Tenesmus was complained of in the majority of instances. The abdomen was tender, and there were marked contraction and thickening of the sigmoid flexure. A concise analysis of these cases is given in Appendix VII.

(3) *Toxic or fulminating type:* This was quite a distinct type. The constitutional disturbance was profound. The stools frequently exceeded fifty in the twenty-four hours, and in many instances their number was uncountable. The material passed consisted of the so-called "meat washings"; faecal material, when present, was generally light green in colour, and often contained sloughs of necrosed mucous membrane. Clots of light pink tenacious mucus, aptly called by Dr. Daniels "frog-spawn stools," were noticed. This form was apt to be accompanied by a tendency to collapse, apparently through exhaustion resulting from the continuous straining, combined with absorption of toxic matter. The temperature in such cases either remained high, or, as in one case, subnormal throughout (Charts 5, 6, 7, 8 and 9). Owing to the constant drain of fluid and the physical strain, these patients became rapidly emaciated, the skin inelastic, the abdomen sunken and acutely tender, and the contracted coils of the sigmoid flexure could be felt with ease through the abdominal wall. Before death the abdominal tenderness became less marked. Twelve of these cases were encountered. Of these the first seven all died, collapse and death taking place suddenly in nearly every instance. In one case, before death, the stools consisted of pure blood. Details of five such cases which improved under treatment are given in Appendix XIV.

From the stools of three of these fulminating cases dysentery bacilli were isolated.

Complications of Acute Dysentery.

Of these, undoubtedly, *pulmonary tuberculosis* is the most formidable; in eleven post-mortems on cases of bacillary dysentery, evidences of extensive pulmonary tuberculosis were found in two. Amongst those cases who recovered

* In many cases amongst the Fijian patients an exceptionally low pulse-rate of 60, and even as low as 48, was registered during the height of the attack and during convalescence. A pulse-rate as low as this is not, however, a racial characteristic. From an examination of 55 individuals (45 males and 10 females) the average normal pulse-rate of adult Fijians was found to be 77.

under treatment, signs of pulmonary tuberculosis were found in seven. One case developed signs at both apices after recovering from a severe attack (Chart 9). This form of dysentery, therefore, appears to be a common terminal affection in natives suffering from any form of tuberculosis.

One Indian, suffering from tertiary syphilis, ankylostomiasis and signs of tuberculosis at both apices, recovered completely under treatment.

One case developed dysentery during an attack of right lobar pneumonia.

Malaria. The spleen was found enlarged in six cases ; these were all Indians and Solomon Islanders but recently settled in Fiji. Malaria parasites were never found in the blood.

Ankylostomiasis. The haemoglobin percentage was roughly estimated by Tallequist's method in every case. In the normal Indians the average was 82 per cent. ; in those harbouring ankylostomes it was 72 per cent. The average haemoglobin percentage in Fijians was 91 per cent., but in those harbouring ankylostomes 86 per cent. Of two severe cases of ankylostomiasis in Indians who were suffering from dysentery, the haemoglobin percentage was 30 per cent. and 40 per cent. respectively. In neither case was the attack of dysentery severe. Improvement was noted immediately appropriate anti-helminthic treatment was undertaken.

Cardiac complications. Systolic bruits, probably of haemic origin, were noted in four cases. They disappeared when convalescence became established. Ankylostome ova were found in the stools in only one of these cases.

Arthritis of dysenteric origin was not noted.

Stomatitis. One case developed acute stomatitis, with the loss of all the incisor teeth. He eventually recovered from the attack of dysentery, which was of the most virulent type.

Intestinal haemorrhage (melaena) was noted in four cases during life. Three of these cases were of the most severe type ; two of them died. In one instance melaena was noted in the stools of a patient convalescent from a mild attack.

Sequelae.

Relapse. One case of relapsing dysentery, apparently of bacillary origin, was noted. A summary of my notes of this case is given in Appendix VIII. (1). The patient had signs of extensive pulmonary tuberculosis.

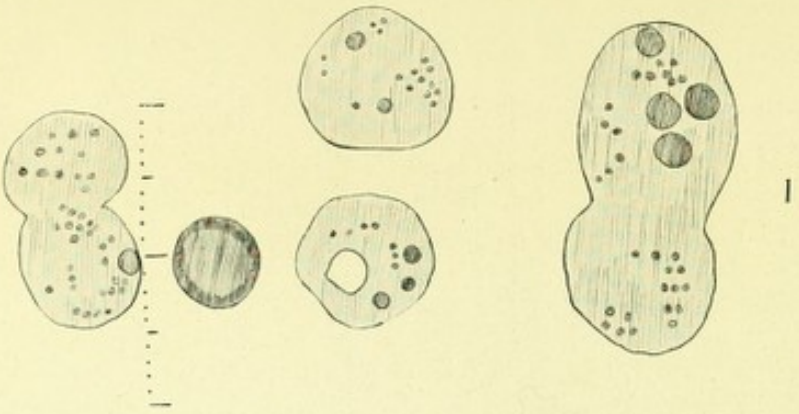
Diarrhoea following dysentery was noted in three cases. The motions averaged about six a day, and consisted of liquid yellow faeces. It was apparently a painless diarrhoea of the lienteric type, and persisted for some months after the other symptoms of dysentery had disappeared.

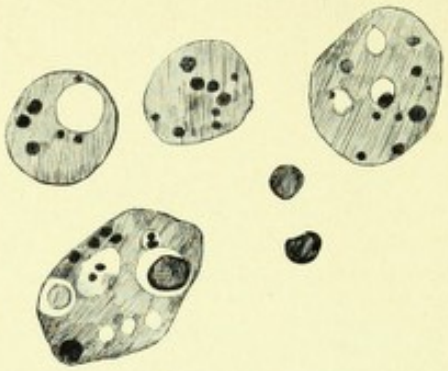
Sprue is a rare disease in Fiji ; nevertheless, one patient, an Indian, after a stay of two-and-a-half months in hospital, developed symptoms resembling those of sprue. A summary of this case is given in Appendix VIII. (2). It is to be particularly noted that no hepatic symptoms were observed in any case during or after the attack of this form of dysentery, save in the sprue case just mentioned.

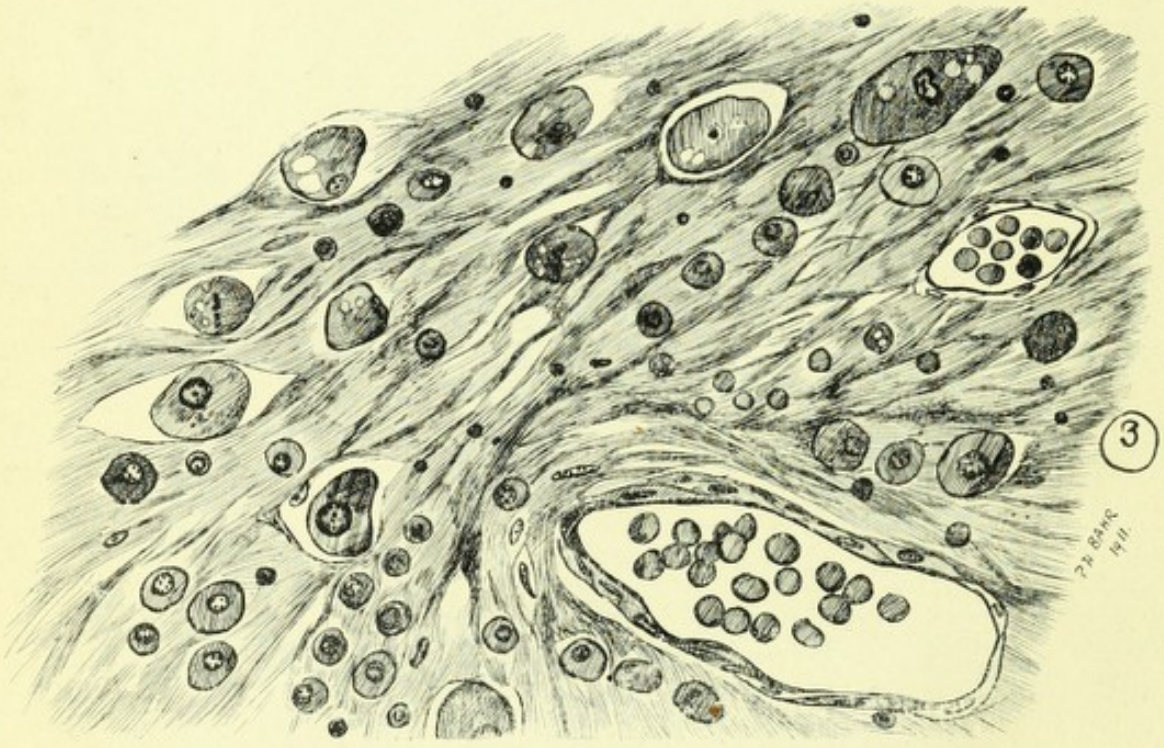
The Microscopical Examination of Stools.

The stools were examined microscopically in every case. Wherever possible the mucus and faecal discharges were examined separately, the former for the presence of amoebae, the latter for ova of intestinal helminthes and flagellates. All cases in which amoebae were found in dysenteric discharges were classified as amoebic dysentery, and will be discussed under that heading.

In the mucous and bloody discharges numbers of red blood corpuscles and pus corpuscles were, of course, found in every case. Refractile, globular bodies of







P. H. Bahr del., 1911.

PLATE I.

1. Refractile vacuolated cells seen in the fresh evacuations in cases of bacillary dysentery. Micromillimetre scale and red blood corpuscle drawn to scale for comparison. —Camera lucida.

2. Similar bodies stained by Schaudinn's method (Heidenhain's haematoxylin and iron alum). One body contains a red blood corpuscle; two red blood corpuscles drawn to scale for comparison. The bodies stained black probably are chromatin masses broken up during process of degeneration.—Camera lucida.

3. Section of submucosa, from a fatal case of bacillary dysentery, showing great numbers of round cells and "mast cells" lying in the inflammatory tissue. Such cells are vacuolated and are similar to the bodies seen in the stools. They are liable to be mistaken for amoebae. Similar bodies have been noted by Jürgens in the Doberitz epidemic and by Dutcher. —Camera lucida.

To face p. 19.

a greenish colour, and often of irregular outline, measuring ten to thirty μ diameter, some of them containing red blood corpuscles (Plate I.) were found on many occasions in the mucus. They contain in their substance granules of various sizes, and are liable to be mistaken for amoebae. Jürgens mentions that these bodies were frequently found in the stools in the Doberitz epidemic. These bodies were found in sixty-two cases in which the stools contained a quantity of mucus. When stained by Schaudinn's method (Heidenhain's haematoxylin and iron alum) the included granules were stained black, and were evidently of chromatic origin. Presumably these bodies represent some degenerating cells derived from the intestinal wall. Similar cells were found in the stools in cases of amoebic dysentery, so that they do not appear to be distinctive of the bacillary form. In sections of the large intestine, large vacuolated cells of a like appearance are found (Plate I.) in the submucosa. These, according to Dutcher, are liable to be mistaken for amoebae in the tissues. They are in all probability, as he suggests, connective tissue cells or "mast cells." It is possible, then, that these bodies have a two-fold derivation, some representing degenerating goblet cells from the intestinal epithelium, while others are derived from the submucosa.

In the stools of the Indian patients, ova of *Ankylostoma duodenale* * were found in 44 per cent., and only once ova of *Ascaris lumbricoides*. Intestinal flagellates were found three times in seventy-nine examinations; these were globular bodies, with large nucleus and terminal flagellum, and resembled most closely the Cercomonas described by Wenyon. In the stools of the Fijian patients, ova of *Tricocephalus dispar* and *Ankylostoma duodenale* were found associated in 35·1 per cent., and ova of *Ankylostoma duodenale* alone in 24 per cent. Small cysts, probably of intestinal flagellates, were encountered five times in fifty-four examinations.

In the stools of the Solomon Islanders, ova of *Ankylostoma duodenale* and *Tricocephalus dispar* were found in 35 per cent.

* More probably ova of *Necator americanus*. Thus, out of 213 adult worms removed from the intestines of Fijians and Indians post-mortem, no fewer than 199 were determined by Dr. Leiper as *Necator americanus*.

IV. Pathology.

(A) Pathological Anatomy.

Post-mortems were procured in eleven cases of bacillary dysentery. Of these nine were adult males, one was an Indian woman, and one a Fijian child of one-and-a-half years of age.

Nine of the cases had been under observation and treatment before death. Two were admitted in a moribund state. Two cases were complicated by extensive pulmonary tuberculosis. In two others there was a terminal pneumonia. In the Indian woman, ankylostomiasis, possibly causing the associated fatty degeneration of the liver, was present, and both kidneys showed chronic inflammatory changes. In one of the cases the left kidney was congenitally absent. In two a few specimens of *Ankylostoma duodenale* and of *Ascaris lumbricoides* were found. No constant change in the mesenteric glands was noted; in one case only were they enlarged and injected.

I did not succeed in cultivating the dysentery bacillus from the bile or heart blood. Putrefaction occurs so rapidly after death in the Tropics that, in the absence of any refrigerating apparatus, the results from such cultures are unreliable. *Bacillus pyocyaneus* was, however, isolated from the bile in one case, and was probably of extraneous origin.

In all these nine examinations the large intestine was the part most severely affected. In five cases there were inflammatory changes of varying degrees

of intensity in the lower part of the ileum, and in one actual gangrene of the mucous membrane.

The large intestine was affected throughout its whole extent in every case, the process involving the rectum. In some cases the dysenteric lesions terminated three inches above the anus; in others they extended to the anus itself.

The parts most affected were the caecum, the hepatic and splenic flexures, and, especially, the sigmoid flexure and first part of the rectum. Actual ulceration, when present, was noted mostly in the transverse folds of the mucous membranes. The appendix participated in the general change in only one case.

Once curious patches of apparently normal mucous membrane, of some two to three inches in extent, were seen at points situated distal to the hepatic and sigmoid flexures.

In the most severe forms the bowel wall was greatly thickened, chiefly owing to oedema and infiltration of the submucosa. The peritoneal coat was normal, save in one instance, where the caecum had been perforated and peritonitis had resulted.

The mucous coat was either of a light green or, in these very severe cases, blackish-green colour, and wholly necrotic (Plate II.). In several instances, where there was extensive sloughing, the necrosed mucous and submucous layers could be removed by the finger; this condition is entirely different from the condition known as diphtheritic dysentery, a term employed by German writers.

The process of necrosis of the mucous membrane appears to start from the solitary follicles; in one case, by holding the specimen up to the light, this could be clearly seen in the descending colon. The surface of the necrosed mucous membrane was rough and nodular to the touch, and was often pitted with holes where portions of membrane had sloughed away. In other cases, where, although only local patches of necrosis had formed, the whole of the surrounding mucosa was discoloured and oedematous.

In cases of this nature ragged ulcers of varying depth had resulted from the sloughing of the necrosed mucous membrane.

Nothing resembling the regular punched-out ulcers with thickened edges, such as are described in amoebic dysentery, was seen.

Small, sinuous undermining ulcers, varying from the size of a sixpence to a shilling, could often be traced by a probe passed under the mucous membrane.

In milder cases the mucous membrane was of a cherry-red colour. There were local patches of necrosis, and, especially on the free edges of the transverse folds, bright red submucous haemorrhages.

In others (Plate III.) the mucous membrane was pink and oedematous, with a lobulated appearance. On the transverse folds were gelatinous red patches, due to submucous haemorrhages. Fine sloughs of a yellow colour and sinuous outline were seen forming on the exposed surface of the folds.

In one, namely a Fijian child who died one week after all symptoms of dysentery had ceased, the mucous membrane of the transverse colon was injected and thickened, and there were a few small green patches; apparently no sloughing of the mucous coat had taken place.

As regards the intestinal contents, these, in the majority of cases, consisted of tenacious, slimy mucus, intermingled with thick, green faecal matter. In two in which the gangrenous process was especially severe, there were no fluid contents, but the intestinal surface was covered with thick mucus and blood resembling pink frog's spawn. The same typical frog-spawn substance had been recognized in the discharges during life. In one case, together with destruction of the most severe type, hard lumps of yellow faecal matter were noted in the caecum.

1 2

3

P. H. Bahr del., 1910.

PLATE II.

Post-mortem appearances of intestines in a fatal case of bacillary dysentery from which Shiga's bacillus was isolated.

1. Lower part of ileum, showing necrosed epithelium and injected Peyer's patch.
2. Transverse colon, showing bright green colour of necrosed mucous membrane.
3. Lower part of rectum, showing patches of bright green necrosed mucous membrane and submucous haemorrhages.

To face p. 20.]

(B) Microscopic.—Pathology.

Sections were cut of the portions of the bowel most affected by the dysenteric lesions. From each post-mortem three or more areas of affected bowel wall were chosen.

The microscopical changes varied according to the severity of the lesions.

A description of the changes noted in sections of the large intestine also applies to the ileum where similarly affected.

In the gangrenous form the epithelial cells could no longer be distinguished, necrosis of the whole of the mucous layer having taken place; here and there, a few vacuolated and distorted cells remained at the fundus of a crypt.

The outstanding feature of the sections is the infiltration of the submucosa and destruction of the nutrient blood-vessels. The inflammatory changes are intense, the blood-vessels dilated ; there are numerous haemorrhages, especially beneath the muscularis mucosae, the fibres of which, though participating in the general change, can still be distinguished (Plate IV.). The appearances are extremely similar to those found by Vaillard and Dopter (*Annales de l'Institut Pasteur*) both of lesions in human subjects and experimental dysentery in rabbits.

In some instances, where the process had not been so acute, the crypts can still be distinguished, but the cells stain badly, are vacuolated and distorted, and the lumen of the crypt distended by a fibrinous exudate containing numerous round and disintegrated epithelial cells. The interstitial tissue between the crypts is infiltrated and the blood-vessels distended, and there are a few haemorrhages. In others the entire epithelial layer has been removed and replaced by granulation tissue. When sloughing and actual ulceration to any marked degree have taken place, the muscularis mucosae can no longer be distinguished, the muscular coats being covered solely by a layer of granulation tissue. In cases of a milder type, or when the process has been a more gradual one, no haemorrhages are seen in the submucosa. The blood-vessels are greatly dilated, the stroma infiltrated with round cells and especially by large mast cells with a prominent nucleus and vacuolated protoplasm. These cells are apt to be mistaken for amoebae (Plate I.), but the character of the nucleus is quite distinctive. In every case sections were stained for the presence of bacilli by carbol thionin. Intestinal organisms were seen crowding the affected surface in great numbers.

It was interesting to note that where the muscularis mucosae remained intact no bacilli were ever found in the submucosa.

Numerous micro-organisms could be distinguished amongst the lymphoid tissue of the solitary follicles ; whether they were dysentery bacilli or not it was impossible to determine from their morphological characters alone. The muscular coats of the intestinal wall are not changed to any marked degree, although dilatation of blood-vessels and round cell infiltration between the muscular fibres were noted.

Haemorrhages into the submucosa and complete destruction of the mucous coat were found in cases dying from other causes, and after what clinically appeared to be but a comparatively mild attack of dysentery.

V. Epidemiology. The spread of acute dysentery in Suva.

The dysenteric excreta undoubtedly act as a source of infection. Shiga considers that the main factors in the spread of acute dysentery are : (1) excreta of normal persons harbouring dysentery bacilli ; (2) excreta of mild cases of dysenteric diarrhœa ; (3) excreta of cases convalescent from dysentery. Cases of diarrhoea due to infection with the dysentery bacillus undoubtedly occur, their dysenteric nature being unsuspected. An epidemic of acute diarrhoea

occurred in the native high school near Suva ; three cases developed dysentery, one had diarrhoea with a concomitant rise of temperature ; seven had diarrhoea with a normal temperature. The serum of five of those with diarrhoea agglutinated Shiga's bacillus in a dilution of 1 : 50.

In Fiji the planters believe that the Indian coolies imported from Calcutta act as reservoirs of the infective agent of dysentery ; dysentery, they say, invariably breaks out amongst the Solomon Island labourers when brought into contact with the Indian coolies on the plantations.

There is no adequate reason for incriminating water as a source of infection in Suva. Suva has a large rainfall, and the water is everywhere excellent. The water supply is conveyed from a reservoir situated four miles from the town at the source of a small river, where faecal contamination is out of the question. It is distributed to all the institutions (the gaols and schools) and to every house, in iron pipes.

Experiments were undertaken to ascertain the viability of the dysentery bacillus in water. Frost and Whitman give one week as the maximum period the bacillus is able to remain alive in water. In Fiji I found the period much longer. Test tubes containing tap water and sterilized distilled water were inoculated with a loopful of a freshly isolated culture of Flexner's bacillus ; samples of the water were from time to time plated out on Conradi-Drigalski medium. From the sterilized water the bacilli could be recovered and cultivated four weeks, and from the tap water six weeks, after inoculation.

There is no evidence, in Suva at least, that any article of diet can be regarded as the direct cause of acute dysentery. The disease occurs amongst all the races in Fiji, though their respective dietaries are entirely dissimilar. The Fijians live for the most part on the farinaceous native foods, the " yam " and the " dalo," and rarely partake of uncooked foods, such as fruit and especially raw vegetables ; whilst the Indians subsist, as elsewhere, on cooked rice and pulse.

There is considerable circumstantial evidence that in Fiji flies (*Musca domestica*) act as the main agents in conveying the infection by transferring it to foodstuffs. Various writers have incriminated the fly as the carrier of the dysentery virus without adducing any direct evidence. Nuttall and Jepson include dysentery in their résumé of diseases spread by *Musca domestica* and give an epitome of the literature up to date. Graham-Smith has isolated a bacillus resembling Flexner's from the intestines of flies caught in Cambridge.

House-flies (*Musca domestica*) constitute a great plague in Fiji. They swarm during the hot weather (November to April) in Suva ; in the sugar districts they are prevalent throughout the year, finding suitable breeding-places in the decomposing vegetation necessarily connected with this industry. In many of the islands the traveller becomes covered from head to foot with these insects immediately he ventures outside his lodging. In the sugar districts life is made endurable only by the provision of fly-proof netting over the doors and windows. In these districts nearly every house is provided with at least one fly-proof room. Flies are specially numerous in the Fijian villages, where they find abundant congenial breeding-places in the refuse heaps.

I need hardly point out that in consequence of this great profusion of flies every article of food is liable to gross contamination by these insects ; and it is my suggestion that the fly season in Suva corresponds exactly with the annual epidemic of dysentery* (*vide* Appendix I.). After the great hurricane in March, 1910, there was a sudden and large increase in the number of cases of dysentery

* It must be borne in mind that in the official returns no distinction is drawn between cases of bacillary and amoebic origin. It may safely be stated, from my experience of the epidemic of 1910, that the majority of the cases occurring in hot weather are of the bacillary form.

P. H. Bahr del., 1910.

PLATE III.

Transverse colon from a case of bacillary dysentery, painted 1½ hours after death. There is intense oedema of the mucous membrane ; actual ulceration has taken place at the exposed transverse folds of mucous membrane, where yellow sloughs are seen forming. There are numerous submucous haemorrhages.

Shiga's bacillus was isolated post-mortem from the mucous surface.

Actual gangrene of the mucous layer has not yet taken place.

To face p. 22.]

concurrently with a vast increase in the number of flies, which found in the rotting vegetation (Appendix I.) resulting from the hurricane favourable breeding grounds.

In the sugar districts (*vide* Appendix II.) there appears to be no definite dysentery season, and there apparently is no seasonal variation in the number of flies.

The infection of patients, who are in hospital suffering from other complaints, with dysentery can best be explained by the agency of flies. A summary of the methods employed for the disposal of dysenteric excreta at the Colonial Hospital, and the opportunities for the fly-borne spread of the infection, is given in Appendix IX. In the same Appendix also the sanitation of the two gaols from which were derived the great majority of the cases which came under observation is detailed.

The phenomenal preponderance of flies in Fiji, the great frequency of dysentery there, the concurrence of the fly season with the dysentery season, the many opportunities supplied by the insanitary conditions obtaining in the villages, plantations and public institutions for contamination of food by infected flies, are distinctly in favour of regarding Fijian epidemic dysentery as a fly-borne disease.

Further evidence in favour of this hypothesis will be found in the following observations and experiments.

I made many attempts to isolate the dysentery bacillus from the intestinal contents of house-flies caught in the dysentery ward.

