Bacillary Dysentery in the City of Bahia

by

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Endemic or epidemic all over the world, dysentery does not spare Brazil and its existence has already been made sure in Rio de Janeiro, São Paulo, Bello Horizonte and in the «Hinterland» of the State of Minas Gerais.

In Bahia up to now no one had demonstrated its existence in a manner worthy of credit.

In Bahia, up to now no one had the disease in that city must date from very far back, for the sanitary registers record 60 cases in 1897, as may be seen in table I, and, since then, there have been considerable epidemics, like that of 1908 which killed 479 persons.

I EPIDEMIOLOGICAL NOTES

1. Number of Cases

A. Predominant Sort of Dysentery.

Table I shows the number of deaths which occurred in the city from 1897 to 1924. In these figures are included the cases of death by the two sorts of dysentery—the bacillary and the amoebic. Amoebic dysentery is infrequent and must contribute a very small contingent to the figures of the death-rate of the town.

<table>
<thead>
<tr>
<th>Years</th>
<th>Deaths</th>
<th>Years</th>
<th>Deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td>1897</td>
<td>60</td>
<td>1911</td>
<td>90</td>
</tr>
<tr>
<td>1898</td>
<td>32</td>
<td>1912</td>
<td>80</td>
</tr>
<tr>
<td>1899</td>
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<td>1913</td>
<td>176</td>
</tr>
<tr>
<td>1900</td>
<td>15</td>
<td>1914</td>
<td>62</td>
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<tr>
<td>1901</td>
<td>19</td>
<td>1915</td>
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<td>1902</td>
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<td>1903</td>
<td>11</td>
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<td>23</td>
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<tr>
<td>1904</td>
<td>17</td>
<td>1918</td>
<td>18</td>
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<tr>
<td>1905</td>
<td>10</td>
<td>1919</td>
<td>51</td>
</tr>
<tr>
<td>1906</td>
<td>10</td>
<td>1920</td>
<td>212</td>
</tr>
<tr>
<td>1907</td>
<td>13</td>
<td>1921</td>
<td>162</td>
</tr>
<tr>
<td>1908</td>
<td>479</td>
<td>1922</td>
<td>71</td>
</tr>
<tr>
<td>1909</td>
<td>177</td>
<td>1923 (1)</td>
<td>148</td>
</tr>
<tr>
<td>1910</td>
<td>93</td>
<td>1924</td>
<td>291</td>
</tr>
</tbody>
</table>

(1)—From April to December.
This assertion is justified not only by the low percentage of deaths from this sort of dysentery as by the fact that it is recognised to be infrequent by practitioners in the town. Out of 281 cases of dysentery occurring in 1924, up to September, the number of cases of amoebic dysentery was restricted to 30 by the practitioners, as may be seen from Table II.

**TABLE II**

| Number of cases of dysentery notified up to September 1924, according to the Statistics of the Public Health Service of the State of Bahia. |
|---|---|---|---|---|
| Month | Amoebic Dysentery | Bacillary Dysentery | Not specified | Totals |
| January | 2 | 6 | 28 | 36 |
| February | 5 | 8 | 20 | 33 |
| March | 0 | 5 | 18 | 23 |
| April | 3 | 6 | 38 | 47 |
| May | 9 | 5 | 35 | 49 |
| June | 4 | 3 | 19 | 26 |
| July | 2 | 3 | 22 | 27 |
| August | 4 | 4 | 16 | 24 |
| September | 2 | 0 | 14 | 16 |

I., for my part, was able to study 23 cases of dysentery, in patients either still ill or after recovery, who were left over from the epidemic. Among these I did not find one with *Entamoeba histolytica*. The only time I saw this amoeba during the three months of my stay, was in faeces sent to DR. HORACIO MARTINS for diagnosis of the parasite.

One more fact comes in support of this view and that is the mortality, which, according to the official figures of the Public Health Service, oscillated between 75 and 100%. These figures do not represent the whole truth of the matter, for the service of notification of infectious diseases is not carried out with regularity in this city.

It is enough to mention the statistics of the Isolation Hospital of Mont-Serrat, where 35 cases were isolated, 11 of which died, that is, 31%. In the 18 cases I studied, the mortality was of 60% for cases of the Shiga type and 16% for those of the Y type, with an average of 38%. The statistics of the Hospicio Sao JOAO DE DEUS also come in my support, showing a death rate which rose to 50, 48, 20, 66 and 54%, respectively in the years 1920, 1921, 1922, 1923 and 1924 (the latter up to September) with an average of 50%.

Amoebic dysentery never gives rise to such a high death-rate.

I was unable to obtain details about the frequency and outcome of complications in the liver, but I believe that they must also be uncommon.

From all this one may draw the conclusion that, even if amoebic dysentery is nothing out of the way in Bahia, it is not at all common and must con-

**TABLE III**

Estimate of number of cases of dysentery allowing a mortality of 80% and consulting the number of deaths registered by the Public Health.

<table>
<thead>
<tr>
<th>Years</th>
<th>Number of cases</th>
<th>Years</th>
<th>Number of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>1897</td>
<td>75</td>
<td>1911</td>
<td>112</td>
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<tr>
<td>1898</td>
<td>40</td>
<td>1912</td>
<td>101</td>
</tr>
<tr>
<td>1899</td>
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<td>1913</td>
<td>220</td>
</tr>
<tr>
<td>1900</td>
<td>19</td>
<td>1914</td>
<td>77</td>
</tr>
<tr>
<td>1901</td>
<td>24</td>
<td>1915</td>
<td>80</td>
</tr>
<tr>
<td>1902</td>
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<td>29</td>
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<tr>
<td>1904</td>
<td>21</td>
<td>1918</td>
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</tr>
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<td>12</td>
<td>1919</td>
<td>64</td>
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<td>12</td>
<td>1920</td>
<td>265</td>
</tr>
<tr>
<td>1907</td>
<td>16</td>
<td>1921</td>
<td>200</td>
</tr>
<tr>
<td>1908</td>
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<td>88</td>
</tr>
<tr>
<td>1909</td>
<td>221</td>
<td>1923</td>
<td>266</td>
</tr>
<tr>
<td>1910</td>
<td>116</td>
<td>1924</td>
<td>533</td>
</tr>
</tbody>
</table>
tribute next to nothing towards the mortality from dysentery. Allowing, as I do, that the most frequent type of dysentery is the Shiga type or toxic type, with its higher mortality and attributing to the cases in Bahia a maximum and overrated mortality of 80%, I was able to construct a table, table III, from which it is possible to judge, although imperfectly, of the extension of the epidemics.

From it one may see that the great epidemic of 1908 attacked about 600 persons, and that the one of last year affected more than 500.

These figures are still far from the truth.

B—Simulated dysentery.

The number of cases of simulated dysentery was great.

The study of the bulletins of the Sanitary Statistical Service of the State (Demographia Sanitaria do Estado) directed my attention towards the number of cases of death from dysentery, and

Table IV

Number of deaths from diarrhoea and enteritis in the City of Bahia in the years 1923 (1) and 1924.

<table>
<thead>
<tr>
<th>Month</th>
<th>1923</th>
<th>1924</th>
</tr>
</thead>
<tbody>
<tr>
<td>January</td>
<td>65</td>
<td></td>
</tr>
<tr>
<td>February</td>
<td>59</td>
<td></td>
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<td>May</td>
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<td>August</td>
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<td>September</td>
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<td>October</td>
<td>74</td>
<td>47</td>
</tr>
<tr>
<td>November</td>
<td>91</td>
<td>55</td>
</tr>
<tr>
<td>December</td>
<td>77</td>
<td>40</td>
</tr>
</tbody>
</table>

Totals    | 580  | 703  |

(1)—From April on.

awakened in me the desire to examine this question more closely although I was not there for this purpose. Besides this statistical evidence I ascertained that the number of deaths from diarrhoea and enteritis was enormous. To have an idea of this it is enough to scan chart B briefly.

In 9 months of 1923 there were 580 deaths from this cause, and in the same number of months 561 in 1924, while during the whole year there were 703. The connection between diarrhoeas and inflammatory disease of the intestine on the one hand and dysentery on the other are now well-known. Many of them, even outside of the time of epidemics, are atypical forms of dysentery.

BAERTHELIN and HÜNEWALD, for instance, were able to isolate dysentery bacilli in 21 out of 72 cases of inflammatory disease of the intestine in children. Post-mortem examination of these cases did not show the typical lesions of dysentery, the lesions being limited to epithelial exfoliation, punctiform haemorrhages in the mucous membrane of the thick intestine which was covered in its whole extension by mucous exudation. These were therefore atypical cases, both clinically and from the pathological point of view, of real dysentery.

STRAUSS obtained positive agglutination in cases in which the disease had always begun in an atypical way (haemorrhagic or not haemorrhagic colitis), developing insidiously and lasting weeks and months; in other cases there were only symptoms classified clinically as "gastro-dyspeptic". FRÆNKEI in 44 of 90 cases studied, with mucosanguinoilent diarrhoea and little fever, found dysentery bacilli in the faeces.

Already in 1903 the American workers DUVAUL and BASSET, studied the aetiology of the diarrhoeas of children and found that out of 63 cases, 52 had dysentery, while MARTHA WOLLS-
TEIN, out of 114 children with the same disease, found that 39 cases were of dysenteric origin. Still about the same time FLEXNER found 299 out 412 cases examined with dysentery.

![Chart B]

Deaths from diarrhoea and enteritis during 1923 and 1924.

In the hot season the number of functional disturbances of the intestine increases as is to be seen from the intestinal flow which is most marked in the diarrhoeas which the Germans call summer-diarrhoeas (Sommer diarhoe).

DUNN, at the same time as DUVAL and BASSET, reported dysentery in 61 out of 160 children with diarrhoea. WEATHER, TUNNICLIFE, HEINE-MANN and MICHAEL also found positive serum-agglutination against dysentery bacilli in many cases of diarrhoea in children. The results of KENDALL and DAY must also be mentioned; out of 103 cases of ordinary diarrhoea 5 were shown to have dysentery, while KENDALL alone studying cases of severe diarrhoea in infants of less than 2 years found that 75 % (39 out of 52 cases) were of dysenteric origin. This
same worker, one year before, had found
dysentery bacilli in 16 out of 146 cases
of diarrhoea, that is 12.3 %.

BOWDITCH also, examining cases of
intestinal disturbances in babies with
symptoms of indigestion or ileocolitis,
found that some were due to dysentery.

Similar observations upon adults or
children are reported by KEUPER,
STRAUSS, FRÄNKEI, LEVADITI and
NICOLAS.

During the years of 1921 and 1922
I made an enquiry, together with DR.
GOMES DE FARI A, as to the cause of
the diarrhoeas of children in Rio de
Janeiro. Two series of cases were stud-
ied: the first was composed of 77
cases, without any selection, with a sim-
ple intestinal discharge containing blood
or not. In this series 15, i.e. 19.2 %,
were found to harbour dysentery bacilli.
In the second series of 31 children only
those cases with a well-defined dysen-
teriform symptomatology were taken and
permitted the isolation of dysentery ba-
cilli from the faeces in 13 cases, i.e.
in 42 %. In the same years in Australia,
BEARE found that ot of 300 cases of
diarrhoea 25 % were of dysenteric ori-

From all this one may judge of the
frequency of atypical forms of dysen-
tery. The technical conditions under
which all this research-work has been
carried out, including ours, may well be
criticised and the results may stand for
only a part of the truth, owing to the
difficulties of putting this sort of work
into practice. Besides laboratory rules,
which are relatively easy to observe, exa-
nmination ought to be repeated in each
case, it ought to be made at the begin-
ning of the disease with early inocula-
tion of culture-media, avoidance of in-
testinal disinfectants, collection of faec-
es directly from the intestine, precau-
tions which have not been carried out
as a whole in any of the works I have
quoted.

