form of a square with the diagonal lengthwise; if the sphincter pylori is not included the incision is continued through its centre. In those cases in which the sphincter is considerably thickened, a wedge is removed on either side of the incision. The sutures are then placed after the manner of a Horsley's pyloroplasty, an inner layer of interrupted through-and-through sutures of No. 1 catgut and a superimposed layer of Lambert sutures of linen or silk. The packs are now removed, and further pockets of exudate mopped up gently with moist gauze. After this toilet of the peritoneum the wound is carefully closed in layers without any local drainage, but the pelvic drainage tube is kept in for twenty-four to forty-eight hours. No drainage at all is required in early cases with little extravasation, and no form of irrigation is attempted.

Immediately after the operation the patient is given a pint of glucose and sodium carbonate solution (5 per cent.) per rectum. If there are indications of shock, as indicated by a weak and rapid pulse and low blood pressure, a subcutaneous or intravenous saline is administered; the patient is returned to bed, maintained in the recumbent position; radiant heat is applied to the abdomen, and the patient is kept under the influence of morphine. The patient is not raised to the Fowler position until the pulse shows a considerable improvement, but on no account is any use made of pituitary extracts.

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TWO APPARENTLY UNRELATED OUTBREAKS OF BACILLARY DYSENTERY.

BY

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A FAIRLY extensive literature has of late accumulated with regard to bacillary dysentery in Great Britain, suggesting that the disease is endemic and that its unusual prevalence during the past few years is probably only the result of more careful investigation.

The last twenty-four years have shown one or more outbreaks of bacillary dysentery in at least eleven areas; even so, the following recital is probably incomplete: 1906, Birmingham¹; 1911, Paddington²; 1915, Blackwell rural district³ (1,000 to 1,700 cases of diarrhoea mainly dysenteric in character associated with pollution of water supply); 1921, Ogmire and Garw urban district⁴ (1,100 cases—probably a water-borne infection); 1922, Lynton urban district⁵ (18 cases due to milk-borne infection); 1923 to 1926, Aberdeen⁶ (about eight outbreaks, totalling 50 cases); 1925, Ayr⁷ (five groups of 12 cases); 1926, St. Andrews (150 cases)⁸; 1928, Smethwick⁹; 1928, Newcastle¹⁰ (141 cases). Further cases are referred to by Nabarro,¹² Clayton,¹³ and Warren.¹⁴

Textbook descriptions of bacillary dysentery postulate epidemicity where sanitation is defective or where water and milk supplies are infected. Endemicity is regarded as the normal in Eastern cities, while the occasional outbreaks in this country are relegated to asylums. The following account of the Flexner and Sonne outbreaks in Scunthorpe will help to refute the foregoing ideas, and will support the statements of other observers (Fraser, etc.,⁶ Wiseman,⁷ Fyfe,⁸ Charles and Warren,¹¹ Nabarro,¹² and Warren¹⁴), regarding the marked variation in virulence and the unrecognized prevalence of bacillary dysentery.

The Aberdeen, Newcastle, St. Andrews, and Ayr outbreaks in particular emphasized the variable virulence both of Sonne and of Flexner infections. Sonne infections appear to have been regarded as the less serious, which

agrees with Thjötta's observation that the resultant diarrhoea is often not serious enough to warrant the attention of a physician. On the other hand, some of the Aberdeen Sonne infections could definitely be subdivided into severe and mild groups—the former again being subdivided into two further groups. The more serious Flexner type of infection at Newcastle proved especially virulent among children, while adults appeared immune. The experience of the Scunthorpe series agrees in detail with these observations. Another curious analogy with the Newcastle^{10 14} cases is that in both areas the outbreaks were at first regarded as food poisoning, several members of a family being taken ill, with fatalities resulting.

Flexner Outbreak.

On February 4th, 1930, a message was received from the doctor in attendance that the four children, aged 3 to 10 years, of a working-class family residing at Ashby, Scunthorpe, were suffering from what was believed to be food poisoning.

The house was at once visited, and one child (Be. G.), aged 3 years, was found to be dying. There was marked dyspnoea, and Kernig's sign was present. No pulse could be felt; no head retraction was observed; and the temperature was 100° F. The history was that the child had first been taken ill four days previously, complaining of abdominal pain, headache, and diarrhoea. Death occurred on February 4th. Ba. G., aged 8 years, was said to have been delirious on the evening of February 3rd, and within twenty-four hours had a temperature of 102° F. with abdominal pain and diarrhoea. A. G., aged 10, had headache on the morning of February 4th, but wanted to go to school. By the evening the pulse was 140 and the temperature was 103° F., with abdominal pain and diarrhoea. S. G. had a similar onset, the symptoms being complicated by whooping-cough of about seven days' duration. Both the father—an ironworker—and mother were quite well.