The technique was as follows: Four to six flies were caught on patients; none were taken on the ward verandahs, where there was a chance of these flies having fed on dysenteric excreta. The insects were chloroformed and dropped into a tube of broth, and were then dissected with sterilized Hagedorn needles on sterile slides. The abdominal segments were separated and the intestines drawn out without touching any other part of the fly—an easy procedure after a little practice. A segment of the intestine was divided by a red-hot platinum loop and inoculated into broth from which Conradi-Drigalski plates were made. The legs and wings were also inoculated into broth and similarly treated. *Bacillus neapolitanus* and other members of the coli group were frequently isolated from these appendages, and on two separate occasions Shiga's bacillus was isolated from the lower intestinal tract. As regards the latter, the flies were caught on the bed of a patient suffering from a very acute attack of dysentery, and from whose stools this same bacillus had been isolated. The insects had probably imbibed the infection from the soiled clothing. These bacilli were subjected to a series of tests, and had the typical morphological and cultural characteristics; they were also agglutinated by Lister Institute anti-serum.

After the isolation of the Shiga bacillus from the intestinal tract of flies, experiments were undertaken to ascertain (1) the length of time the fly remains infective, and (2) whether the bacillus undergoes any variation by its passage through the intestinal tract of the insect. These experiments, over forty in number, were begun in Fiji and continued at the London School of Tropical Medicine.

Technique.

The works of Graham-Smith on this subject in the Reports of the Local Government Board were consulted, and his method, with modifications, has been closely followed throughout.

Fly larvae were collected from rubbish, and the resulting pupae selected, cleaned, and placed in sterilized wooden cages covered with gauze. It was impossible by such means to obviate previous contamination of the larva by

extraneous organisms, and possibly by the dysentery bacillus itself; but the work of Graham-Smith on larvae infected with *Bacillus typhosus* and *B. enteritidis* renders the transmission of infection from larva to imago extremely improbable. He was able to recover only the spore-bearing organisms from insects infected through the larva,* and *B. dysenteriae* does not belong to this category of bacteria.

Attempts at breeding the larvae from ova under sterile conditions proved futile, since a fermentation is necessary for their development, as suggested by Jepson.

The experimental flies were hatched and kept in sterile cages and fed on sterile food—bread soaked in glucose broth and changed twice daily. A number of control experiments were made with flies similarly hatched and fed.

The experiments were conducted in a glass house from which other flies and insects were rigorously excluded. Before commencing the interior of the house had been cleansed with lysol. The cages were placed on a layer of cotton wool, which was kept constantly damp with lysol solution. The temperature of the house simulated tropical conditions as closely as possible, and in August varied from 68° F. during the night-time to 91° F. at mid-day.

Ten of these flies were removed at a time and killed with chloroform; their intestines were then dissected out, as described above, and emulsified in broth. The resulting emulsion was spread on Conradi-Drigalski plates. In this way some idea was obtained of the bacterial flora of the intestinal tract of newly-hatched insects, and also of those which had been kept for varying periods.

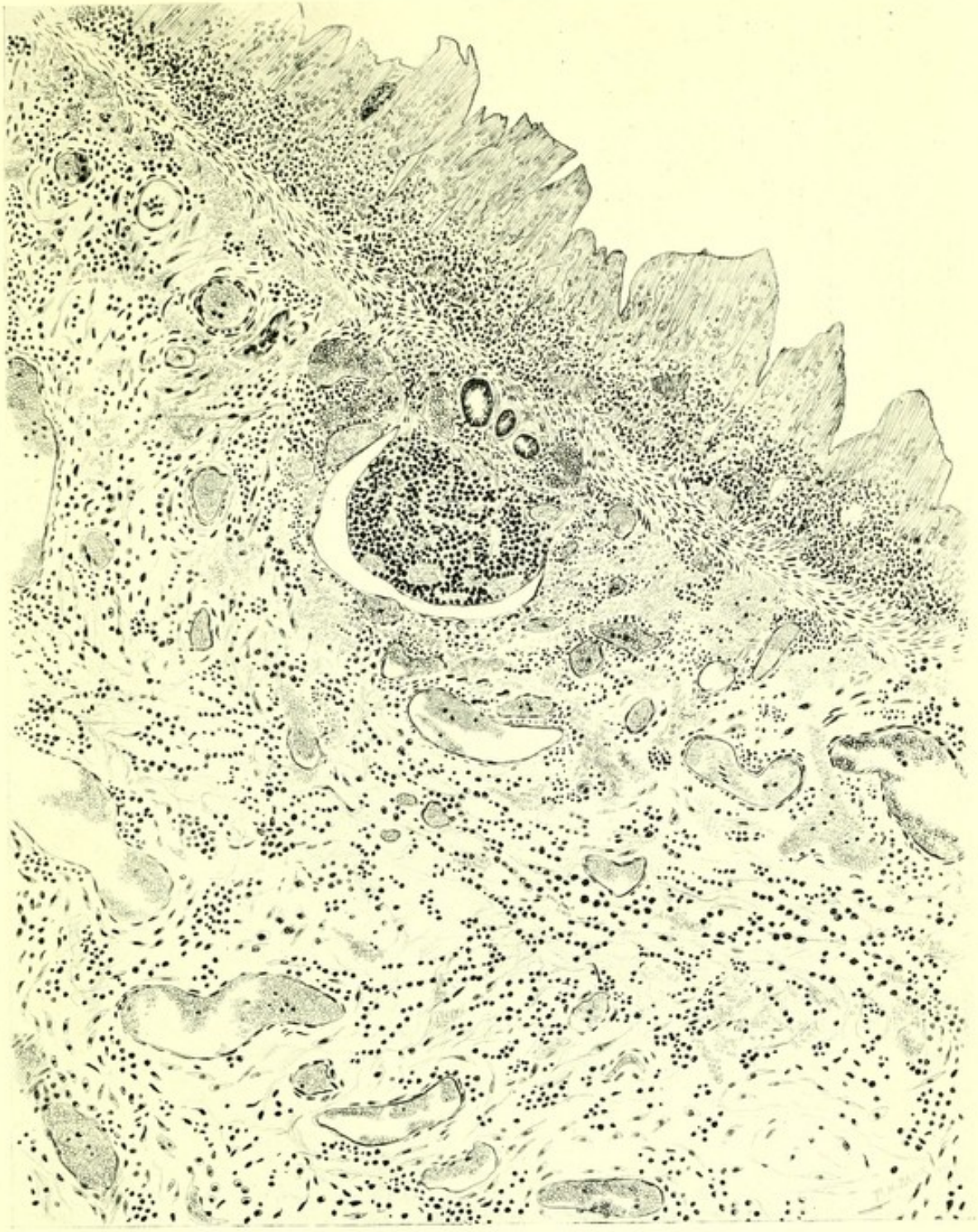
Out of twenty such examinations, all of flies recently hatched, in nine the emulsion proved sterile on culture. In the other eleven flies the organism most commonly encountered was a small non-Gram-staining streptococcus. Gram-staining strepto- and staphylococci were also encountered, but no bacilli at all resembling those of the coli group.

Other flies were infected with the dysentery bacillus in the following way: A watch-glass, containing sterile bread soaked in broth cultures of the dysentery bacillus which had been incubated for twenty-four hours, was placed in a sterilized cage containing twenty flies. After feeding on this material for twenty-four hours, the flies were removed in test tubes to glass jars similar to those enployed by Graham-Smith. The jars were provided with gauze tops, through which the food could be introduced and removed. Metal spoons which could be sterilized frequently and easily were found the easiest and most convenient way of introducing the food. The food was changed every twenty-four hours. The jars were appropriately labelled and placed on cotton wool soaked in lysol. On successive days two to three of the infected flies were removed and dissected. The intestines were drawn out and removed on platinum wires in the manner already described, an emulsion made in a small quantity of broth, and the whole then poured on to a Conradi plate.

By following the technique just described I never recovered the dysentery bacillus from the intestine of any fly after the fifth day of infection. Similarly, working with non-spore-bearing organisms, *Bacillus typhosus* and *enteritidis*, Graham-Smith found that flies do not remain infective for a longer period than seven days, and further that the bacilli on the legs and wings perish in even a few hours.

I subjected the bacilli recovered from the plates to a series of identification

* The house-fly of Fiji has been identified for me by Col. A. Alcock, I.M.S., C.I.E., F.R.S., as the same *Musca domestica* as occurs in England. Lately, Bacot (*Parasitolgy* Vol. IV., No. 1, 1911, p. 68) has isolated *B. pyocyaneus* and *B. typhosus* from the pupal contents of *Musca domestica* infected by these organisms in the larval state.



P. H. Bahr del., 1911

PLATE IV.

Microscopic section through the sigmoid flexure in a fatal case of bacillary dysentery, from which Shiga's bacillus was isolated post-mortem, showing the necrosed mucous layer and the greatly infiltrated submucous layer. The blood-vessels are dilated and there are numerous haemorrhages. The section has passed through a solitary lymphoid follicle.

To face p. 24.

tests. In addition to careful scrutiny of their morphological character, they were tested with Lister Institute anti-serum in a dilution of 1 : 50, and were cultivated in the various sugars in peptone water.

The stock cultures of dysentery bacilli utilized for the purpose of infection were selected from cultures which, after four separate trials extending over a period of several months, had constantly given the same sugar reactions (Appendix IV.). They were undoubtedly the true non-acid Shiga's bacillus (type A) and the true acid bacillus (type D).

In the majority of instances the bacillus recovered from the intestinal tract of the flies gave the same sugar reactions as the original bacillus. All observations on the sugar reactions were extended over a period of ten days' incubation in each instance.

In several instances bacilli, which were undoubtedly derived from the bacillus originally fed to the flies, gave slightly different sugar reactions. These variants were morphologically identical with the original bacillus, and were all agglutinated by anti-serum in a dilution of 1 : 200. Thus, from type A a bacillus was obtained which fermented maltose (Appendix X.); while from flies fed on bacillus type D bacilli were obtained which fermented maltose and saccharose, in addition to their power of fermenting mannite and dextrose.

One bacillus in particular is of great interest. It was tested on three separate occasions, and always gave the same reactions. Its power of fermenting mannite had apparently become diminished, for neither mannite nor maltose was fermented by it till the fourth day of incubation. Thus this mannite-fermenting power, on which a distinction between the acid and non-acid types of dysentery bacilli has been made, varies with environment. Other colonies, selected from the same plate as this particular organism referred to, gave the same reactions as the original bacillus.

On some other occasions different colonies from the same plate gave variable sugar reactions.

These changes in the fermentation properties of the bacilli have not remained constant on further subculture on agar. After two months' subculture bacilli of the non-acid group (Appendix III., type A) gave the reactions of the original strain, and no longer fermented maltose; while a bacillus of the acid group, which fermented maltose and saccharose after the same period of subculture, only did so on being incubated for fifteen days.

Lastly, a bacillus derived from the acid group (Appendix III., type D), after one month's subculture, produced a feebly acid reaction with mannite after twenty-four hours, but after ten days' further incubation the medium became definitely alkaline. After culture for fifteen days in maltose it once more gave the original acid reaction. After a still longer period of subculture this bacillus gave the same reactions as the type from which it was derived, and produced acid from mannite and dextrose after twenty-four hours' incubation.

The formation of indol by these bacilli, thus recovered from flies, was found to vary greatly and within wide limits. In some, indeed, a marked indol reaction was produced after twenty-four hours' incubation in peptone water, while in others (giving the same sugar reactions) no indol was formed even after incubation extending over a period of ten days.

VI. Treatment.

In a disease of such varying severity as bacillary dysentery it becomes an especially difficult task to estimate the effect and value of any particular line of treatment. In dealing with Europeans, even though they may be under the

most favourable conditions for observation, it is almost impossible. This difficulty was greatly increased in our dealings with Fijians and Indians, for, in addition to the irresponsible behaviour of the former while under treatment, one must bear in mind the tendency to malingering, especially amongst the Indians, to which allusion has already been made.

As regards diet, accurate supervision of the food supply was another difficulty. The hospital diet of patients under treatment consisted of a generous allowance of milk only, but to this sago and arrowroot were added on convalescence. Not infrequently, however, the friends and relations of patients contrived to smuggle in other articles, amongst which jam and pickles were especial favourites.

The routine medical treatment adopted in the Colonial Hospital, Suva, in dysentery cases was as follows : A dose of castor oil and opium (ol. ricini 1 oz., tinct. opii m. xv.) was given immediately on admission, after which sodium sulphate 1 drachm was given every hour for the first twenty-four hours, and subsequently four-hourly. In a certain limited number of cases rectal lavage with warm, weak boracic solution was resorted to, mostly, however, in Europeans.

Ipecacuanha was only given in one case towards the termination of the attack. In this the stool contained great numbers of intestinal flagellates, and the tongue continued furred. After the exhibition of pulv. ipecac. in twenty-grain doses the tongue became clean and the flagellates disappeared from the stool.

Of the cases of dysentery observed by me, the first series, consisting of fifty-three consecutive cases, were treated according to the hospital routine treatment just described.

Thirty-one, or 58·5 per cent., were suffering from the mild or catarrhal type.

Seventeen, or 32 per cent., were suffering from the acute or ulcerative type.

Five, or 9·4 per cent., were of the fulminating or toxic type.

Of these seven, or 13·2 per cent., died. In four only dysenteric lesions alone were found post-mortem ; in two pulmonary tuberculosis was also present, and one was complicated by severe tertiary yaws. Details of three of these cases are given in Appendix XI.

The 106 cases constituting the next series were treated by intestinal antiseptics by the mouth, and intravenous injections of anti-dysenteric serum supplied by the Lister Institute. In thirty-four consecutive cases treated by cyllin and salines alone there were no deaths. The remaining seventy-two were treated with cyllin by the mouth, and by injections of anti-serum in addition to the routine hospital treatment with salines. There were two deaths, but neither of these could be ascribed to the dysenteric lesions. The dysentery in each case was not of the severest clinical type. One, an Indian woman with chronic nephritis and ankylostomiasis, died in uraemic convulsions ; the other, a Fijian child, died from broncho-pneumonia, a week after the stools had become normal. (Details of these cases are given in Appendix XII.)

The mortality in this series was 1·8 per cent.

Sixty-one of these cases, or 57·6 per cent., were classified as belonging to the clinically mild type of dysentery.

Forty, or 37·7 per cent., were classified as belonging to the acute type.

Five, or 4·7 per cent., were classified as belonging to the fulminating or toxic type.

No deaths occurred in patients suffering from the most severe or toxic type of infection, while under treatment with anti-serum (Appendix XIV. and Charts 6, 7, 8 and 9).

As the condition of the stools affords some indication, and probably the only reliable one, of the progress of cases under treatment, an attempt was made to

determine whether the rapid amelioration in the condition of the stools was in favour of any particular line of treatment. A table constructed with this in view is given in Appendix XIII., attention being paid to the rapidity with which the stools became entirely faeculent, and the length of the patient's stay in hospital. The figures obtained are slightly in favour of the treatment with cyllin and anti-serum.

Cyllin Treatment.

The treatment of acute dysentery by intestinal antiseptics has been extensively used in India. Vaughan, Mackie and others report in favour of the treatment with izal in large doses as decreasing mortality and shortening the attack. The cyllin utilized in my cases was prepared by Messrs. Jeyes in two forms, and was given in large doses. One consisted of pure cyllin enclosed in gelatine capsules, each capsule containing three minims of cyllin. The other preparation, known as cyllin syrup, containing five per cent. pure cyllin, was given in doses of one drachm to children and half an ounce three times a day to adults. Twenty and even thirty of the gelatine capsules (sixty to ninety minims of pure cyllin), or a corresponding amount of the syrup, were given daily to cases of exceptional severity. The drug was generally given for three to four days, though in some instances its administration was continued for over a week, and occasionally, in cases of the fulminant type, for as long as a fortnight. No untoward effects were ever noted. It is difficult to say whether this drug actually had an effect on the course of the disease. Stools of foul odour and green colour rapidly ameliorated during a course of this treatment. A series of thirty-four consecutive cases were treated in this way, in addition to the routine hospital treatment. There were no deaths (Appendix XIV., Chart 5).

Anti-serum Treatment.

Anti-serum, in the majority of cases, was given intravenously. Adults were given twenty cc., children ten cc. or less. For injection a Roux's syringe, holding 10 cc., was used. One injection was given into the median basilic vein on each side. In several instances, especially in those cases suffering from the severest type of infection, the administration of 50 to 70 cc. of anti-serum on three or four consecutive occasions was followed by marked improvement. In some few cases in which the veins were not prominent, especially in women and in children, the anti-serum was injected subcutaneously. A rise of temperature of from 2° to 3° F. was noted in a few instances after the injection, and in one case in particular there was a rigor ; otherwise no untoward after-effects were noted.

To prevent deterioration of the serum by climate, the stock was kept in an ice chest at about one degree above freezing point.

In cases of collapse, continuous subcutaneous transfusion with normal saline, along with the anti-serum, was given, and often was employed with marked benefit.

If I might be allowed to draw conclusions from my limited number of cases, the combined method of anti-serum injection, together with the administration of salines and cyllin, gives the best results.

The details of four severe cases of dysentery which recovered under this line of treatment are given in Appendix XIV., and Charts 6, 7, 8 and 9. The improvement which took place, not only in the general condition of the patient, but also in the number and character of the stools, was very apparent (Appendix XIV., Chart 7).

The combination of these three lines of treatment seems to be a rational one. The lesions of acute dysentery are local at first, and are mostly confined

to the large intestine, whence the toxins are absorbed into the blood and cause severe constitutional effects. To counteract the deleterious effect of the toxins of the dysentery bacillus, anti-serum is injected. The administration of sodium sulphate tends to cleanse the large bowel of faecal matter, thereby hindering undue putrefaction, and consequently accelerates the healing of the ulcerated surface. Cyllin in large doses has been found to exert a marked influence on the intestinal flora, and thus most probably prevents the multiplication of the dysentery bacillus and other intestinal organisms in the intestinal contents.

Shiga states that since the introduction of anti-dysenteric serum the case mortality from bacillary dysentery has fallen from thirty-five per cent. to nine per cent. Good results from polyvalent anti-dysenteric sera have been recorded by Coyne and Auché, Vaillard and Dopter in France, by Rosenthal in Moscow, and Lüdke in Germany.

In instituting therapeutic comparisons, especially in diseases of such variable severity as dysentery, it is necessary to specify the type of cases treated, and especially the proportion of exceptionally severe ones.

VII. Prophylaxis.

The question of prevention of epidemic dysentery in Suva is of vital importance to the whole Colony. Epidemic dysentery constitutes the only epidemic scourge of importance interfering with the increasing prosperity of the islands.

At present there is no drainage system in Suva; it would seem that the comparatively healthy condition of the town is attributable to the frequent cleansing effected by the exceptionally heavy rainfall.

There is an extensive pail system of disposing of the nightsoil. The benefit of this as regards dysentery is, to say the least, doubtful, for the pail system necessarily affords facilities for flies to breed in numbers, and to contaminate themselves in faecal matter, as has been proved to be the case under actual experiment. From what I have observed I am convinced that the spread of infection by house-flies affords the most plausible explanation of the incidence and prevalence of dysentery in Suva, and in great measure in the villages and plantations in the Fiji Islands. A complete drainage system is about to be installed in Suva. It is to be hoped and expected that, with this and improved sanitation generally, the number of house-flies will diminish, and with them the seasonal occurrence of epidemics of bacillary dysentery.

Bacillary Dysentery.

In preparation of the foregoing report on bacillary dysentery, the following works have been consulted :—

Abe.—Arch. f. Hyg. LXV., 1908, Vol. II., p. 107.
Amako.—Ztsch. f. Hyg. u. Inf. LX., 1908, Vol. I., p. 93.
Aveline, Boycott & Macdonald.—Journ. of Hyg. VIII. 1908, No. 3, p. 309.
Blackham.—Journ. Roy. Army Med. Corps., X., 1908, No. 4, p. 436.
Blasi, de.—Cbl. für Bakt., Pt. 1, XXXVI., 1904, No. 2, p. 161.
Brunwin.—Journ. Trop. Med. 1908, Sept. 15, p. 278.
Buchanan.—B. M. J., 1901, I., p. 881, and 1902, II., p. 843.
Castellani.—Journ. Ceylon Branch B.M.A., 1904.
Coyne & Auché.—Compt. Rend. de la Soc. de Biol. LXV., 1908, p. 629.
Deycke.—Deutsch. Med. Wochensch. 1907, No. 18, p. 726.
Dutcher.—Journ. Amer. Med. Assoc. 1903, No. 8, p. 476.
Duval.—*ib.* 1904, II., p. 381.
Duval & Bassett.—Am. Med. 1902, IV., p. 417.
Duval & Shorer.—Ann. Rockefeller Inst. 1904, Vol. II., p. 42.

Escherich.—Cbl. für Bakt. XXVI., 1899, No. 13, p. 385.
Eyre.—B. M. J. 1904, I., p. 1,002, and Ed. Med. Journ., 1904, p. 489.
Firth.—Journ. Roy. Army Med. Corps, Dec. 1903.
Fisher.—Journ. Med. Res., May, 1907, p. 181.
Flexner.—Univ. Penna. Med. Bull., Aug. 1901, p. 1, and B. M. J. 1901, II., p. 786.
Frost & Whitman.—Cbl. für Bakt. XXXVII., 1906, Nos. 11, 14, p. 359.
Gay & Duval.—Univ. Penna. Med. Bull., 1903, XVI., p. 177.
Graham-Smith.—Rep. Loc. Govt. Board (New Series), No. 40, 1910, I., p. 1; No. 53, 1911, 4, p. 31.
Hirsch, C.—Ed. Med. Journ., 1900, p. 52.
Hiss.—Journ. Med. Res. XIII., 1904, No. 1, p. 1.
Jennings.—Ind. Med. Gazette, 1905, p. 265.
Jürgens.—Ztsch. für Exp. Path. u. Therap. IV., 1908, Vol. III., p. 769.
Kemp.—Ztsch. für Hyg. u. Inf. LVII., 1907, Vol. III , p. 489.
Kruse.—Deutsch. Med Wochensch., 1900, No. 40, p. 637.
— ,, ,, ,, ,, 1901, No. 23, p. 360; No. 24, p. 386.
— ,, ,, ,, ,, 1903, No. 12, p. 201.
— ,, ,, ,, ,, 1907, No. 8, p. 292; No. 9, p. 338.
— ,, ,, ,, ,, Klin. Jahr. XIX., 1908, Vol. IV.
Kruse, Ritterhaus, Kemp & Metz.—Ztsch. für Hyg. u. Inf. LVII., 1907, Vol. III., p. 417.
Kühn, Gildmeister & Woithe.—Art. a.d. Kaiser. Gesundh. XXXI., Vol. II., 1911, p. 394.
Lentz.—Ztsch. für Hyg. u. Inf. XLI., 1902, Vol. III., p. 540.
Lentz, O.—*ib.* XLIII., 1903, Vol. III., p. 480.
Lewis.—Journ. Roy. Army Med. Corps, VII., 1906, p. 510.
Liefmann.—Münich Med., Wochensch. 1906, p. 2,097.
Loghem & Schüffner.—Geneest. Tijdsch. v. Ned. Ind. XLIX., 1909, I., p. 16.
Lüdke.—Deutsch. Med. Wochensch. 1906, No. 5, p. 181.
Macalister.—B. M. J. 1910, II., p. 1,506.
Mackie.—Ind. Med. Gazette, 1905, p. 260.
Manson.—Tropical Diseases, 1907.
Marshall.—Journ. Roy. Army Med. Corps, 1909, Vol. XII., p. 556.
Martini & Lentz, O.—Ztsch. für Hyg. u. Inf. XLI., 1902, Vol. III., p. 540.
Mense.—Handbuch der Tropenkr. Vol. III., pp. 219-274.
McWeeney.—B. M. J. 1906, I., p. 1,564.
Morgan.—Journ. of Hyg. 1911, Vol. XI., No. 1, p. 1.
Müller.—Cbl. für Bakt. XXXI., 1902, p. 558.
Nuttall & Jepson.—Rep. Loc. Govt. Board (New Series), 1909, No. 16, 4, p. 20.
Ohno.—Phil. Journ. Sci. Vol. I., No. 9, Nov. 1906, p. 951.
Penfold.—Journ. of Hyg. Vol. II., No. 1, Mar., 1911, p. 30.
Rogers.—Ind. Med. Gazette, 1903, XXXVIII., No. 2, p. 53.
Rosenthal.—Cbl. für Bakt. XXXIV., 1904, Nos. 16-17, p. 503.
Sandwith.—Lancet, 1907, II., Dec. p. 1,589.
Saundby.—B. M. J. 1906, I., p. 1,325.
Scheube.—Krankheiten der warmen Länder, 1910, pp. 850-874.
Shiga.—Cbl. für Bkt. XXIII., 1898, No. 14, pp. 599; XXIV., 1898, pp. 817, 870, 913.
— Deutsch. Med. Wochensch., 1901, No. 43, p. 741; No. 44, p. 765; No. 45, p. 783.
— Ztsch. für Hyg. u. Inf. XLI., 1902, II., p. 355.

