EPIDEMIC DYSENTERY IN THE NURSING STAFF DUE TO *BACILLUS DYSENTERIAE* (SONNE).

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(With Plate VI.)

An extensive outbreak of a mild dysenteroid nature occurred among the nursing staff at Guy's Hospital during February 1930. About one hundred of the staff were affected.

It is of extreme interest and at the same time very gratifying to note that, although many affected nurses continued their duties in the wards, no single case was observed among the hospital patients. This suggests that the scrupulous cleanliness of the nurses was sufficient precaution against the spread of infection.

There was no difficulty in establishing the *Bacillus dysenteriae* (Sonne) as the causal organism; and the evidence, although not conclusive, pointed to infection of food in the kitchens by a carrier.

The majority of recorded epidemics have been of this mild nature—acute diarrhoea and toxic symptoms usually followed by a rapid and complete recovery except in children who may die from the toxaemia.

B. dysenteriae (Sonne) is now a well-defined organism which, although first satisfactorily established as pathogenic by Sonne in 1915, had been previously isolated by other investigators who either failed to realise its significance or, owing to unobserved delayed fermentations of lactose and litmus milk, placed it amongst the Flexner Group as an inagglutinable strain. It has been shown to have an extensive distribution in Scandinavia, but was not definitely recognised in the British Isles until 1924, when Smith (1924) described a small outbreak in Aberdeen affecting four children. Since this date a steadily increasing number of isolated cases and epidemics have been recorded and, according to Gardner (1930) and a personal communication from Dr W. M. Scott of the Ministry of Health, there has been a much larger number of unrecorded outbreaks throughout Great Britain.

The evidence would suggest that the organism has travelled from Scandinavia viâ Scotland to England unless it is presumed that it had previously been endemic but undetected. Some support is given to the latter hypothesis by Nabarro (1927), who claims that bacilli which he isolated in 1921 from cases of summer diarrhoea and called "inagglutinable B. Flexner" were identical with *B. dysenteriae* (Sonne). At the same time he protests against

separate names being given to such organisms of the Flexner group because of slight differences in biological characters, and suggests the group name of *B. coli dysenteroides*. We contend, however, that the sharply defined serological reactions and the extensive clinical distribution of this organism are ample reasons for a separate name.

Epidemics and cases have now been described in Australia, Brazil, Denmark, Egypt, France, Germany, Japan, India and U.S.A.—a world-wide distribution.

The incidence rate is difficult to assess, as in an epidemic many infected individuals are not sufficiently ill to deem it necessary to seek medical advice.

The outbreak.

One isolated case (Nurse E.) occurred 15. ii. 30, 5 clear days before the main wave. This nurse had more severe symptoms than the majority. It was thought that she might in some manner have been responsible for the outbreak, and a careful enquiry was made into her history. As far as could be ascertained she had had no contact with any illness of this nature, but in January (6 weeks previously) she had had diarrhoea and vomiting for 3 days. There was then no pyrexia, and the patient stated that the attack was in no way comparable to her present illness. In any case it was impossible to find any likely method by which she could have contaminated food used by the other nurses either directly or indirectly.

The main wave, estimated at about one hundred cases, occurred within the next 7 days (20–28. ii. 1930), but only half of these reported sick.

No cases, except the two recorded below, could be described as severe; the onset was commonly marked by giddiness and malaise, followed in a few hours by abdominal discomfort and diarrhoea. Pyrexia, if present, was never higher than 101° F., falling to normal within 48 hours.

Two cases occurred a few days after the main wave and both showed unusual and severe symptoms.

(1) *Sister M.*

The general toxaemia was severe and the illness was accompanied by an excruciating headache. Although the organisms rapidly disappeared from the stools, convalescence was slow.

(2) *Nurse B. W.*

This patient became ill on March 4th, with diarrhoea, but little constitutional disturbance and no pyrexia. Her stools contained a little blood and mucus for the first week, but we failed to isolate Sonne's bacillus. Examinations for other pathogenic organisms or amoebae were also negative. The diarrhoea persisted, on the average about four stools being passed per diem. Repeated examinations of the stools were negative. After 5 weeks, during which she had been completely apyrexial, the temperature commenced to show slight

evening rises to 99° or 99·5° F., and the frequent motions continued. At the end of two more weeks (7 weeks from the onset) the condition was unchanged, and it was decided to give anti-dysenteric serum (B. W.'s polyvalent). Four intramuscular injections of 5000 units were given on consecutive days. These injections produced neither reactions nor apparent change in her condition. Seven days later a fifth injection of 5000 units was given. Within 48 hours the temperature had become normal and remained so, although the number of motions did not decrease for another 2 weeks, when the patient made a rapid recovery.