In Bahia I was able to make sure
that the Shiga type of dysentery pre-
vails, so that malignancy and consequent
mortality of the disease is at its highest.
The diarrhoeas and enterites produced
by this bacillus, atypical forms of dysen-
tery, but nevertheless genuinely of
dysentery (dysenteria vera), show the
same coefficients of mortality as typical
dysentery. The sanitary statistics of the
state do not show the number of cases of
disease corresponding to the deaths
from these causes.

Allowing a monthly average of 60
cases, like that of the first 3 months of
1924, for the term of 1923 which I did
not obtain (Table IV), we should have
580 + 180 = 760 as the probable number
of deaths from these intestinal affections
in 1923.

By calculation of the corresponding
coefficients the following is obtained:
in 1923 there was a mortality of 2.3 for
every thousand inhabitants, if we accept
the population of the town as being fi-
xed at 320,000, as is done by the De-
partment of Statistics of the Public He-
alth Service of the State, and in 1924
of 2.5 for every thousand inhabitants.

Consequently there was a rise in the
coefficients as compared with the pre-
vious year, just as in the case of dysen-
tery the coefficients of which were of
0.046 in 1923 and of 0.09 in 1924 for
every thousand inhabitants.

The graphic showing the mortality
through diarrhoea and enteritis accom-
panies that of dysentery, which justifies
my point of view in establishing a con-
nection of cause in the appearance of
these diseases.

If there is no doubt as to the con-
nection between the cases of diarrhoea
and dysentery, it is evident that the
number of cases of dysentery should
be increased, by adding up to it a cer-
tain percentage of the cases of diarrhoea
and enteritis.
The statistics furnished by the Health Department of the State give no information as to the clinical characters of the diseases causing death through diarrhoea and enteritis. We must therefore take it that diarrhoea stands for syndromes of various causes, including dysentery and making up a group similar to the group of 77 cases which it has been said that I studied and consequently I will allow that these cases are of dysentery in the same proportion as those that I studied, that is in 19.2, or, practically, in 20% of the cases. For the enterites which imply an inflammatory state of the intestinal mucous membrane we may allow the coefficient 1 obtained for the second group studied of 31 cases, with dysenteriform symptoms which was of 42, or, practically, of 40%.

As the diseases were not sorted out according to their clinical characters in the evidence furnished by the Public Health Department, they must obviously be of a mixed nature: so we may adopt a mean percentage between the two averages of 20 and 40%, obtained in our results, or, in other words, that of 30%. This average, as has already been pointed out, refers to children, but so does the greater number of deaths from diarrhoea and enteritis in the city. Of the 580 cases shown in Table V, 454 were of children of less than 2 years of age or of children in the first stage of infancy and 126 from this age on; of the 561 of 1924 (up to September), 436 were of children in their first childhood and 125 of this age or more.

This once established, I summed up 30% of the 760 cases of death from diarrhoea and enteritis during 1923, that is, 228 cases, which taken together with the 148 notified as dysentery make up 376; and 30% of the 703 of 1924, or, in other words, 210 summed up with the 291 cases notified as dysentery bring up to 501 the probable number of deaths from dysentery during this year.

Making use again of the overrated coefficient of mortality of 80% we obtain as 6266, the probable number of cases of dysentery occurring during 1924.

During my stay in Bahia I asserted that the epidemic of dysentery appeared to me more considerable than that of typhoid fever, although dissimulated.

The points I have just exposed do not seem to confirm this opinion of mine, which I take back in all good faith.

2—The epidemics.

Table I shows the great increase in number of cases of dysentery since 1908. This increase made itself felt repeatedly in relatively short lapses of time, 1908, 1913, 1920 and 1923—1924.

During the two years following an epidemic, the number of cases is still considerable.

Uncertain conditions brought about the great epidemic of 1908.

It is easily understood that the patients fill a town with germ-carriers, who are often persons having overcome an infection and who go on spreading the disease and keeping up the virus between the times of epidemics. Possibly the majority of germ-carriers may be children, whose severe and frequent diarrhoeas and enteritis are in part caused by dysentery. Chart C show a narrow correlation between diarrhoeas and enterites and dysenteries. Enterites and diarrhoeas increase before the dysenteries, as if heralding them. These benignant or atypical cases are possibly responsible for the spreading of the virus, as in yellow fever, malaria and other diseases, in which it is children that bring this about. There can be no doubt as to the frequency of sporadic cases of dysentery, leaving its imprints on the blood, as was shown by LÖ-
Dotted line—Deaths from diarrhoea and enteritis.
Unbroken line—Deaths from dysentery.
WENTHAL, who found them 130 times in 628 serums which he tested and which had been brought to his laboratory for various diagnoses during 1911, a year in which only 5 cases of dysentery had been notified in the town. The number of germ-carriers, left over from epidemics or appearing sporadically is very variable, as is shown by statistics.

In contrast with typhoid fever, in which the bacillus is ordinarily harboured in the gall-bladder of carriers, the dysentery bacilli are kept up in ulcerations of the intestine.

Dysentery is very wide-spread among the poorer population in Bahia.

The blood of inhabitants of the Açude (Dam) do Queimado, all of them poor people, workmen, day-workers, newspaper-sellers, servants, enabled me to obtain positive agglutination in many cases when tested with dysentery bacilli.

Many of these persons who gave positive serum-reactions, reported no history of a typical dysentery, whilst others had had some simple diarrhoeas and yet others repeated colites. I tried several times to isolate the bacillus from the intestine of some of them, but, without success, either because there were no more in the intestines or because the moment was not opportune.

Table IX shows that the total number of agglutinations was higher for the Shiga Bacillus, which shows that this is the most wide-spread and proves that it was also responsible for the epidemics before 1924.

3—Means of Transmission

It is difficult to reconstruct the sanitary conditions of the town during the great epidemic of 1908, when the only evidence at hand is the number of deaths. In any case, the great number of deaths, which permits one to suppose that there must have sickened 600 persons and the fact that at the time there was no regular water-supply in the city, which obtained its water from the ancient and exposed Açude (Dam) do Queimado, supported by that of Matta Escura and distributed by many public fountains, make it possible that the epidemic should have been due to the water. I have no other grounds for assertion.

In the epidemic of 1924 the water does not seem to have played a part in the transmission. If the water should have been at fault, it appears to me that that could only have been at the Açude do Queimado. Round this reservoir, which had been abandoned, but which, on account of special circumstances, came again into use in that year, there lives a population of about 1500 persons, unenlightened and poor, and dwelling in huts without canalisation or any other sanitary fittings. This population contaminated the waters of the reservoir with typhoid bacilli and provoked an epidemic of more than 800 cases in the town. A study of the distribution of the cases of this epidemic, showed that the parts of the town which did not make use of this water suffered nothing or next to nothing from the disease. The same did not happen with the cases of dysentery as may be seen from table V.

In the quarters of Penha and Mares, for instance, which were spared from typhoid fever, as they did not make use of the water which had been contaminated, 24 and 25 cases respectively of dysentery were registered. From the suburban zone 19 cases of dysentery where notified, although the contaminated water was not made use of and only 1 case of typhoid fever was furnished, which came from Pirajá and was admitted to the Hospital S. Isabel. Establishments like the Hospicio São João de Deus, which dispose of their own wells, did not report a single case of typhoid fever, but the Hospicio São João de Deus alone
### TABLE V

Distribution of cases of dysentery according to Districts

<table>
<thead>
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<th>MONTHS</th>
<th>SÉ</th>
<th>S. Pedro</th>
<th>Sant’Anna</th>
<th>Conceição</th>
<th>Pilar</th>
<th>Rua Passo</th>
<th>Sto. Antônio</th>
<th>Victoria</th>
<th>Brotas</th>
<th>Penha</th>
<th>Mares</th>
<th>Nazareth</th>
<th>Suburban</th>
<th>Hosp. Sta. Isabel</th>
<th>H. Isolamento</th>
<th>H. S. João Deus</th>
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</tr>
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<td>—</td>
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<td>1</td>
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<td>1</td>
<td>—</td>
<td>7</td>
<td>7</td>
<td>24</td>
<td></td>
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<tr>
<td>September</td>
<td>1</td>
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<td>3</td>
<td>3</td>
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<td>5</td>
<td>1</td>
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<td>2</td>
<td>—</td>
<td>16</td>
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<tr>
<td>October (Up to 11)</td>
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<td>—</td>
<td>3</td>
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<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>6</td>
<td></td>
</tr>
</tbody>
</table>

**Totals**: 15 8 7 2 11 3 45 34 25 24 24 8 19 27 1 34 286

Contributed 34 cases of dysentery up to September. Up to this date, the Hospital S. Izabel had 23. In this hospital I am informed that all the cases of typhoid fever became ill outside and were then admitted; the same cannot be said with regard to dysentery. The appearance of dysentery in patients admitted for other diseases I was able to ascertain in the case of the patient MARC. FONS. This case I had examined in the Isolation Hospital of Mont Serrat; his observation is the following:

"MARC. P. FONS., male, 37 years of age, of brown colour, Brazilian nationality and residing at the rua da Calçada. He says that he was admitted to the Hospital S. Izabel on Aug. 4th, with an affection of the skin of the back of the hand like a rupia and extending to the dorsal surface of the middle finger. Besides this he has destructive lesions of the uvula which remind one of leishmaniosis.

On the 12th he awoke with intestinal colics, which became more frequent during the day, at which time nausea, head-ache and fever set in. The faeces
at once became muco-sanguinolent and were expelled with contractures (tenesmus). As his condition did not improve next day he was removed to the Isolation Hospital where I found him three days later.

Examination shows, besides the two lesions already spoken of, an emaciated subject, shrivelled and dry-skinned, with a depressed facies, hollow-eyed, with an excavated abdomen which is sensitive to pressure. The descending colon feels hardened and there is a thickening of the lower right side of the abdomen, where gurgling noise is to be perceived. The patient was in a state of great depression and refused nourishment.

The temperature chart (Chart D) showed that there was fever, which persisted, the pulse was weak and rapid.

I did not return to the Isolation Hospital until this man's death which took place on the 26th of the same month and on the 14th day of illness.

The term of incubation of dysentery is very short and does not usually exceed 3 days. As the patient was admitted to Hospital on Aug. 4 and only felt ill on Aug. 12th, it is evident that he must have got contaminated during the 8 days he spent in hospital on account of a very different affection.

Hospital infections have been seen before in the case of dysentery. DreSEL, for instance, reports an epidemic among the staff of a hospital. Agglutination tests practised on the serum of 66 members of the staff that had not sickened, gave positive results in 33%, i.e. in 21 amongst them. One

Diagram of the dysentery epidemic during the years 1923-1924.
In white—cases notified.—In black—deaths.
of the nurses, whose serum gave a positive reaction and who felt perfectly well, became ill with dysentery a month later. It is possible that the patient studied should have been another case of protracted incubation, but it seems far more likely, that he should have got contaminated within the hospital, since there were patients of dysentery in it enough to infect the remaining patients.

Against the origin of the epidemic from water-supplies are also the gradual curve of the disease (chart E) and the number of cases (Table VII).

**TABLE VI**

Number of cases notified and of deaths by dysentery from April to December 1923 and during 1924, in Bahia.