Shiga.—Deutsch. Med. Wochensch., 1903, No. 7, p. 113 ; No. 18, p. 327.
— Phil. Journ. Sci., I., No. 5, June, 1906, p. 425.
— Ztsch. für Hyg. u. Inf. LX., 1908, No. 1, pp. 75, 120.
Torrey.—Journ. Exp. Med., 1905, VII., pp. 365 and 385.
Twort.—Proc. Roy. Soc. Lond., Vol. LXXIX., 1907, p. 329.
— Cbl. für Bakt. XL., 1907, p. 508.
Vaillard & Dopter.—Ann. de l'Institut Pasteur, 1903, June, p. 463 ; 1907, XXI., No. 4, p. 242.
Vaughan.—Ind. Med. Gazette, 1904, Oct., p. 361.
Vedder & Duval.—Journ. Exp. Med., Vol. VI., No. 2, Feb. 1902, p. 181.
Wenyon.—Quart. Journ. Microscop. Sci., Part II., June, 1910, p. 241.
Woolstein & Dewey.—Ann. Rockefeller Instit., 1904, Vol. II., p. 42.

B.—Amoebic Dysentery.

Historical.—Amoebae were discovered in association with dysenteric stools by Lösch in 1875. In 1886 Koch and Kartulis found similar protozoa very commonly in dysenteric excreta in Egypt, and by rectal injections of amoebae-containing liver pus the latter produced dysenteric symptoms in a cat. The assumed pathogenicity of these amoebae was, however, placed in doubt by Kruse and Pasquale in Italy, who found similar organisms in the stools of twenty out of thirty-five healthy persons examined.* It was recalled also that Lewis and Cunningham had found amoebae in the stools of cholera patients in India.

In 1891, in their well-known essay, Councilman and Lafleur described the lesions of the type of dysentery associated with amoebae, and now known as Amoebic Dysentery, as quite distinct from any other affection of the bowel, and demonstrated that the histological picture produced by the action of the amoeba in the tissues was entirely different from that produced by a bacterial invasion.

Jürgens, in 1902, studied an outbreak of dysentery in troops from abroad on their return to Germany. Both amoebic and bacillary forms were found. He contended that the amoeba was able to cause dysenteric bowel lesions without bacillary intervention.

Shortly afterwards Schaudinn's work appeared, and seemed definitely to settle the problem. He described two forms of amoebae—a harmless and a pathogenic. He stated that, in addition to distinctive differences in structure of their respective vegetative stages, the method of reproduction in the two forms was also entirely dissimilar. The two forms were specifically distinct. The pathogenic form he called *Entamoeba histolytica*, the non-pathogenic *Entamoeba coli*.

The chief characteristics of the two species were given as follows : *E. coli.*—The vegetative form when at rest shows no distinction between endo- and ectoplasm. The nucleus is prominent and visible in the fresh specimen. In specimens stained by Schaudinn's method (iron alum and Heidenhain's haematoxylin) the nucleus is centrally placed, has a well-marked nuclear membrane, and a distinct central karyosome. Multiplication takes place both by simple fission and by spore formation. As preliminary to the latter the amoeba encysts; then the nucleus divides into two pairs, which subsequently conjugate,

* Authors differ in regard to the proportion of healthy individuals harbouring amoebae. Musgrave and Clegg found them in 4 per cent. of cases not suffering from dysentery or diarrhoea. After the administration of Rochelle salts, Craig found them in 65 per cent. of cases examined ; Vedder in 70 per cent. of Filipinos and 50 per cent. of American soldiers examined in the Philippines ; whilst Hoyt in 1908 found them in 76 per cent. Filipinos and 32 per cent. of men in the American navy.

each pair forming a cell with two nuclei. These in turn again divide, and thus eight young amoebae are formed within the parent cyst. The nuclei retain their distinctive characters, namely, a nuclear ring and a central karyosome.

E. histolytica has a definite refractile homogeneous ecto- and a granular endoplasm. The nucleus cannot be distinguished in the living amoeba. In stained specimens the nucleus is excentrically placed, is poor in chromatin elements, and contains no definite network. This amoeba multiplies by fission, and also by a peculiar process of budding, or sporogony. The young amoebae, each containing part of the chromatin of the original nucleus, are budded off in this way and measure three to seven μ in diameter. They possess a membrane which is extremely dense and refractile.

Schaudinn never found amoebae in bacillary dysentery stools, and was of opinion that the dysentery bacillus killed off amoebae and other intestinal protozoa.

Craig's results, published shortly afterwards, confirmed Schaudinn's statements, especially as regards the morphological differences of the vegetative form of the two types of amoeba. These minute distinctions, though accepted by most German writers, have not received confirmation at the hands of others who have worked at this subject, notably Musgrave and Clegg, in Manila. Recently, however, Walker, working in Manila, has confirmed Schaudinn's statements in every particular and maintains that both *E. coli* and *E. histolytica* are obligatory parasites, the latter being the germ cause of amoebic dysentery.

In 1907 Viereck described a new amoeba, which he considered belonged to the same family as *E. coli*, but was distinguished by having only four nuclei in the cystic stage. Quite independently, Hartmann described the same amoeba in cases of amoebic dysentery occurring in Africa. This species is now known as *E. tetragena*. The nucleus of the vegetative form is intermediate in character between that of *E. coli* and *E. histolytica*. It possesses a well-marked intranuclear ring of chromatin. In the cystic condition the chromatin of the nucleus is separated into two distinct masses, situated at the periphery of the nucleus. In 1908 Werner found the same amoeba in cases of amoebic dysentery at Hamburg, and confirms the distinctions noted by the previous observers. Both he and Hartmann were able to infect cats by rectal injection; the latter also succeeded in causing hepatic abscess in these animals.

Tetragena cysts have been found in normal stools by Hartmann in Africa, and by Kuenen in Java.

Lately, Von Prowazek has described yet another amoeba found in the normal stool of Samoans; this he proposes to call *E. williamsi*. In the cystic stage there are eight nuclei clustered together in the centre of the cyst; these nuclei have a distinct nuclear ring and a well-marked central karyosome.

The experimental production of dysentery in animals, notably cats, has been effected by a number of workers. Kruse and Pasquale were able to infect cats by rectal injections of dysenteric stools, and also with sterile liver pus containing amoebae. Harris produced dysentery and liver abscess in puppies by the same means. Schaudinn produced dysentery in cats by feeding them with faeces containing amoeba cysts only, whilst with faeces containing only amoebae he failed to convey the disease. Sanduchean, by the intravenous injection of sterile liver pus containing amoebae, was able to produce dysenteric symptoms in a dog.

The culture of intestinal amoebae is attended by considerable difficulties. No one has yet succeeded in growing them in artificial media in pure culture and free from extraneous organisms. Apparently, for their propagation in artificial culture, lower forms of life, notably bacteria, must be present. A

symbiotic bacillus is essential. Hence the term " pure mixed culture " employed by Frosch and utilized by Musgrave and Clegg and other workers. The medium generally employed for these " pure mixed cultures " is a weak solution of agar containing salt and beef extract.

Musgrave and Clegg claim to have produced dysenteric symptoms in cats by the injection of amoebae cultivated from water, earth, and vegetables, as well as of amoebae obtained from stools and from ulcers of the human intestine. They produced amoebiasis in a man who swallowed a culture grown from an amoebic dysentery stool, but they experienced considerable difficulty in cultivating amoebae from liver-abscess pus. According to them, harmless saprophytic amoebae may become pathogenic when injected into animals; an observation confirmed in 1909 by Noc, who cultivated what he regards as pathogenic amoebae from the water supply of Saigon, but opposed by Walker in Manila.

Notwithstanding these observations, the view held by Kuenen, Bensen, Vedder and others is gradually gaining ground, namely, that the real intestinal amoebae of man have not yet been cultivated on artificial media, and that those described as being derived from human excreta really belong to another species *Amoeba limax*, a harmless saprophyte extremely common in water and soil, and consequently liable to occur in human excreta. It is pointed out that neither the vegetative form nor the cysts bear any resemblance to the original intestinal amoebae from which they were also said to be descended. The cysts depicted by Musgrave and Clegg had a tough refractile cyst membrane, not at all resembling that of *Amoeba coli*. The same conclusions have been arrived at by Greig and Wells in the course of a recent investigation published in the Scientific Memoirs of the Government of India.

Pathology of Amoebic Dysentery.

As regards the pathology of amoebic dysentery, the monograph of Councilman and Lafleur is still a classic ; while regarding the amoeba as the essential factor, they are careful to express their belief that amoebic lesions, unmodified by the action of intestinal bacteria, are never seen.

According to these authors, to Wooley and Musgrave and many others, the whole or part of the large intestine in amoebic dysentery may be affected by the characteristic lesions. The typical ulcer is recognized by the undermining of its edges, the consequence of lack of resistance on the part of the submucous layer to the invading amoebae.

Kuenen states that, although there are great variations in the appearances of amoebic lesions owing to secondary infections, yet the amoebic form can always be distinguished macroscopically from the bacillary. In the amoebic form, he says, the ulcers are definitely circumscribed, have raised undermined margins, the mucous membrane between the ulcers being apparently normal ; whereas in bacillary dysentery inflammation of the mucosa is diffuse, and there is disintegration of the epithelium, the so-called diphtheritic or croupous form being due to a gradual transformation of the inflamed mucosa into a layer of fibrin containing numerous pus cells.

As noted by Musgrave and Lenz, lesions of the appendix are common in amoebic dysentery, and may give rise to symptoms of appendicitis during life.

Observations on Amoebic Dysentery in Fiji.

During the routine microscopical examination of dysenteric discharges in Fiji, active amoebae were found in nine cases. In two others amoebae were found in the intestinal lesions post-mortem. A summary of these eleven cases is given in Appendix XV. (Charts 10, 11, and 12).

As regards racial incidence, two of the patients were Europeans, two Solomon Islanders, six Indians and only one Fijian.

There appeared to be no marked tendency of the disease to occur at any particular season.

In most instances the amoebae were very numerous in the mucus ; in two instances, however, they were only found at the second microscopical examination of the discharges. As regards their characters, considerable variation was met with. In size they averaged about twenty to thirty μ in diameter. In no instance could the nucleus be clearly distinguished in the fresh specimen. The protoplasm was, in many instances, vacuolated and contained red blood corpuscles. Although in some the granular endo- and clear ecto-plasm were clearly distinguishable, in others this character differentiation was not obvious. In one stool only were amoebic cysts seen. These were large cysts, twenty to thirty μ in diameter, and containing eight nuclei, in this respect resembling *Entamoeba coli*.

Specimens of amoebae from every case were fixed and stained by Schaudinn's method. These specimens have all been critically examined by Dr. C. M. Wenyon, to whom I am greatly indebted for this assistance. All resemble the *Entamoeba histolytica* type of Schaudinn in certain features, namely, the excentric position of the nucleus and the absence of a definite nuclear ring of chromatin.

Cultures on Conradi-Drigalski medium from the freshly-passed mucus were made in every case, but no organisms at all resembling the dysentery bacillus were ever isolated.

The clinical histories, as far as I could procure them, of my cases of amoebic dysentery, differed widely from those of bacillary dysentery. Dysenteric symptoms had been present for a considerable time, and in the case of the two Europeans, whose statements could be relied on, there had been several relapses before admission to hospital. Hepatic symptoms were present in four instances. In one there was definite inflammation and enlargement of the liver, a marked rise in temperature and a leucocytosis. One had a large abscess of the liver, concomitant with an attack of amoebic dysentery, whilst two others had tenderness and enlargement of the liver without a corresponding rise in temperature. In a fifth instance, no hepatic symptoms were recognized during life, but post-mortem, together with ulceration of the gut, numerous small liver abscesses were found.

In the remaining cases not much assistance could be obtained for the differential diagnosis of the two varieties of dysentery from a physical examination of the patient.

In six out of ten cases of amoebic dysentery temperature was above normal* (charts illustrating different cases of amoebic dysentery are given in Appendix XV. and Charts 10, 11 and 12). There was the same abdominal pain and tenderness, located especially in the region of the caecum and of the sigmoid flexure, and the thickened, contracted coils of the latter could easily be felt through the abdominal wall.

Eight of the cases were acutely ill. In one instance only was the attack unaccompanied by abdominal tenderness or tenesmus.

One case ran a very rapid course, death taking place after eleven days in hospital. He was passing blood continuously during that time. No amoebae were found in the discharges during life, but, post-mortem, amoebae were found in sections of the ulcerated gut and in the liver. Cases resembling this one in acuteness and in the absence of amoebae in the stools have been quoted by Kuenen.

* In one case as high as 102 deg. F.

No agglutination reaction with cultures of Flexner's or Shiga's bacilli in a dilution of 1 : 50 was obtained in any of my cases of amoebic dysentery.

In the differential diagnosis of the bacillary and amoebic forms no definite assistance could be obtained from the macroscopic examination of the stools. Sometimes the intestinal discharges consisted of green-coloured faecal matter, over which clots of pink mucus were scattered, identical with the stools of bacillary cases. At other times the faecal matter had the most offensive odour, and was intimately mixed with slimy mucus and quantities of blood, the general appearance resembling strawberry jam.

A method of treatment different from that employed for bacillary dysentery was adopted in amoebic dysentery. All, in whom a diagnosis of amoebic dysentery was made during life, were given powdered ipecacuanha, some in gradually increasing doses from five to thirty grains, others in decreasing doses from thirty to five grains on successive nights, according to Manson's method.

When the vomiting was excessive, coating the ipecacuanha pills with salol, or enclosing them in keratin capsules, as suggested by Rogers, proved successful.

The rapid improvement which followed the administration of ipecacuanha left no doubt in my mind as to its specificity for the amoeba. The beneficial action of the drug was well exemplified in cases of hepatitis, in which an immediate reduction of temperature with a disappearance of symptoms soon took place. I think it can be fairly claimed that not only does it avert the development of hepatic abscess, as suggested by Rogers, but actually exerts some specific action on the parasite.

I have quoted in Appendix XV. (6) the case of an Indian who was operated on for a large abscess in the right lobe of the liver, and amoebae were found in the pus. The abscess cavity became secondarily infected. The stools contained blood and mucus and many amoebae. Subsequently, another abscess formed in the left lobe of the liver and a pneumo-thorax developed on the right side. On full doses of ipecacuanha he completely recovered after a protracted illness, and was able to return to India.

One European had been dosed with sulphates for three months without any permanent improvement. On full doses of ipecacuanha he was discharged, after three weeks' stay in hospital, passing normal stools.

The other European, on failing to take the ipecacuanha pills with which he was provided, relapsed immediately.

A post-mortem was secured on a Solomon Islander, Appendix XV. (4), after apparent recovery from an attack of amoebic dysentery. Death was due to pulmonary tuberculosis. Ipecacuanha had been given in full doses. He died eleven days after the stools had become normal. The condition, found post-mortem, in the large intestine may, perhaps, explain the action of ipecacuanha on the amoebae. Numerous scars of healed ulcers were found in the large intestine ; the whole surface was covered by a greenish-yellow diphtheritic membrane, which could be removed with ease, leaving a granulating surface behind. In microscopical sections amoebae, many of them dead and undergoing degenerating changes, were found in a dense layer of fibrin and round cells surmounting the epithelial layer of the mucous membrane (Pl. V.).

No amoebae were ever found in the stools after the administration of ipecacuanha. Intestinal flagellates which had been present also disappeared.

Post-mortems were secured in three cases of amoebic dysentery. One specially deserves mention. This was a Fijian who died suddenly from intestinal hæmorrhage (Appendix XVI.). The large intestine, from sigmoid to anus, was studded with large, deep ulcers of irregular shape ; the adjacent mucous membrane appeared healthy. In microscopical sections of the ulcers, amoebae were seen in numbers invading the submucous layer at the edge of the ulcer. This layer

P. H. Bahr del., 1911.

PLATE V.

Microscopic section of the transverse colon from a case of amoebic dysentery which had been treated for a lengthy period with ipecacuanha. The patient died subsequently from widespread tuberculosis after all clinical symptoms of dysentery had disappeared. A false membrane is seen covering the epithelial layer, which is only partially disintegrated. In this membrane numbers of amoebae undergoing disintegration are lying.

To face p. 34.

was swollen and oedematous, the blood-vessels were dilated, but there were no hæmorrhages. The amoebae were surrounded by fibroblasts and round-cell infiltration. Amoebae were seen also in the adjacent intact mucous membrane, lying amongst the interstitial tissue between the crypts and actually in the lumen of the crypts themselves. Many of the amoebae, in sections stained with eosin, were seen to contain red blood corpuscles. The structure of the nuclei was similar to that already described (Pl. VI.).

In Appendix XV. (10) is given a summary of a post-mortem on a Solomon Islander who died after a very acute attack. No amoebae were found in the stools. The case was regarded as of bacillary origin, and a number of injections of anti-serum were given without the least benefit. Ulcers were found throughout the whole of the large intestines ; they were irregular in shape, and ramified in every direction. The rest of the mucous membrane appeared healthy. In microscopical sections the circular muscular fibres were seen to be laid bare, except for a thin layer of necrotic tissue, in which a few vacuolated amoebae were found. There was no evidence to show that the amoebae alone could be held responsible for damage of such an extensive nature. The assumption that a secondary infection with intestinal bacteria had probably supervened, and that in the subsequent destruction of the mucous membrane all the amoebae had been swept away, seems justifiable. In this case the liver weighed sixty ounces, and throughout its substance many small abscesses were scattered, none larger than a small pea. In microscopical sections of the liver stained by iron haematoxylin, amoebae in small numbers were found in the centre of the necrotic tissue. There was marked absence of reaction on the part of the surrounding tissue, the liver cells appearing to have undergone a gradual necrosis. These amoebae, as far as could be ascertained from specimens in section, bore the morphological characters of *E. histolytica*. No amoebae were found in the healthy liver tissue.

A summary of the third post-mortem, to which reference has been made, is given in Appendix XV. (4).

A series of normal Fijian stools were examined for the presence of amoebae. The specimens were obtained from several native schools, where the boys were under supervision and an accurate collection of stools possible. All were collected from young individuals of from 7 to 18 years of age. Out of 154 such examinations, vegetative amoebae and amoebic cysts were found in 9·2 per cent.

In the majority of instances these cysts resembled those of *Entamoeba coli*, and contained eight nuclei ; they measured 20 to 30 μ in diameter. Smaller cysts were commonly present ; these contained only four nuclei. The stained specimens agree with the published descriptions of the cysts of *Entamoeba tetragena*. The cysts with eight closely-aggregated nuclei, with well-marked central karyosomes, correspond to the figures of *Entamoeba williamsi* of Prowazek.

The vegetative forms, in stained specimens, had a definite nuclear ring, and corresponded with the descriptions of *Entamoeba coli*.

The percentage infection with amoebae was found to vary in different localities. In one school, on a remote island, it was as high as 23 per cent., while in another, close to Suva, it was as low as 2 per cent.

On the whole the percentage infection with *E. coli* in Fijians is much lower than that given by the American workers for Manila. This can be explained, perhaps, by the diet of the two peoples, which, amongst the Fijians, consists almost entirely of cooked farinaceous food, raw vegetable material being rarely consumed.

Intestinal flagellates, probably *Trichomonas intestinalis*, were found in 31 per

cent. of the stools; a great many small round and oval cysts 7 to 15 μ in diameter, possibly the cystic stage of this flagellate, were commonly encountered.

A few words may be added about certain other conditions which, from the characters of the stools passed during life, were liable to be confounded with amoebic dysentery.

An Indian woman, passing dysenteric-like stools containing blood and mucus, was found post-mortem to have a new growth of the right suprarenal capsule, which had extended into the hepatic flexure and caused ulceration.

Stools containing slimy mucus are passed in tubercular ulceration of the intestine, and resemble in their macroscopic appearances those of amoebic dysentery. One such case, in a Solomon Islander, was met with: post-mortem. In addition to extensive pulmonary tuberculosis, there was tubercular ulceration of the small intestine.

A post-mortem on an Indian child who had passed stools containing quantities of blood and slime revealed a phagedaenic condition of the vagina and rectum, the latter being occupied by green sloughs. The large intestine was dotted with large, deep undermined ulcers of a peculiar green colour; the caecum was the seat of a large ulcer, which had perforated the appendix. In microscopical sections great numbers of micrococci, but no amoebae, were found. The condition resembled the disease known as gangrenous rectitis, described by Dr. Daniels as occurring in Fiji.

REFERENCES. AMOEBIC DYSENTERY.

Bensen.—Archiv für Schiffs- und Tropenhyg., Vol. XII., pt. 20, 1908, p. 661.

Councilman & Lafleur.—Johns Hopkins Hospital Reports, II., 1891.

Creig & Wells.—Scientific Memoirs, Government of India (New Series), No. 47, 1911.

Dutcher.—Journal of the American Medical Association, 1903, No. 8, p. 480.

Frosch.—Centralblatt für Bakt., XXI., 1897, p. 926.

Harris.—Journal of the American Medical Association, 1903, No. 8, p. 476.

Jürgens.—Ztsch. für Exp. Path., IV., 1908, p. 769.

Kartulis.—Virchow's Archiv CV., Vol. III., 1886, p. 521.

Kuenen.—Janus, 1909, XIV., No. 7, p. 542 & No. 8, p. 627.

,, —Archiv für Schiffs- und Tropenhyg., Vol. XIII., pt. 7, 1909, p. 7.

Lenz.—Mense's Archiv., XIV., 1910, p. 352.

Musgrave & Clegg—Bureau of Government Laboratories, Manila, 1904, No. 18.

Musgrave & Clegg.—Philippine Journal of Science, 1906, Vol. I., No. 9.

Noc.—Annales de l'Institut Pasteur, XXIV., 1909, p. 177.

Von Prowazek.—Archiv für Protist., XXII., 1911, pt. 3, p. 385.

Rogers.—B. M. J., I., 1903, p. 1,315.

Gaudcheau ~~Sandchehar~~.—Journal of Tropical Medicine, 1906, p. 52.

Schaudinn.—Arb. a.d. Kais. Gesundt., XXIX., pt. 3, 1903, p. 547.

Strong.—Bureau of Government Laboratories, Manila, 1905, No. 32.

Vedder.—Journal of American Medical Association, March, 1906, II., p. 870.

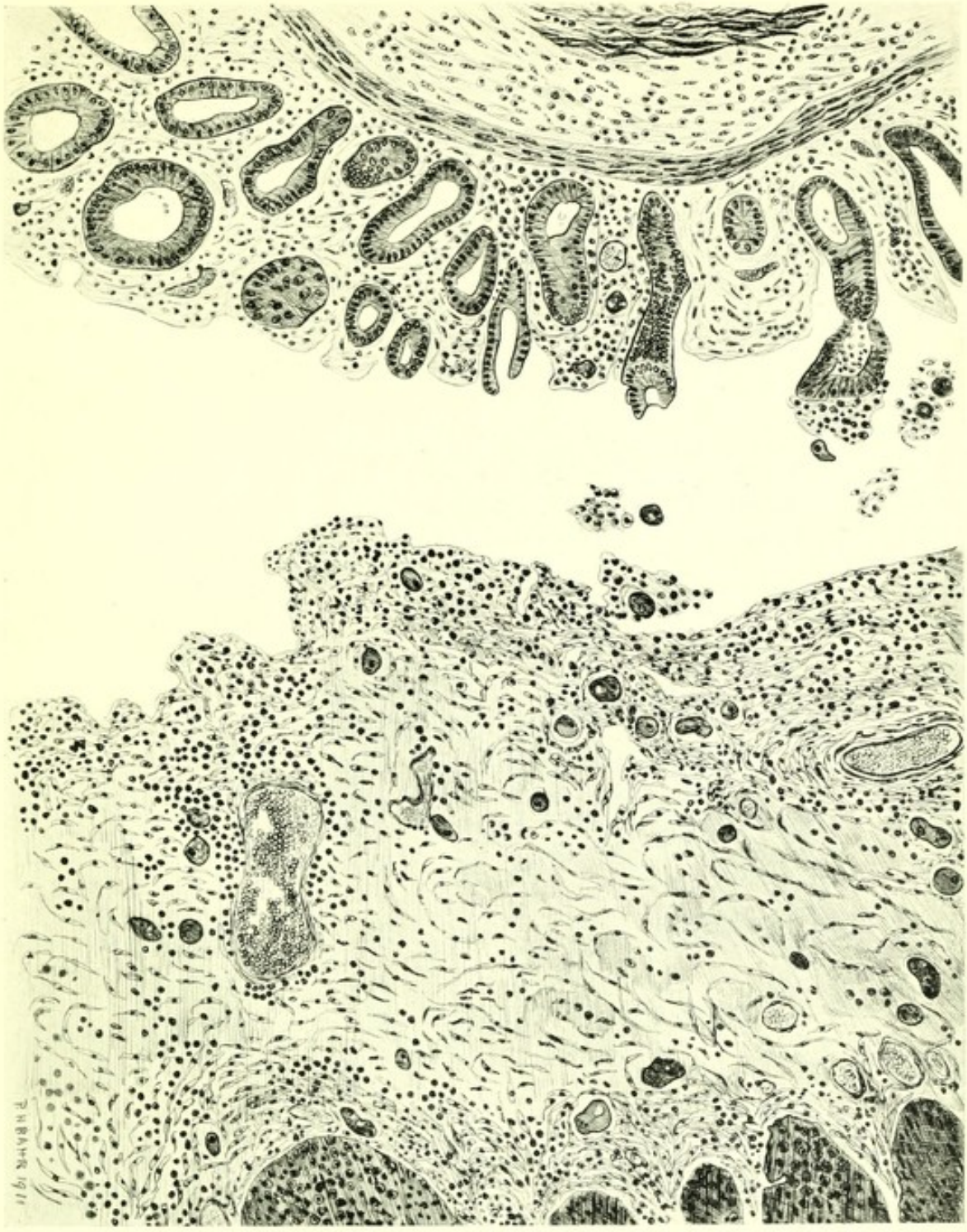
,, —Journal of Tropical Medicine, June, 1907, p. 190.