A proctoscopic examination made late in the course of the illness showed granular changes, but no ulceration of the mucosa.

It is debatable whether this was an infection with *B. dysenteriae* Sonne, as the illness differed so much from that in the other cases. At no time was a known pathogenic organism isolated from the stools and no agglutinins to Sonne, Shiga or Flexner bacilli were found in her blood.

The part played by the anti-dysenteric serum in her recovery is doubtful.

INVESTIGATIONS.

Laboratory investigations were commenced on February 24th, and by this time a considerable number of cases were well on the way to recovery. Attempts were made to secure stools from all known infected persons, and blood was taken for agglutination. As will be seen in the bacteriological notes *B. dysenteriae* Sonne was isolated from sixteen cases out of forty-two examined. About 30 per cent. of the cases showed a small amount of blood and mucus in the early motions.

In all investigated cases *B. dysenteriae* Sonne had disappeared from the stools within a few days.

Full details of the organisms and agglutinins present are given below.

DISCUSSION OF CAUSE.

As soon as the nature of the epidemic was appreciated investigations into the possible cause were commenced. After discussion with the matron and sisters it became clear that all cases had occurred solely in persons who fed in the nurses' dining room. The sisters have a separate dining room, supplied from a different kitchen, and only those sisters who supervised the nurses' dining room and had their meals there were affected.

No article of food or drink was purchased and/or supplied solely to this kitchen. The large number of cases, and their occurrence over a period of 14 days, made it impossible to suspect that only one part of the milk or food supply contained the organism on arrival at the hospital.

Another possible source of infection was the water supply. This, unfortunately, was not investigated. Again, however, it would be difficult to explain the limitation of infection.

It was therefore presumed that infection took place during the preparation

of food not previously contaminated. Stools and blood were therefore obtained from every one who handled or came in contact with the food in the kitchen, when it was found that two kitchen maids gave positive cultures from the stools and one showed agglutinins in the blood. Neither had been ill and a careful enquiry into their history and contacts for the last 6 months failed to disclose any illness or contact with illness that could be interpreted as a Sonne infection.

These two maids were removed and placed in the Isolation Ward. No further cases occurred after this step, but it is only fair to note that the epidemic was already showing signs of exhaustion or intermission.

The faeces of the two kitchen maids became negative in 3 days and, upon repeated negative examinations, they were allowed to return to work.

It must be concluded that, despite the rapid disappearance of the organism from the stools, one or both of these maids infected the food.

A CASE OF LABORATORY INFECTION.

During the bacteriological investigations one of the laboratory assistants (F. M.) who was employed exclusively on this work became infected from a culture; he had a typical illness lasting a week, followed by complete recovery. *B. dysenteriae* (Sonne) was recovered from his stools.

BACTERIOLOGICAL NOTES.

The following investigations were carried out on the sixteen strains of Sonne's bacillus isolated from the forty-two cases investigated, as well as one strain isolated from a laboratory infection, and one strain from the intestinal mucosa of a rabbit which died as the result of artificial feeding with cultures of the organism.

Isolation.

All specimens of faeces were plated out on nutrose litmus agar—the Guy's Hospital modification of Conradi-Drigalski. After 24 hours' incubation at 37° C., non-lactose fermenting colonies were picked and planted on to Andrade's indicator agar (phenol red), thence into "sugars" if no change occurred.

In all positive cases the number of non-lactose fermenting colonies on the nutrose plates was fairly large, forming more than 20 per cent. of the total number of colonies. In about half the cases more than 50 per cent. were non-lactose fermenters, while, in three, Sonne's bacillus was present practically in pure culture. Provided specimens are received in the first 24 hours of the disease, there is no difficulty in isolating the organism. It soon becomes rare, however, and in only one case was it present longer than 3 days after the onset of symptoms.

Colony appearances.

