A HOSPITAL OUTBREAK OF TYPHOID FEVER. BACTERIOLOGICAL AND SEROLOGICAL INVESTIGATIONS

By A. C. JONES, M.B., B.S.

From the Public Health Laboratory, Royal Salop Infirmary, Shrewsbury

CONTENTS

	PAGE
I. Introduction	335
II. Diagnosis of the outbreak	335
III. Isolation of <i>Salmonella typhi</i> from the faeces	336
IV. Search for carriers among kitchen workers	337
V. Search for excreters among staff and patients	338
VI. Identification of incubating cases ('precocious carriers')	340
VII. Identification of symptomless excreters	340
VIII. Exclusion of a chronic typhoid carrier as a cause of the outbreak	343
IX. Exclusion of connexion with typhoid cases at another hospital	343
X. Secondary infection of contacts	344
XI. A typhoid incident investigated by Moore's sewage-pad technique	345
XII. Discussion	346
Summary	347
References	348

I. INTRODUCTION

Bacteriological findings in the typhoid outbreak at the Oswestry Orthopaedic Hospital, where 135 persons are known to have been infected, may be of general interest in so far as they throw light on the epidemiology of enteric infection. Full epidemiological data are published in a preceding article (Bradley, Evans & Taylor, 1951). In this paper little attempt is made to deal with the bacteriology of clinically typical cases of typhoid fever as these were dispersed throughout the Midlands. Discussion is confined to initial diagnosis, the diagnosis of incubating cases ('precocious carriers') and symptomless excreters, and to an assessment of the importance of various other carriers discovered in the course of investigations.

II. DIAGNOSIS OF THE OUTBREAK

On 8 September 1948, a specimen of blood from a nurse (G.D.), who had suffered unexplained pyrexia for 3 days, was received at the laboratory. Serum agglutinins were present to the following titres: *typhi* H, 1/25; *paratyphi* B H, 1/125. No O agglutinins were found. These results aroused suspicion of enteric infection, and further specimens of blood were examined from the nurse and from six other persons who had been ill for periods varying from 1 to 5 days. On 9 September serum agglutinins against typhoid antigens to a 'diagnostic titre' were reported in all specimens. On 10 and 11 September, *Salmonella typhi* was isolated from the blood and faeces of some of these early cases. The results of the Widal reaction in the first ten cases to be examined are listed in Table 1.

The case of G.D. (1*a*, 1*b*) is of interest inasmuch as the first specimen indicated a possible paratyphoid-B infection. Two days later, however, the typhoid H agglutinins had risen to a far higher titre than those of *paratyphi B* H. The patient gave no history of previous enteric infection, T.A.B. inoculation, or of unexplained pyrexia. It is conceivable that she may have had a previous subclinical *Salm. paratyphi B* infection. The findings in this case illustrate the importance of isolating the organism from faeces or blood in order to establish the true diagnosis.

Table 1. *Serological findings in the first ten cases*

Case	Date of collection of blood	Day of illness	Titre of agglutination			
			<i>typhi</i> H	<i>typhi</i> O	<i>para-typhi B</i> H	<i>para-typhi B</i> O
1 <i>a</i> G.D.	7. ix. 48	3	1/25	—	1/125	—
1 <i>b</i> G.D.	9. ix. 48	5	1/1280+	1/1000	1/320	1/125
2 J.M.	9. ix. 48	3	1/80	1/250	1/40	1/50
3 E.We.	9. ix. 48	5	1/160	1/250	—	1/125
4 J.S.	9. ix. 48	3	1/1280+	1/250	—	1/125
5 L.H.	9. ix. 48	3	1/320	1/250	—	1/125
6 S.L.	9. ix. 48	1	1/80	1/125	—	1/25
7 E.Wi.	9. ix. 48	2	1/1280	1/500	—	1/25
8 W.M.	10. ix. 48	6	1/1280	1/500	—	1/25
9 E.F.	10. ix. 48	6	1/5000+	1/250	.	.
10 J.D.	12. ix. 48	1	1/160	—	.	.

Table 1 shows that *typhi* H and O titres much higher than those normally regarded as diagnostic may be obtained in the serum of cases of typhoid fever well within the first week of onset of symptoms. O agglutinations were carried out by Dreyer's technique; readings were taken after 18 hr. incubation in a 50° C. water-bath; still higher readings might well have been obtained by the more sensitive technique of Felix using round-bottomed tubes with 2 hr. incubation at 37° C. followed by storage for 22 hr. in the refrigerator. Some of the cases developing later in the outbreak seemed to conform more closely to the classical description of the disease where the 'Widal' is said to show a diagnostic titre 8–14 days after onset of illness. 'Positive agglutination tests may be obtained towards the end of the first week but are rare and weak before that' (Scott, 1941). It should be noted, however, that Felix (1924, 1930) emphasized the special importance of O agglutinins in the early diagnosis of cases when he introduced his serological method for the diagnosis of enteric fevers.