Walker.—Phil. Journ. Sci., Vol. VI., No. 4, October, 1911, p. 259.

Werner.—Archiv für Schiffs- und Tropenhyg., Vol. XII., pt. 2, 1908, p. 7.

Wooley & Musgrave.—Bureau of Govt. Laboratories, Manila, 1905, No. 32, III.

Wenyon.—Archiv für Protist., Festband Rich. Hertwig, p. 169.



P. H. Bahr del., 1911.

PLATE VI.

Transverse section through the base of an amoebic ulcer. Amoebae are lying in the submucous layer and invading the interstitial tissue between the crypts, where the section passes through an overhanging fold of apparently healthy mucous membrane.

To face p. 36.

APPENDIX I.

TABLE showing number of patients admitted to the Colonial Hospital, Suva, suffering from dysentery. The maximum incidence of the disease corresponds with that of the highest temperature and of the largest rainfall.

	1908.				1909.				1910.			
	Number admitted	Max. mean temp.	Min. mean temp.	Rainfall.	Number admitted	Max. mean temp.	Min. mean temp.	Rainfall.	Number admitted	Max. mean temp.	Min. mean temp.	Rainfall.
				Ins.				Ins.				Ins.
January ..	39	85·0°F.	73·9°F.	5·03	68	87·5°F.	72·6°F.	11·63	69	87·9	73·3	6·916
February ..	23	86·8°F.	73·8°F.	12·48	51	88·6°F.	73·0°F.	15·32	39	88·7	74·4	11·570
March	24	86·0°F.	73·7°F.	12·0	42	87·9°F.	73·6°F.	11·76	16	87·8	73·7	12·043
April	23	83·5°F.	73·6°F.	13·34	44	84·7°F.	71·4°F.	7·45	31*	85·8	73·9	3·871
May	25	81·7°F.	71·4°F.	20·32	32	82·2°F.	70·8°F.	8·33	49	83·1	71·2	14·123
June	17	78·8°F.	68·9°F.	3·3	20	80·5°F.	70·7°F.	2·13	40	79·1	69·1	9·894
July	14	78·0°F.	67·0°F.	6·7	11	79·5°F.	68·0°F.	2·63	36	81·8	68·6	3·134
August ..	8	78·4°F.	68·6°F.	9·3	7	78·8°F.	67·4°F.	4·95	18	78·7	67·5	8·959
September .	8	78·8°F.	64·5°F.	3·5	2	81·4°F.	69·9°F.	4·25	20	78·9	69·1	12·914
October ..	14	79·3°F.	67·2°F.	4·9	21	82·3°F.	69·7°F.	9·16	11	81·2	70·6	19·328
November ..	9	82·0°F.	71·9°F.	5·2	19	83·4°F.	72·3°F.	21·74	12	83·6	72·0	12·725
December ..	31	86·8°F.	72·0°F.	8·6	11	86·3°F.	72·7°F.	12·902	25	86·6	74·4	9·586
Total ..	235			104·5	328			112·25	366			125·063

* This rise took place immediately following the hurricane on March 24th. A great plague of house-flies also followed this event.

APPENDIX II.

Showing the Mortality from Dysentery at the Colonial Hospital, Suva. Compiled from the Statistical Returns of the Last Six Years.

	Europeans.	Fijians.	Solomon Islanders.	Indians.	Miscellaneous.	Total.
1904	12	38	6	94	○	153
Deaths	1	2	1	4	3	8
	Death rate—5·2 per cent.					
1905	5	32	7	78	1	123
Deaths	1	2	1	7	1	12
	Death rate—9·7 per cent.					
1906	19	43	15	70	2	149
Deaths	4	4	○	10	1	19
	Death rate—12·8 per cent.					
1907	8	26	11	78	1	124
Deaths	○	3	3	7	○	13
	Death rate—10·4 per cent.					
1908	25	67	50	99	5	253
Deaths	1	5	1	5	○	12
	Death rate—4·7 per cent.					
1909	27	89	28	136	5	285
Deaths	3	7	8	8	○	26
	Death rate—9·1 per cent.					

The returns given above are taken from the Government Blue Book statistics and show a somewhat low and variable mortality. It must be remembered that in these returns no distinction is made between amoebic and bacillary dysentery, and also that many cases with diarrhoea, admitted for observation, but who never developed dysentery, are included under this heading. It must be borne in mind, too, that all these patients are treated under modern conditions and under trained nurses. Amongst the native Fijians the mortality is much higher; in 1908 an epidemic, of which there are accurate records, occurred on the Rewa river. In three months there were 38 cases with 11 deaths—a mortality of 25·7 per cent. At the same time there were 139 cases amongst the indentured Indians in the plantation hospital with a mortality of 2·8 per cent. These Indians are nursed under modern conditions and are admitted immediately they show signs of the disease.

APPENDIX II.—*continued.*

Returns from Provincial Hospitals (Sugar Districts) in Fiji, showing the Mortality and Seasonal Incidence.—Nearly all Patients are Indians working under Indenture on Plantations.

Locality.	Greatest Seasonal incidence.	Number of cases.	Deaths.	Mortality per cent.
1908—				
Lautoka ..	May, June, July. ..	40	1	2·5
Rewa	July, August... ..	139	4	2·8
Navua ..	December.	61	5	8·1
Ba & Tavua ..	August, September. ..	74	1	1·3
1909—				
Lautoka ..	March, April. ..	45	1	2·2
Rewa (Indians)..	February, March, April, May.	236	8	2·5
,, (Fijians) ..	January.	65	6	9·2
Navua ..	February, March, ..	100	4	4
Ba & Tavua ..	July.	71	○	○

APPENDIX II.—*continued.*

Returns from Provincial Hospitals showing Death-rate amongst the Native Fijians.

	District.	No. of Cases.	Deaths.	Mortality.
1905—	Ba	7	1	
	Bua	1	○	
	Cakondrove	6	○	
	Kadavu	7	○	
	Macuata	1	○	
	Nadroga	7	3	
	Ra	5	3	
	Rewa	7	○	
		41	7	17 per cent.
1906—	Ba	11	1	
	Cakondrove	3	2	
	Kadavu	2	○	
	Macuata	1	○	
	Nadroga	1	○	
	Ra	7	1	
	Rewa	4	1	
		29	5	17 per cent.
1907—	Ba	5	○	
	Cakondrove	2	○	
	Kadavu	15	○	
	Macuata	13	1	
	Nadroga	3	2	
	Ra	12	2	
	Rewa	9	○	
		59	5	8 per cent.
1908—	Ba	4	1	
	Bua	1	○	
	Cakondrove	3	1	
	Kadavu	10	○	
	Macuata	4	○	
	Nadroga	15	1	
	Ra	3	○	
	Rewa	31	2	
		71	5	7 per cent.
1909—	Ba	2	○	
	Cakondrove	28	3	
	Kadavu	56	16	
	Macuata	22	1	
	Nadroga	5	1	
	Ra	6	4	
	Rewa	65	6	
		191	31	16 per cent.

APPENDIX III.

Dysentery Bacilli isolated in Fiji.

Reactions after twenty-four hours' incubation at 37° C. in litmus peptone water to which 1% of sugars had been added.

Type and No. oftimes isolated.	Mannite	Lactose	Saccharose	Maltose	Dulcite	Dextrose	Indol (peptone water)	Milk	Agglutination with Lister Inst. Anti-serum	Clinical form of case	Nationality of cases
A. Shiga, Type I. (Original dysentery bacillus) 12 times, twice P.M. 8 times from stools, twice from intestines of flies caught in dysentery ward	—	—	—	—	—	+	Nil.	Acid at first, returned to alkaline generally on 2nd–4th day, once on 6th	Dilution 1 : 50 + (1 hour)	2 classified as mild 5 classified as acute 3 classified as toxic	2 Europeans 5 Indians 2 Fijians 1 Solomon Islander
B. Once from stool	—	—	+	+	—	+	Nil.	Acid at first, returned to alkaline on the 4th day	Dilution 1 : 50 + (1 hour)	Classified as acute	Indian
C. Once from stool Shiga's bacillus also isolated from same stool	—	+	+	+	—	+	+	Permanent acidity; slight clot	Dilution 1 : 50 — (1 hour)	Classified as toxic	Fijian
D. Shiga type II. (Ohno type G) 5 times from stools	+	—	—	—	—	+	Variable + —	Acid at first, returned to alkaline 3rd & 4th day, once on 7th	Dilution 1 : 50 + (1 hour)	1 classified as acute 4 classified as mild	2 Indians 2 Fijians 1 Solomon Islander
E. 5 times from stools	+	—	—	+	—	+	Variable + —	Acid at first, returned to alkaline 2nd–4th day; one remained permanently acid.	Dilution 1 : 50 + (1 hour)	1 classified as mild 2 classified as acute 2 classified as toxic	1 Indian 3 Fijians 1 Solomon Islander
F. Once from stools	+	+	—	—	—	+	+	Acid returned to alkaline after 7 days	Dilution 1 : 50 + (1 hour)	Classified as acute	Indian
G. (Ohno variety M) three times from stools	+	+	—	+	—	+	Variable + —	Acid returned to alkaline on the 4th day	Dilution 1 : 50 + (1 hour)	1 classified as acute 2 classified as mild	2 Fijians. 1 Solomon Islander

APPENDIX IV.

Showing the varying reactions of dysentery bacilli of the different types mentioned in Appendix III. on subculture. One bacillus from each group has been selected. 1% of the sugars in 1% peptone water was used throughout. Observations extend over a period of ten days at 37°C. on each occasion.

Type of dysentery bacillus.	Mannite	Lactose.	Saccharose.	Maltose.	Dulcite.	Dextrose.	Indol.	Milk.	Agglutination with Lister Institute Anti-serum.
A. 1 Reaction when first isolated; incubated for 24 hours at 37°C.	—	—	—	—	—	+	—	Acid. Alk. 4th day	1 : 50+
2 Reaction after 7 months' subculture; incubated for 24 hours ,,	—	—	—	—	—	+	—	Acid. Alk. 4th day	
,, ,, 7 ,, ,, ,, ,, 10 days ,,	—	—	—	—	—	+	—	Alkaline	
3 Reaction after 8 months' subculture; incubated for 24 hours ,,	—	—	—	—	—	+	—	Acid. Alk. 4th day	
,, ,, 8 ,, ,, ,, ,, 10 days ,,	—	—	—	—	—	+	—	Alkaline	
4 Reaction after 10 months' subculture; incubated for 24 hours ,,	—	—	—	—	—	+	—	Acid. Alk. 4th day	1 : 50+
,, ,, 10 ,, ,, ,, ,, 10 days ,,	—	—	—	—	—	+	—	Alkaline	
B. 1 Reaction when first isolated; incubated for 24 hours ,,	—	—	+	+	—	+	—	Acid. Alk. 4th day	1 : 50+
2 Reaction after 6 months' subculture; incubated for 24 hours ,,	—	—	—	—	—	+	—	Acid. Alk. 4th day	
,, ,, 6 ,, ,, ,, ,, 10 days ,,	—	—	+	—	—	+	—	Alkaline	
3 Reaction after 7 months' subculture; incubated for 24 hours ,,	—	—	—	—	—	+	—	Acid. Alk. 4th day	
,, ,, 7 ,, ,, ,, ,, 10 days ,,	—	—	—	—	—	+	—	Alkaline	
4 Reaction after 9 months' subculture; incubated for 24 hours ,,	—	—	—	—	—	+	—	Acid. Alk. 4th day	1 : 50+
,, ,, 9 ,, ,, ,, ,, 10 days ,,	—	—	—	—	—	+	—	Alkaline	
D. 1 Reaction when first isolated; incubated for 24 hours ,,	+	—	—	—	—	+	+	Acid. Alk. 4th day	1 : 50+
2 Reaction after 7 months' subculture; incubated for 24 hours ,,	+	—	—	—	—	+	+	Acid. Alk. 4th day	
,, ,, 7 ,, ,, ,, ,, 10 days ,,	+	—	—	—	—	+	+	Alkaline	
3 Reaction after 8 months' subculture; incubated for 24 hours ,,	+	—	—	—	—	+	±	Acid. Alk. 4th day	
,, ,, 8 ,, ,, ,, ,, 10 days ,,	+	—	—	—	—	+	+	Alkaline	
4 Reaction after 9 months' subculture; incubated for 24 hours ,,	+	—	—	—	—	+	±	Acid. Alk. 4th day	1 : 50+
,, ,, 9 ,, ,, ,, ,, 10 days ,,	+	—	—	—	—	+	+	Alkaline	

Type of dysentery bacillus.		Mannite	Lactose.	Saccharose.	Maltose.	Dulcite.	Dextrose.	Indol.	Milk.	Agglutination with Lister Institute Anti-serum.
E. 1	Reaction when first isolated; incubated for 24 hours at 37°C.	+	—	—	+	—	+	+	Remained acid	1 : 50+
2	Reaction after 6 months' subculture; incubated for 24 hours ,,	+	—	—	+	—	+	+	Acid	
	,, ,, 6 ,, ,, ,, ,, 10 days ,,	+	—	—	+	—	+	+	,,	
3	Reaction after 7 months' subculture; incubated for 24 hours ,,	+	—	—	+	—	+	+	,,	
	,, ,, 7 ,, ,, ,, ,, 10 days ,,	+	—	—	+	—	+	+	,,	
4	Reaction after 9 months' subculture; incubated for 24 hours ,,	+	—	—	+	—	+	+	,,	
	,, ,, 9 ,, ,, ,, ,, 10 days ,,	+	—	—	+	—	+	+	,,	1 : 50+
F. 1	Reaction when first isolated; incubated for 24 hours ,,	+	+	—	—	—	+	+	Acid. Alk. after 7 days	1 : 50+
2	Reaction after 4 months' subculture; incubated for 24 hours ,,	+	+	—	—	—	+	+	Acid. Alk. 4th day	
	,, ,, 4 ,, ,, ,, ,, 10 days ,,	+	+	—	—	—	+	+	Alkaline	
3	Reaction after 5 months' subculture: incubated for 24 hours ,,	+	+	—	—	—	+	+	Acid. Alk. 4th day	
	,, ,, 5 ,, ,, ,, 10 days ,,	+	+	—	—	—	+	+	Alkaline	
4	Reaction after 7 months' subculture; incubated for 24 hours ,,	+	+	—	—	—	+	+	Acid. Alk. 4th day	1 : 50+
	,, ,, 7 ,, ,, ,, ,, 10 days ,,	+	+	—	—	—	+	+	Alkaline	
G. 1	Reaction when first isolated; incubated for 24 hours ,,	+	+	—	+	—	+	+	Acid. Alk. 4th day	1 : 50+
2	Reaction after 3 months' subculture; incubated for 24 hours ,,	+	+	—	—	—	+	+	Acid. Alk. 4th day	
	,, ,, 3 ,, ,, ,, ,, 10 days ,,	+	+	—	—	—	+	+	Alkaline	
3	Reaction after 4 months' subculture; incubated for 24 hours ,,	+	—	—	+	—	+	+	Acid. Alk. 4th day	
	,, ,, 4 ,, ,, ,, ,, 10 days ,,	+	—	—	+	—	+	+	Alkaline	
4	Reaction after 6 months' subculture; incubated for 24 hours ,,	+	—	—	+	—	+	+	Acid. Alk. 4th day	1 : 50+
	,, ,, 6 ,, ,, ,, ,, 10 days ,,	+	—	—	+	—	+	+	Alkaline	

There are two variants; bacillus type B has lost its power of fermenting saccharose and maltose, while bacillus of type G has lost its power of fermenting lactose, and temporarily lost its power of fermenting saccharose.

APPEN-

TABLE showing reaction of patients' sera, at different periods of the disease from other cases but belonging to other types, according to their microscopic method of agglutination used. Time limit allowed in

Type to which bacillus isolated from patient's stool belongs, and form of dysentery from which patient was suffering.	Day of illness on which the tests were made.	Agglutination with patient's own serum.
A.—Clinically acute case	11th day	Dilution 1 : 200 +
A.—Clinically acute case	8th day	Dilution 1 : 200 +
A.—Clinically mild case	5th day 9th day	Dilution 1 : 100 — Dilution 1 : 50 —
A.—Clinically acute case	5th day 8th day	Dilution 1 : 100 — Dilution 1 : 100 +
A.—Clinically acute case	6th day	Dilution 1 : 500 +
A.—Clinically fulminant or toxic case ..	5th day 12th day	Dilution 1 : 200 + Dilution 1 : 500 +
A.—Clinically mild case	4th day	Dilution 1 : 100 +
B.—Clinically acute case	7th day	Dilution 1 : 200 +
C.—Clinically fulminant or toxic case ..	5th day	Dilution 1 : 200 +
D.—Clinically mild case	7th day	Dilution 1 : 100 +
E.—Clinically acute case	8th day	Dilution 1 : 500 +
E.—Clinically acute case	9th day	Dilution 1 : 500 +
E.—Clinically acute case	11th day	Dilution 1 : 100 +
E.—Clinically mild case	4th day	..
F.—Clinically mild case	5th day	..
G.—Clinically mild case	4th day	Dilution 1 : 50 —
G.—Clinically acute case	4th day	Dilution 1 : 100 —

In the case of natives it is difficult to ascertain the exact date of the onset of
inspected and are sent to hospital

DIX V.

towards bacilli isolated from their own stools, and also towards bacilli isolated fermentation reactions. Only 24-hour cultures in peptone broth and each case being one hour.

Agglutination tests of sera of other patients suffering from bacillary dysentery.	Reaction of patient's serum towards dysentery bacilli obtained from other sources and belonging to other types.	Day of illness on which these tests were made.
Of 26 other patients— in 13 positive reaction in dilution 1 : 100 in 9 positive reaction in dilution 1 : 200 in 4 negative reaction in dilution 1 : 100	With bacillus type A 1 : 200 + ,, ,, ,, E 1 : 200 +	5th day.
Of 4 other patients— in 3 positive reaction in dilution 1 : 100 in 1 positive reaction in dilution 1 : 200	With bacillus type A 1 : 100 +	4th day.
Of 10 other patients— in 6 positive reaction in dilution 1 : 100 in 2 positive reaction in dilution 1 : 200 in 2 negative reaction in dilution 1 : 100	With bacillus type E 1 : 50 — ,, ,, ,, A 1 : 50 — ,, ,, ,, E 1 : 50 —	5th day. 8th day.
Of 5 other patients— in 4 positive reaction in dilution 1 : 100 in 1 negative reaction in dilution 1 : 100	With bacillus type A 1 : 100 + ,, ,, ,, E 1 : 100 —	5th day.
Of 16 other patients— in 5 positive reaction in dilution 1 : 100 in 4 positive reaction in dilution 1 : 200 in 7 negative reaction in dilution 1 : 100	With bacillus type A 1 : 100 + ,, ,, ,, D 1 : 500 +	6th day.
Of 1 other patient— dilution 1 : 100, negative reaction	With bacillus type A 1 : 200 + ,, ,, ,, D 1 : 200 +	5th day.
Of 1 other patient— dilution 1 : 100, positive reaction	With bacillus type A 1 : 100 + ,, ,, ,, E 1 : 100 —	4th day.
..	With bacillus type A 1 : 200 + ,, ,, ,, E 1 : 200 —	7th day.
..	With bacillus type A 1 : 200 + ,, ,, ,, D 1 : 200 +	5th day.
Of 10 other patients— in 3 positive reaction in dilution 1 : 100 in 1 positive reaction in dilution 1 : 500 in 6 negative reaction in dilution 1 : 100	With bacillus type A 1 : 100 — ,, ,, ,, D 1 : 100 +	7th day.
Of 7 other patients— in 2 positive reaction in dilution 1 : 50 in 1 positive reaction in dilution 1 : 100 in 4 negative reaction in dilution 1 : 100	With bacillus type A 1 : 200 — ,, ,, ,, A 1 : 200 + ,, ,, ,, E 1 : 200 +	3rd day. 8th day.
Of 2 other patients— in 1 positive reaction in dilution 1 : 50 in 1 negative reaction in dilution 1 : 50	With bacillus type A 1 : 100 + ,, ,, ,, E 1 : 50 +	7th day.
Of 2 other patients— in 1 positive reaction in dilution 1 : 50 in 1 negative reaction in dilution 1 : 100	With bacillus type A 1 : 100 + ,, ,, ,, G 1 : 100 —	6th day.
Of 2 other patients— in 1 positive reaction in dilution 1 : 100 in 1 negative reaction in dilution 1 : 100	With bacillus type A 1 : 100 — ,, ,, ,, D 1 : 100 —	4th day.
Of 5 other patients— in 3 positive reaction in dilution 1 : 100 in 2 negative reaction in dilution 1 : 100	With bacillus type A 1 : 100 — ,, ,, ,, D 1 : 100 —	5th day.
Of 3 other patients— in 1 positive reaction in dilution 1 : 50 in 2 negative reaction in dilution 1 : 50	With bacillus type A 1 : 50 — ,, ,, ,, E 1 : 50 —	5th day.
..	With bacillus type A 1 : 100 + ,, ,, ,, F 1 : 100 +.	4th day.

dysentery. Most of the cases, however, were of prisoners whose stools are daily immediately they show signs of this disease.

APPENDIX VI.

Agglutination Tests with sera of Patients under observation and suffering from symptoms of Bacillary Dysentery.

Sera of 112 cases were tested. A positive agglutination with Shiga's bacillus in dilutions of from 1 : 100 to 1 : 200 were obtained in 83, or 74·1 per cent., of the cases. Only twenty-four hour cultures of the bacillus in peptone broth were used. Observations were made in hanging drops in welled slides. The time assigned for the test was one hour in each case.

A series of thirty-two cases was tested with cultures of Shiga's bacillus and Flexner's bacillus (fermenting mannite, dextrose and maltose, and belonging to type E, *vide* Appendix III.); these were obtained from Professor R. T. Hewlett and Dr. F. W. Twort,* and brought out to Fiji; they had been subcultured for several years.

Shiga maintains that cultures of bacilli are only suitable for agglutination tests immediately after they have been isolated. In view of this statement, not much reliance was placed upon the results obtained with bacilli subcultured over a long period.

These cases, from which the sera were obtained, have been subdivided according to their clinical severity.

There were 14 *mild or catarrhal cases.* Of these—

9 agglutinated Shiga's bacillus (type A) in a dilution of 1 : 200.
4 agglutinated Flexner's bacillus (type E) in a dilution of 1 : 200.
One case failed to agglutinate either.

There were 14 *acute or ulcerative cases.* Of these—

10 agglutinated Shiga's bacillus (type A) in a dilution of 1 : 200.
2 agglutinated Flexner's bacillus (type E) in a dilution of 1 : 200.
4 cases failed to agglutinate either.
Only one case agglutinated both types of bacilli.

There were 4 *toxic or fulminant cases.*

All agglutinated Shiga's bacillus (type A) in a dilution of 1 : 200.
One agglutinated Flexner's bacillus (type E) as well, in a dilution of 1 : 200.