All plates showed two types of colony, which on macroscopic appearances could be described as rough and smooth, the latter, however, being far more

numerous. Smooth colonies were somewhat larger and more opaque than typical Shiga or Flexner colonies, while the rough were much larger, flatter and with spreading serrated edges. (See Plate VI, figs. 1–3.)

There is a distinct sweetish smell about the cultures resembling that noted in culture of *B. dysenteriae* Shiga, which has been described as spermatic.

Fermentation reactions.

Both rough and smooth colonies were used in order to ascertain if any difference in fermentative activity existed between the two forms. There was marked permanent acidity in litmus milk. Indol was not produced in any case.

Table I. *Fermentation reactions of the smooth and rough types isolated from each individual case.*

Case no.	Type of colony	Lactose	Dextrose	Maltose	Saccharose	Mannite	Glycerin	Inulin	Arabinose	Litmus milk	Indol
1	S.	+ (11)	+	+	+ (14)	+	0	0	+	+	0
	R.	+ (10)	+	+	+ (14)	+	0	0	+	+	0
2	S.	+ (4)	+	+	+ (4)	+	0	0	+	+	0
	R.	+ (9)	+	+	+ (15)	+	0	0	+	+	0
3	S.	+ (4)	+	+	+ (10)	+	0	0	+	+	0
	R.	+ (8)	+	+	+ (14)	+	0	0	+	+	0
4	S.	+ (5)	+	+	+ (4)	+	0	0	+	+	0
	R.	+ (8)	+	+	+ (15)	+	0	0	+	+	0
5	S.	+ (5)	+	+	+ (14)	+	0	0	+	+	0
	R.	+ (8)	+	+	+ (15)	+	0	0	+	+	0
6	S.	+ (12)	+	+	+ (4)	+	0	0	+	+	0
	R.	+ (8)	+	+	+ (15)	+	0	0	+	+	0
7	S.	+ (4)	+	+	+ (7)	+	0	0	+	+	0
	R.	+ (7)	+	+	+ (14)	+	0	0	+	+	0
8	S.	+ (7)	+	+	+ (14)	+	0	0	+	+	0
	R.	+ (5)	+	+	+ (15)	+	0	0	+	+	0
9	S.	+ (7)	+	+	+ (4)	+	0	0	+	+	0
	R.	+ (4)	+	+	+ (15)	+	0	0	+	+	0
10	S.	+ (4)	+	+	+ (9)	+	0	0	+	+	0
	R.	+ (7)	+	+	+ (14)	+	0	0	+	+	0
11	S.	+ (10)	+	+	+ (4)	+	0	0	+	+	0
	R.	+ (9)	+	+	+ (14)	+	0	0	+	+	0
12	S.	+ (5)	+	+	+ (5)	+	0	0	+	+	0
	R.	+ (5)	+	+	+ (14)	+	0	0	+	+	0
13	S.	+ (12)	+	+	+ (11)	+	0	0	+	+	0
	R.	+ (7)	+	+	+ (9)	+	0	0	+	+	0
14	S.	+ (6)	+	+	+ (8)	+	0	0	+	+	0
	R.	+ (8)	+	+	+ (15)	+	0	0	+	+	0
15	S.	+ (4)	+	+	+ (12)	+	0	0	+	+	0
	R.	+ (4)	+	+	+ (10)	+	0	0	+	+	0
16	S.	+ (4)	+	+	+ (3)	+	0	0	+	+	0
	R.	+ (7)	+	+	+ (10)	+	0	0	+	+	0
17	S.	+ (12)	+	+	+ (3)	+	0	0	+	+	0
	R.	+ (5)	+	+	+ (14)	+	0	0	+	+	0

+ = Acid reaction. 0 = No change. The figures in parentheses denote the day of culture on which the acid reaction was first observed.

It is generally accepted that the rough form of this organism has more active fermentative powers than the smooth. As will be seen, however, from Table I, the variation in lactose was in no way consistent. In the majority of strains there was a difference in the time taken for acid production, but as frequently as not the smooth showed the shorter interval. In the case of saccharose there is a definite lag on the part of the rough in producing acid.

Diagnosis.

As lactose takes some days to be fermented, it is unsatisfactory to await a diagnosis on the fermentation reactions alone.

The diagnosis was made by agglutination of the organism with a specific serum kindly supplied to us by Dr W. M. Scott. This serum agglutinated all strains up to a titre of 1 in 1000.