On revisiting the patients with the family doctor in the evening the three surviving children were found to be extremely ill with almost constant diarrhoea, the motions being greenish black in colour, resembling spinach, and emitting a peculiar smell which, later, was realized to be almost pathognomonic. There was no obvious blood or mucus, and apparently no vomiting. No splenic enlargement could be discovered, and no rose spots were seen. Specimens of faeces were obtained from each surviving child. A tentative diagnosis of food poisoning was made, but an hour's close questioning failed to suggest any incriminated foodstuff.

The routine procedure suggested by the Ministry of Health was followed, and four food samples were sent to the Ministry's laboratory for examination. In view of the earlier results obtained from the examination of faeces the examination of foodstuffs was not considered necessary. A post-mortem examination was carried out on the fatal case (Be. G.) on February 5th by the patient's doctor, and evidence of an enteritis was found. The following specimens were sent to the Ministry's laboratory: (a) small intestine, (b) kidney, (c) spleen, (d) section of liver, (e) stomach. Unfortunately no part of the large intestine was sent. On February 5th the condition of the patients was critical, especially that of the youngest child, S. G. Specimens of blood were obtained from the parents and the two children A. G. and Ba. G. and dispatched to the Ministry's laboratory.

The house involved was one of nine scattered old houses forming an isolated courtyard, with a common water supply from a shallow well. Immediate instructions were given to boil all water before use. Four pail closets were in use at the onset of the outbreak. The property generally speaking was old, but in fair habitable condition. Only one house was overcrowded by two families of four adults and nine children. New accommodation was found for the subtenants.

Detailed inquiries were made at all the remaining eight houses, but no history of illness was obtained except from the overcrowded cottage where the five children of the

K. family were said to have had bloodstained diarrhoea about the beginning of January. The children when seen on February 5th were quite fit, and no doctor had been called in during their illness.

Pasteurized milk was in use at three houses (including the G. and K. families) and raw milk from another dealer at four houses, while the two remaining houses used milk from both sources.

The condition of the three sick children of the G. family remained much the same on February 6th, with continuation of marked diarrhoea, which showed bright red blood on the evening of that day. There was some remission of the pyrexia.

On February 7th a telegram was received from the Ministry stating that W type Flexner dysentery bacilli had been found in the faecal specimens of two of the three G. children. A. G. and B. G. were at once removed to an infectious diseases hospital, but the toxic condition of S. G. precluded removal. Polyvalent anti-dysenteric serum was wired for.

Further inquiries were then instituted for dysentery carriers, and an indefinite history was obtained from Mr. K., whose children had been ill in January. Blood specimens were sent to the Ministry's laboratory from both parents and from two of the five children (E. K. and B. K.) who had been the most seriously affected. Normal agglutinations were given by the parents' bloods, while those of the children gave slight but definite evidence of Flexner infection (titre 80 in 160). E. K.'s faeces on February 9th were normal and negative, although she was said to have had slight diarrhoea on February 8th.

Eighty c.cm. of antidyenteric serum was administered to the surviving sick child (S. G.) on February 8th, but death occurred later in the day.

Prophylactic doses of 10 c.cm. were given to three of four women who had taken turns in nursing the sick children and 30 c.cm. to the fourth (Mrs. M.), who was found to be in bed ill with diarrhoea. A specimen of faeces was obtained prior to this patient's removal to hospital as a suspected case. The report received subsequently showed the presence of a few colonies of Flexner W.

Following the receipt of the Ministry's telegram circulars were sent to the medical practitioners of the district requesting notification of any suspicious cases—dysentery being notifiable under the Public Health (Pneumonia, Malaria, and Dysentery) Regulations, 1919. As a result two groups of cases—(a) Dr. E. and S. T., and (b) Mrs. R.—were investigated in different parts of the district. Although blood-stained diarrhoea had occurred in each case the agglutinations were all negative.

No further developments took place until February 10th, when Mrs. B., who had assisted in the nursing of the G. children, complained of diarrhoea with mucus and blood. The stools when seen presented the classical "red currant jelly" appearance. Culture from a specimen obtained on February 11th showed numerous colonies of Flexner W, and removal to hospital was effected on the same day. Prophylactic doses of serum were given to the remaining members of the B. household.

As soon as the true nature of the outbreak was realized the Ministry of Health was requested to send assistance, with the object of trying to locate the source of infection. Dr. N. R. Beattie visited the district on February 12th and 13th, but in spite of the closest investigation no definite source of infection could be found. An attempt was made, however, to link up the infections between the K. and G. families. The members of the K. family were the only unsatisfactory tenants in this particular group of houses, and it was found that they had been allocated a pail closet for their own use, while the remaining eight families shared the other three. One of the elder K. children admitted having used the pail closet allocated to the G. family. In view of the fact that the K. children had suffered from undiagnosed dysentery in January it may be possible that the infection was transmitted in this way to the G. children. On the other hand, the family of subtenants (including three young children) who lived with the K. family from January 12th to February 11th,

suffered from no suspicious symptoms. A specimen of faeces could not be obtained from Mr. G. (who had given an indefinite history of dysentery during the South African and European wars) until February 19th. This proved to be negative, and the outbreak was by then considered to have subsided.