A series of 80 cases was tested with broth cultures of bacilli freshly isolated from dysenteric stools and from the intestines, post-mortem. These cases are again subdivided according to their clinical severity.

42 *were mild or catarrhal cases.* Of these—

28 agglutinated Shiga's bacillus (type A).
16 up to a dilution of 1 : 100.
12 up to a dilution of 1 : 200.

Flexner's bacillus, belonging to two varieties, was used, namely, types D and E, *vide* Appendix III.

18 cases were tested with cultures of bacillus type D.
10 agglutinated the bacillus.
8 in a dilution of 1 : 100.
1 in a dilution of 1 : 200.
1 in a dilution of 1 : 500.
9 cases agglutinated Shiga's bacillus (type A) as well as bacillus D.

* The original culture from which this bacillus was obtained was type 4 of Firth, and gave the same reactions, as stated above, when first isolated.

12 cases were tested with cultures of bacillus type E.
9 agglutinated the bacillus.
8 in a dilution of 1 : 50-100.
1 in a dilution of 1 : 500.
Only one case of this series failed to agglutinate a dysentery bacillus.

35 *were acute or ulcerative cases.*
29 agglutinated Shiga's bacillus (type A).
16 in a dilution of 1 : 100.
13 in a dilution of 1 : 200.
1 in a dilution of 1 : 500.

7 cases were tested with culture of bacillus type D.
5 agglutinated the bacillus.
2 in a dilution of 1 : 100.
1 in a dilution of 1 : 200.
2 in a dilution of 1 : 500.
3 agglutinated Shiga's bacillus (type A) as well as type D.

16 cases were tested with cultures of bacillus type E.
7 agglutinated the bacillus.
5 in a dilution of 1 : 50-100.
2 in a dilution of 1 : 500.
6 agglutinated Shiga's bacillus (type A) as well as bacillus E.

4 cases were tested with cultures of bacillus type F.
2 agglutinated the bacillus in a dilution of 1 : 100.
2 cases agglutinated Shiga's bacillus as well as type F.
7 cases of this series failed to agglutinate a dysentery bacillus.

3 *were toxic or fulminant cases.*
All agglutinated cultures of Shiga's bacillus (type A).
1 in a dilution of 1 : 100.
1 in a dilution of 1 : 200.
1 in a dilution of 1 : 500.
All agglutinated Flexner's bacillus (type E).
1 in a dilution of 1 : 100.
1 in a dilution of 1 : 200.
1 in a dilution of 1 : 500 .
All agglutinated Shiga's bacillus as well as type E.

APPENDIX VII.

ANALYSIS OF CLINICAL CASES.

(1) Mild or Catarrhal Form of Dysentery.—An Analysis of 90 Cases.

The temperature was raised only at the commencement of the attack, and fell to normal on the second day in 44·6 per cent. In only 7·7 per cent. was a temperature of over 100° F. registered—in one case it was 105° F.

The pulse rate was not accelerated above normal in a number of instances. In only 20 per cent. of the cases was a pulse rate of over 100 per minute recorded.

The tongue remained clean throughout the attack in 15·6 per cent.

Tenesmus on defaecation was noted in 62·2 per cent.

Abdominal tenderness, especially in the left iliac fossa, over the sigmoid flexure, was found in 60 per cent. (Tenderness over the caecum and ascending colon was noted only four times.) The contracted coils of the sigmoid flexure could be palpated as a definite movable cord in 23·3 per cent. of the cases.

Stools.—The first stools passed consisted of blood and mucus without the addition of faecal matter in 23·3 per cent. One case passed a normal stool at the commencement, followed by others consisting of pure blood and mucus. In 7·7 per cent. the motions were solid and faecal, but coated with blood and mucus.

In the remaining cases the stools consisted of liquid yellow faecal material, together with blood and mucus. In a few instances the colour of the faeces was green.

(2) Acute or Ulcerative Form of Dysentery.—An Analysis of 54 Cases.

The temperature was raised above normal at the commencement in every case but six. In 74 per cent. the temperature registered above 100° F., extending over a period of from two to nine days. The corresponding pulse rate was accelerated over 100 per minute in 24 per cent.

The tongue was furred in every case.

Tenesmus on defaecation was noted in 92·5 per cent.

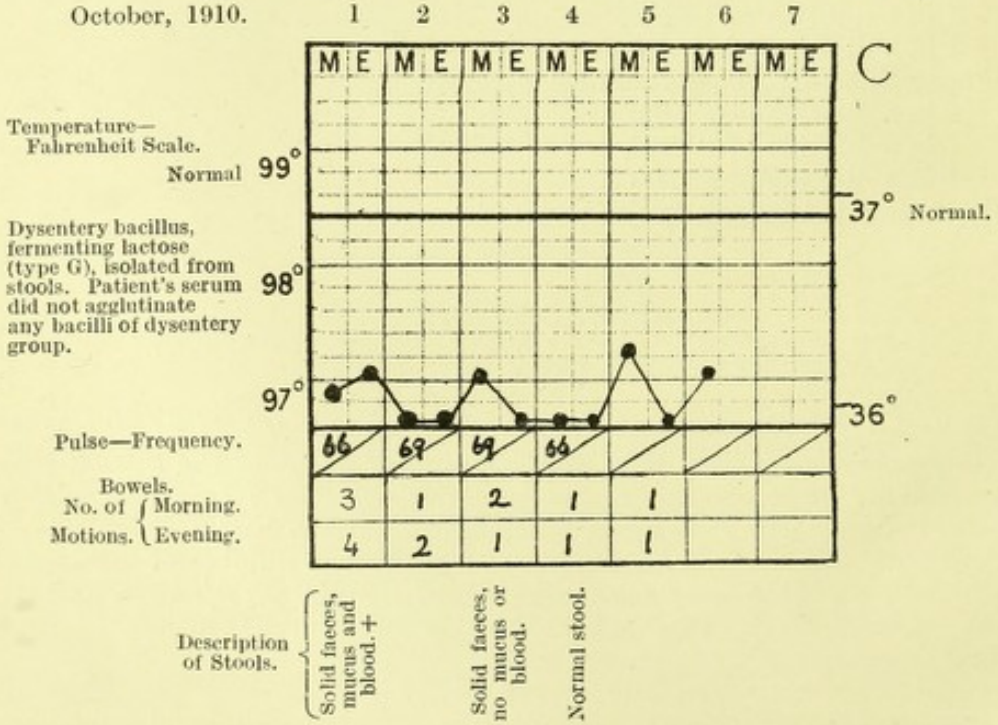
Abdominal tenderness over the sigmoid flexure was present in 68·5 per cent.; the contracted coils of the sigmoid could be felt through the abdominal walls in 40·7 per cent.

Stools: In 29·6 per cent. the stools first passed consisted entirely of blood and mucus, but in the remaining cases of yellow and green faecal material, often containing undigested curds.

(3) For a Further Account of Toxic or Fulminant Cases, *vide* Appendix XIV.

Appendix VII.—CHART 1.

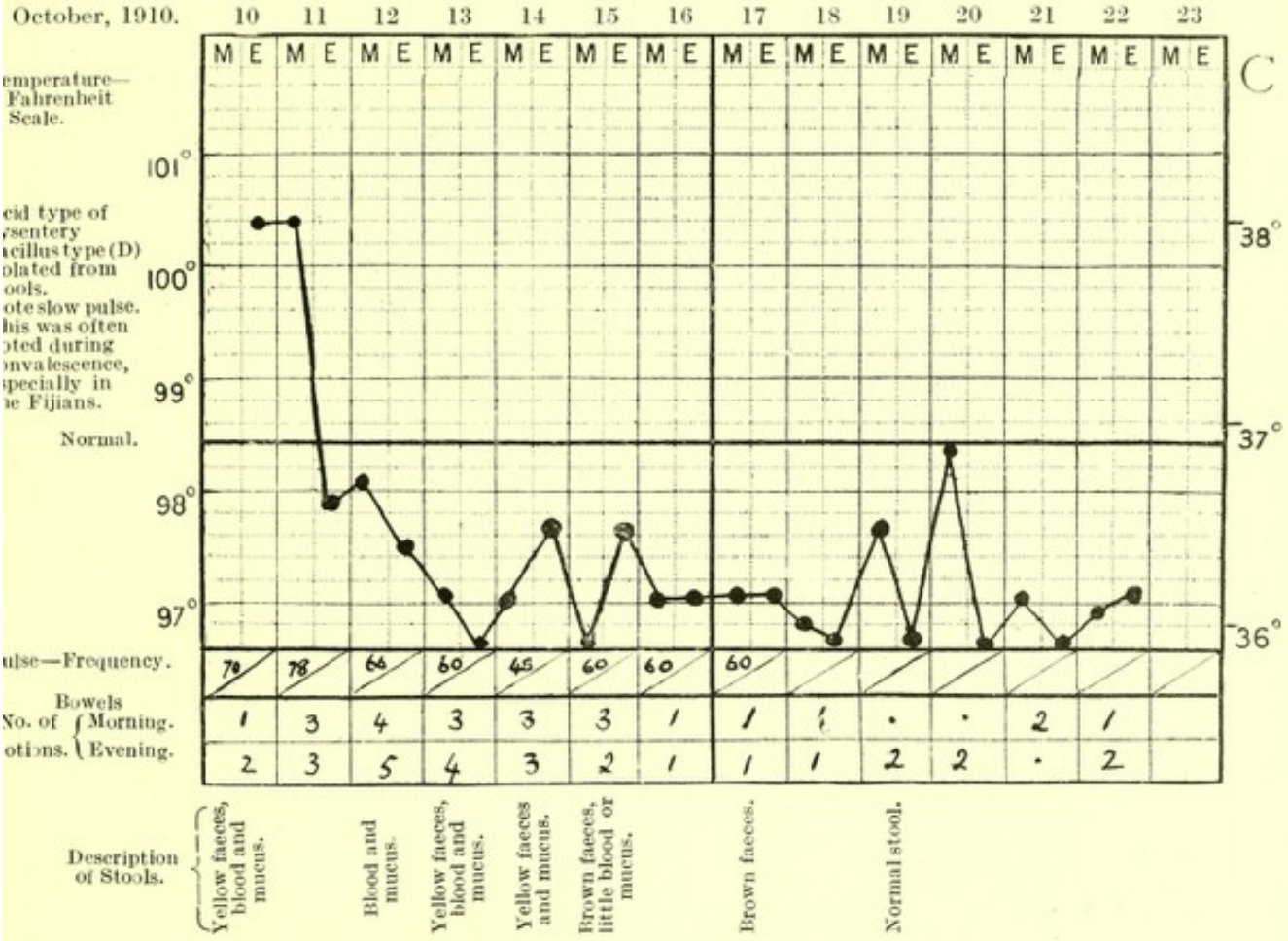
Disease: Mild or catarrhal form of bacillary dysentery, with no rise of temperature.
Name: Andriano ♂. *Race*: Fijian. *Age*: 26. *Diet*: Milk.



Appendix VII.—CHART 2.

Disease: Bacillary dysentery; mild or catarrhal form, with initial rise of temperature.

Name: Eparama. *Race*: Fijian ♂. *Age*: 24. *Diet*: Milk.

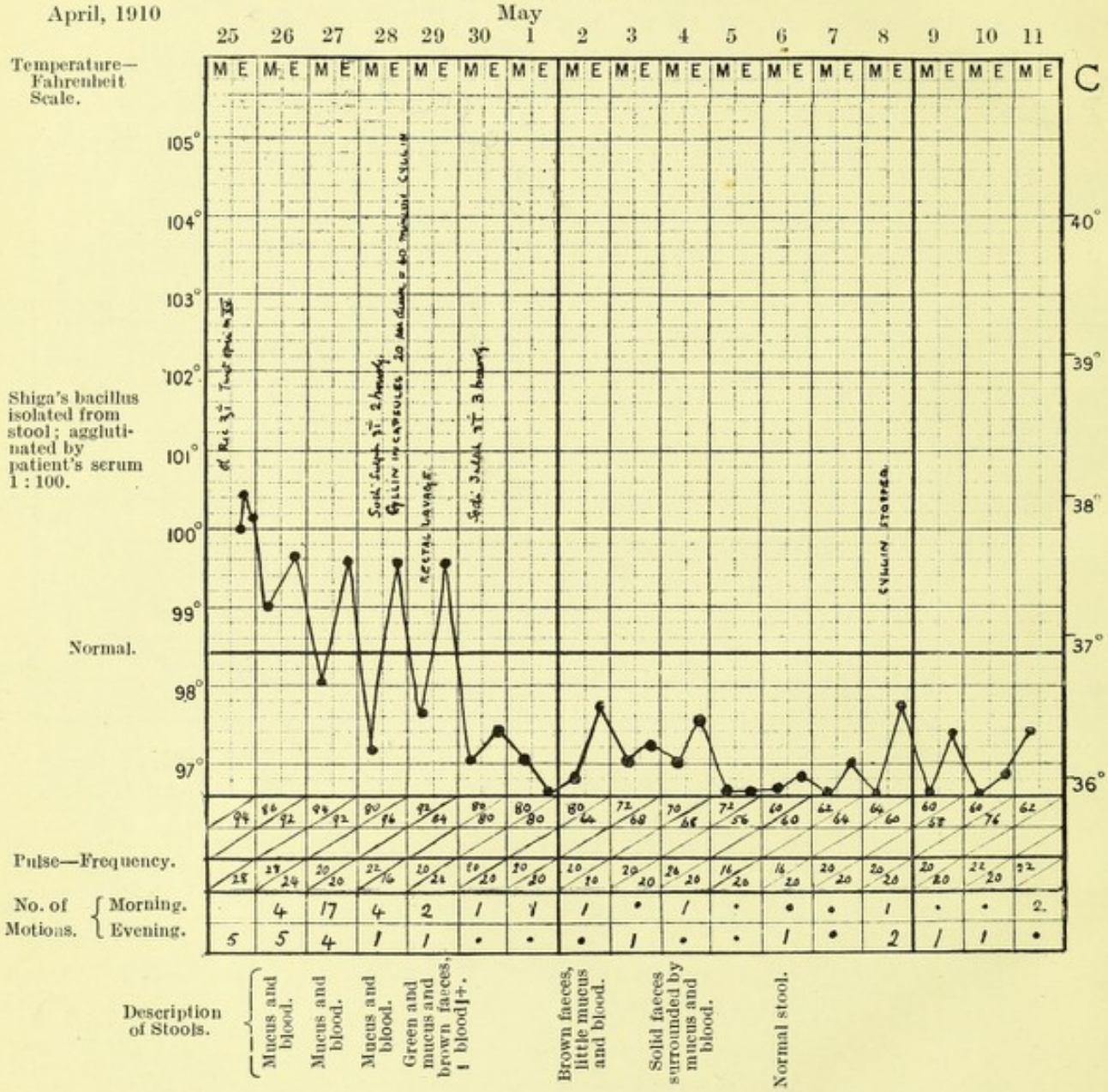


Acid type of dysentery bacillus type (D) isolated from stools. Note slow pulse. This was often noted during convalescence, especially in the Fijians.

Normal.

Appendix VII.—*continued.*—CHART 3.

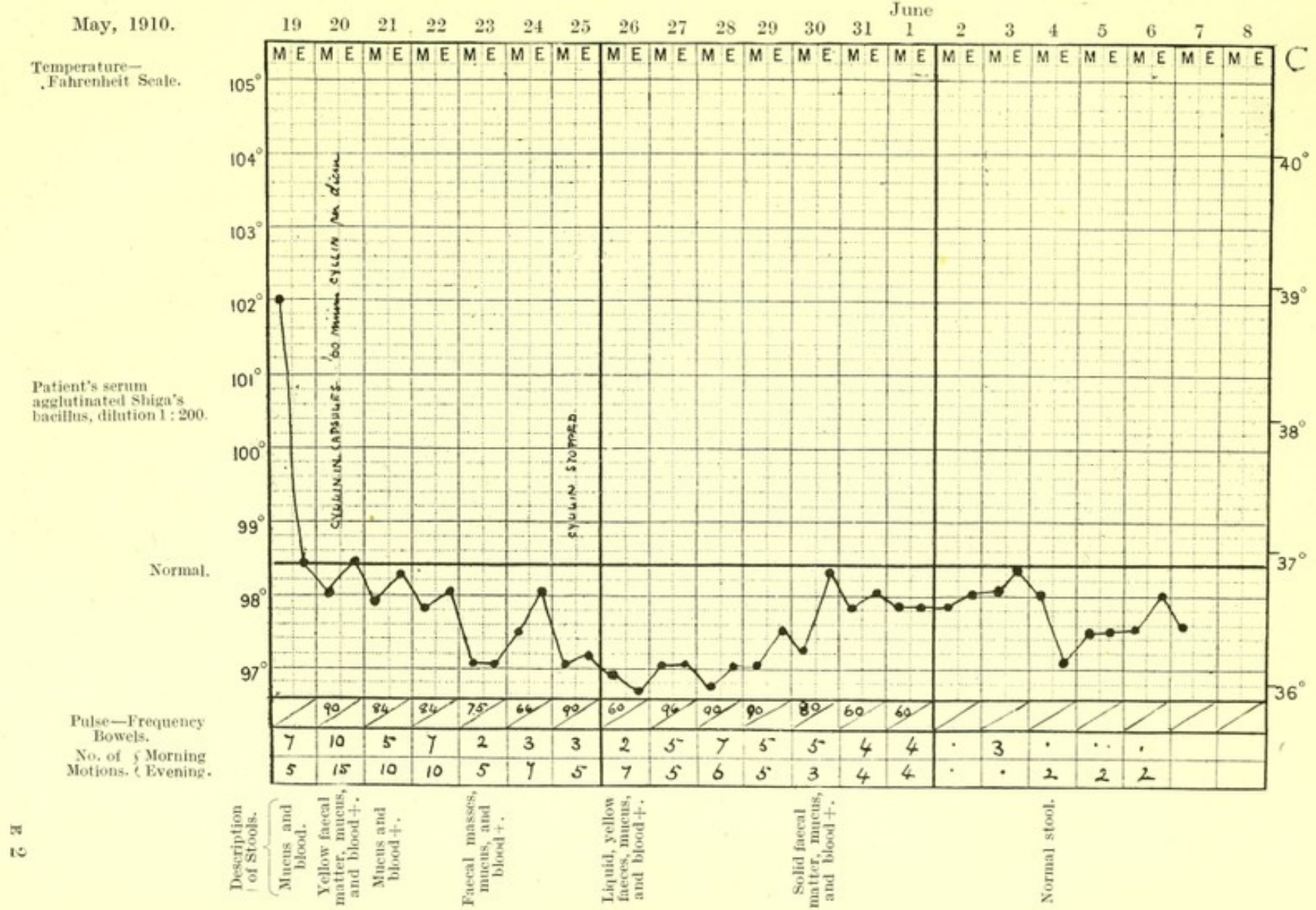
Disease: Bacillary dysentery; acute form. *Name*: V. A., Esq. *Race*: European (planter).
Age: 26. *Diet*: Milk.



Appendix VII.—*continued.*—CHART 4.

Disease: Bacillary dysentery; acute form. Marked symptoms and frequent stools. No marked rise of temperature.

Name: Alinale. *Race*: Fijian ♂ (constable from gaol). *Age*: 26. *Diet*: Milk.



APPENDIX VIII.

(1) Details of a Case of Relapsing Dysentery apparently belonging to the Bacillary Form.

An Indian male, aet. 29, was admitted to hospital in October, 1910, with all the symptoms and signs of an acute attack of dysentery. He passed on an average fifteen to twenty stools in the twenty-four hours ; these contained a great deal of adhesive mucus, rarely with the addition of blood. The temperature was raised to 100° F. for the first two days after admission. There was considerable abdominal tenderness, and the sigmoid could be distinctly palpated in the left iliac fossi. There were signs of consolidation, probably of tubercular origin, at both apices, especially at the left. His haemoglobin percentage was 70. The stools contained numerous Ankylostome ova, red blood corpuscles and swollen vacuolated epithelial cells. His physical condition was very poor ; he was very emaciated, but improved rapidly on sodium sulphate and cyllin treatment. After seventeen days' stay in hospital he was discharged. He was then passing normal stools, had no abdominal pain, and had put on flesh. His serum agglutinated Shiga's bacillus in a dilution of 1 : 200.

Three weeks later he was readmitted, passing stools of a similar nature, containing quantities of mucus, but no blood. The pulmonary condition had seemingly not advanced, the abdomen was tender, and the sigmoid could be distinctly palpated in the left iliac fossa ; he again improved on treatment with sodium sulphate and cyllin, and was discharged a month later in good condition passing normal stools. His serum still agglutinated Shiga's bacillus. No amoebae were ever found in the stools, though examined frequently with this object in view.

(2) Details of a Case of Acute Dysentery followed by Symptoms resembling those of Sprue.

An Indian male, aet. 59, was admitted to hospital with a typical attack of acute dysentery. Temperature 101° F. The tongue was furred and abdomen tender ; he was passing numerous stools, containing quantities of mucus and blood, and accompanied by tenesmus. There was considerable pain on palpation over the sigmoid flexure,which could be distinctly felt. He was in hospital altogether two and a half months, and during the whole of that period, though the mucous discharges were frequently examined microscopically, no amoebae were ever found ; large vacuolated cells containing chromatin masses were always present.

Though frequently tested, his serum never reacted towards the dysentery bacillus. He continued to pass frequent bulky stools on an average of five to ten a day. He was treated with cyllin and sodium sulphate, and after the first week he was passing no more mucus or blood. A few days later the abdominal tenderness had disappeared, and there was no tenesmus ; pain was complained of over the liver, which was distinctly tender on pressure and definitely enlarged ; there was no concurrent rise of temperature or leucocytosis. Three courses of ipecacuanha treatment were given, beginning with five and working up to thirty grains of the powder, but no definite improvement was noted. The stools were still pale, frequent, bulky, of sour odour, and fermenting. They still averaged about seven a day. The pain over the hepatic area still continued, but his tongue was quite clean.

Two months after admission he began to complain of sore patches at the sides of his tongue; glazed, raw, sensitive areas were noticed on the edges of that organ. The patient was now emaciated, and had dyspeptic symptoms and marked abdominal distension. On attempting to restrict his diet solely to milk, he ran away, so that his subsequent fate could not be ascertained. I may mention that the occurrence of sprue amongst Europeans is considered rare in Fiji.

APPENDIX IX.

Disposal of Excreta of Dysentery Patients in the Colonial Hospital, Suva, Fiji.

The patients defaecate on an open verandah adjoining the dysentery ward. The receptacles provided are pans with movable lids. These are primarily intended to prevent the access of flies to the stool. In many instances these lids do not fit properly; in others, actual holes have rusted through the tin.

Many patients, especially when acutely ill with dysentery, defaecate over the sides of these pails, and even through a crack in the boards of the verandah, soiling the ground below, thus making dissemination of the infective material an easy matter. After inspection by the medical officer, the stools are carbolized and are then emptied by an attendant into a large pail, also fitted with a lid; during this process the flies have easy access to the interior of this large receptacle. These pails are subsequently collected by a sanitary gang and dumped out at sea. The pans themselves are washed underneath a tap and the washings pass into a drain, the outlet of which, conveying infective material, is situated but twenty-four yards away from the ward, and opens into a clump of grass. The ground around is saturated, offensive, and swarms with flies.

The main Indian ward is situated eighty yards, and the Fijian ward 150 yards, away up the hill. A number of patients were infected with dysentery in these wards whilst in hospital suffering from other complaints. The Fijians were especially affected; their habit of eating their food spread out on a common dish would give easy access to infected flies, which settle on the food in great numbers.

The post-mortem room is another possible source of infection; there the pipe carrying infective material from the post-mortem table opens out on the hillside but twenty yards away.

Enquiry into the Spread of Acute Dysentery in the Native Gaols, Suva, Fiji.

The main gaol, Suva. This gaol accommodates some 200 prisoners, for the most part Indians; there were also a few Solomon Islanders and Fijians. It is situated about one and a half miles from the town, on the shores of the harbour, and accommodates also thirty female prisoners, though on an average about a dozen are detained there.