Other sera, Flexner (V, W, X, Y and Z) and Shiga, failed to agglutinate even in dilutions of 1 in 20. As cultures of rough forms failed to agglutinate with our original serum, only smooth colonies were used for diagnosis. This serum was presumably prepared from smooth strains only.

Presence of agglutinins in blood of infected cases.

In order to ascertain to what extent, if any, agglutinins were produced to smooth and rough varieties, the sera of infected cases were put up against their own organisms, both smooth and rough forms isolated from the original stool. The macroscopic method of agglutination was used, readings being taken after 3 hours in the water bath at 56° C., and again after 24 hours at room temperature. As agglutination of non-flagellated organisms of the colon-typhoid group is of the fine granular variety, it was found convenient to read results in a concave mirror, as the finest agglutination could thereby be seen and any difference between smooth and rough forms noted. For dilution purposes 0·9 saline was used, and here it must be mentioned that there was no evidence of salt sensitivity of the rough forms, a point which was investigated and will be discussed later on. The agglutination of the rough variety was indistinguishable from that of the smooth and there was no evidence of spontaneous agglutination in the control tubes.

Notes on smooth and rough variations.

It was thought that further investigations into the agglutinogenic properties of the two colonial forms would not be without interest, so it was decided to carry out further experiments. We use the term "variation" and "variants," not because there is proof that these forms are true bacterial variants, but for lack of a better descriptive term. This doubt exists in the case of our rough colonies which, as we shall see, do not fulfil all the requirements of the true rough variant.

Smooth and rough colonies were picked off on to agar plates and subcultured every 24 hours until apparently pure cultures of both forms were obtained. The rough forms were easily separated and maintained in pure culture, and have shown no tendency to revert to a smooth type. The smooth colonies, however, gave rise to a few rough colonies, which soon disappeared after five or six subcultures, and these too have maintained their form for at least 2 months, but eventually rough forms reappeared. Each form

was then seeded into 100 c.c. of nutrient broth, incubated for 24 hours at 37° C., and then heated in the water bath at 60° C. for 1 hour and sufficient formalin added to make a concentration of 0·2 per cent. This was done to ensure stable suspensions for agglutination purposes. The smooth forms gave a uniform turbidity in broth while the rough forms completely settled down, leaving a clear supernatant medium which, on shaking, however, became uniformly turbid.

This sedimentation of the rough form is in no way akin to agglutination as no clumping, even of the fine granular variety, could be seen microscopically, and this is confirmed when the rough suspension is agglutinated by an appropriate rough serum, when true agglutination takes place and clumping is obvious.

Analysis (see Table II).

Four patients showed no demonstrable agglutinins of either kind (Nos. 7, 8, 10 and 17), four agglutinins to the smooth variant only (Nos. 4, 11, 12 and 15),

Table II. *Agglutination of the smooth and rough variants by the respective homologous human sera.*

Case no.	Type of colony	Titre of serum 1/20	1/40	1/80	1/100	1/150	1/200	Control
1	S.	+	+	+	±	−	−	−
	R.	+	+	+	−	−	−	−
2	S.	+	+	−	−	−	−	−
	R.	+	+	−	−	−	−	−
3	S.	+	−	−	−	−	−	−
	R.	+	+	±	−	−	−	−
4	S.	+	±	−	−	−	−	−
	R.	−	−	−	−	−	−	−
5	S.	−	−	−	−	−	−	−
	R.	+	±	−	−	−	−	−
6	S.	+	+	+	+	±	−	−
	R.	+	−	−	−	−	−	−
7	S.	−	−	−	−	−	−	−
	R.	−	−	−	−	−	−	−
8	S.	−	−	−	−	−	−	−
	R.	−	−	−	−	−	−	−
9	S.	−	−	−	−	−	−	−
	R.	+	+	−	−	−	−	−
10	S.	−	−	−	−	−	−	−
	R.	−	−	−	−	−	−	−
11	S.	+	+	−	−	−	−	−
	R.	−	−	−	−	−	−	−
12	S.	+	+	±	−	−	−	−
	R.	−	−	−	−	−	−	−
13	S.	+	+	+	+	−	−	−
	R.	+	±	−	−	−	−	−
14	S.	+	−	−	−	−	−	−
	R.	+	+	±	−	−	−	−
15	S.	±	−	−	−	−	−	−
	R.	−	−	−	−	−	−	−
16	S.	+	+	+	−	−	−	−
	R.	+	+	−	−	−	−	−
17	S.	−	−	−	−	−	−	−
	R.	−	−	−	−	−	−	−

+ = Complete agglutination. ± = Partial agglutination. − = Absence of agglutination.

https://doi.org/10.1017/S0022172400010883 Published online by Cambridge University Press

two to the rough variant only (Nos. 5 and 9), while seven showed agglutinins to both forms (Nos. 1, 2, 3, 6, 13, 14 and 16).