On March 1st E. K. was reported to be passing blood and mucus, the only symptoms being slight abdominal pain. On the following day a small quantity of almost pure blood and mucus was passed, and a specimen showed the presence of numerous colonies of Flexner W. The child undoubtedly had a "relapse," in view of the definite agglutination given by her blood on February 7th and the negative faeces of February 9th. Hospital removal was therefore effected on March 5th, and a negative stool was obtained by March 11th, the stools in fact having become quite normal in appearance by March 3rd before hospitalization.

On March 19th Mrs. K. was reported to be ill, and blood and mucus were passed on the 21st. Specimens of stools were sent for examination, and showed an abundant growth of Flexner W. In view of the negative agglutination of February 7th the patient had, curiously, escaped infection during the January infections in her own home, and again during E. K.'s relapse of March 1st. E. K. had returned from hospital on March 17th. A specimen of blood from Mrs. K. on April 2nd showed a "suspicious" agglutination against Flexner W in a titre of 30. A previous specimen on February 7th had shown normal agglutination only.

On April 8th J. K. was said to have passed blood and mucus, although the child was not ill and had a normal temperature. On the following day there was slight mucus but no blood, and a specimen showed Flexner W to be present. On receipt of the report the child was removed to hospital, where no treatment was necessary, and she was discharged on April 17th, following a negative stool. Although no serological evidence had been previously obtained J. K.'s case was undoubtedly a "relapse."

This group of Flexner W infection therefore comprises 11 cases, 7 of which gave definite bacteriological evidence, and 2 serological, while the remaining 2, although negative bacteriologically, were quite definite clinically—one in fact being fatal. Of the 11 patients, 3 were adults (all females) and 8 were children (7 females and 1 male). In addition, two other patients were removed to hospital as suspects, but were subsequently found not to be suffering from bacillary dysentery. The population at risk in this isolated group of houses consisted of 21 adults (7 male and 14 female) and 14 children (13 females and 1 male).

The cases are interesting inasmuch as the infection "bred true" throughout; in view of the greatly increased virulence in the children of one family (2 deaths); the occurrence of two "relapses"; and because no source of infection could be discovered—an experience typical of all other outbreaks, except those due to infected water or milk.

Diagnosis was confirmed by faecal culture tests, while agglutination tests were only found to be useful accessories (see Charles and Warren¹¹). For successful faecal tests the specimens had to be obtained early. One negative stool was considered sufficient for discharge from hospital.

Sonne Outbreak.

On April 10th a message was received from a doctor that there had been suspicious diarrhoea affecting a household next door to one where he was visiting. This house was also situated in Ashby, but at a considerable distance from the venue of the Flexner cases. A visit disclosed that a child, E. M., aged 2, had complained of diarrhoea with blood and mucus about the end of March. It was stated that she had experienced similar attacks previously.

The father, an ironworker at the same works as Mr. G. of the Flexner series, had been taken suddenly ill while at work about the beginning of April. Blood and mucus had been passed, but no medical attention had been sought. No connexion could be made out between Messrs. G. and M.

TABLE OF LABORATORY INVESTIGATIONS.
Flexner Cases.