<table>
<thead>
<tr>
<th>Month</th>
<th>1923 Notification</th>
<th>1923 Deaths</th>
<th>1924 Notification</th>
<th>1924 Deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td>January</td>
<td></td>
<td>36</td>
<td></td>
<td>36</td>
</tr>
<tr>
<td>February</td>
<td></td>
<td>33</td>
<td></td>
<td>32</td>
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<tr>
<td>March</td>
<td></td>
<td>23</td>
<td></td>
<td>22</td>
</tr>
<tr>
<td>April</td>
<td>29</td>
<td>26</td>
<td></td>
<td>47</td>
</tr>
<tr>
<td>May</td>
<td>36</td>
<td>34</td>
<td></td>
<td>49</td>
</tr>
<tr>
<td>June</td>
<td>16</td>
<td>16</td>
<td></td>
<td>26</td>
</tr>
<tr>
<td>July</td>
<td>9</td>
<td>9</td>
<td></td>
<td>27</td>
</tr>
<tr>
<td>August</td>
<td>8</td>
<td>8</td>
<td></td>
<td>24</td>
</tr>
<tr>
<td>September</td>
<td>6</td>
<td>4</td>
<td></td>
<td>16</td>
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<tr>
<td>October</td>
<td>5</td>
<td>4</td>
<td></td>
<td>11</td>
</tr>
<tr>
<td>November</td>
<td>21</td>
<td>21</td>
<td></td>
<td>8</td>
</tr>
<tr>
<td>December</td>
<td>35</td>
<td>26</td>
<td></td>
<td>12</td>
</tr>
<tr>
<td><strong>Totals</strong></td>
<td><strong>165</strong></td>
<td><strong>148</strong></td>
<td><strong>312</strong></td>
<td><strong>291</strong></td>
</tr>
</tbody>
</table>

Chart E shows that there were many cases and that these spread over several months and that they were more pronounced in the first months of the year. As dysentery is a disease as infectious as typhoid fever when placed under the same conditions, it is easy to understand that if the water from the Açude do Queimado had distributed dysentery through the whole town like it distributed typhoid fever, the epidemic of dysentery should have taken place in the same way as that of typhoid fever, that is, with a sudden outbreak and a great number of cases within a short time. This was not the case, as chart E will show.

The transmission of bacillary dysentery by water is very infrequent, not to say exceptional. VINCENT (quoted from LENTZ) asserts that the bacilli may be kept alive 10 to 12 days in distilled and sterile water, while in impure water they die off in 2 to 6 days. KARLIŃSKI (quoted from LENTZ) kept them alive in water taken from the tap during 71 days; his results were confirmed in part by WINTER.

The infrequency of epidemics of dysentery from the contamination of water had already been pointed out by LENTZ. This author states that such epidemics as are caused by the contamination by water and in which it is possible to isolate the bacilli from the water, are isolated facts referring mostly to small streams, polluted by receiving faeces as a whole.

It is well known that the small volume of water in brooks makes a process of natural purification very difficult on account of the rapidity with which the polluted water is taken to the dwellings; on this account the sanitary conditions of these small streams are always very deficient.

It is only in one particular that I think that water may play a part in bringing about a certain number of cases, and that is in the case of water kept for domestic use. The shortage of water, which had up to then been distributed to the population, obliged the people to make use of clay receptacles. When there was more than one of these, as was frequently the case, they would be lined up in the kitchen in rows, into which the cook would dip her can every now and then as the need arose, and into which the remainder of the household would dip a ladle made from
the shell of the cocoanut when they wished to drink. The poor people do not usually have separate vessels.

When typhoid fever made its appearance the newspapers made intense propaganda in favour of the use of boiled water. This was easily accepted and pretty nearly wide-spread, and it is a measure which it seems to me must greatly reduce the number of cases of dysentery.

Chart VI already shows this fall, towards the last months of 1924, as compared with the same term of 1923 and just in the hot weather when the number of cases always increases.

Once the question of drinking water is laid aside I might rapidly scan the other channels of indirect infection so as to come afterwards to the direct contagion.

Flies have been incriminated with the transmission of dysentery. It would be unnecessary to quote writers to demonstrate this. The amount of flies in Bahia is small however as compared to the number of them in Rio de Janeiro and Sao Paulo, a fact which struck me right at the beginning of my stay. There a great many of them near the incinerating furnace where a good deal of rubbish is kept waiting to be burnt and in the stables. In the town I saw no flies. I went through many parts of the Low City and the High City and only found them in the afore-mentioned places. There was some trouble about obtaining flies when I wanted to attempt some experiments as to the part played by flies in the transmission of typhoid fever. Even if it were allowed that they might be responsible for a certain number of cases, and I do not think this could be done for many cases, flies could hardly be held responsible for the cases in the quarters where there are very few of them.

Vegetables are very unlikely to aid in the transmission of the disease. If water plays an unimportant part in it, vegetables are of still less importance and RUSS & WEIHE and JEHLE merely say that they might be thought of (denkbar) in the transmission of dysentery. Dysentery bacilli are particularly sensitive to heat; while it is necessary to keep typhoid bacilli at 58 to 60°C, during an hour for sterilisation, the dysentery bacilli, above all the Shiga bacillus, are unable to hold out more than 10 or 15 minutes at this temperature. The healing of the souces would be sufficient to kill them. Besides this they are also less resistant than typhoid bacilli against drying. The use of raw vegetables is not very wide-spread among the people of Bahia, as I am able to testify from an enquiry made within their homes with a view to ascertaining the origin of typhoid fever.

There remains direct contagion, if we take it that this means infection through food-stuffs polluted by the contaminated hands of patients and germ-carriers or by the hands of healthy persons that have touched particles of faeces or objects soiled with faeces containing dysentery bacilli.

The multiplicity of these pollutions is very great and the repetition of the contagion enormous. That dysentery is more wide-spread among the poor and unenlightened is well-known; like exanthematic typhus, it is considered a disease of filth.

Besides this, contagion in this manner is not only frequent but also immediate, i. e., the germs find themselves again in the intestines shortly after having been expelled and before they have undergone the sanitary influence of the outside world.

Another factor which makes it easy for a great number of cases to arise in this way is the repetition of the contagion which increases the number of bacilli swallowed. As it is known that the conditions of individual resistance
fluctuate, the repetition of contagion allows of infection at the moments of less resistance.

It has already been seen shown, when speaking of germ-carriers how wide-spread the disease is among the poor classes of the town.

Among the inhabitants of the banks of the Açude of Queimados there are people employed as cooks, waiters, washerwomen and so forth in town. It is easy to understand how these may act in the spreading of the disease to the collectivity.

II TECHNICAL METHODS.

A. Conditions of Research

My work was directed towards the bacteriologic demonstration of dysentery in the faeces and blood of the patients. In reconvalescents I limited myself to serum-tests for diagnosis.

The usual technical methods for the demonstration of dysentery bacilli in the faeces of patients with symptoms of the disease have given discouraging results in the last years. Some writers have even ventured to doubt the value of these methods, while others have attributed the cases they studied to other causes of sickness, such as heat, spoilt food-stuffs or other unknown causes. I might quote as an instance the paper of FRIEDMANN and STEINBOCK (1916). These writers in 335 examinations they carried out obtained positive results only in 29, i.e. in 8.6 %, a fact which they attributed to various reasons. ARONSON'S results are also quoted in literature with its 1133 examinations and 299 i.e. 26 % positive results. Another writer, who obtained precarious results in his work and tried to incriminate an acidophile capsulated bacillus for enteritis, was CZAPLEWSKI (1917). FRIEDMANN, in 1917 also, mentions very feeble percentages obtained by HIRSCH, HISS, GALAMBOS, KOLLE and DOREMBERG, besides his own work during 1916 and 1917, which gave results fluctuating between 5 and 11 % and rising up to 20 and 25 % during summer.

KOLLE and DORENDOFF, in over 1,000 attempts only cultivated dysentery bacilli 6 times, and they even cast doubt on the responsibility of these bacilli for the epidemic of dysentery in Galicia where they worked.

MEYER, ARNETH, HANDMANN, KÖHLICH all of them report poor results in 1916.

The greater part of these workers attributes the insuccess of their work to deficient collection and transportation of the material, to unrepeated examination and so forth.

FRIEDMANN himself, however, allows that with faeces taken directly from the rectum, he always obtained positive results, and he even thinks that this is an indispensable condition for success.

The repetition of examination was successful very often in the hands of SCHILD (1914). This writer mentions a case in which only the fifth attempt ensured successful cultivation of the germ. The best results he obtained in the first days of the disease and when the material was inoculated in culture media immediately after its collection. He also mentions the work of JACOB who obtained 1 50 % positive results in the first days of the disease and only 11 % later.

A low temperature keeps up the vitality of the bacilli. LENTZ states that in winter the bacilli hold out longer than in summer and quotes SCHMIDT'S experiment, keeping the bacilli alive for two months on ice. STREMPPEL (1920) is of the same opinion and quotes the observations of GRUBER and SCHADEL, who were able to isolate dysentery bacilli from faeces sent by post but kept sufficiently cool. He also mentions the experiments of BREINL and
SCHWERINGER who left dysenteric faeces in the incubator and in the refrigerator, and obtained 24 hours of life more in the latter than in the incubator, as well as those of HANDMANN and STARCKER who had similar results. I have more than once examined faeces sent by post without cooling with constantly negative results, although they came from a zone in the centre of the State of Minas Geraes in which there was an epidemic outbreak, and, where at the same time and place DR. COSTA CRUZ had isolated the Shiga bacillus, on the spot.

I was able to isolate on ENDO and DRIGALSKI media in Peri dishes a lot of colonies of the Flexner bacillus by inoculating the mucous substance from an intestine with the lesions of dysentery which had been kept in the refrigerator of the autopsy building of the Hospital S. Francisco de Assis in Rio de Janeiro and which I made use of at the request of the Pathology Department of this Institute. The colonies were as plentiful as if they had been isolated from dysenteric faeces. Another factor which is given a great deal of importance is inoculation of culture-media immediately after the collection of material. By inoculation of culture-media at the bedside, immediately after the discharge of faeces, BREINL obtained 95% positive results in the 70 patients examined. FRIEDMANN obtained similarly favourable results.

Nevertheless, some cases were failures with this method and STREMPHEL attributes this fact to the bactericidal properties of the blood which was plentiful in those cases. In some cases of dysentery haemorrhage reaches such a point that the patient discharges almost pure blood. In these cases of intestinal bleeding the examination suffers. Twice I have examined faeces in this state, one case being from the Hospital Pedro II and the other from the Policlinic for children, with negative results in spite of the fact that both cases were still in the first days of illness. Another case from Bahia, a patient of DR. ADRIANO PONDE had faeces in this state and from them I was only able to obtain strains of a slow-fermenting Bacillus coli of which I will say more anon. I was unable to obtain serum to test the GRUBER-WIDAL phenomenon, nor any more faeces, as the patient died soon afterwards. The patient had all symptoms of dysentery, including the lowering of temperature, which may be considered one of the most characteristic symptoms of the disease and which follows a term of fever.

It is believed that it is in the struggle for life against the other germs present in the intestine, that the dysentery bacilli die out more or less rapidly in the faeces. Cooling inhibits the activities of these competitors.

The examination in the first days of the disease is very important. SCHWERINGER examining the faeces in the first 4 days obtained 57% positive results; from the 4th. to the 12th. day this percentage fell to 28 and only after this to 12%. JACOB, quoted from STREMPHEL, obtained dysentery bacilli in 50% of the cases and later only in 11%.