The most noticeable features shown are:

1. Agglutinins may be absent altogether in some cases.

2. When present, they are so in low titre. This may be due to the fact that in this epidemic, the disease was of a very mild type with little toxaemia and therefore little antibody response.

3. Agglutinins to the smooth form have a tendency to be more common and in greater amount.

4. Agglutinins may be present for both varieties at the same time. This last feature needs discussion as it presents possibilities which are difficult to understand. That there should be agglutinins in the blood to both forms would seem to indicate that both smooth and rough forms have invaded the intestinal mucosa. Both rough and smooth colonies were present on the original plates from the faeces, therefore both forms were simultaneously present at the time of the invasion. As will be seen later, there is little difference in the virulence between the two forms, therefore it is reasonable to suggest that their agglutinogenic properties do not vary widely. This also is confirmed by subsequent experiments. It is most regrettable that insufficient serum was obtained to allow absorption tests to be carried out as these would have helped to confirm our opinion that agglutinins of both varieties are due to infection or invasion of the intestinal mucosa with both variants. That either is capable of producing antibodies to itself alone is borne out by the fact that agglutinins of one type alone may be present. The practical bearing that this has lies in the fact that both smooth and rough suspensions should be used when attempting to demonstrate agglutinins in the blood of Sonne infections, as otherwise a number of sera containing rough agglutinins only would be missed.

Production of specific agglutinins to smooth and rough forms in rabbits.

Twenty-four hour agar cultures were used. Saline suspensions containing 500 million organisms per c.c. were heated for 1 hour at 60° C. and 1 c.c. was injected intravenously at weekly intervals for 3 or more weeks, three injections being usually sufficient to produce titres of 1 in 2000. In this way smooth and rough anti-sera were produced without difficulty. In order to produce a serum containing both smooth and rough agglutinins 0·5 c.c. of each suspension was injected simultaneously. The results of subsequent agglutination are shown in Tables III, IV. Each serum was put up against both smooth and rough suspensions.

Agglutination by specific sera.

Serum dilutions were used in the following strengths of 1/20, 1/40, 1/80, 1/100, 1/500, 1/1000, 1/2000 and 1/3000. The smooth type was agglutinated by the smooth serum up to 1/2000, while the rough organisms were not agglutinated in any dilution. The rough serum agglutinated the rough organism

up to the same titre (1/2000) while seven of the sixteen strains of the smooth type were agglutinated either wholly or partially in dilutions of 1/20 only.

Agglutination by specific smooth and rough sera combined.

The serum was diluted as above and put up against suspensions of both smooth and rough organisms from each case. Both forms were agglutinated up to 1/1000.

These agglutinations prove the specificity of smooth and rough sera. Where the rough serum has agglutinated a smooth suspension it has done so in such a low titre as to be of little significance. This may be due to the difficulty of obtaining pure cultures of the smooth form. Eight of the sixteen smooth suspensions were agglutinated by the rough serum in a dilution of 1 in 20 either fully or partially, representing a percentage of one of the full titre, and this is probably due, in these instances, to a smooth suspension containing a few rough forms.

It is interesting to note that the agglutinogenic properties are approximately equal, and that sera can be prepared which will agglutinate both forms to nearly the same titre.

Absorption experiments.

In order to enhance the value and to confirm the opinion that these two colonial forms differ serologically, it was decided to try absorption experiments of this mixed serum with first the smooth form and then the rough.

The mixed smooth and rough serum was absorbed by adding 5000 million smooth organisms (previously heated at 60° C. for 1 hour) to 1 c.c. of serum and this mixture incubated at 37° C. for 2 days. The organisms were then separated by high speed centrifugalisation and the serum pipetted off and tested for sterility.

The results of agglutination are given in Tables III, IV.