III. ISOLATION OF *SALMONELLA TYPHI* FROM THE FAECES

In recent years diagnosis of cases and carriers of typhoid has been made easier by the introduction of various selective and enrichment media for faeces culture. Fry (1944), using Müller and Kauffmann's tetrathionate broths, found that two out of every three positive results were obtained with those media alone 'even when using so selective a medium as Wilson and Blair for primary plates'. Hobbs & Allison (1945) found 'Wilson and Blair' to be the best solid medium for the isolation of *Salm. typhi*, and desoxycholate citrate agar to be very satisfactory.

They recommended the use of both media to obtain the highest percentage of positive isolations. They concluded that selenite F was the best enrichment medium for isolating *Salm. typhi*, stressing its simplicity of preparation, and stability when kept for indefinite periods at room temperature as compared with other liquid enrichment media. Knox, Gell & Pollock (1943) claimed that careful timing of subcultures from tetrathionate broth increased the proportion of positive results. Under epidemic conditions such careful timing may be difficult to arrange owing to the large number of specimens under test.

At the Shrewsbury Public Health Laboratory 4158 specimens of faeces and 3529 specimens of urine were examined in connexion with the Oswestry outbreak during the first 4 months of investigations.

Table 2. *Isolation of Salmonella typhi from faeces*

Part 1. Comparison of three methods						Total no. of
D+ T+ S+	D- T+ S+	D- T+ S-	D- T- S+	D+ T- S+		positive faeces
20	25	1	68	2		116
Part 2. Comparison of two liquid media						
T+ S+	T+ S-	T- S+				Total no. of
87	11	210				positive faeces
						308

+, *Salm. typhi* isolated; -, *Salm. typhi* not isolated; D, direct plating on Leifson's desoxycholate citrate agar (Hynes's modification); T, enrichment in Kauffmann's tetrathionate broth overnight and subculture on desoxycholate citrate agar; S, enrichment in selenite F medium overnight and subculture on desoxycholate citrate agar.

In Table 2 the results of examination of faeces of cases, convalescents, and carriers for *Salm. typhi* by the methods in routine use at the Shrewsbury Public Health Laboratory are compared. Part 1 of the table shows that in the examination of 116 specimens the organism was isolated from 115 by selenite F enrichment, from 46 by tetrathionate enrichment, and from 22 by direct plating on desoxycholate citrate agar.

In the later stages of the outbreak direct plating was abandoned except in special cases; Table 2, part 2, compares the results obtained using the two-liquid media; *Salm. typhi* was isolated from 297 out of 308 specimens after enrichment through selenite F, but from only 98 out of 308 specimens after tetrathionate broth enrichment.

In this outbreak, therefore, *Salm. typhi* was isolated approximately three times as frequently through selenite F as compared with Kauffmann's tetrathionate broth, and approximately six times as frequently as compared with direct plating on desoxycholate citrate agar.

IV. SEARCH FOR CARRIERS AMONG KITCHEN WORKERS

At the onset of the outbreak meals at the hospital were prepared in three kitchens; the Nurses' Home kitchen catered for the nursing staff and the night staff; the Main Hospital kitchen supplied the wards and domestic workers; a canteen

provided meals for out-patients, visitors, and non-resident workers. Of the 28 suspected cases reported by 9 September, 26 either fed at the Nurses' Home dining-hall or else had easy access to food from the Nurses' Home kitchen; the remaining two were patients in the children's ward which was the nearest ward to the Nurses' Home. It was conceivable, although never established, that food might, on occasion, be carried across to this ward from the Nurses' Home. The staffs of the Nurses' Home kitchen, Main Hospital kitchen and Hospital food stores were interrogated on 9 September. Repeated specimens of excreta were requested, and a rectal swab and blood specimens were collected.

On 12 September, *Salm. typhi* was isolated from the faeces of the lady cook (M.B.) in charge of the Nurses' Home kitchen. This finding was confirmed by examination of a rectal swab and further specimens of faeces. Her blood serum, however, contained no agglutinins against typhi H, O, or Vi antigens. This person, aged 21, was trained at a domestic science college and appeared to be an intelligent worker, well trained in the principles of kitchen hygiene. Careful inquiry disclosed no previous history of enteric fever or of unexplained pyrexia. At this stage three possibilities were considered:

(1) *That M.B. was the carrier responsible for the outbreak.* This was the most tempting conclusion to draw. The absence of any previous history of enteric fever did not, in itself, rule out this possibility. Klinger (1909) found that 163 out of 431 proven carriers gave no previous history of typhoid fever. M.B. had been on a camping holiday a few weeks before the outbreak and it was possible, for example, that her symptomless infection might have been acquired at that time.