Out of a total of 157 cases of acute dysentery under observation at the Colonial Hospital, no less than eighty were prisoners from the gaol, and of these fifty-six were Indians. These figures, I think, indicate the prevalence of acute dysentery at this institution. The warders are Fijians, of whom several were also infected. The quarters for the women are situated on higher ground; their cooking and sanitary arrangements are quite separate from those of the men. No cases of dysentery occurred amongst them; they never leave the

gaol, whereas the men are at work in gangs during the day and undertake a great deal of the public work of the Colony. The surface drainage is excellent, and the gaol itself is kept very clean. There are proper latrines for the men ; the dry-earth system is adopted, each closet is provided with a wooden lid, and the faeces are covered with lime immediately by an attendant. The night stools are passed in a sort of iron cage leading off from the dormitory into which the men are locked every night. The excreta fall into an open drain, out of which they are flushed every morning into a closed drain-pipe, which frequently becomes blocked ; the habit of the prisoners in using a kind of reed to cleanse themselves with also adds to the obstruction. This drain is frequently out of order ; the ground around is soiled, and probably constitutes one of the main foci of infection in the gaol.

A new fly-proof kitchen is being built, but the old one is a dirty open shed. The food is served in another open shed, and in both of these places is liable to contamination by flies.

The gaol generally is particularly free from these insects, but they collect in numbers round both the day and night latrines and the kitchen.

The food is good, and consists of beef, one pound of rice, one pound of bread, pumpkin and beans, *ad libitum*, per man. The water supply is good, and is the same as provided at the town of Suva, namely, by pipes from reservoirs situated four miles from the town.

The excreta, removed daily from this dry-earth system by a sanitary gang of convicts, are placed on barges and dumped out at sea. No cases of dysentery occurred amongst the sanitary gang, which also performs the same service at the residences of the European Government Officers.

All the Hindoos wash their hands before meals, and have two baths a day ; they are kept much cleaner and receive much better fare than the coolies in the plantations.

The Fijian gaol, Nasova, Suva. This is a small prison to accommodate thirty Fijian prisoners and two constables. It is situated in the grounds of Government House ; several of the most severe cases of dysentery amongst the Fijians occurred in this institution. The quarters are constructed in the shape of Fijian houses, or *buris*.

There is a dry-earth latrine, which is infested with flies. The excreta are covered with ashes after deposition. The kitchen is a tin shed, not screened, and flies abound and have free access to the food. The night-soil system is similar to that adopted in the large gaol ; the receptacle is a bucket, which is emptied in the morning.

The police barracks, situated in the vicinity, accommodate forty native constables, who sleep in native huts. The latrines are on the dry-earth system, and are cleanly kept.

Out of fifty-four Fijians suffering from acute dysentery, and who came under observation, seventeen were prisoners and twelve were constables living in this gaol.

A complete drainage scheme has now been completed, and when installed the hospital and gaols will be linked up in the general system, and then these possible foci of infection will cease to exist.

APPENDIX X.

TABLE showing variation in fermentative reactions of acid and non-acid groups of dysentery bacilli after passage through the intestinal tract of the house-fly (*Musca domestica*). The period of incubation has been extended over a term of ten days in each instance. The reactions have remained the same throughout the period of incubation save where otherwise stated. The two strains of dysentery bacilli used for the purpose of infecting the flies were just those which remained constant, as regards their fermentative reactions with the sugars, over a period of nine months.

1. NON-ACID GROUP.		Mannite	Lactose.	Saccharose.	Maltose.	Dulcite.	Dextrose.	Litmus milk.	Indol (after 10 days' incubation in peptone water).
REACTION OF THE ORIGINAL BACILLUS (SHIGA) ISOLATED FROM STOOL.		○	○	○	○	○	+	Acid returned to alk. on 3rd day	Nil.
Number of the Experiment.	Period elapsing between time of infection of flies and recovery of the bacillus from the intestinal tract of the fly.								
33	2 days	○	○	○	○	○	+	Acid returned to alk. on 4th day	Nil.
33 (After 7 days' incubation)	2 days	○	○	○	+	○	+	Acid returned to alk. on 4th day	Nil.
33 (Two other colonies from same plate after 24 hours' incubation)	2 days	○	○	○	+	○	+	Acid returned to alk. on 4th day	Nil.
34 (2 colonies) (Another colony from the same plate gave reactions of original bacillus)	3 days	○	○	○	+	○	+	Acid returned to alk. on 3rd day	Nil.

Thus, apparently from the original *Shiga's bacillus*, a variety fermenting maltose as well as dextrose has been obtained. This corresponds to type B of Ohno (Port Arthur).

All these bacilli were agglutinated by anti-dysenteric serum in a dilution of 1 : 200.

APPENDIX X.—*continued.*

2. ACID GROUP.		Mannite	Lactose.	Saccharose.	Maltose.	Dulcite.	Dextrose.	Litmus Milk.	Indol (after 10 days' incubation in peptone water).
REACTION OF THE ORIGINAL BACILLUS (FLEXNER) ISOLATED FROM STOOL.		+	○	○	○	○	+	Acid returned to alk. on 3rd day	+
Number of the Experiment.	Period elapsing between time of infection of flies and recovery of the bacillus from the intestinal tract of the fly.								
24	2 days	+	○	+	○	○	+	Acid returned to alk. on 3rd day	+
24 After 2 days' incubation		+	○	+	+	○	+	Acid returned to alk. on 3rd day	+
29	4 days	+	○	○	○	○	+	Acid returned to alk. on 4th day	—
29 After 7 days' incubation		+	○	○	+	○	+	Acid returned to alk. on 4th day	+
31	2 days	+	○	○	+	○	+	Acid returned to alk. on 4th day	+
37* (Another colony from same plate gave reactions of original bacillus)	4 days	○	○	○	○	○	+	Acid returned to alk. on 3rd day	+
After 4 days' incubation (Tested on three subsequent occasions with same result)	4 days	+	○	○	+	○	+	Acid returned to alk. on 4th day	+

From an acid type of bacillus (Shiga type II., Ohno type G), type D in Appendix III., varieties corresponding in their reactions to type E and saccharose-fermenting varieties (Ohno types I and J) have been obtained.

* Lastly a bacillus was isolated which possessed but a feeble power of fermenting mannite, a positive acid reaction in that medium not appearing till the 4th day of incubation.

All these bacilli were agglutinated by anti-dysenteric serum in a dilution of 1 : 200.

APPENDIX XI.

Abstract of three Cases, belonging to the Toxic or Fulminating form of Acute Dysentery, who died after treatment with sodium sulphate; together with a summary of the post-mortem findings.

(1) *Fijian male, aet.* 30, admitted with a temperature of 100° F., great abdominal rigidity and pain. He passed over thirty stools per diem, consisting of green faecal matter, together with a great quantity of pink mucus, and accompanied by considerable tenesmus. The tongue was covered with a dense white fur.

Treatment consisted of sodium sulphate, one drachm every hour for the first twenty-four hours, and then four-hourly. The temperature remained raised for four days and then became subnormal.

Two days before death he became collapsed and passed a quantity of melaena. The stools were still very frequent, the pulse 130 per minute and very weak, the abdomen very tender, especially in the left iliac fossa. The heart sounds became hardly audible, and the patient collapsed and died.

Post-mortem. Thorax. All organs were healthy; there were adhesions at the apices of both lungs, but no signs of tuberculosis.

Heart. All cavities contained dark, fluid blood. The right ventricle was dilated.

Abdomen. The liver, kidneys and pancreas appeared healthy. The gall bladder was distended; the spleen was small, dark and diffluent.

The alimentary canal. The stomach and duodenum were healthy.

The small intestine. In the upper part of the jejunum six specimens of *Necator americanus* were found. The lower part of the ileum was injected and contained black, blood-stained mucus.

The large intestine. The caecum and the whole of the large intestine were in a similar condition; the intensity of the process increased as the rectum was approached. The intestinal contents consisted of mucus and a great deal of black melaena.

The intestinal walls were greatly thickened, the whole of the mucous membrane being gangrenous and of a bright green colour, covered with thick, blood-stained mucus. In places the necrotic layer had sloughed away. The process was most severe in the lower part of the rectum. No actual ulceration was seen. The mucous membrane of the appendix was healthy.

(2) *Fijian male, aet.* 20, was under observation seventeen days before death. He was admitted for severe tertiary yaws ulcerations, but contracted dysentery while under treatment in hospital. For the first ten days the temperature rarely rose above normal, but became irregular (up to 100° F.) during the last week.

The stools consisted for the most part of foul green faeces, containing large masses of pink mucus and blood which resembled frog's spawn and numbered about twenty per diem. At first there was no abdominal tenderness, but this became very marked towards the end.

The pulse throughout remained thin and weak. Death took place suddenly, apparently from collapse. Treatment consisted of sodium sulphate, one drachm, for the first two days, then the same dose four-hourly.

At the post-mortem.—Thorax. All the organs were healthy.

Abdomen. Liver, kidneys and pancreas appeared healthy. The spleen was small, dark and diffluent. The stomach and duodenum were healthy.

The small intestine was perfectly healthy throughout its extent; no parasites were found.

The large intestine. On opening the caecum, ragged irregular-shaped ulcers were seen spreading over the surface. The solitary lymphoid follicles were large and distinct; the ulcers apparently had their origin from these points. The intervening mucous membrane appeared healthy.

Ascending, transverse and descending colons contained numerous ulcers and submucous haemorrhages; the surface was lined with tenacious strawberry-coloured mucous. The contents consisted of liquid green faecal material.

The sigmoid flexure was most affected, being of great thickness and studded with small ulcers, the intervening mucous membrane being greyish-green in colour and the blood-vessels dilated. There was a small patch of apparently healthy mucous membrane in the pelvic colon. The most intense ulceration was situated in the rectum, where sinuous ulcers could be traced under the mucous coat. The mucus membrane of the appendix was normal.

(3) *Indian male, aet.* 45, was under observation eight days before death. On admission he was in a very emaciated state. The tongue was dry and furred; abdomen sunken and acutely tender. The sigmoid flexure could be distinctly palpated in the left iliac fossa.

The temperature registered 100° F.; the stools numbered over thirty per diem and were very foul, consisting of green faecal matter and large quantities of blood-stained mucus. There was considerable tenesmus on defaecation.

In a few days the stools contained more and more melaena; the patient collapsed, continually passing stools of the foulest description. He gradually sank and died.

At the post-mortem.—Thorax. All organs were healthy.

Abdomen. The right iliac fossa contained a quantity of bright yellowish-green pus. There were recent adhesions between the neighbouring coils of intestines. Two small perforations were found on the ventral aspect of the caecum.

Liver, kidneys and pancreas were healthy. The spleen was small, hard and dark. The mesenteric glands were not enlarged. Stomach and duodenum were normal.

The small intestine was normal throughout the whole extent, and contained quantities of bile-stained mucous.

The large intestine. The caecum contained a large ragged ulcer extending through all the intestinal coats, and perforation had occurred in two places. The rest of the large intestine was occupied by large ragged ulcers with greatly thickened margins; in places the mucous and submucous coats had been stripped away, leaving the muscular coat bare. The rectum was much thickened and the mucous membrane consisted of a thick yellow slough. A collection of pus was found occupying the recto-vesical fascia.

APPENDIX XII.

Details of two cases, treated with anti-serum injections combined with the administration of cyllin by the mouth, and who died while under treatment; together with a summary of the post-mortem findings.

(1) *Indian female, aet.* 20, found on the road in an exhausted condition. On admission her temperature was 98·8° F.; pulse very weak and irregular. There was oedema of both ankles, and the urine contained an eighth part albumen. The apex beat was situated outside the nipple line, and the cardiac impulse was hardly perceptible. The heart sounds were weak, and there was a well-marked haemic murmur.

The patient was hardly conscious ; the tongue was thickly furred, the abdomen not tender ; ten stools were passed in the first twenty-four hours consisting of foul green faeces and quantities of mucus and blood. Microscopically numerous ova of *Ankylostoma duodenale*, pus cells and red blood corpuscles were found. The haemoglobin was found to be 50 per cent.

An injection of anti-serum, twenty cc., was given subcutaneously, immediately after admission, cyllin thirty minims per diem and sodium sulphate one drachm every four hours.

Two days after admission the patient became unconscious, and the pulse irregular (120 to the minute), the respirations unequal. Typical uraemic convulsions set in, starting in the right arm and gradually working over the whole body. The breath was ammoniacal ; there was incontinence of urine and faeces. Strychnine grs. 1-22 was injected, normal saline transfused, and 40 cc. anti-serum given subcutaneously and intravenously. The patient never regained consciousness, and died.

The post-mortem was performed one hour after death.

Thorax. All the organs were healthy.

Heart. There was dilatation of the right ventricle, and some hypertrophy of the left.

Abdomen. The liver, bright yellow in colour, weighing thirty-seven ounces, was very fatty and friable. The spleen was small, dark and diffluent. The kidneys were large, pale and fatty, and there was no demarcation between cortex and medulla. The capsule stripped easily. The pancreas was normal in appearance. The oesophagus, stomach and duodenum were healthy.

Small intestine. The jejunum was perfectly healthy, and contained numerous ankylostomes and one large female *Ascaris lumbricoides*. Of the ileum, the upper part was healthy, the lower three feet were injected, especially round the Peyer's patches ; just above the ileo-caecal valve there was a small patch of superficial ulceration.

In the ascending colon the mucous membrane was quite healthy ; the transverse colon, on the other hand, was much thickened; the mucous membrane was strawberry-red in colour, with patches of superficial ulceration here and there. The necrotic margins were of a bright yellow colour. The whole epithelium was very oedematous, and haemorrhages in the submucous coat were visible ; the deepest ulceration occurred on the exposed transverse folds of mucous membrane. The intestinal contents consisted of bright yellow liquid faeces. No gangrenous epithelium of a green colour was seen. In the neighbourhood of the splenic flexure a patch of apparently normal mucous membrane was found.

In the ascending colon and sigmoid flexure the change was very marked: the mucous membrane presented an appearance like claret jelly; the rectum, save for a few patches of ulceration in the upper part, was normal ; both ovaries were cystic. Shiga's bacillus was isolated from scrapings of the transverse colon post-mortem.

(2) *Fijian child, male, aet.* $1\frac{1}{2}$, was admitted with a temperature of 99° F. and abdominal pain ; he was still being fed on the breast ; the tongue was thickly furred, and there was great tenderness on palpation over the left iliac fossa. The stools numbered about ten a day and consisted of green faecal matter, together with mucus and blood. Anti-serum was injected subcutaneously on three occasions, in doses from two to seven cc. ; cyllin syrup (three minims pure cyllin) was given by the mouth. The temperature remained raised and irregular, often reaching 104° F. at night-time ; sponging was resorted to. He was transfused with normal saline on three occasions.

The stools became normal and consisted of yellow faecal matter. Three

days before death signs of consolidation were found in both lungs. The child died in convulsions. A post-mortem examination was performed immediately after death, and the following condition found :

Thorax. There was broncho-pneumonic consolidation of the lower lobes of both lungs. In the purulent exudate numbers of pneumococci were found.

Abdomen. Liver, spleen, kidneys and pancreas were healthy. Mesenteric glands were injected and enlarged.

The jejunum and upper part of ileum were healthy and contained liquid yellow faecal matter. The lower part of the ileum was thickened and injected; the mucous surface was ragged; yellow superficial sloughs could easily be detached.

The mucous membrane of caecum and appendix was much thickened and oedematous ; sago-like patches studded the whole of the colon, about the size of a millet seed. This appeared to be due to an effusion into the submucosa. In the sigmoid a small patch of necrotic epithelium, about six inches long, was seen. There was injection of the mucous membrane of the rectum, but no actual ulceration. No dysentery-like organisms were cultivated from scrapings of the affected bowel post-mortem.

Neither of these cases came under the category of the most severe or toxic type of dysentery.

According to the classification I have adopted, they belong to the acute clinical form of dysentery. In neither case could the cause of death be actually ascribed to the dysenteric process.

In the first, death was probably due to uraemia, consequent on parenchymatous nephritis, complicated by severe ankylostomiasis In the second case there is little doubt that the actual cause of death was broncho-pneumonia and that the actual dysenteric lesions were in the process of healing.

APPENDIX XIII.

	Day on which stools became entirely faeculent.	Day on which stools became normal and patient discharged from hospital.
(1) Sodium Sulphate Treatment.		
Mild cases—Average of 32 ..	3rd day	7th day
Acute cases— ,, ,, 15 ..	8th ,, ...	15th ,,

(2) Cyllin by the Mouth, Doses 60–90 Minims per diem, in Addition to Treatment with Sodium Sulphate.

	Day on which stools became entirely faeculent.	Day on which stools became normal and patient discharged.
Mild cases—Average of 19 ..	2·7th day	6·5th day
Acute cases— ,, ,, 14 ..	6·5th ,, ...	13·6th ,,
Toxic cases—1 case	8th ,, ...	19th ,,

(3) Cases Treated by Intravenous Injections of Anti-Dysenteric Serum (Lister Institute), together with Cyllin and Sodium Sulphate by the Mouth.

	Day on which stools became entirely faeculent.	Day on which stools became normal and patient was discharged.
Mild cases—Average of 41 ..	3·5th day	7·8th day
Acute cases— ,, ,, 22 ..	5·2nd ,, ...	11·7th ,,
Toxic cases— ,, ,, 4 ..	14·25th ,, ...	29·25th ,,

APPENDIX XIV.

Abstract of five Toxic or Fulminating Cases of Bacillary Dysentery which recovered under treatment with Injections of Anti-serum intravenously, and Cyllin by the mouth.

(1) *Indian, male, aet.* 20, admitted in a collapsed state with a temperature of 102° F. The pulse was weak and rapid, over 130 per minute; the tongue thickly furred, the stools very frequent; these were green in colour, very offensive, and containing quantities of mucus and blood. There was considerable tenesmus on defaecation.

On the 4th day from the commencement of the illness the abdomen was very tender on examination; the coils of the contracted and thickened sigmoid flexure could be felt as a tense, movable cord.

Microscopical examination of the stools revealed no ova, but quantities of red blood corpuscles, pus cells and disintegrating epithelial cells. The haemoglobin percentage was 80.

He was treated with sodium sulphate, one drachm, for the first two days, then three times a day. Cyllin syrup was given as an intestinal antiseptic in half-ounce doses three times a day (30 minims of pure cyllin).

He was in hospital altogether for seventeen days. For the first four days the temperature was raised and irregular, reaching a maximum of 102° and 103° F. He lay on the floor prostrated, involuntarily passing stools of the foulest description, but too weak to pass them into the pail provided for the purpose.

These stools consisted almost of pure mucus and blood, but contained foul-smelling faecal matter and undigested clots of casein. Sloughs of mucous membrane were passed and were examined microscopically.

The patient became extremely emaciated, the abdomen sunken. He was in a very weak condition, evidently from want of rest and from physical exhaustion. On the sixth day he was given cyllin in capsules, in four doses of five capsules each, a total amount of sixty minims of cyllin a day.

A marked improvement was noted after this; the tongue began to clean, and the faecal matter in the stools changed from a green to a yellow colour. The number of stools gradually diminished, so that on the tenth day from the commencement of the illness, he was passing but four a day. On the twelfth day they contained no more mucus and blood.

The patient began to put on flesh at a remarkable rate; by the fifteenth day of his illness the stools were normal and the tongue quite clean.

The serum of this patient agglutinated both Shiga's and Flexner's bacilli in a dilution of 1 : 100.

(2) *A Solomon Islander, aet.* 40, admitted to hospital suffering from a severe attack of dysentery. He was under observation twenty-nine days altogether. The temperature was for the most part subnormal, but on three occasions was slightly raised.

The history of this case exhibits some of the difficulties experienced in attempting to keep a more or less accurate record of cases amongst these natives. On one occasion he ran away and was brought back nearly dead, and on another he refused altogether to take his medicine.

The stools at first averaged over twenty a day and consisted of foul-smelling green faecal material and quantities of mucus and blood. On microscopical examination ova of *Tricocephalus dispar* and *Ankylostoma duodenale* were found, besides great numbers of red blood corpuscles and pus cells. His haemoglobin percentage was normal. 20 cc. of anti-serum were injected into the

median basilic vein and he was given sodium sulphate, 1 drachm four-hourly. The tongue was very furred and the patient was in a very weak and emaciated state.

The abdomen was not at first tender to palpation, though the sigmoid flexure could be distinctly felt ; later, however, the tenderness in this area was marked.

On the fourth day after coming under observation he ran away, but was brought back after wandering two miles and having partaken of a large meal of farinaceous native food. He was then almost pulseless and was given a second injection of 10 cc. of anti-serum intravenously. The stools were still very numerous, and of the foulest description ; the faecal material, when present was of a bright green colour.

On the seventh day, since the patient remained in a very collapsed and exhausted state, a third injection of 20 cc. of anti-serum was given, and on the ninth day after admission great improvement was noted. The abdomen was relaxed and no longer tender, though the sigmoid could still be felt as in the left iliac fossa.

Henceforward improvement was rapid and he put on flesh, and on the twentieth day from the commencement of the attack he was passing normal stools.

A month after his discharge he was seen again ; he was then in excellent condition, and was acting as a porter on the wharf and carrying heavy loads.

The serum of this patient agglutinated Shiga's bacillus in a dilution of 1 : 100.

(3) *Fijian, male, aet.* 24, admitted in a critical state with a temperature of 100·2° F., a rapid pulse and great abdominal tenderness. He was passing stools continuously ; these consisted solely of blood and mucus. The number of stools actually passed was uncountable, so that the patient became exhausted with straining. The tongue was thickly furred, and there was great tenderness in the left iliac fossa.

Microscopical examination of the mucus passed revealed a great number of vacuolated epithelial cells, red blood corpuscles and pus cells.

He was given an injection of anti-serum intravenously and cyllin in capsules up to 60 minims a day, a dose which was afterwards increased to 90 minims ; sodium sulphate was also given by the mouth, 1 drachm every two hours. On the third day after admission he was given a second injection of anti-serum, 20 cc., and on that day a small amount of green faecal matter appeared in the motions. On the fourth day he began to improve ; the tongue became cleaner ; on the fifth the motions contained yellow faecal matter for the first time.

Acute stomatitis then set in, commencing round the alveolar margins. The whole of the buccal mucous membrane became involved and of a green colour ; the breath was most offensive. Eventually, all the incisor teeth dropped out. This process was controlled by frequently cleansing the mouth with a carbolic mouth wash. There was oedema of the lips and of the face consequent upon this inflammation. The abdominal tenderness disappeared, but the sigmoid flexure could still be distinctly felt. By the twelfth day the motions became normal, the patient had begun to put on flesh and was discharged from hospital at his own request, but was readmitted later for a further period of convalescence owing to inability to perform his work ; his motions were quite normal. A dysentery bacillus (type E) was isolated from the stools, and was agglutinated by the patient's serum in a dilution of 1 : 500.

(4) *A Solomon Islander, male, aet.* 20, admitted with a temperature of 102° F., a small, rapid pulse, and great abdominal tenderness was most marked in the left iliac fossa, where coils of the sigmoid flexure could be distinctly felt through the abdominal wall. He was passing over twenty stools a day, accompanied by marked tenesmus. These consisted of green faeces and quantities of blood and mucus.

On microscopical examination vacuolated epithelial cells, red blood corpuscles and pus cells were seen.

Anti-serum, 20 cc., was administered intravenously, and cyllin in capsules up to 60 minims a day was given by the mouth, together with sodium sulphate, 1 drachm, every two hours. Later a second injection of anti-serum was given intravenously, as the temperature remained irregular and the patient's condition did not materially improve.

On the seventh day after admission the stools became less frequent and contained a considerable amount of yellow faecal matter, together with quantities of blood and mucus. On the tenth day the abdomen was no longer tender, though the sigmoid could still be felt. On the twelfth day the tongue cleaned and the stools became half formed, and from this date onwards contained no more mucus or blood.

An acid type of dysentery bacillus (type E) was isolated from the stools, and was agglutinated by the patient's serum in a dilution of 1 : 100.

(5) *Fijian, male, aet.* 24, a constable from the Fijian gaol, was admitted with a temperature of 100·4° F., a rapid pulse and great abdominal tenderness, especially marked in the left iliac fossa. The motions actually numbered, as far as could be ascertained, over fifty in the twenty-four hours. They consisted for the most part of blood and mucus ; faecal matter, when present, was foul-smelling, of a green colour, and contained no ova of intestinal parasites.

Great numbers of pus cells and red blood corpuscles were seen on microscopical examination. Anti-serum, 20 cc., was administered intravenously, and 60 minims of cyllin in capsules were given by the mouth, together with sodium sulphate, 1 drachm, every two hours. A second injection of anti-serum (20 cc.) was given on the second day, and the dose of cyllin was increased to 90 minims a day. On the fourth day after admission he completely collapsed, the pulse became imperceptible at the wrist, the heart sounds just audible, and the patient very restless.

Tinct. opii in 5-minim doses was given every two hours for three days by the mouth, and two pints of normal saline were transfused into the pectoral muscles.