Table III. *Agglutination by combined smooth and rough sera absorbed with smooth organisms.*

Case no.	Type of colony	Titre of serum 1/20	1/40	1/80	1/100	1/200	1/500	1/1000	1/2000	1/3000	Control
1, 4, 6, 7, 9, 13,	S.	+	+	+	+	−	−	−	−	−	−
14, 15, 16	R.	+	+	+	+	+	−	−	−	−	−
2, 3, 8, 10, 11,	S.	+	+	+	+	+	−	−	−	−	−
12	R.	+	+	+	+	+	+	−	−	−	−
5	S.	+	+	+	+	−	−	−	−	−	−
	R.	+	+	+	+	−	−	−	−	−	−

Table IV. *Agglutination by combined smooth and rough sera absorbed with rough organisms.*

Case no.	Type of colony	Titre of serum 1/20	1/40	1/80	1/100	1/200	1/500	1/1000	1/2000	1/3000	Control
1, 2, 3, 4, 5, 6,	S.	+	+	+	+	+	+	−	−	−	−
8, 11, 13	R.	−	−	−	−	−	−	−	−	−	−
7, 9, 12, 14, 15,	S.	+	+	+	+	+	−	−	−	−	−
16	R.	−	−	−	−	−	−	−	−	−	−
10	S.	+	+	+	−	−	−	−	−	−	−
	R.	−	−	−	−	−	−	−	−	−	−

https://doi.org/10.1017/S0022172400010883 Published online by Cambridge University Press

Absorption with rough organisms was more successful than absorption with smooth, as the latter suspensions were still agglutinated to a third of their original titre. At the same time the titre for rough suspensions was reduced to some extent, though this remained higher than that for smooth. In the case of absorption with rough organisms, however, all rough agglutinins were absorbed successfully, though here again the titre for smooth suspension was reduced.

Why absorption with rough forms should be more successful is difficult of explanation, unless there is greater affinity between rough agglutinins and their respective organisms than that existing between the smooth varieties at 37° C. which was the temperature used during absorption. The series is too small a one on which to base any definite conclusions and the titre of the sera insufficiently high. These somewhat anomalous results led us to investigate the question whether we were dealing with rough variants in the strict sense of the word. With this object in view it was decided to treat our rough cultures with alcohol and chloroform by the method advocated by White (1927).

Fifteen c.c. of absolute alcohol were added to each plate of rough cultures and the "suspensions" heated for 30 minutes in the water bath at 56° C., after which 10 c.c. of chloroform were added and the "suspension" left at 56° C. for 4 hours with frequent shaking. The organisms were then centrifugalised, washed and re-washed in alcohol and suspensions made up to 2000 millions per c.c.

As all our organisms were of one serological strain, only three were picked out at random and treated thus. These three rough bacilli, Nos. 4, 5 and 6, which previously were agglutinated by the rough serum up to a titre of 1 in 2000 were now not agglutinated. As a control the corresponding smooth cultures were similarly treated and this caused all agglutination to disappear. According to White the smooth variant should retain its agglutinability after such treatment. Our contradictory result invalidated the differential value of the test. This and the absence of salt sensitivity are the only two arguments against this particular rough strain of Sonne being a typical rough variant. On the other hand, the colonial appearances, the serological reactions and the pathogenicity for rabbits suggest strongly that this particular strain is a true rough variant. Of course, one could postulate an intermediate form showing some characters and not others, but in our opinion such an assumption, in view of the lack of sufficient data on the serology of Sonne infections, particularly of the two types of agglutinins referred to in the literature, is unwarranted.

Pathogenicity for rabbits.

Feeding experiments were first tried. A batch of four rabbits were fed on bran with which had been mixed living broth cultures of the smooth form of Sonne's bacillus, four more were fed on rough cultures and four kept as controls. Successful infection was not expected as the rabbits' gastric juice was thought to be sufficiently lethal to bacteria to prevent infection. One of

the number fed on smooth cultures, however, became ill, severe diarrhoea developing on the second day after feeding and death resulting on the fifth day. At autopsy the whole of the colonic mucosa was acutely inflamed and haemorrhagic, the peritoneum was injected, but all the other serous cavities appeared normal. Sonne's bacillus was recovered from the intestinal mucosa, but cultures from the heart and from the peritoneum remained sterile. All the other animals remained well.