Date.	Patient's Initials.	Specimen.	Result.
1930.			
Feb. 5	A. G.	Faeces	Flexner W in considerable numbers.
"	Ba. G.	"	No B. dysenteriae.
"	S. G.	"	Flexner W in small numbers.
"	Be. G.	P.M. Organs	No B. dysenteriae (large intestine not sent).
"	Mr. G.	Blood	No agglutination against Flexner W.
"	Mrs. G.	"	Agglutinated Flexner W 1 in 40 (? significance).
"	A. G.	"	No agglutination Flexner W.
"	Ba. G.	"	Insufficient serum.
Feb. 7	Mr. K.	"	Normal agglutination.
"	Mrs. K.	"	Ditto.
"	E. K.	"	Agglutinated Flexner W 80 in 160 (slight but definite).
"	B. K.	"	Ditto.
Feb. 8	Mrs. M.	Faeces	Flexner W, a few colonies.
Feb. 9	E. K.	"	No B. dysenteriae.
Feb. 11	Mrs. B.	"	Flexner W, numerous colonies.
"	Mrs. E.	"	No B. dysenteriae.
Feb. 13	"	Blood	No agglutination to Flexner W.
"	Dr. E.	"	Ditto.
"	S. T.	"	Ditto.
Feb. 17	"	Water sample	Colonies on agar D 37° C. = 12 per c.cm. Presumptive B. coli plus in 5 c.cm. No dysentery bacilli on plating 800 c.cm.
Feb. 19	Mr. K.	Faeces	No B. dysenteriae.
March 2	E. K.	"	Flexner W, numerous colonies. (See reports on Feb. 7th and 9th.)
March 11	E. K.	"	No B. dysenteriae (hospital clearance).
"	Mrs. B.	"	Ditto
March 23	Mrs. K.	"	Flexner W (abundant).
March 31	M. H.	"	No B. dysenteriae.
April 2	Mrs. K.	Blood	Flexner W agglutinated 1 in 40. (Compare with blood of Feb. 7th—that is, suspicious).
April 9	Mrs. K.	Faeces	No B. dysenteriae (hospital clearance).
"	J. K.	"	Flexner W present.
April 15	J. K.	"	No B. dysenteriae (hospital clearance).
<i>Sonne Cases.</i>			
April 12	Mr. M.	Blood	Haemolysed on arrival at laboratory.
"	F. M.	Faeces	B. dysenteriae Sonne (pure culture).
April 23	F. M.	"	No B. dysenteriae (hospital clearance).
April 26	Mrs. M.	"	Ditto.
May 2	"	Water sample	No B. dysenteriae in 100 c.cm.

F. M., aged 6, commenced with similar symptoms on April 10th, and a faecal specimen obtained on the 12th showed Sonne bacilli in pure culture. A blood specimen taken from Mr. M. at the same time was unfortunately haemolysed on arrival at the Ministry's laboratory. F. M. was admitted to hospital on receipt of the bacteriological finding; there was no apparent illness while in hospital, and no treatment was given. A negative stool was obtained on April 23rd, after which he was discharged (April 26th).

On April 24th Mrs. M. complained of violent abdominal pain for about two hours, diarrhoea (but no apparent blood and mucus), and vomiting. Temperature and pulse were normal when seen, and a faecal specimen obtained on April 26th was negative. The family also included another child, P. M., and a baby. Both were said to have had very slight similar symptoms. Although Sonne dysentery was only bacteriologically proved in one child (F. M.), there appears little doubt that the whole family

of six were affected at one time or another. Mr. V., who lived next door and had been attended by the doctor who had drawn attention to the M. family, had also suffered from diarrhoea at the beginning of April. Unfortunately no bacteriological tests could be carried out.

No possible connexion could be traced with the Flexner series. The milk supply was the same pasteurized supply as that used by some of the Flexner series, but in view of its large sale in the area and the absence of any explosive outbreak it was assumed not to be the source of infection. The shallow well water supply to the houses occupied by the M. and G. families showed gross contamination on bacteriological analysis, but no dysentery bacilli were isolated from 100 c.cm.

The accompanying table shows the specimens examined at the Ministry's laboratory.

I would gratefully acknowledge the co-operation of Dr. J. H. Bellamy of Ashby and of Dr. A. H. G. Johnston of Scunthorpe, of Dr. Neil R. Beattie of the Ministry of Health, and particularly of Dr. William Scott, pathologist to the Ministry, who carried out the whole of the laboratory investigation.

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THE DIAGNOSIS OF BRAIN ABSCESS.

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THE diagnosis of brain abscess is often a matter of great difficulty to the surgeon; in some cases while he may be pretty certain of the presence of an abscess he may fail to locate it. For diagnostic purposes brain abscesses may be classified into two groups:

1. Those in which the classical symptoms of abscess of the brain are present. This group may be subdivided as follows: (a) Those cases in which localizing symptoms are present, where the abscess is caused by direct extension of the infective process. The best examples of this group are the otogenic abscesses, which occur as a result of middle-ear disease and are known as temporo-sphenoidal when they are found in the temporal lobe, and as cerebellar abscess when the cerebellum is the seat of the disease. Another instance of this type of abscess may be found in the frontal area of the brain following suppuration in the frontal or ethmoidal sinuses or injuries in that region of the skull. (b) Those in which localizing symptoms are absent—namely, those cases in which the cause is situated at some distance from the abscess and the infective agent is carried by the blood stream. Metastatic abscesses are examples of this kind and are the most difficult to locate. It is well to remember that sometimes otogenic abscesses are formed at some distance from the original focus of infection, and have even been found in the opposite side of the brain.

2. Abscesses of the symptomless or latent type, in which there is an entire absence of symptoms and in some cases an apparent absence of cause. In this latter type the fate of the patient is sealed before an absolute diagnosis is made, owing to the abscess bursting into the lateral ventricles, or, on the other hand, owing to the increase in tension or acute inflammation which results in a breaking down of adhesions formed during the course of the disease, and spread of pus between the dura and brain, causing fulminating meningitis and encephalitis.