The patient then fell into a deep sleep, after which his condition became much improved and the pulse gained in strength. He still passed a great number of stools, resembling the so-called "meat washings," and these on several occasions contained sloughs of mucous membrane, which were examined microscopically. The temperature remained irregular.

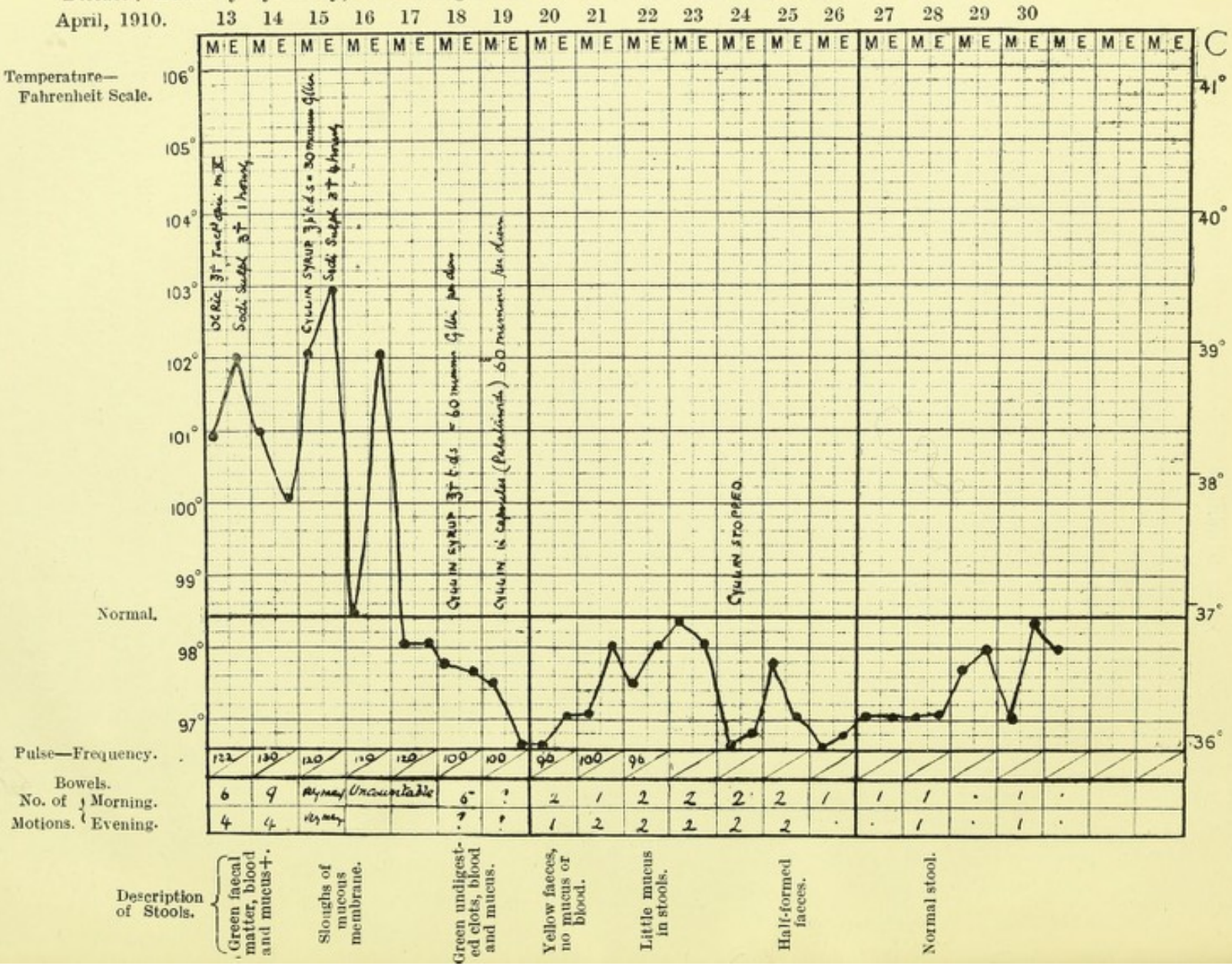
On the twelfth day of the illness, after having induced his attendant to give him a cold bath, he once more collapsed and became pulseless ; his temperature fell to 95·6° F. The pulse remained irregular and weak for three days, during which time the heart sounds were hardly audible. Stimulants were given and normal saline, one pint, transfused. Two injections of anti-serum (30 cc.), given intravenously and subcutaneously, were followed by great improvement in the general condition of the patient. For some time past he had been strange in his manner and speech ; his mental condition did not improve till convalescence was fairly established.

He was now absolutely emaciated, abdomen sunken, the skin shrunken and inelastic, so on the sixteenth day another injection of normal saline was given and was absorbed with great rapidity. From this time forward improvement was rapid and pronounced, and the patient began to put on flesh. Concurrently with the general improvement, the tongue became clean and the stools yellow and faecal, so that three weeks from the commencement of the attack he was passing normal stools. Signs of pulmonary tuberculosis at both apices were now discovered, as the temperature still remained of a hectic type. Shiga's bacillus was isolated from his stool, and was agglutinated by his serum up to a dilution of 1 : 500.

Appendix XIV.—CHART 5.

Severe case of bacillary dysentery treated with cyllin.

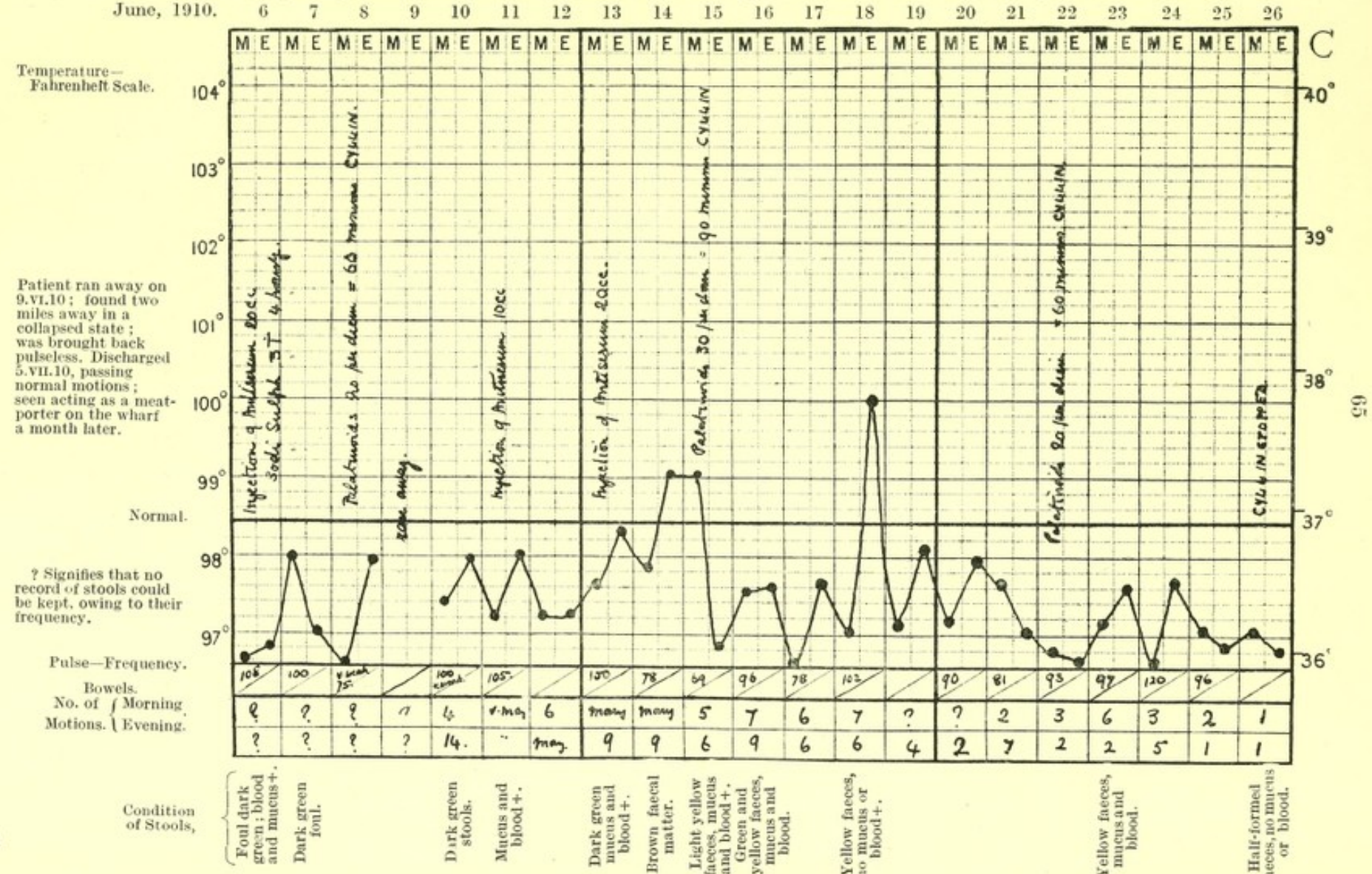
Disease: Bacillary dysentery, fulminating form. *Name*: Judnath, ♂. *Race*: Indian. *Age*: 20. *Diet*: Milk.



Appendix XIV.—*continued.*—CHART 6.

Severe case of bacillary dysentery treated with intravenous injections of anti-serum as well as large doses of cyllin by the mouth.

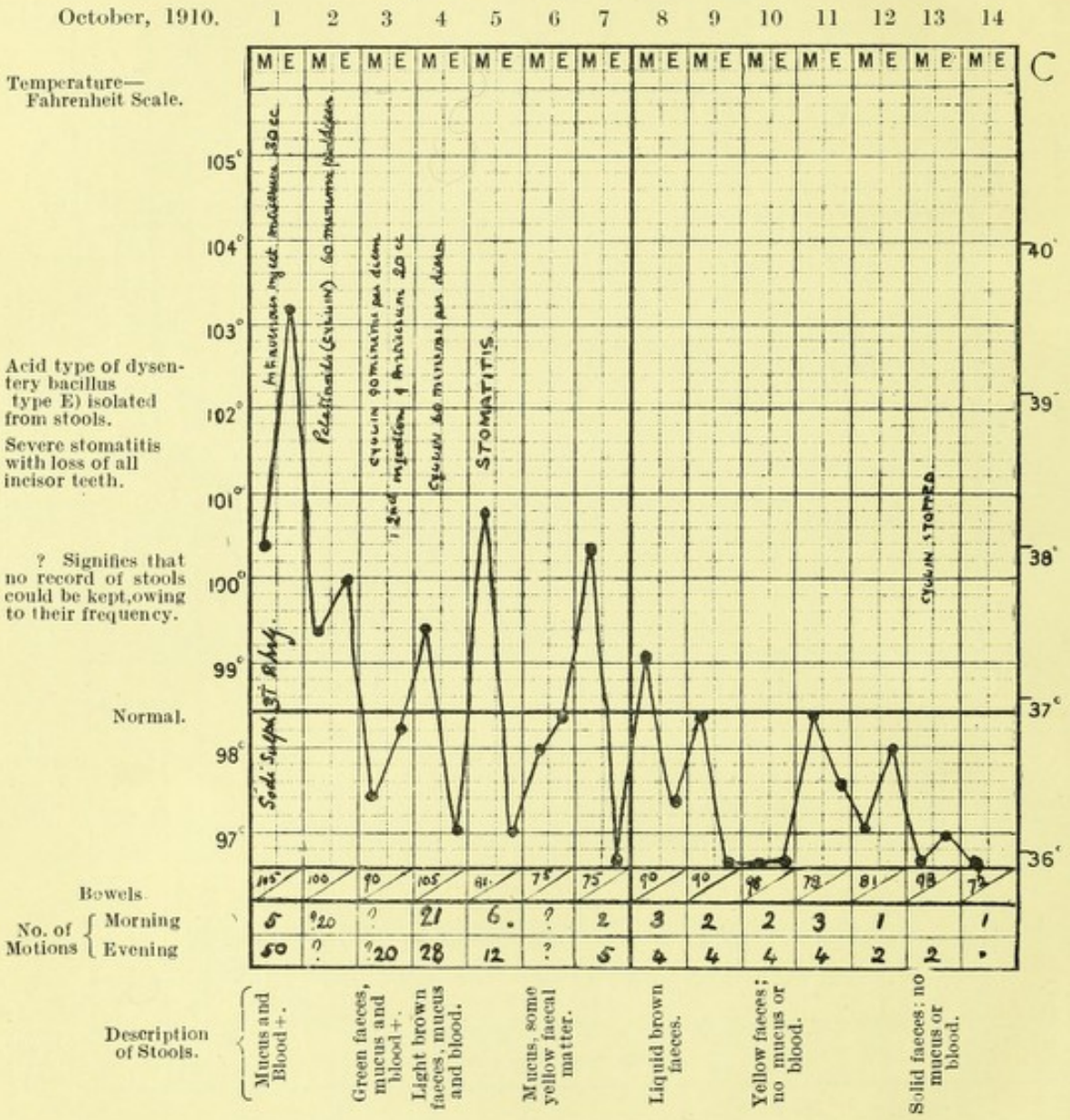
Disease : Bacillary dysentery ; toxic or fulminating form. *Name* : Udu ♂. *Race* : Solomon Islander. *Age* : 40. *Diet* : Milk.



Appendix XIV.—*continued.*—CHART 7.

Severe case of bacillary dysentery treated with anti-serum intravenously and with cyllin by the mouth.

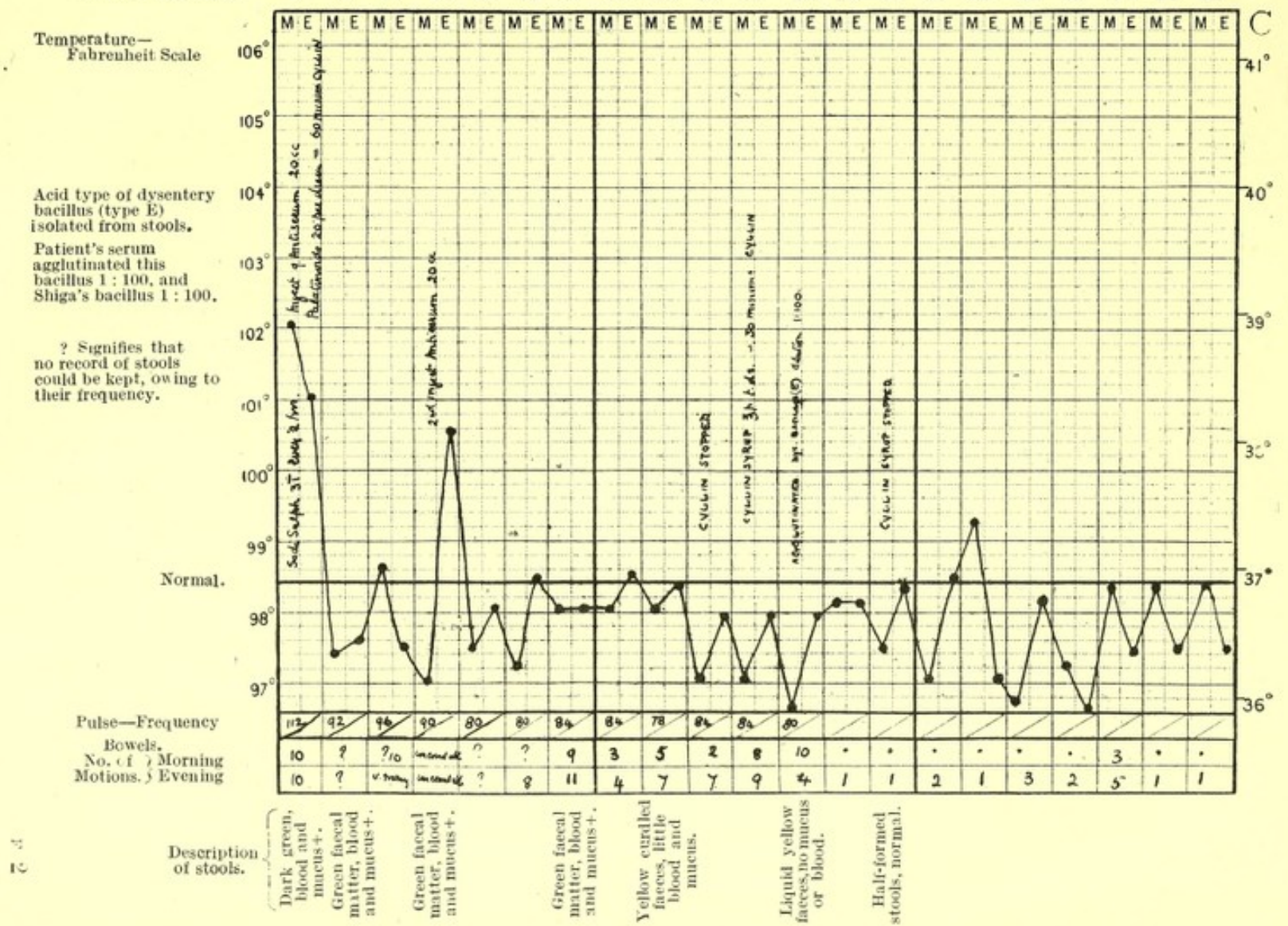
Disease: Bacillary dysentery; toxic or fulminating form. *Name*: Tevita ♂. *Race*: Fijian. *Age*: 24. *Diet*: Milk.



Appendix XIV.—*continued.*—CHART 8.

Severe case of bacillary dysentery treated with anti-serum intravenously and cyllin.

Disease: Bacillary dysentery; toxic or fulminating form. *Name*: Arofia ♂. *Race*: Solomon Islander. *Age*: 20. *Diet*: Milk.

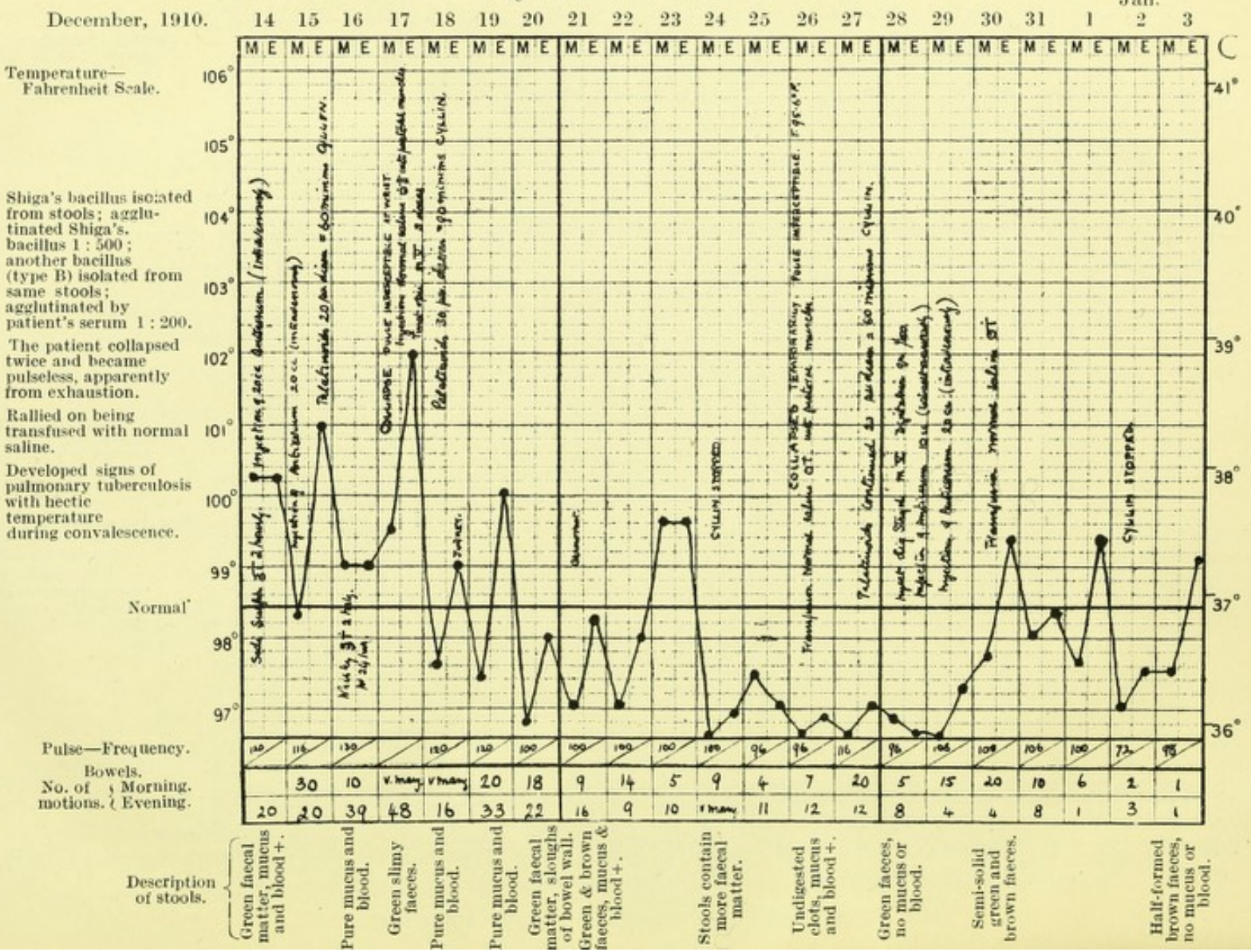


Appendix XIV.—*continued.*—CHART 9.

Severe case of bacillary dysentery treated with anti-serum intravenously and with cyllin by the mouth.

Disease : Bacillary dysentery ; toxic or fulminating form. *Name* : Veroniki ♂. *Race* : Fijian (constable).

Age : 24. *Diet* : Milk.



APPENDIX XV.

Summary of cases of Amoebic Dysentery.

(1) *Indian male, aet.* 25, had blood and mucus in stools for two weeks before admission to hospital. Stools numbered twelve to eighteen per diem, consisting almost entirely of pure blood and mucus. His temperature was 101·5° F., his tongue was furred ; there were rigidity and tenderness over the sigmoid flexure and slight tenesmus on defaecation. A great number of active amoebae were found in the mucus ; these measured 20 to 30 μ in diameter. In the unstained state the nucleus was not visible, but the endoplasm was very granular ; ova of *Ankylostoma duodenale* were also found in the stools.

Mucus from the freshly-passed stool was cultured on Conradi-Drigalski medium, but no bacilli resembling the dysentery bacillus were isolated.

Agglutination tests with cultures of Shiga's and Flexner's bacilli in a dilution of 1 : 50 gave a negative result. After the administration of four doses of pulv. ipecac. (grs. 10-30) he was passing formed yellow stools.

(2) *Indian male, aet.* 18, had blood and mucus in stools for four days before admission to hospital. Stools numbered six to eight per diem, and consisted of brown faecal matter, fresh mucus and blood. On microscopical examination of the stools, ova of *Tricocephalus dispar*, *Ankylostoma duodenale* and *Ascaris lumbricoides* were found. Great numbers of active amoebae and flagellates, probably *Trichomonas intestinalis*, were also present. The nuclei of the amoebae were not visible in the unstained specimens ; the endoplasm was very granular.

The temperature was normal on admission ; five days after he had a rigor with marked hepatitis ; the liver was enlarged and tender. The temperature remained raised for two days, and there was a leucocytosis of 10,000.

On treatment with pulv. ipecac. (grs. 30) the stools rapidly became normal and all signs of hepatitis disappeared.

After discharge he had no more symptoms of dysentery ; he was examined two months after and found to be quite normal.

The serum of this patient was tested against a dilution of Shiga's bacillus 1 : 100 with a negative result (Chart 10).

(3) *European male, aet.* 33, had been ill for three months before admission, passing blood and mucus in his stools. He had been treated during this period with salines, but although he improved temporarily a relapse always occurred when he partook of a more extended dietary. His temperature on admission was 102° F., intermittent in type, and remaining raised for eleven days.

His tongue was thickly furred and he was acutely ill. The abdomen was very tender to pressure, especially over the sigmoid flexure. The stools, which consisted of very offensive liquid brown faecal matter and a quantity of mucus and blood, averaged about ten to eighteen in number per diem.

Numerous active amoebae were found in the mucus ; these measured 20 to 30 μ in diameter ; the nuclei were not visible in the fresh state, and the endoplasm was granular. After the administration of pulv. ipecac. (in doses of from 5 to 30 grains) over a period of fourteen days, the temperature rapidly fell to normal, and the stools became healthy and averaged one a day. After three weeks' stay in hospital he was discharged.

The serum of this patient did not agglutinate Shiga's bacillus.