MARTIN and WILLIAMS, quoted from BEARE, inoculated culture media with faeces in 1918; when making use of faeces of the 4th. to 10th. days they obtained 17% positives, from the 11th. to the 15th. days 6.3%; from the 16th. to the 50th. 0.3%.

BEARE in 1921—1922, counted up to the 4th. day 82%; up to the 8th. 37%; up to the 12th. 33%; up to the 16th. 17%; up to the 20th. 14% and up to the 24th. 13% positive results.

B. CULTURE-MEDIA

Culture-media employed in the isolation of dysentery bacilli do not differ much from those used for the typhus
group, with which dysentery bacilli are in many ways connected.

The most used media up to now have been those of DRIGALSKI and of ENDO.

The former must not contain chris-
tal-violet as this dye has a certain inhibi-
tory action on dysentery bacilli.

In the work done up to now I have always had satisfactory results from the use of these media. Besides these I have also availed myself of the medium of HOLT-HARRIS and TEAGUE, without cane-sugar. If this sugar is placed in the medium, as is advised by HOLT-
HARRIS and TEAGUE, it becomes im-
possible to isolate certain types of the Bacilli described by MORGAN, on ac-
count of their fermentative action on cane-sugar which renders them indistin-
guishable from the B. coli. These ba-
cilli offer a certain interest to bacterio-
logists studying intestinal pathology.

I had not up to then made a compar-
ative systematic study of these dif-
ferent media, so that I could make no definitive assertions as to the advan-
tages of any of them. The work carried out in Bahia permits me to advance something on this score, which I will do further on.

It is now well known that the most important point in the cultivation of bacteria, in their preservation, toxin-pro-
duction and so forth is the hydrogen-ion concentration of the medium, expressed by its pH.

This concentration had not been tit-
trated up to now for dysentery bacilli, but I have already for some time regu-
lated my culture media at pH 7.5 with satisfactory results in the cultivation and pres-
ervation of dysentery bacilli. The method used for the titration of the hy-
drogen-ion concentration was the inter-
polation method of GODOY, which sol-
vess the problem simply by the use of formulæ, once the reaction of the me-
dium against phenolphthalein and methyl-
orange is known.

With this concentration I prepared several media for the isolation of bacilli of the typhus group, which I then need-
ed, and I made use of the same for the dysentery group.

These culture media included, be-
sides the three that have been mentioned, the agar-bile salts medium of MC CON-
KEY, which is meant specially for the typhus group.

The appearance of the colonies on PETRI dishes with MC CONKEY's me-
dium is entirely similar to that of the typhus bacillus, for which they may be mistaken. There does not seem to be any advantage in making use of this medium, which does not however involve any technical difficulties. The isolation of dysentery bacilli on bile culture media cannot cause any surprise since NO-
WICK isolated, from the blood, bacilli of the Y type in culture media enriched with bile. BEARE had used the same medium in 1922 for the same purpose. But if KNORR's experience is to be taken into account the use of culture media with bile should not be advisable.

In an attempt to cultivate dysentery ba-
cilli on bile alone, KNORR obtained constant multiplication only with the Y type; the remainder, including the Shiga bacillus, would grow a certain number of times, but would mostly die off.

What I would like to emphasize is the absolute similarity of results on all these culture media, which, it seems to me, is entirely due to the uniform hydrogen-ion concentration of the media, the pH of which was 7.5 as has been stated.

If one medium must be selected, preference should be given to that of HOLT-HARRIS and TEAGUE, the prepa-
ration of which is very economical. The difficulty about this medium is that of the methylene-blue, upon the quality of
which depends the appearance of the medium after it is poured into the PE-
TRI dishes. I have tried several brands of methylene-blue and have obtained
good results only with those of HOE-
CHST and of POULENC FRÈRES, the
latter when it has «pour la micro-
copie» on the label. Some other makes
of methylene-blue pre-cipitate when pou-
red into the agar.

The following is a summary of the prepara-
tion of this medium:

Meat broth. . . . . . 2,000 c. c.
(or 20 grms. of beef-extract in 2,000 c. c.
of distilled water) (1).

Peptone. . . . . . . . . . 20 gms.

Sodium Chloride. . . . . . . . . 10 gms.

Agar. . . . . . . . . . . . 60 gms.

Dissolve while heating, reduce the
acidity with a little soda, heat up to
120°C. during 5 minutes, so as to pre-cip-
itate the salts and stabilise the ion-
concentration, fix the hydrogen-ion con-
centration at 7.5; interrupt the high tem-
perature; add white of egg and heat up
to 110°C. for 10 minutes to clear the
medium; strain filler through flannel.
Distribute in balloons of 200 c. c. and
sterilise at 115°C. during 15 minutes;
keep in the dark. When about to use the
medium, place 2 grms. of lactose in
each balloon and heat up to 108°C. for
5 minutes, so that the agar is melted
and the lactose sterilised (2). Next 4
c. c. of a 0.5 % solution of methylene-
blue and 4 c. c. of a 2 % solution of
yellow eosin are added. These solutions
need not be prepared with sterilised wa-
ter and will last indefinitely.

C Diagnosis of the Isolated Germ:

Three orders of more usually prac-
tised examinations are sufficient for the
determination of dysentery bacilli. These
are as follows: a) study of the colonies
grown in PETRI dishes on suitable me-
dia, including observation of their mor-
phology, colours. and the morphology
and motility of the bacilli which make up
these colonies; all this might be cal-
ed evidence of probability in the de-
termination; b) the testing of the biolo-
gical properties of the isolated germ;
and e) agglutinating properties when mixed
with specific sera.

1. EVIDENCE OF PROBABILITY.

The appearance of the colonies of
dysentery bacilli does not differ prac-
tically from that of the typhus group on
the media commonly used for the iso-
lation of these bacteria.

The concomitant presence of dysen-
tery and typhus which has already been
seen, although rarely, or that of more
than one type of dysentery bacillus, or
that of read dysentery and para-dysen-
tery bacilli, bacilli of MORGAN and so
on, which are relatively frequent, be-
sides the possibility of a colony being
contaminated with B. coli, for instance,
make it advisable to isolate more than
one colony from the PETRI dish. The
most typical and the colonies standing
isolated should be preferred.

Another important factor in this part
of the diagnosis is the motility, about
which it is sometimes difficult to make
sure. Dysentery bacilli are not motile,
but owing to their small size, specially
in some samples, they show very vivac-
cious brownian motion, which may be
mistaken for motility.

Besides this, certain organisms,
which do not ferment the culture media
used for isolation, are very little motile,
that is, only a few of those under ob-
servation betray their motility. On this
account it is advisable to examine both the periphery and the centre of the hanging-drop, and to run through many microscopic fields before making an assertion.

Here it should be pointed out that there may appear colonies of Bacillus coli or B. lactis aerogenes, in every way similar to dysentery bacilli and which do not modify or ferment the sugar of the culture medium used for isolation in the first 24 hours after inoculation, but only after 48 hours or more. These are 'colt bacilli of slow fermentation'. These germs are of interest and have their value in making it probable that there should be or that there should have been of late concomitant dysentery bacilli in the intestine from which they were isolated. Besides their non-motility, they co-agglutinate to a large degree, so much so that they may be the cause of a false result for the examination, the more so if the study of the germ was not completed by lengthy observation of the biological tests.

The importance of the knowledge and study of such organisms and its bearing on the accurateness of diagnosis are self-evident, as is the necessity of repeating tests on the faeces and serum of the patients until positive proof is obtained.

I will undertake the study of these organisms further on.

2. BIOLOGICAL PROOFS.

I am in the habit of subjecting the samples isolated to a series of tests as to their characters when cultivated on well-known diagnostic culture-media, which are previously prepared in the laboratory and which are as follows:

1. ROTHBERGER's medium, modified by OLDEKOP.
2. Sterilised milk.
3. Peptone water (for the indol test).
4. Media with sugars (glucose, lactose, saccharose, mannite and maltose) added to agar with yeast.
5. PETRUSCHKY's milk-serum.

Besides this I inoculate a tube with simple slanting agar so as to prepare a suspension for agglutination.

So as to obtain enough material for so many tubes and thereby speed up diagnosis, I make a suspension of each colony in about 2 c. c. of sterilised saline solution and distribute 2 drops in each of the tubes of the list I have given.

After 24 hours the results as to fermentation of the sugar and ROTHBERGER's medium are read; the latter ensures the separation of the pseudo-dysentery and paratyphus bacilli. With the suspension from the tube of agar serological tests are made, so that diagnosis is generally ready before the 48 hours which are usually required.

Growth in milk, in PETRUSCHKY's medium and the indol test are complementary proofs. The fermentation of sugars clears up the diagnosis as to the type of dysentery bacillus and the fermentation of lactose excludes the Bacillus coli.

I have already mentioned the finding of Bacillus coli strains fermenting this sugar slowly, so that a lengthier observation of the action on this sugar is necessary, specially when the sample of bacillus is non-motile. Even among the dysentery group there may be found differences as to fermentative action which impair bacteriological diagnosis.

I have mentioned the existence of the HISS and RUSSELL bacilli which do not ferment mannite, when recently isolated and thereby simulate the Shiga bacillus. These organisms after the 1st or 2nd. reinoculation on this medium begin to ferment it, at times slowly, so that fermentation in 24 hours is obtained only after several passages.

This inertia as to fermentation I have also found in the sample of the
STRONG bacillus of the collection of the Institute, obtained from the United States, and which only ferments cane-sugar after several reincubations.

Most bacteriologists who have worked with dysentery bacilli are well acquainted with their ability to ferment or leave unfermented the different sorts of sugars. This is not a very malleable property and as to it I can only record some types of HISS and RUSSELL which transformed themselves into FLEXNER, but these two are so closely related that I consider them to be the same germ. Besides this character of altering their action on sugars and their biological characters, as to which they are quite alike, the serum which is agglutinating for one has the same action on the other.

**TABLE VII**

<table>
<thead>
<tr>
<th>No.</th>
<th>Name</th>
<th>Date</th>
<th>Milk</th>
<th>Robbiger Odaton Medium</th>
<th>Glucose</th>
<th>Lactose</th>
<th>Saccharose</th>
<th>Maloase</th>
<th>Mannite</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>J. Bez.</td>
<td>7-IV-923</td>
<td>No coag.</td>
<td>--</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>Ferments mannite after 48 hours in the 2nd passage. Agg. Shiga serum at 1.640 and Flexner Serum at 1.280</td>
</tr>
<tr>
<td>2</td>
<td>M. H. Mout.</td>
<td>26-III-923</td>
<td>*</td>
<td>--</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>Ferments mannite after 48 hours in the 2nd passage; after this in 24 hours. Agg. Shiga serum at 1:80 and Flexner serum at 1:800</td>
</tr>
<tr>
<td>3</td>
<td>4.031 Hosp. Pedro II</td>
<td>16-V-923</td>
<td>*</td>
<td>--</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>Ditto. Agg. Shiga serum at 1:320 and Flexner at 1:1.280</td>
</tr>
</tbody>
</table>

In table VII are indicated some experiments with bacilli which behaved in this way.

However when HISS and RUSSELL bacilli are thus disguised it is not difficult really to show them up, because almost always they agglutinate at a higher titre with anti-Flexner or Y serum than with anti-Shiga. Sometimes the Shiga bacillus may behave in the same way and strains of this type fermenting mannitol are accepted, which complicates still more bacteriological research, but as a rule this co-agglutination only produces very fine clumps.