(2) *That M.B. was incubating typhoid.* The absence of any serum agglutinins favoured this theory. She remained afebrile, however, throughout the 7 weeks during which she was known to be excreting *Salm. typhi*, and failed to develop any typhoid antibodies.

(3) *That M.B. was a 'symptomless excreter'* infected by the vehicle responsible for the main outbreak. As will be described later, 19 symptomless excreters were discovered in this community when investigations were extended to include all staff and patients.

V. SEARCH FOR EXCRETORS AMONG STAFF AND PATIENTS

On 12 September, the laboratory was asked to examine personnel handling food in the Nurses' Home dining-hall. Food handling by some of these was limited to carving of meat, serving of vegetables, etc. The duties of others included bread cutting, and preparation of sandwiches. Rectal swabs, repeated specimens of excreta, and blood were obtained, and the results of faeces examinations may be summarized as shown in Table 3.

This finding of six faecal excreters of *Salm. typhi* among the 13 members of the staff of the Nurses' Home dining-hall remaining on duty caused considerable concern. By this time detailed study, at the Central Enteric Reference Laboratory, of the strain of *Salm. typhi* isolated from the earlier cases had led Dr Felix to the conclusion that the strain was of relatively low virulence and that the proportion

of symptomless excreters and ambulant cases might be greater than would be expected in a typhoid outbreak due to an organism of average virulence. The situation at the hospital was considered by Dr Felix to be comparable to a paratyphoid-B outbreak where a relatively large number of ambulant excreters constitutes an increasing source of potential infection (Felix & Anderson, 1951).

Table 3. *Excreters among food handlers at work*

	Total staff	Already ill	Faecal excreters of <i>Salm. typhi</i>		
			Symptomless	Later developing typhoid fever	Faeces negative
Nurses' Home dining-hall	20	7	1	5*	7

* Blood taken from one of these cases showed a 'diagnostic' *typhi* H titre before the faeces examination was completed.

Meanwhile, the total number of cases and suspected cases at the hospital had risen to 80, of whom only 16 were hospital patients. It was decided to examine repeated specimens of excreta and blood from all members of the staff who had any close contact with the patients, i.e. all nursing staff, physiotherapists, domestic staff, ward orderlies, etc. It was hoped that these examinations would identify incubating cases and symptomless excreters who could then be isolated. This measure, it was felt, would be a valuable supplement to the strict hygienic precautions which had been instituted throughout the kitchens and hospital wards.

At the request of the hospital authorities the search for faecal excreters was later extended to include all hospital patients, and the results of the examinations of excreta of the various groups within the hospital are tabulated in Table 4.

Table 4. *Search for excreters*

Group	Date of first 'screening' by bacteriological methods	No. examined	Faecal excreters of <i>Salm. typhi</i> eventually discovered in the group	
			Symptomless	Later developing typhoid fever
Nurses' Home kitchen staff	9. ix. 48	6	1	0
Main kitchen staff	10. ix. 48	14	0	1
Canteen staff	10. ix. 48	12	0	0
Stores staff	10. ix. 48	3	0	0
Nurses' Home dining-hall staff	12. ix. 48	13	1	5
Maids' dining-hall staff	12. ix. 48	9	0	0
All nursing and domestic staff	27. ix. 48	208	9	8
Patients	16. x. 48	170	8	1
Total		435	19	15

VI. IDENTIFICATION OF INCUBATING CASES ('PRECOCIOUS CARRIERS')

Ledingham & Arkwright (1912) refer to the isolation of *Salm. typhi* from the faeces in the incubation period by Conradi (1907), Mayer (1910) and Prigge (1909). The last-named, in a survey of a population of 10,841, discovered 84 carriers, three of whom developed typhoid fever 18, 19 and 22 days after isolation of *Salm. typhi* from the faeces. Wilson (1938) described a milk handler who was found to be excreting *Salm. typhi* 4½ months before he suffered a severe attack of typhoid fever, and was regarded as the probable source of infection of 10 other cases before he developed symptoms.

In Oswestry, using highly selective media, 15 incubating cases were diagnosed by the repeated examination of faeces in an institutional outbreak. It is possible that a larger number of such cases would have been identified had it been practicable to have carried out a comprehensive survey of the community at an earlier stage in the epidemic.