As far as we can ascertain this is the first case of spontaneous infection of a rabbit during feeding experiments. Bamforth (1924), Kerrin (1928), and Ray (1930), have made attempts to do so without any successful case of infection.

Intravenous injections produced results very similar to those noted when *B. dysenteriae* (Shiga) is employed. Thus 2000 million dead bacilli generally caused severe diarrhoea and wasting in 24 hours and death in 4 to 8 days. Four rabbits injected with rough suspensions died after one injection and two more after the second injection a week later. Three out of six infected with a similar dose of smooth suspensions died, and three more after a third dose at weekly intervals. Doses of 500 to 1000 millions of dead bacilli of either smooth or rough forms caused diarrhoea with wasting of varying severity which sooner or later cleared up. On the whole there is little difference between the two forms, if anything, the rough form was found to be slightly more toxic.

CONCLUSIONS.

1. Diagnosis depends primarily on finding the organism in the stools and agglutination with a specific serum. Both rough and smooth sera should be employed for their respective variant.

2. High titre agglutinating rough serum can be produced with as equal ease as a similar smooth serum.

3. In our series the patients' sera showed agglutinins present only in low titre, and in some cases they were absent altogether. Two cases had agglutinins to the rough organism only. A negative result is of little diagnostic value.

4. The smooth and rough variations of *B. dysenteriae* (Sonne) require further investigation, especially the agglutinogenic properties of each variant.

SUMMARY.

1. An epidemic of a dysenteroid nature, largely ambulatory, due to *B. dysenteriae* (Sonne), affecting some 100 nurses without transference to any hospital case in the wards served by this section of the nursing staff, is described.

2. Sonne's bacillus was isolated from sixteen out of forty-two cases examined.

3. The probable cause was infection of food during preparation in the kitchens by a carrier.

4. A case of infection from laboratory cultures is described.

5. Notes are given of investigations upon the colonial appearance, biochemical and agglutinogenic reactions and pathogenicity to rabbits of the organism isolated, and also upon the presence of agglutinins in the blood of infected cases.

6. One case of infection in a rabbit by feeding experiments is described.

We would express our sincere thanks to Prof. J. Eyre for his advice, especially with the preparation of this paper, and to Dr W. M. Scott of the Ministry of Health for assistance in identification and the supply of agglutinating sera.

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EXPLANATION OF PLATE VI.

Bacillus dysenteriae (Sonne). Colonies in plate cultures.

Fig. 1. Rough type of colonies.
Fig. 2. Mixed rough and smooth types of colonies in first subculture from faeces.
Fig. 3. Smooth type of colonies.

(*MS. received for publication* 12. XII. 1930.—Ed.)

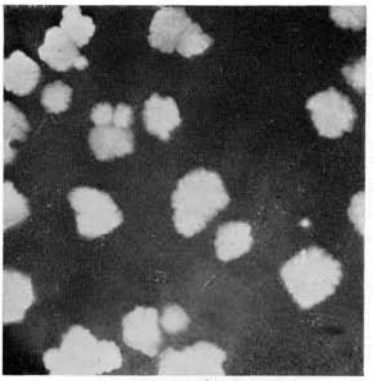

Fig. 1

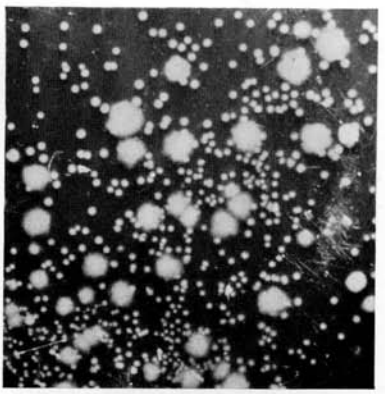

Fig. 2

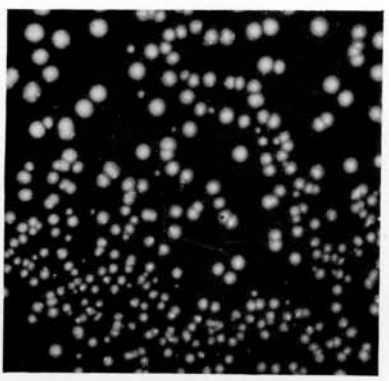

Fig. 3

https://doi.org/10.1017/S0022172400010883 Published online by Cambridge University Press