In table VIII may be seen the results of the cultivation of dysentery bacilli producing little toxin (oligotoxic), isolated from faeces and cultivated during five months, without any change in their fermentative characters.
### TABLE VIII

Experiments as to the immutability of fermentation of sugars by dysentery bacilli

<table>
<thead>
<tr>
<th>No.</th>
<th>NAME</th>
<th>Date of isolation</th>
<th>Glucose</th>
<th>Lactose</th>
<th>Saccharose</th>
<th>Mannite</th>
<th>Maltose</th>
<th>Anti Shiga</th>
<th>Anti Flexner</th>
<th>Re-inoculated on Mannite and Maltose on</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>X.</td>
<td>16-IV-923</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>0</td>
<td>1.600</td>
<td>Dec. 27-923 Jan. 31-924 Feb. 25-924 April 5-924</td>
<td>It continued unchanged in May of the same year.</td>
</tr>
<tr>
<td>2</td>
<td>And.</td>
<td></td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>320</td>
<td>1.280</td>
<td>»</td>
<td>Do.</td>
</tr>
<tr>
<td>3</td>
<td>Aur. Ros.</td>
<td>5-IV-923</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>640</td>
<td>1.280</td>
<td>»</td>
<td>Do.</td>
</tr>
<tr>
<td>4</td>
<td>M. Conc.</td>
<td>14-IV-924</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>800</td>
<td>»</td>
<td>»</td>
<td>Do.</td>
</tr>
<tr>
<td>5</td>
<td>Alv. Pon.</td>
<td>31-X-924</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>640</td>
<td>1.280</td>
<td>»</td>
<td>Do.</td>
</tr>
<tr>
<td>6</td>
<td>Sampaio</td>
<td></td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>640</td>
<td>1.280</td>
<td>»</td>
<td>Do.</td>
</tr>
</tbody>
</table>
It would be of great value to find out whether the strains of SHIGA bacilli described as fermenting mannite were not really HISS and RUSSELL bacilli similar to the ones that have just been spoken of.

Still as concerns the fermentation of sugars I have found three strains which behaved like the SHIGA type as to fermentation, but agglutinating more with FLEXNER serum, and which I thought to be like the Y types of belated fermentation, so that I undertook the tests with this end in view, as may be seen in table VIII.

These strains were obviously more closely connected with the Y or FLEXNER type, because they produced indol and agglutinated better with anti-FLEXNER serum, which removes them from the SHIGA type as also from that of SCHMITZ, which only agglutinated with its own serum. In practice this tendency to variation is of secondary importance, since these types are all variations from the great group of the types which produce little toxin (oligotoxic), and which cause a much smaller mortality than the toxic type. It is probably due to little-known actions of their environment when in the intestines, where by successive passages from one individual to another, or perhaps under the influence of diet, they acquire or lose their characters.

3. SEROLOGICAL TESTS.

I always made use of the specific agglutinating sera so as to confirm the tests as to the changes brought about by the growth on the series of special culture-media for diagnosis used in the series.

For this purpose I used only agglutinating sera against the SHIGA bacillus and against the FLEXNER, as the latter is known to agglutinate indifferently the FLEXNER or the HISS and RUSSELL bacilli.

The existence of bacilli with certain characteristics enabling them to pass for dysentery bacilli in the series of cultural tests is also seen as regards the agglutinating properties.

It must be observed that these characters are generally evanescent and will disappear after a few reinculations on culture-media. The knowledge of these facts is not by any means new. RIMPAU in 1912 reports strains of the Bacillus coli isolated from the faeces of a patient suspected of dysentery and whose serum agglutinated the FLEXNER-bacillus at 1:100. These strains were agglutinated by the anti-FLEXNER serum and were given the name of «coliflexner bacillus» by the writer who described them, who points out that they lost this property of theirs with reinculation on culture-media.

Similarly CZYLARIZ and NEUSTÄDT in 1914 found strains which gave blue colonies on the DRIGALSKI medium and which subsequently behaved like B. coli and which they called «Paracolis» or «Paracolibacillus», but which were not agglutinated by the serum of the patient himself. These bacilli were made out in about 20 % of the cases.

In 1919 ALMAGIA found a coli which was agglutinated by the serum of patients with the symptoms of dysentery in dilutions of 1:100 and 1:200 and was also agglutinated by anti-FLEXNER serum. From these patients he was in every case unable to isolate dysentery bacilli.

SOMME also described in 1914 a similar bacillus which he took to be an atypical dysentery bacillus, and which was again found subsequently by TJOTTA in Norway and BEARE in Australia in 1922.

I have already mentioned that I found similar sorts of bacteria while engaged in other work, carried out before this one.
<table>
<thead>
<tr>
<th>No.</th>
<th>NAME</th>
<th>Date</th>
<th>Lactose</th>
<th>Glucose</th>
<th>Succharose</th>
<th>Mannite</th>
<th>Maltose</th>
<th>Broth</th>
<th>Petruschky indol</th>
<th>Rothberger Oldekop</th>
<th>Milk</th>
<th>Serum Shiga</th>
<th>Serum Flexner</th>
<th>Patient's Serum</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Mar. Em.</td>
<td>15-1-924</td>
<td>+</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Coag. 1280</td>
<td>160</td>
<td>80</td>
<td>The strain ferments slowly the isolation medium and the lactose and gives yellow pigment on agar.</td>
</tr>
<tr>
<td></td>
<td>(Slow)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>X. (patient of</td>
<td>27-XII-924</td>
<td>+</td>
<td></td>
<td>+</td>
<td></td>
<td></td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>Coag. 2500</td>
<td>80</td>
<td></td>
<td>Do.</td>
</tr>
<tr>
<td></td>
<td>Prof. Figueira)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(Slow)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Luc. Teix.</td>
<td>22-1-924</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td></td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>Coag. 320</td>
<td>160</td>
<td></td>
<td>The culture becomes yellowish with age.</td>
</tr>
<tr>
<td>4</td>
<td>Autopsy 2173</td>
<td>2-XII-924</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td></td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>Coag. 320</td>
<td></td>
<td></td>
<td>Agglutination with delicate clumps. Rapid fermentation of isolation medium.</td>
</tr>
<tr>
<td>5</td>
<td>EURIC. Per.</td>
<td>24-1-924</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td></td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>Coag. 160</td>
<td>160</td>
<td></td>
<td>From this case Morgan bacilli were isolated</td>
</tr>
</tbody>
</table>
It is true that up to now I never made a systematic study of this question, but once and again when the opportunity occurred, I would isolate, at the same time as the colonies of dysentery bacilli, a colony of *Bacillus coli* which would be subjected to agglutination with the same sera as the dysentery bacilli.

In table IX may be seen some of these samples I had isolated in the course of previous work and showing a fairly high titre. In one case (that of no 1) I was able to find agglutinins in the serum of the patient, while in the others no serum was obtained for this test. This sample fermented cane-sugar, which allows it to be classed with the *Bacillus coli communitor*; it did not ferment malt-sugar which shows that it was an atypical strain.

I have observed the existence of other bacilli resembling the dysentery bacilli by their characters. There is some doubt about these bacilli, principally as to their pathogenicity which is of course what matters most from the practical point of view.

A certain number of them have already been separated and more closely studied, while others, although with reservations, are still kept in the dysentery group, as for instance the SCHMITZ bacillus.

Among the better-known, the bacilli of MORGAN take a prominent place. This author brought them together in 1905 on account of a certain number of characters which are common to them and ascribed to them the causation of summer dysenteries. As a matter of fact there does seem to be a correlation between their number in the intestines of children with diarrhoea and the increase of summer diarrhoeas. Evidence supporting this idea is also gained from the great percentage of flies infected with these bacilli during the hot seasons, which, as is well-known, favour the development of flies, and by the reduced percentage of flies infected in places where there were no cases of diarrhoea or only very few. Besides this a rapid increase of flies has been observed preceding epidemic increases of diarrhoea.

These bacilli are often found in healthy children though in a much smaller degree than in those suffering from diarrhoea.

These facts have been observed by many British research-workers.

At the time that I carried out, together with my chief, DR. GOMES DE FARIA, an enquiry into the aetiology of the dysentery of the children of Rio de Janeiro, I was able to confirm, although in a smaller number of cases than those of the British statistics, the observations of MORGAN and his successors, as to the frequency of the bacilli in diarrhoeas of children and the percentage here obtained. I will reproduce here the average figures I obtained in Rio de Janeiro, compared with those of MORGAN and published in 1908 (table X). I also found some types which did not agree with those of MORGAN and which are set apart.

Bacteriologists should be familiar with the MORGAN bacilli, as well as with the different bacilli described either as atypical dysentery bacilli or as non-dysentery bacilli, because it is not always possible to distinguish them from real dysentery bacilli within the first 48 hours.

The colonies, for instance present the same appearance as colonies of dysentery bacilli on the culture-media used for isolation; many of these bacilli are non-motile, do not stain with GRAM’s process, grow in fine delicate cultures in agar, ferment the sugars like the dysentery bacilli and only sometimes produce a yellow pigment, after some time, in this medium, a character which is shared by some strains of dysentery bacilli; some strains do not ferment RO-
## TABLE X

Percentage of Morgan's bacillus as isolated in England (1905, 1906 and 1907) by Morgan Ledingham and in Rio de Janeiro (1921 and 1922) by Gomes de Faría and the writer.

<table>
<thead>
<tr>
<th>Types of Morgan's Bacillus</th>
<th>England</th>
<th>Rio de Janeiro</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Type I</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>40</td>
<td>2.6</td>
</tr>
<tr>
<td>1 A</td>
<td>0.16</td>
<td>11.1</td>
</tr>
<tr>
<td>2</td>
<td>3.4</td>
<td>2.8</td>
</tr>
<tr>
<td>3</td>
<td>7.5</td>
<td>2.8</td>
</tr>
<tr>
<td>4</td>
<td>2.45</td>
<td>0</td>
</tr>
<tr>
<td><strong>Flexner Type</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4 A</td>
<td>7.1</td>
<td>4.2</td>
</tr>
<tr>
<td>4 B</td>
<td>0.96</td>
<td>0</td>
</tr>
<tr>
<td>4 C</td>
<td>0.96</td>
<td>0</td>
</tr>
<tr>
<td>5</td>
<td>6.7</td>
<td>1.4</td>
</tr>
<tr>
<td><strong>Type V</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5 A</td>
<td>1.20</td>
<td>1.4</td>
</tr>
<tr>
<td>5 B</td>
<td>0.16</td>
<td>0</td>
</tr>
<tr>
<td>6</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td>7</td>
<td>5.5</td>
<td>1.4</td>
</tr>
<tr>
<td>7 A</td>
<td>1.8</td>
<td>0</td>
</tr>
<tr>
<td><strong>Gaertner Type</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7 B</td>
<td>1.9</td>
<td>0</td>
</tr>
<tr>
<td>7 C</td>
<td>1.2</td>
<td>0</td>
</tr>
<tr>
<td>8</td>
<td>0.56</td>
<td>0</td>
</tr>
<tr>
<td>9</td>
<td>0.56</td>
<td>0</td>
</tr>
<tr>
<td>10</td>
<td>3.2</td>
<td>4.2</td>
</tr>
<tr>
<td>11</td>
<td>4.8</td>
<td>0</td>
</tr>
<tr>
<td><strong>Type X</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11 A</td>
<td>2.9</td>
<td>1.4</td>
</tr>
<tr>
<td>11 B</td>
<td>0.96</td>
<td>1.4</td>
</tr>
<tr>
<td>11 C</td>
<td>1.4</td>
<td>2.8</td>
</tr>
<tr>
<td><strong>Type XII</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>1.1</td>
<td>0</td>
</tr>
<tr>
<td>13</td>
<td>3.5</td>
<td>0</td>
</tr>
<tr>
<td>13 A</td>
<td>1.9</td>
<td>0</td>
</tr>
<tr>
<td>14</td>
<td>4.18</td>
<td>8.4</td>
</tr>
<tr>
<td>14 A</td>
<td>4</td>
<td>19.7</td>
</tr>
<tr>
<td><strong>Type XIV</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>14 B</td>
<td>3.16</td>
<td>2.8</td>
</tr>
<tr>
<td>14 C</td>
<td>2.28</td>
<td>8.4</td>
</tr>
<tr>
<td>14 D</td>
<td>0.16</td>
<td>8.4</td>
</tr>
<tr>
<td><strong>Type XV</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>15</td>
<td>0.5</td>
<td>1.4</td>
</tr>
<tr>
<td>15 A</td>
<td>1.6</td>
<td>0</td>
</tr>
<tr>
<td>15 B</td>
<td>2.1</td>
<td>1.4</td>
</tr>
</tbody>
</table>

**NOTE** — Besides these, we found other types which could not be placed among those described by Morgan.
THBERGER's culture medium, coagulate milk slowly or not at all, and ferment PETRUSCHKY's medium, some of them slowly.