(4) *Solomon Islander, male, aet.* 25, admitted to hospital with a temperature of 102° F. and a dirty tongue. He was evidently suffering from pulmonary tuberculosis, of which there were definite signs at the right apex ; his spleen was enlarged, probably owing to long-standing malaria. There was considerable anaemia ; the haemoglobin index was only 40 per cent. His stools were very

numerous, twelve to eighteen a day; these consisted of foul faecal matter intimately mixed with mucus and blood, bearing a remarkable resemblance to strawberry jam. On microscopical examination, numbers of active amoebae were found; these measured about 15 μ in diameter. The nucleus was not visible in a fresh state, and the endoplasm contained no granules. A number of amoebic cysts, measuring 20 to 30 μ in diameter, were also seen; they contained eight nuclei (in this respect resembling the cysts of *Amoeba coli*). Great numbers of active trichomonas and small cysts were also present, in addition to ova of *Ankylostoma duodenale*. His serum was tested against a culture of Shiga's bacilli with a negative result.

After a few days in hospital there was acute tenderness over the hepatic area, and the liver was definitely enlarged. The sigmoid flexure was acutely tender on pressure. After the administration of pulv. ipecac. (grs. 5 to 30) his stools became normal, averaging but one or two per diem, and no more amoebae could be found. The abdominal tenderness disappeared.

The patient succumbed to pulmonary tuberculosis eleven days after the stools had become normal.

At the post-mortem evidences of widespread tuberculosis were found; there were miliary tubercles in both lungs; tubercles were found in the mesenteric glands, suprarenals, pelvis of both kidneys, and also in the liver. The spleen was large, and contained well-marked caseating nodules. The small intestine was healthy and contained numbers of ankylostomes. The large intestine, however, was covered with a definite greenish-yellow diphtheritic membrane, which could be removed with ease, leaving behind a pink granulating surface. Numbers of healing ulcers, situated in the long axis of the bowel, were also found; cicatrizing ulcers of a large and irregular shape were found in the ascending colon. The sigmoid flexure and upper part of the rectum were healthy.

In the lower third of the rectum, snail-track ulcers with yellow superficial sloughs were found running parallel to the transverse folds of mucous membrane.

In microscopical sections of the large intestine numbers of amoebae, often in a partially disintegrated state, were seen in a layer of fibrinous exudate, superficial to the epithelial layer (Plate V.).

(5) *European male, aet.* 23, had had an attack of chronic dysentery in Demerara two years previous to admission, in addition to two attacks in the last three months. He had never recovered from the last attack; the stools, averaging six to seven a day, contained blood and slimy mucus.

On admission the temperature was subnormal and remained so during his stay in hospital. His tongue was dirty; there was no abdominal tenderness. The stools averaged seven to twelve a day; these bore a very foul odour and peculiarly greasy appearance. Flecks of peculiarly tough mucus and a small quantity of blood were present.

On microscopical examination amoebae were found; they averaged about 20 μ in diameter, the nucleus was not visible in fresh preparations, the endoplasm was markedly granular.

His serum, tested against Shiga's and Flexner's bacilli, gave a negative result. He was treated with pulv. ipecac. (grs. 5 to 30) over a period of four days and the stools rapidly became normal. At first he vomited frequently after the powder, but this was well tolerated when enclosed in keratin capsules. A fortnight after discharge he was re-admitted, complaining of considerable pain in the left iliac fossa. His stools then averaged seven or eight a day and contained mucus and blood.

After a further course of ipecacuanha in keratin capsules he was discharged in a fortnight's time, passing normal stools. Subsequently, after a further period of three months, he was examined again, but had had no return of symptoms.

(6) *Indian male, aet.* 40, was admitted in a very wretched condition, very emaciated and anaemic (haemoglobin 40 per cent.); there was marked oedema of both ankles.

His temperature was irregular, varying from 99° F. to 101° F., but his tongue was fairly clean. There was a large bulging swelling on the right side of the thorax, which was very painful on pressure. The liver was enlarged upwards to the fourth rib in the nipple line, and downwards two finger-breadths into the abdomen. Definite consolidation at the base of the right lung was found.

The heart's apex beat was displaced two inches external to the nipple line.

On examination of the blood the red cells numbered 2,300,000 per cubic millimetre, and there was a polymorphonuclear leucocytosis of 13,000 (polymorphs 80 per cent.). His serum did not agglutinate a culture of Shiga's or Flexner's bacilli.

The abscess was explored; liver pus, which was sterile on culture, was obtained. Before an operation could be performed the patient absconded, but was brought back ten days after in an almost moribund condition. The liver dulness now extended up to the third rib in the nipple line.

At operation two ribs were resected and four pints of thick curdy liver pus evacuated. The cavity in the right lobe, which was a large one, appeared to extend into the left lobe as well.

He was passing six to eight stools a day, containing a quantity of mucus and blood, in which numbers of amoebae were found. The nuclei of these amoebae were not visible in the unstained specimens.

Ova of *Ankylostoma duodenale* were also present in the stools in great numbers.

The second day after operation, amoebae bearing the same morphological characters were also found in the liver pus. On continuous doses of ipecacuanha (5 to 30 grs.) the stools became rapidly normal. To prevent vomiting, the powder was given in pill form, coated with salol, with good results.

The after-history of this man is most interesting. He developed a pyopneumonothorax on the right side, which caused a marked displacement of the heart; there was great respiratory distress, the abscess cavity in the liver became septic, and he almost died from septic absorption. Another abscess then developed in the left lobe of the liver; this was opened under local anaesthesia. Eventually, however, the patient recovered entirely and returned to India, though, naturally, the pneumonothorax still persisted.

(7) *Indian male, aet.* 45, admitted with a history of dysentery of nine weeks' duration. His temperature was normal, and was never raised during his whole stay in hospital. The stools were frequent, twelve to fourteen a day; they consisted of semi-solid faecal matter coated with mucus and blood. For the first week in hospital he was treated with frequent doses of sodium sulphate, but without any marked improvement in the number and nature of the stools, which still contained quantities of blood and mucus. He was very emaciated and anaemic (haemoglobin 50 per cent.) and his tongue was furred.

Ova of *Ankylostoma duodenale* were numerous in the stools; active amoebae were found also in great numbers; these measured 15 to 22 μ in diameter; their nuclei could not be distinguished in the fresh specimens, nor was the endoplasm markedly granular. They were so numerous that twenty or more could be found in one field of the microscope with a high-power lens. Some small cysts, probably of some intestinal flagellate, were also present in great numbers.

There was no abdominal tenderness and the liver was not enlarged. His serum did not agglutinate cultures of Shiga's or Flexner's bacilli.

Pulv. ipecac. (grs. 5 to 30) was given over a period of ten days with great benefit; the patient rapidly put on flesh, the stools became normal and averaged

but one a day. On discharge, after three weeks in hospital, he was in good condition. There was then a considerable amount of thickening round the sigmoid flexure, which could easily be felt through the abdominal wall.

(8) *Indian male, aet.* 35, a confirmed opium eater, admitted to hospital with a history of chronic dysentery of three months' duration. His temperature remained subnormal during his stay in hospital. Six to eight stools a day were passed, consisting of foul-smelling faecal matter and a considerable amount of mucus and blood. The tongue was thickly furred ; the abdomen was acutely tender and there was considerable pain over the hepatic area, but the liver was not definitely enlarged, though tender on pressure. Dulness on percussion was found at the base of the right lung.

Great numbers of amoebae were found in the mucus passed in the stools; their nuclei could not be clearly distinguished in the living state ; their endoplasm was not granular.

The serum of this patient failed to agglutinate cultures of Shiga's or Flexner's bacillus.

Treatment consisted of pulv. ipecac. (grs. 5 to 40) for fourteen days, after which all pain over the hepatic area had disappeared. Thickened coils of the large intestine could easily be palpated through the abdominal wall.

He was discharged after a month's stay in hospital; he was then passing normal stools and had gained considerably in weight.

(9) *Indian male, aet.* 30, admitted with a history of dysentery of two months' duration. His temperature was subnormal on admission; only on two occasions during his stay in hospital was a slight rise of one degree noticed. His tongue was thickly furred.

The stools consisted of green faecal matter intermingled with a quantity of mucus and blood, and numbered from nine to sixteen a day. Numbers of amoebae and ova of *Ankylostoma duodenale* were found in the stools. These amoebae were very numerous and active ; great variation in their size was noted, but no nucleus could be distinguished in the living examples. The contrast between the granular endoplasm and the clear ectoplasm was marked in them. Some contained red blood corpuscles.

Pulv. ipecac. (grs. 5 to 30) was given over a period of eight days. After the exhibition of this drug, evident improvement in the general appearance of the patient was apparent. The stools rapidly became normal in appearance and averaged about one per diem on his discharge (Chart 11).

(10) *Solomon Islander, male, aet.* 30, was admitted in a very collapsed state ; the pulse was just perceptible and the heart sounds were just audible.

There was a history of having passed blood for a fortnight previous to admission. The temperature was raised (101° F.) and of a hectic type (Chart 12). His tongue was thickly furred, and there were sordes on his lips.

The abdomen was not tender to palpation, but the coils of the thickened sigmoid could be plainly felt through the abdominal wall.

Stools were being constantly passed, accompanied by great pain and tenesmus ; their number was uncountable. They consisted almost entirely of thick blood, with the addition of a little mucus. Under the influence of continuous saline transfusion, he rallied a little. No amoebae were found in the stools after frequent examination.

His serum did not agglutinate either Shiga's or Flexner's bacilli.

Being under the impression that I was dealing with a case of bacillary origin, I injected anti-dysenteric serum on seven occasions (120 cc. being given altogether), and cyllin was administered by the mouth, but without any beneficial result.

He collapsed frequently, but rallied after transfusion with normal saline,

and died on the eleventh day after admission. The abdomen was acutely tender before death, and the stools still consisted almost entirely of blood. *At the post-mortem* the thoracic organs were all healthy.

Abdomen. The liver weighed sixty ounces and was very pale ; on section, numerous miliary abscesses were scattered throughout its substance ; none were larger than a small pea.

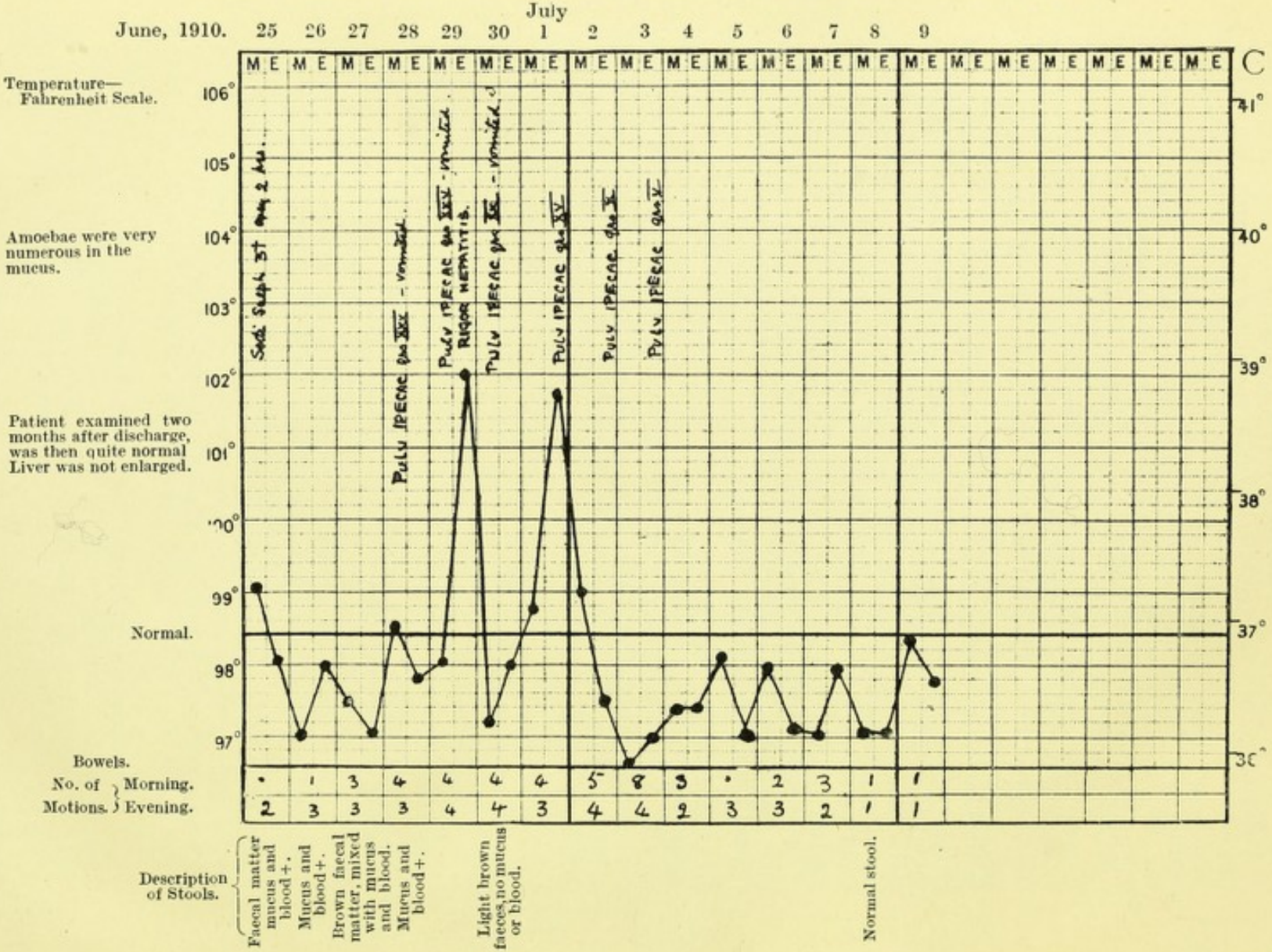
Intestines. Some old adhesions were found uniting the hepatic flexure of the colon to the peritoneum. The small intestine was normal in appearance. The large intestine was ulcerated throughout its whole extent. Large ragged ulcers extended down to the muscular coat, which in places was almost perforated. The caecum and the rectum were most intensely affected. The margins of the ulcers were haemorrhagic, but the mucous membrane in between the ulcerated areas appeared healthy. The intestinal contents consisted of grey purulent material.

Amoebae were found in numbers in the hepatic abscesses in microscopical sections ; they were also found in sections through the intestinal ulcers, in the submucosa, and in the substance of the circular muscular coat. The mucous and submucous coats appeared to have been swept away ; the circular muscular coat was covered by a thin layer of necrotic tissue, in which a few amoebae could be recognized ; probably, however, the pathological picture had been masked by an intense secondary bacterial infection.

Acute cases of this description have been recorded by Kuenen in Java ; in these cases, also, it is instructive to note, no amoebae were found in the discharges during the patient's lifetime.

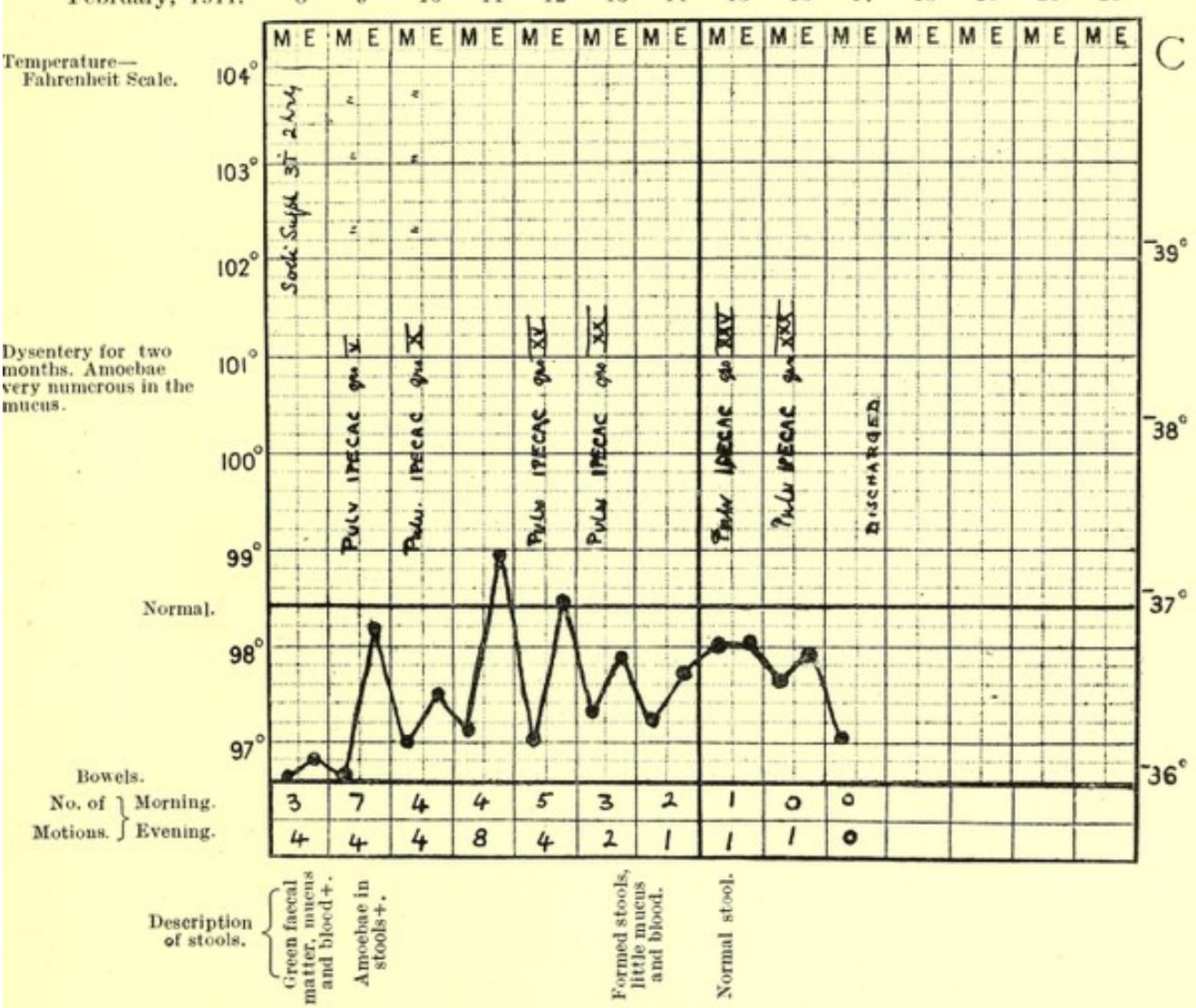
Appendix XV.—*continued.*—CHART 10.

Disease: Amoebic dysentery with hepatitis. *Name*: Kumbil Agab. *Race*: Indian. *Age* 18.



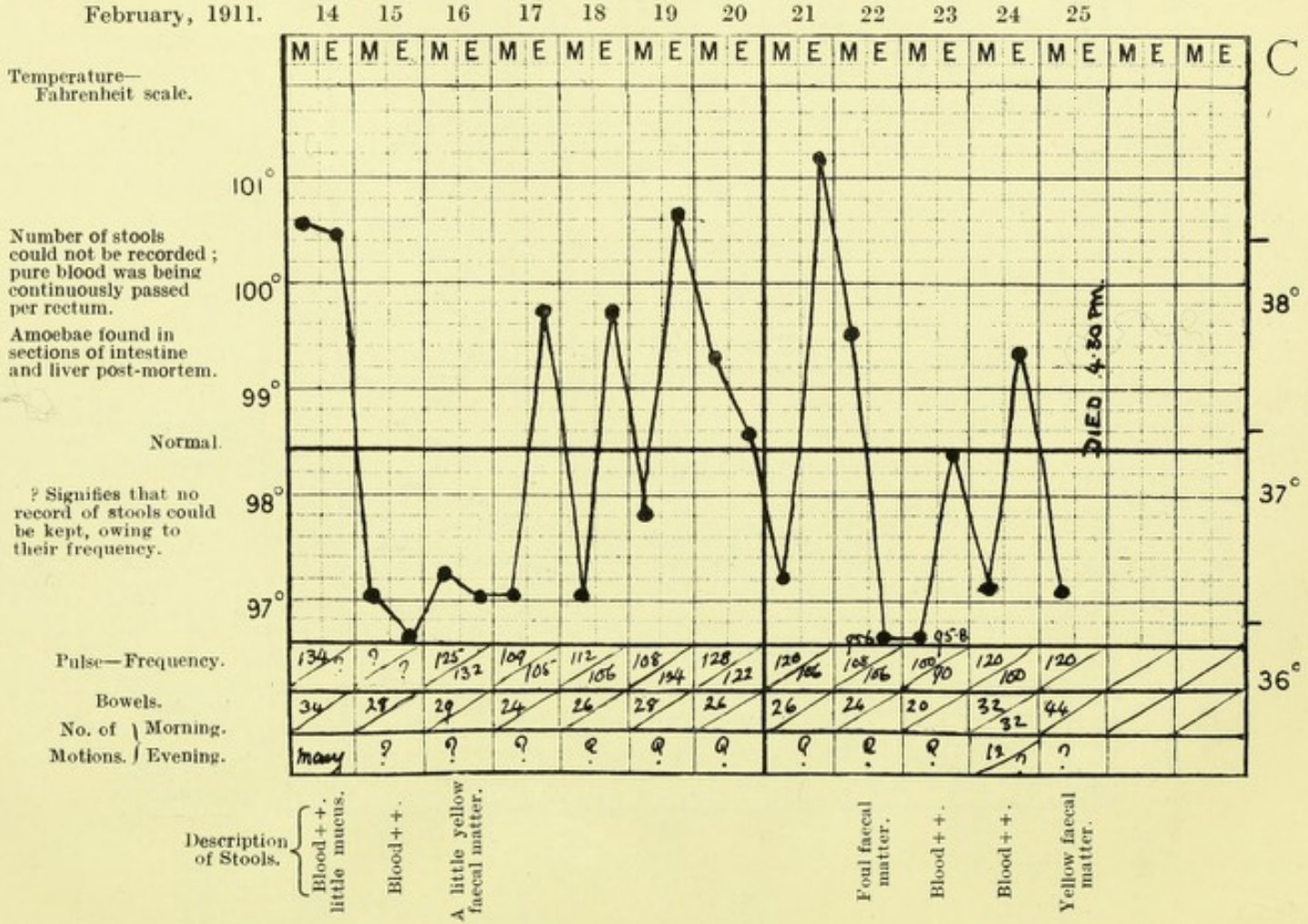
Appendix XV.—*continued.*—CHART 11.

Disease: Amoebic dysentery. *Name*: Gulab. *Race*: Indian. *Age*: 30.



Appendix XV.—*continued.*—CHART 12.

Disease: Amoebic dysentery; multiple liver abscesses. *Name*: Jack. *Race*: Solomon Islander. *Age*: 30.



APPENDIX XVI.

Summary of post-mortem of a Fijian who died from intestinal haemorrage consequent on Amoebic Dysentery.

A well-nourished Fijian, about 40 years of age, was admitted with a history of dysentery of some duration. Almost immediately after admission he vomited quantities of blood, and dark blood was passed per rectum. In the melaena, ova of *Necator americanus* were found, but no amoebae. Death took place after a severe bout of haematemesis

At the post-mortem :—

Thorax. A quantity of pus was found in both pleural cavities. The lungs were healthy ; the bronchi were full of aspirated blood.

Abdomen. The oesophagus, stomach and intestines were full of black blood. A number of *Necator americanus** were found in the ileum.

The large intestine, the caecum, ascending and transverse colons, were healthy. In the descending colon numerous old and cicatrizing ulcers were seen. The sigmoid was intensely ulcerated ; these ulcerations were deep and extended down to the muscular coat ; the edges of the ulcers were raised, rolled and bile-stained. They were of irregular outline, and measured one to one-and-a-half inches in length.

In the upper part of the rectum other ulcerated areas were found which extended to the anus itself. On microscopical section through these ulcers, great numbers of amoebae were found invading the submucous coat ; they were lying in a mass of chronic inflammatory tissue. Many of them were of irregular outline, and contained red blood corpuscles (Plate VI.).

The subjacent muscular coats also showed inflammatory changes. In the intact mucous membrane in the neighbourhood of the ulcer a few amoebae could be seen invading the interstitial tissue between the crypts, and actually lying within the lumen of the crypts themselves (Plate VI.).

The kidneys were large, pale and fatty, due to chronic parenchymatous nephritis.

Strong, of Manila, has described similar cases of amoebic dysentery in which intestinal haemorrhage took place with fatal result.

* For the identification of these specimens, I am indebted to Dr. R. T. Leiper.

1933-1