BACILLARY DYSENTERY (SHIGA) CONTRACTED IN ENGLAND.

BY

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DYSENTERY in this country before the war, with the exception of a few isolated outbreaks, occurred in the form of asylum dysentery, and in those cases in which bacteriological examinations revealed the presence of dysentery bacilli the organisms were almost invariably of the mannite-fermenting (Flexner) type. References to the occurrence of cases caused by the Shiga bacillus are few. In 1904 Eyre¹ isolated the bacillus from six out of nine cases of acute asylum dysentery, and since then two sporadic cases have been described, one by Saundby and Hewetson in 1906,² and one by Saundby and Wynn in 1908.³

The following are the particulars of a case of dysentery caused by the Shiga bacillus, which was traced to contact with a convalescent soldier who was found to be excreting large numbers of the organism in his faeces.

Mother.

The patient, a woman aged 50, under the charge of Dr. May of Wakefield, to whom I am indebted for the particulars, was in her usual health up to February 11th, 1916, when she was attacked by symptoms of acute dysentery. The motions, which consisted chiefly of mucus and blood, were very frequent, sometimes numbering fourteen in the course of the night during the earlier stages of the illness, and were accompanied by marked tenesmus. There was a considerable degree of fever, and the patient showed a good deal of prostration. Previous to the onset of her illness the patient is said to have been liable to attacks of diarrhoea. At the time of the onset of the illness the patient's son, who had contracted dysentery at the Dardanelles, had been six days residing in the house, which is a small one, consisting of two living rooms and two bedrooms, with an outside closet, used, of course, in common by all members of the family.

A specimen of faeces from the patient was obtained by Dr. May on February 18th, and was examined on the same day. It was found to be very fluid, of a reddish colour due to admixture with blood, and contained a large quantity of slimy mucus and a few very small yellowish granules. On standing it separated into two layers, the upper being clear and watery and the lower containing the mucus. On microscopic examination the material was found to be composed almost entirely of pus and red blood corpuscles so far as cellular elements were concerned. No amoebae or flagellates could be found. Organisms were very scanty, and were seen mostly within the pus cells, some of which contained three or four Gram-negative bacilli.

Plate cultures were made on MacConkey's medium modified by the addition of crystal violet; after incubation for twenty-four hours numerous non-lactose fermenting colonies of a non-motile, Gram-negative bacillus, varying somewhat in length, but generally shorter and thicker than *B. typhosus*, developed. The organism produced acid but no gas in glucose, laevulose, galactose, and mannose, and no change in lactose, saccharose, dulcitol, mannite, adonite, inulin, salacin, sorbite, raffinose, maltose, or glycerine. In litmus milk it produced a slightly acid reaction but no clot, and the acidity remained permanent, though it was slightly less marked at the end of seven days. It did not form indol in peptone water in seven days (Ehrlich's test), and did not liquefy gelatine. With dysentery (Shiga) serum it was distinctly agglutinated, but the limit of agglutination fell far short of the titre of the serum tested with a standard strain.

Son.

The patient's son, aged 22, was seen on February 19th by Dr. Gibson, M.O.H. Wakefield, who kindly furnished me with the following history: He was sent to the Dardanelles in September, 1915, where he was attacked with dysentery about the end of November. He was then removed to a hospital on one of the islands of the Eastern Mediterranean, and was afterwards sent home to England. He was admitted to hospital on January 8th, and discharged on February 5th, and returned to his home; six days after his arrival there his mother's illness commenced. He remained at home until February 14th, when he proceeded to a military rest camp. When seen by Dr. Gibson the man was not free from symptoms of dysentery, as he was still having frequent slimy motions, often containing blood, and there was a certain amount of tenesmus. His condition varied, but at times he had as many as three to four motions in quick succession, and observed that this was most liable to occur directly after meals.

A specimen of faeces was obtained by Dr. Gibson on February 20th, and was examined by me the same day. The specimen consisted partly of dark solid material and partly of thick blood-stained mucus, some of which was of a distinctly yellowish colour, and resembled thick tenacious pus. Microscopically, the mucous portion of the stool showed the same characters as in the case of the mother, except that organisms were present in very large numbers. Cultures were made from the mucus, and large numbers of organisms developed which

gave the same cultural and agglutinating characters as the organism isolated from the mother.