Greater confusion is added by the serological tests, which are once and again positive, because there may be a considerable amount of coagglutinins. During the time I worked with these bacilli I found once and again strains which agglutinated up to 1:5,000 when mixed with SHIGA or FLEXNER serum, as may be seen in table XI which shows some of them, obtained in 1920.

**TABLE XI**

Results of agglutination of Morgan's bacilli isolated from the faces of children with diarrhoea or with dysentery, in Rio de Janeiro when mixed with dysenteric agglutinating sera.
formation of large clumps, and with anti-Flexner up to 1:320 ± 1:640, with formation of fine clumps. This sample had been obtained from the clinical service of PROF. FERNANDES FIGUEIRA, at the Policlinic for Children, and from this case I isolated on Dec. 29, 1923 the SHIGA bacillus which agglutinated with anti-Shiga serum at 1:2,500, and ± at 1:5,000 and with anti-Flexner serum at 1:1,280 and ± at 1:2,500.

Not all samples isolated from the same case show the same properties. In case 1, table XI, for instance, I isolated 2 samples which fermented slowly the culture-medium used for isolation, agglutinated with the specific sera and had other identical properties; 2 others were also isolated which fermented rapidly the culture medium used for isolation and did not agglutinate with the specific sera.

4—Results

The study of table VIII shows that following this technic for isolation, in 10 patients with dysentery, the research was carried out in:

3 on the 3rd. day of disease, with positive results.

1 on the 4th. day of disease, with positive results.

3 on the 5th. day of disease, with positive results.

3 in the 2nd. week of disease, with 2 positives and 1 negative.

1 in the 3rd. week of disease, with 1 positive.

1 in the 4th. week, with negative results.

1 after the 4th. week, with positive results.

3 at a time not ascertained, with 1 positive and 2 negative results.

Working in the first week of the disease I obtained 100% positive results in 7 cases, making only one attempt in each case; in the 2nd. week 66% in 3 cases; in the 3rd. 1 case with positive results, from the 4th. week on, 4 cases with 2 positives or 50%. Out of a total of 16 cases I isolated dysentery bacilli in 12 or 75%, in spite of making only one attempt and inoculating the culture media with material kept for 1 or 2 hours at the ordinary temperature.

In 12 cases SHIGA bacilli were isolated in 7 or 43.7%. HISS and RUSSELL bacilli in 5 or 31.2%. The work was not repeated and the lapse of time between the obtaining of material and its inoculation on culture media did not usually exceed 2 hours. Negative results were obtained in 25% of the cases (in 4 cases). Of these 4 cases, 3 gave serum-agglutination for the SHIGA bacillus, and 1 did not.

These results endorse the efficiency of the methods used and were obtained in spite of the fact that the conditions were not ideal. They also keep up the credit of the aetiological importance of dysentery bacilli in the epidemics of dysentery and show the necessity of methods and conditions of research that make possible a sure diagnosis.

The strains isolated, all of them without exception, gave typical reactions in the series of biological tests and agglutinated very well as may be seen from table XIII. The strain of _faecalis_ did not agglutinate with the sera.
**TABLE XII**

Results of the research for dysentery bacilli with different culture media for isolation in the 
faeces of patients in the city of Bahia during 1924.

<table>
<thead>
<tr>
<th>No.</th>
<th>NAME</th>
<th>Origin</th>
<th>Day of illness</th>
<th>Date of research</th>
<th>Media inoculated</th>
<th>Results</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Mar. Ad.</td>
<td>Isolated</td>
<td>17</td>
<td>Aug. 17-924</td>
<td>+</td>
<td>Shiga Bacillus</td>
<td>Patient is unable to inform the day upon which he sickened, but states that it is more than a month ago.</td>
</tr>
<tr>
<td>2</td>
<td>Marc. P. Fon.</td>
<td>Isolated</td>
<td>5</td>
<td>Aug. 17-924</td>
<td>+</td>
<td>Shiga Bacillus</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Ir. Souz.</td>
<td>Isolated</td>
<td>+30</td>
<td>Aug. 17-924</td>
<td>+</td>
<td>Shiga Bacillus</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>F. D.</td>
<td>Private Practice</td>
<td>4</td>
<td>Sept. 12-924</td>
<td>+</td>
<td>Hiss &amp; Russell Bacillus</td>
<td>Serum agglutination positive, with Shiga bacillus</td>
</tr>
<tr>
<td>5</td>
<td>Man. M. Sant.</td>
<td>Isolated</td>
<td>30</td>
<td>Sept. 16-924</td>
<td>+</td>
<td><em>Bacillus fecalis alcaligenes</em></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>Orl. Ac.</td>
<td>Private Practice</td>
<td>5</td>
<td>Sept. 17-924</td>
<td>+</td>
<td>Shig aBacillus</td>
<td>The record of case does not state when illness began. Do. Serum agglutination negative.</td>
</tr>
<tr>
<td>7</td>
<td>Fort. San.</td>
<td>Hospital S. Izabel</td>
<td>5</td>
<td>Sept. 20-924</td>
<td>+</td>
<td>Hiss &amp; Russell Bacillus</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>Ped. S. Mel.</td>
<td>Hospital S. Izabel</td>
<td>5</td>
<td>Sept. 20-924</td>
<td>-</td>
<td>Negative</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>M. I. O.</td>
<td>Private Practice</td>
<td>5</td>
<td>Sept. 23-924</td>
<td>+</td>
<td>Hiss &amp; Russell Bacillus</td>
<td>Serum agglutination positive with Shiga bacillus</td>
</tr>
<tr>
<td>10</td>
<td>Graz. Cou.</td>
<td>Private Practice</td>
<td>3</td>
<td>Sept. 24-924</td>
<td>+</td>
<td>Shiga Bacillus</td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>Cos. Rib.</td>
<td>Private Practice</td>
<td>3</td>
<td>Oct. 3-924</td>
<td>+</td>
<td>Hiss &amp; Russell Bacillus</td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>At. B. Sout.</td>
<td>Private Practice</td>
<td>3</td>
<td>Oct. 3-924</td>
<td>+</td>
<td>Hiss &amp; Russell Bacillus</td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>M. Leonc.</td>
<td>Isolated</td>
<td>16</td>
<td>Jul. 25-924</td>
<td>-</td>
<td>Negative</td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>Jard.</td>
<td>Isolated</td>
<td></td>
<td>Jul. 25-924</td>
<td>-</td>
<td>Negative</td>
<td></td>
</tr>
<tr>
<td>15</td>
<td>M. Frei.</td>
<td>Hospital S. Izabel</td>
<td>10</td>
<td>Aug. 13-924</td>
<td>+</td>
<td>Shiga Bacillus</td>
<td></td>
</tr>
<tr>
<td>16</td>
<td>A. Pe. Frei.</td>
<td>Private Practice</td>
<td>13</td>
<td>Sept. 3-924</td>
<td>+</td>
<td>Shiga Bacillus</td>
<td></td>
</tr>
</tbody>
</table>

**+= Result positive**  
**-= Result negative**  

A clear space indicates that the medium was not made use of.
### TABLE XIII

Results of agglutination of strains isolated from faeces of patients

<table>
<thead>
<tr>
<th>No.</th>
<th>NAME</th>
<th>Agglutinating Serum SHIGA titre 1:40.000</th>
<th>Agglutinating Serum FLEXNER titre 1:20.000</th>
<th>Serum of patient SHIGA BACILLUS</th>
<th>Serum of patient FLEXNER BACILLUS</th>
<th>DIAGNOSIS</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M. Franc.</td>
<td>1.200</td>
<td>40</td>
<td>80</td>
<td>80</td>
<td>Dysentery Shiga type</td>
</tr>
<tr>
<td>2</td>
<td>M. Adel.</td>
<td>2.500</td>
<td>320</td>
<td>1.280</td>
<td>40</td>
<td>Dysentery Shiga type</td>
</tr>
<tr>
<td>3</td>
<td>Marc. Fon.</td>
<td>2.500</td>
<td>320</td>
<td>160</td>
<td>0</td>
<td>Dysentery Shiga type</td>
</tr>
<tr>
<td>4</td>
<td>Irin. Sou.</td>
<td>2500</td>
<td>320</td>
<td>160</td>
<td>40</td>
<td>Dysentery Shiga type</td>
</tr>
<tr>
<td>5</td>
<td>F. D.</td>
<td>320</td>
<td>2500</td>
<td>—</td>
<td>—</td>
<td>Dysentery Y type</td>
</tr>
<tr>
<td>6</td>
<td>Orl. Ac.</td>
<td>5.000</td>
<td>640</td>
<td>2.500</td>
<td>40</td>
<td>Dysentery Shiga type</td>
</tr>
<tr>
<td>7</td>
<td>Fort. Sant.</td>
<td>5.000</td>
<td>10,000</td>
<td>—</td>
<td>160</td>
<td>Dysentery Y type</td>
</tr>
<tr>
<td>8</td>
<td>M. I. O.</td>
<td>160</td>
<td>2500</td>
<td>—</td>
<td>—</td>
<td>Dysentery Y type</td>
</tr>
<tr>
<td>9</td>
<td>Graz. Cout.</td>
<td>2.500</td>
<td>640</td>
<td>—</td>
<td>—</td>
<td>Dysentery Shiga type</td>
</tr>
<tr>
<td>10</td>
<td>Cos. Bar.</td>
<td>640</td>
<td>2500</td>
<td>—</td>
<td>—</td>
<td>Dysentery Y type</td>
</tr>
<tr>
<td>11</td>
<td>At. B. Sou.</td>
<td>320</td>
<td>2500</td>
<td>—</td>
<td>—</td>
<td>Dysentery Y type</td>
</tr>
</tbody>
</table>

### III SEROLOGICAL DIAGNOSIS OF THE DISEASE.

Neither should the diagnosis of dysentery limit itself to the examination of faeces, nor should the test for the GRUBER-WIDAL phenomenon be forgotten even if the examination of faeces should have confirmed the clinical diagnosis.

In many cases it is only through this test that diagnosis can be made, and this is especially so in chronic dysenteries. Its value is that of complementary evidence, in those cases in which the bacillus has already been isolated from the faeces, or that of exclusive proof of the diagnosis when isolation has not been possible.

Besides this the agglutination of dysentery bacilli through the serum of the patient permits an *a posteriori*, or retrospective diagnosis, which may be of great value in diagnosing an epidemic.

The minimum titre of agglutination to indicate the influence of the bacillus on the organism which has furnished the serum is of 1:100 for the SHIGA bacillus and of 1:150 for the oligotoxigenic types. KRUSE, DRESEL, and MARCHAND allow of a lower titre for the SHIGA bacillus; they are satisfied with 1:50.

The existence of normal agglutinins in persons who apparently never suffered from dysentery is reported by FRANKEL, who was able to show them even at dilution of 1:140 for the HISS and RUSSELL bacillus. This titre was not exceeded. MULLER (quoted from
DRESEL and MARCHAND) also found these agglutinins, although rarely, in new-born babies. The existence of agglutinins, at a titre lower than those I gave as the minimum for diagnosis, is reported by a great many authors. JACOBITZ also reports some observations of the GRUBER-WIDAL phenomenon in persons not vaccinated and who had always been healthy. The same has been done by LENTZ and MARTINI, ARNHEIM, KRUSE, LIEFMAN and LISTER, with titres of 1/60 to 1/200, which led LENTZ to require a minimum agglutinating titre of 1/400 for the oligotoxic types.

The occurrence of such cases has often been seen among persons who have been in contact with dysentery patients.

UMNOS is of the opinion that every intestinal infection in which the faeces and serum have been tested with negative results is not a case of bacillary dysentery.

As a rule agglutinins appear after the eighth day of illness, at which time dilutions higher than 1:200 are ineffective, while in the second week dilutions go up to 1:800. After two months from the time of convalescence the agglutinins disappear. These figures, as well as the time during which the blood retains its agglutinating properties, are very subject to variation. In vaccinated subjects, 6 months. Very often agglutinins do not make their appearance or else remain at a very low titre. LEVADITI and NICOLAS report cases of dysentery with all types of dysentery bacilli and with a titre not exceeding 1:100.

From table XIV it may be seen that out of three cases in which I tried to show agglutinins in the first week of illness, in two I found them in a dilution sufficient to permit diagnosis and in the third agglutinating only for the patient's own strain of bacillus. Out of 13 patients tested for agglutinins 12 proved to have them at a titre warranting diagnosis and which agreed in all cases with the bacteriologic diagnosis. The only case in which the test failed (n. 9) did not have dysentery. Number 12 only agglutinated with the patient's own strain.

In 1915 DÜNNER called attention to the form of agglutination of the dysentery bacilli, which he breaks up into two types: one in which delicate clumps are formed and the other which is coarsely grumous; only the latter does he consider to be of specific value.

The grumous clumps, resulting from the conglomeration of bacilli and which make up the GRUBER-WIDAL phenomenon, are coarse and fall through their weight to the bottom of the tube, leaving the fluid clear and transparent, after a few hours' rest. When agglutination is weak, or unspecific, according to DÜNNER, the clumps are small and delicate and the fluid is not made entirely clear by being allowed to stand. DÜNNER considers agglutination with delicate clumps as a coagglutination phenomenon. This type is very different in cases of typhoid fever, in which the clumps are flocculent and float in the fluid mass like the clouds known as cirrus.

KUTSCHER observed during the war that the appearance of agglutinins against dysentery bacilli was frequently to be seen in the serum of persons vaccinated against typhoid fever and cholera. The titre did not usually exceed 1:50, however and this was confirmed by STEINBOCK (quoted from JACOBITZ) not long after.

JACOBITZ also observed the same fact with the HISS and RUSSELL bacillus, only in the dilution of 1:50 in vaccinated, but also in unvaccinated subjects, so that it would seem to him that the agglutinins were normal agglutinins,
### TABLE XIV

Results of tests for Gruber-Widal phenomenon in the serum of dysentery patients from the city of Bahia

<table>
<thead>
<tr>
<th>No.</th>
<th>NAME</th>
<th>Date</th>
<th>Days of illness</th>
<th>Agglutination with a suspension of</th>
<th>Patient's own strain</th>
<th>NOTES</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Jard.</td>
<td>25-VII-924</td>
<td></td>
<td>2.500</td>
<td>80</td>
<td>It was impossible to find out when they sickened, but the blood was taken while the infection lasted.</td>
</tr>
<tr>
<td>2</td>
<td>M. Leonc.</td>
<td>25-VII-924</td>
<td></td>
<td>320</td>
<td>40</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>M. Franc.</td>
<td>13-VIII-924</td>
<td>10</td>
<td>80</td>
<td>80</td>
<td>160</td>
</tr>
<tr>
<td>4</td>
<td>M. Adel.</td>
<td>17-VIII-924</td>
<td>17</td>
<td>1.280</td>
<td>40</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>Marc. Fon.</td>
<td>17-VIII-924</td>
<td>5</td>
<td>160</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>Irin. Sou.</td>
<td>17-VIII-924</td>
<td>over 30</td>
<td>160</td>
<td>40</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>Aid. P. Frei.</td>
<td>3-IX-924</td>
<td>13</td>
<td>320</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>M. M. Sant.</td>
<td>19-IX-924</td>
<td>30</td>
<td>320</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>Ped. S. Mel.</td>
<td>20-IX-924</td>
<td></td>
<td>—</td>
<td>—</td>
<td>The records of these cases do not inform when they sickened but the blood was taken while the infection lasted.</td>
</tr>
<tr>
<td>10</td>
<td>Fort. Sant</td>
<td>20-IX-924</td>
<td></td>
<td>—</td>
<td>160</td>
<td>320</td>
</tr>
<tr>
<td>11</td>
<td>Ori. Ac.</td>
<td>23-IX-924</td>
<td>5</td>
<td>2.500</td>
<td>40</td>
<td>1.280</td>
</tr>
<tr>
<td>12</td>
<td>F. D.</td>
<td>25-IX-924</td>
<td>4</td>
<td>—</td>
<td>—</td>
<td>160</td>
</tr>
<tr>
<td>13</td>
<td>Vic. Pac.</td>
<td>27-IX-924</td>
<td>over 15</td>
<td>320</td>
<td>—</td>
<td></td>
</tr>
</tbody>
</table>
It was therefore of interest to see whether the same thing was to be seen in patients of typhoid fever. In Bahia I undertook systematic research testing the serum of almost all the cases of typhoid fever for agglutination of dysentery bacilli at the same time as for agglutination of bacilli of the typhoid group. In the same way I tested the serum of dysentery patients for agglutination of the typhoid group.

In spite of the limited number of cases, three times the phenomenon was observed, in the cases shown on table XV.

**TABLE XV**

Cases in which the Gruber-Widal phenomenon was obtained as regards typhus and dysentery bacilli.

<table>
<thead>
<tr>
<th>NAME</th>
<th>Typhus B.</th>
<th>Shiga B.</th>
<th>Flexner B.</th>
<th>NOTES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Veris. Max</td>
<td>1200</td>
<td>—</td>
<td>—</td>
<td>40  80  Agglutination with delicate clumps</td>
</tr>
<tr>
<td>Ped. Theod.</td>
<td>160 320</td>
<td>80</td>
<td>—</td>
<td>40  80  The Castellani test showed that both infections were present.</td>
</tr>
<tr>
<td>Theop. Bisp.</td>
<td>320 640</td>
<td>160 320</td>
<td>80</td>
<td>Do.</td>
</tr>
</tbody>
</table>

The first case was a typhoid patient and the other two probably convalescents from this disease as they had suffered from prolonged fever a few months earlier. In their history they did not report having had intestinal troubles but they were unenlightened persons unable to give a clear account.

The fixation of agglutinins or CASTELLANI’S test and the observation of many other cases of dysentery in persons living under the same conditions in common dwelling-places indicate the previous coexistence of the two infections or the superimposition of one of them secondarily. The existence of typhoid fever with dysentery has already been seen more than once, and in 1922 KNORR quotes two similar observations of GOHN and ROMAN.

As to the value of the test as a proof of diagnosis, there is no doubt about its importance and all workers have made use of it with results.

At times agglutination with the patient’s serum gives no result, even when the examination of faeces has given positive results.

It might be that the standard strains selected, which should be readily agglutinable, contribute towards this, since there is, as is well known, a good deal of variation in this particular.

In the cases in Bahia I made use
<table>
<thead>
<tr>
<th>No.</th>
<th>NAME</th>
<th>Date</th>
<th>Residence</th>
<th>Days of recovery</th>
<th>Agglutination with suspension of Shiga B.</th>
<th>Flexner B.</th>
<th>NOTES</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Alc. Silv.</td>
<td>8-IX-924</td>
<td>Hospicio S. João de Deus</td>
<td>+60 days</td>
<td>-</td>
<td>80</td>
<td>Sickened during first half of 1924.</td>
</tr>
<tr>
<td>2</td>
<td>Eug. Conc.</td>
<td>8-IX-924</td>
<td></td>
<td>2 to 3 months</td>
<td>160</td>
<td>80</td>
<td>Do.</td>
</tr>
<tr>
<td>3</td>
<td>Jan. Sant.</td>
<td>8-IX-924</td>
<td></td>
<td></td>
<td>-</td>
<td>-</td>
<td>Do.</td>
</tr>
<tr>
<td>4</td>
<td>Titt. Conc.</td>
<td>8-IX-924</td>
<td></td>
<td></td>
<td>40</td>
<td>40</td>
<td>Do.</td>
</tr>
<tr>
<td>5</td>
<td>Paul. Costa.</td>
<td>8-IX-924</td>
<td></td>
<td></td>
<td>320</td>
<td>-</td>
<td>Do.</td>
</tr>
<tr>
<td>6</td>
<td>La. Gon.</td>
<td>8-IX-924</td>
<td></td>
<td></td>
<td>-</td>
<td>-</td>
<td>Do.</td>
</tr>
<tr>
<td>7</td>
<td>Em. Germ.</td>
<td>8-IX-924</td>
<td></td>
<td></td>
<td>-</td>
<td>-</td>
<td>Do.</td>
</tr>
<tr>
<td>8</td>
<td>Sylv. R. Vasc.</td>
<td>19-IX-924</td>
<td>Rua do Ouro</td>
<td>1 year</td>
<td>320</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>Greg. C. Nev.</td>
<td>19-IX-924</td>
<td>Rua do Falcão</td>
<td>1 year</td>
<td>320</td>
<td>80</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>Jan.</td>
<td>20-IX-924</td>
<td>Margem do Açude do Queimado</td>
<td>1 year</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
</tbody>
</table>
of a strain of SHIGA bacillus from the collection of the Institute and of one of the FLEXNER bacillus which had been obtained from the United States.

The results obtained always agreed with the bacteriological examination of the faeces, in all the cases from which I obtained serum, except in that of the patient F. D., as may be seen in table XVI.

DÜRNER and LAUBER in a paper on the type of agglutination and the variability of strains, come to the conclusion that dysentery bacilli vary very much as to this particular, and advise the use of strains which have proved to agglutinate with many different sera.

For retrospective diagnosis of cases from the epidemic I made use of this means. The results may be seen in table XVI, where the cases which had the disease are discriminated, including those of the Asylum S. João de Deus.

In this asylum during many years there have been severe epidemic outbreaks of dysentery, the coefficients of mortality of which have been indicated in chapter I; up to now the dysentery bacilli had not been isolated.