Comparison of Strains.

The strains from both mother and son were compared as to their cultural characters, virulence, and agglutination with a standard strain of *B. dysenteriae* (Shiga) obtained from the Lister Institute, and also with a strain (*B. dysenteriae*, Shiga, M.) which I previously isolated from the faeces of an officer who had been in charge of cases of sickness on a transport returning from the Dardanelles, and who developed symptoms of dysentery just after his arrival in England. All four strains gave the same reactions in the carbohydrates already mentioned, produced no indol, and gave an acid reaction but no clotting in litmus milk. The only cultural difference noticed was that *B. dysenteriae* (Shiga, Lister Institute) did not grow so profusely on agar as the other strains.

The organisms were tested for virulence by the intraperitoneal inoculation of guinea-pigs of 350 grams weight with 1 c.cm. of a twenty-four hours broth culture of each bacillus. The organisms isolated from the patient and from her son caused death within twenty-four hours, *B. dysenteriae* (Shiga, M.) caused death in forty-eight hours, while *B. dysenteriae* (Shiga, Lister Institute) did not cause death in three days.

The agglutination reactions were tested with three dysentery (Shiga) serums: (1) Lister Institute serum; (2) a serum prepared by immunizing a guinea-pig with *B. dysenteriae* (Shiga, Lister Institute); and (3) Burroughs, Wellcome and Co.'s serum. The tests were performed macroscopically, and the results noted after two hours' incubation at 37° C. Controls were made in each case, and in most of the tests the tubes were again examined after standing twenty-four hours at room temperature to exclude the possibility of spontaneous agglutination. In the three recently isolated strains the agglutination limit was never higher than 1 in 200, and even with the standard strain the titre was only 1 in 800 to 1 in 1,600. It was noticed that in all cases the clumps were small and deposited slowly, in marked contrast to the large flocculent clumps characteristic of agglutination of the motile organisms of the typhoid-paratyphoid group with their corresponding serums. Ledingham, Penfold, and Woodcock⁴ found that the strains isolated by them were all agglutinated in high dilutions (1 in 1,500 to 1 in 4,000) by the specific Shiga serum used, so that sharper results might have been obtained by more powerful serums. On the other hand, it may be remarked that the serum of Burroughs, Wellcome and Co. was stated to have a titre of 1 in 2,000 to 1 in 4,000 tested with five strains of living Shiga. Unless the serum had deteriorated to some extent since being tested, the results rather suggest that there is a certain specificity among Shiga strains.

The case is of considerable interest as it shows that the disease can be readily contracted under ordinary circumstances in England, although it is possible that in this instance the patient was rendered more susceptible owing to the fact that she was subject to attacks of diarrhoea prior to the onset of the dysentery.

REFERENCES.

- ¹ Eyre, BRITISH MEDICAL JOURNAL, 1904, April 30th, p. 1002.
² Saundby and Hewetson, BRITISH MEDICAL JOURNAL, June 6th, 1906, p. 1325.
³ Saundby and Wynn, BRITISH MEDICAL JOURNAL, January 4th, 1908, p. 20.
⁴ Ledingham, Penfold, and Woodcock, BRITISH MEDICAL JOURNAL, November 13th, 1915, p. 704.

ON THE CURVE OF THE EPIDEMIC.

SUPPLEMENTARY NOTE.

BY JOHN BROWNLEE, M.D., D.Sc.

IN the issue of the BRITISH MEDICAL JOURNAL of May 8th, 1915, I published a short note on possible epidemic forms. These were calculated on various hypotheses, but the only form which could be said to describe the course of an epidemic (Pearson's Type IV) was left completely unexplained. As this curve gives a fairly close fit to the majority of epidemics, no theory which does not account for its origin can be said to be of much practical importance. The form, however, immediately arises, if it be assumed that as the number of infecting centres increases there is more than an equivalent increase in the combined infecting power.

Curiously, though this idea was considered from time to time it was always rejected, why I cannot say. It seems, however, highly probable that such a phenomenon exists. Suppose an individual receive an amount of infection slightly smaller than is necessary to induce the disease. Suppose, also, a second infection is received within a certain time afterwards. There is probably a minimum interval within which two such infections can act together and produce the disease. Outside this interval escape will follow on both occasions. If such a condition exists it is obvious that in the presence of a large number of centres