In the note on cases having occurred in this Institution and labelled as enteritis, and which was compiled, thanks to the courtesy of DR. MURILLO DOS SANTOS, Assistant of the Asylum, from the registers of the establishment, there is an indication that among these are included cases diagnosed clinically as dysentery, but not proved by laboratory tests undertaken by DR. ARMANDO SAMPAIO TAVARES.

### TABLE XVII

Cases of enteritis occurring in S. João de Deus Asylum from 1920 to 1924.

<table>
<thead>
<tr>
<th></th>
<th>1920</th>
<th>1921</th>
<th>1922</th>
<th>1923</th>
<th>1924 (Up to September)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Men</td>
<td>10</td>
<td>16</td>
<td>40</td>
<td>28</td>
<td>45</td>
</tr>
<tr>
<td>Women</td>
<td>8</td>
<td>9</td>
<td>33</td>
<td>26</td>
<td>34</td>
</tr>
<tr>
<td>Totals</td>
<td>18</td>
<td>25</td>
<td>73</td>
<td>54</td>
<td>79</td>
</tr>
</tbody>
</table>

As a matter of fact these were real cases of dysentery, as is demonstrated not only by our own results in convalescents from these cases of enteritis, indicated in table XVII, but also by the high mortality, which may be seen by comparing tables XVI, XVII and chart E with table XVII.

### TABLE XVIII

Number of deaths corresponding to cases shown in Table XVII.

<table>
<thead>
<tr>
<th></th>
<th>1920</th>
<th>1921</th>
<th>1922</th>
<th>1923</th>
<th>1924 (1)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Men</td>
<td>7</td>
<td>7</td>
<td>6</td>
<td>11</td>
<td>27</td>
</tr>
<tr>
<td>Women</td>
<td>2</td>
<td>5</td>
<td>9</td>
<td>24</td>
<td>15</td>
</tr>
<tr>
<td>Totals</td>
<td>9</td>
<td>12</td>
<td>15</td>
<td>35</td>
<td>42</td>
</tr>
</tbody>
</table>

Mortality: 50 %, 48 %, 20 %, 66 %, 54 %

(1) Up to September.
These two factors indicate that the dysentery was of the SHIGA type, the only kind according to tests on serum of convalescents. The following note, furnished together with the information above, also speaks in favour of these enteritis being due to the bacillus: «a few post-mortem examinations carried out by the late PROF. DR. OSCAR FREIRE, on patients sent from this Asylum and having died of enteritis, showed him lesions which he took to be due to bacillary dysentery». This means that what had not been demonstrated by bacteriological investigation was pointed out by pathological inspection.

On table XV are noted the results of tests for the GRUBER-WIDAL phenomenon on 6 insane and one attendant of the Asylum (Hospício São João de Deus). The latter, ALC. SIL. by name, had had symptoms of diarrhoea about two months before, with colics and mucus-sanguinolent faeces, tenesmus and fever which did not exceed 38°C. and was limited to the first two days of illness, disappearing on the third. Recovery within five days. In this case no agglutinins were found, probably owing to their having disappeared already. As has been pointed out, the majority of cases were of unenlightened people who would not
subject themselves to have their blood examined later or who had already moved out, which is often the case among those classes. Furthermore our investigation was only carried out many months after the epidemic had been at its height.

The patients numbers 8 and 9 gave typical reactions, which cannot be said for that of n. 10.

Case n. 8 was that of a young man of eighteen, mulatto, shoe-maker and resident at the Rua do Ouro, number unknown. He suffered from the disease in November of 1923 and was ill for more than 8 days. Number 9 was a tailor, mulatto, 30 years of age, resident at the Rua do Falcão, in S. Antonio. He was ill with symptoms of dysentery from October 29 to November 23, 1923. Soon after, his mother had a similar disease during many days, but is unable to say exactly how many.

Finally the 10th. patient had, in April of 1923, diarrhoea with very frequent muco-sanguineous faeces, accompanied by colics and tenesmus lasting more than 15 days. The same disturbance repeated itself during August of the same year, after the patient had eaten very spicy and fat food, and lasted two or three days. He also mentions that an old person in his family had later the same disease and had been in a serious condition for some time. The latter refused to allow her blood to be taken and in the patient himself agglutinins were no longer to be found.

What is not to be doubted is that dysentery of the SHIGA type did exist in the epidemic of 1924 and was very probably responsible for the majority of cases observed.

IV CASES STUDIED.

a. Frequency

Altogether research-work was carried out on 17 patients of dysentery. Of these 6 belonged to the Isolation-Hospital of Mont-Serrat, 3 to the Hospital S. Izabel, 7 to private practises and one was a pauper without residence and who refused to be admitted to hospital.

The patients of the Isolation-Hospital and of the Hospital, including also n. 13 of table IX, who appeared at my house, in Barra, (VIC. PAC.), and from which I only obtained the blood, all belonged to the indigent classes.

Of these 80 % had dysentery of the SHIGA type, 10 % had dysentery of the Y type and 10 % did not have bacillary dysentery. In these I found a streptococcus in the faeces. Of the patients from private practises, 57 % had dysentery of the Y type and 43 % of the SHIGA type.

From this it may be concluded that the SHIGA type of dysentery or toxic type predominates among the poor classes and that it is less frequent among the more favoured classes. The epidemic just appears to have predominated among the poor classes. Out of 287 cases (up to October 11), 28 were at the Hospital S. Izabel and at the Isolation-Hospital of Mont-Serrat, whither only the indigent go, as isolation in the private dwelling-houses is permitted by the Sanitary Authorities of the State. If we add to these, 34 from the Asylum S. João de Deus, 4 from the Asylo da Mendicidade and 19 from the rural zone, where the indigent population of the town lives, a sum total of 85 is obtained, or 30% of the cases.

b. Symptomatology.

The clinical symptoms seen made up the usual picture of dysentery: sudden appearance of colics, with fever up to 40°c., lasting for a short while, usually not longer than a week. There is further, frequent evacuation of muco-sanguineous faeces, tenesmus, fever, headache
and, sometimes, a rapid wasting and loss of weight.

I was unable to obtain detailed observations of the cases except in a few instances, thanks to the courtesy of the practitioners.

The evolution of the temperature in some cases from the hospital S. Izabel, of which I obtained the graphics charts, show the well-known low temperature of the disease following the usually short febrile period. In this sense the temperature of dysentery may be considered to have two phases; a first with a usually high temperature and a second phase in which the temperature is below the normal, and lasting during convalescence, but interrupted once and again by a rise of short duration, which probably indicates complications, such as secondary infection of the ulcers, absorption of toxic products from the intestines through the damaged mucosa or by the passing of bacteria into the blood for the same reasons.

In some cases this lowering of temperature was considerable: it fell to 33.8°C in patient M. FRANC. of the Hospital S. Izabel.

The observation of this patient un-
dertaken by the student EDGARD FAL-
CÃO, shows that the patient was a fe-
male, white, brazilian, single, 40 years 
old, working as a servant and living 
in the quarter of Tororó.

She was admitted to Hospital on 
Aug. 8, 1924, complaining that some 
five days before, after heavy indigestion 
she had continued to expel mucus and 
blood, with the faeces, which were ex-
pelled frequently. This state of affairs 
did not disappear until after she had 
been admitted.

The only important illnesses in the 
patient's history are malaria which she 
acquired at the beginning of the year 
and two extensive varicose ulcers, one 
on each leg and with dilatation reaching 
down to the feet.

The clinical symptoms of dysentery 
showed nothing of special interest except 
the extreme wasting of the patient, due 
in great part to poverty and the pre-
vious illnesses. The temperature as seen 
in Chart F shows the great lowering of 
temperature which was the most con-
siderable of all the patients of which I was 
able to obtain information.

The patient received anti-dysenteric 
serum in insufficient doses as there was 
not a sufficient supply in the Hospital 
and large doses of saline solution.

With this treatment there was some 
improvement which afterwards disappea-
red so that the patient died on the 20th. 
day of illness.

This was the only case on which I was able to make a post-mortem exa-
mination. I found continuous ulcerations, 
with suffusion of blood along the whole 
length of the thick intestine, extending 
early to the rectum, with a red colour 
which formed a strong contrast with the small intestine, which was pale and 
showed no appreciable lesion.

Another point of interest clinically 
is the pulse, chiefly as regards the rate.

In this particular it may be said 
that dysentery is the opposite of typhoid 
fever. In the latter there is a disparity 
between the pulse and the temperature 
so that while the temperature rises, the 
pulse remains more or less unaltered 
and does not accompany the rise of tem-
perature as is usual in fever.

In dysentery there is a disparity, 
but in the opposite sense, for the tem-
perature falls, at a time that might be cal-
led the algid phase of the disease and 
the pulse increases in number, generally 
the more the temperature falls the more 
the pulse-rate increases.

This phenomenon can be seen with 
evidence on charts H, I and J. It is 
pointed out in this paper so that it may 
be better observed in some Hospital Ser-
vice which I did not dispose of at the 
time.

C. Evolution.

The evolution of the cases was very 
variable. I was only able to make out 
the outcome of the disease in all cases, 
shown in table XIX.

Of the patients dysentery of the 
SHIGA type, 5 number, 3 died, which 
gives a mortality of 60 % for the cases. 
Of the six patients with dysentery of 
the type enumerated, one alone died 
which gives a mortality of 16 %. Of the 
SHIGA cases only 3 received anti-dy-
<table>
<thead>
<tr>
<th>N.o</th>
<th>NAME</th>
<th>Date on which patient sickened</th>
<th>Outcome</th>
<th>Date of outcome</th>
<th>Dysentery type</th>
<th>NOTES</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Orl. Ac.</td>
<td>Sept. 12-924</td>
<td>Cured</td>
<td>23-IX-924</td>
<td>Y</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>M. I. O.</td>
<td>Sept. 13-924</td>
<td>Cured</td>
<td>26-IX-924</td>
<td>Y</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>M. Leonc.</td>
<td>July 18-924</td>
<td>Died</td>
<td>27-VII-924</td>
<td>Shiga</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>Jard.</td>
<td>July 10-924</td>
<td>Cured</td>
<td>26-VII-924</td>
<td>Shiga</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>F. D.</td>
<td>Sept. 8-924</td>
<td>Cured</td>
<td>18-IX-924</td>
<td>Y</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>Aid. P. Frei.</td>
<td>Aug. 23-924</td>
<td>Cured</td>
<td>18-IX-924</td>
<td>Shiga</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>At. B. Sou.</td>
<td>Sept. 30-924</td>
<td>Cured</td>
<td>7-X-924</td>
<td>Y</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>M. Franc.</td>
<td>Aug. 3-924</td>
<td>Died</td>
<td>23-VIII-924</td>
<td>Shiga</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>Cos. B. Rib.</td>
<td>Sept. 30-924</td>
<td>Cured</td>
<td>3-X-924</td>
<td>Y</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>Fort. Sant.</td>
<td>July 20-924 ?</td>
<td>Died</td>
<td>24-IX-924</td>
<td>Y</td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>Marc. Fon.</td>
<td>Aug. 12-924</td>
<td>Died</td>
<td>26-VIII-924</td>
<td>Shiga</td>
<td></td>
</tr>
</tbody>
</table>
senteric serum and of these only number 8 died, who was so wasted as not to be able to resist the consequences of the disease.

The disease lasted more than two months in the cases of the SHIGA type, except for one case of a patient who died on the ninth day of disease. It lasted much less in the cases of the Y type from 3 to 15 days.