Table 5. *Cases of typhoid fever diagnosed in the incubation period*

	No. of days febrile	Interval (in days) between first isolation of <i>S. typhi</i> and onset of fever	No. of positive faeces before onset of fever	Highest recorded temperature in ° F.
H.I.R.	1	3	1	100
S.S.	1	2	1	100
J.B.	2	4	1	101
I.C.	3	18	1	101
I.B.	5	1	1	100
A.B.	7	3	1	103·2
M.Ma.	7	3	1	100·4
J.P.O.	7	9	2	102
O.B.H.	8	5	2	100·8
R.D.	12	3	2	103
S.P.	14	2	1	100
H.J.	15	3	1	103·2
M.Mc.	15	10	1	102
M.H.	18	3	1	102
J.D.	50	1	1	104

VII. IDENTIFICATION OF SYMPTOMLESS EXCRETORS

As in other infectious diseases, the occurrence of cases of symptomless infection in outbreaks of typhoid fever has long been recognized. Klinger (1909) found that 56 % of carriers of *Salm. typhi* of less than 3 months' duration, and 20 % of carriers of over 3 months' duration gave no history of previous typhoid fever. In referring, however, to the finding of occasional symptomless excretors by Semple & Greig (1908) and Tsuzuki (1910), Ledingham & Arkwright (1912) made the comment that 'much fuller evidence is demanded before we can admit that such cases have never at any time exhibited symptoms'.

In the course of the Oswestry outbreak *Salm. typhi* was isolated from the faeces of nineteen persons who remained symptom free. From the outset precautions

Table 6. *Bacteriological and serological findings in nineteen symptomless excretors*

Case	No. of positive faeces	Known duration of excretion of <i>Salm. typhi</i>	Serum agglutinin titres			
			Date	<i>typhi</i> H	<i>typhi</i> O	<i>typhi</i> Vi
1. M.E.	30	28. ix. 48-12. i. 49 (106 days)	11. x. 48	1/200	—	.
			21. x. 48	1/200	1/100	.
			1. xi. 48	1/100	1/50	.
			7. xii. 48	1/25	—	—
2. M.B.	15	10. ix. 48-8. xi. 48 (59 days)	10. ix. 48	—	—	—
			14. ix. 48	—	—	—
			26. ix. 48	—	—	—
			29. ix. 48	—	—	—
			11. x. 48	—	—	—
			21. x. 48	—	—	—
			1. xi. 48	—	—	—
3. J.Gr.	12	16. x. 48-8. xi. 48 (23 days)	25. x. 48	1/50	—	.
			29. x. 48	—	—	.
			8. xi. 48	—	—	.
			29. xii. 48	—	—	—
4. M.W.	9	9. x. 48-11. xi. 48 (33 days)	8. x. 48	1/200	1/100	—
			19. x. 48	1/50	—	.
			3. xi. 48	—	—	.
			24. xii. 48	—	—	—
5. H.M.W.*	8	28. ix. 48-5. xi. 48 (38 days)	28. ix. 48	—	—	.
			11. x. 48	—	—	.
			17. x. 48	1/50	1/125	.
			23. x. 48	1/50	1/500	.
			12. xi. 48	1/25	1/125	.
6. J.Ga.	7	19. x. 48-9. xi. 48 (21 days)	15. xii. 48	1/25	1/25	—
			25. x. 48	1/200	1/100	.
			30. x. 48	1/125	1/50	.
			9. xi. 48	1/125	1/50	.
			25. xi. 48	1/125	1/25	.
			7. xii. 48	1/25	—	1/20
			10. i. 49	1/50	—	1/20
7. A.W.	7	18. x. 48-1. xi. 48 (14 days)	25. x. 48	1/100	1/100	.
			2. xi. 48	—	—	.
			2. xii. 48	—	—	.
			11. xii. 48	—	—	—
8. J.S.*	6	28. ix. 48-19. x. 48 (21 days)	11. ix. 48	1/50	—	.
			18. ix. 48	1/50	.	.
			11. x. 48	1/25	.	.
			21. i. 49	1/50	—	—
9. E.L.J.	5	27. ix. 48-26. x. 48 (29 days)	9. x. 48	—	—	.
			3. xii. 48	—	—	—
10. M.P.D.	5	15. xi. 48-2. xii. 48 (17 days)	15. xi. 48	—	—	.
			14. i. 49	—	—	—
11. J.Fo.*	4	17. x. 48-29. x. 48 (12 days)	25. x. 48	1/50	1/50	.
			3. xi. 48	1/50	—	.
			29. xii. 48	1/25	—	—
12. P.G.	4	21. x. 48-31. i. 49 (102 days)	2. x. 48	—	—	.
			4. x. 48	1/25	—	.
			27. x. 48	1/200	1/100	.
			9. xi. 48	1/50	1/25	.
			24. xi. 48	1/25	1/25	.

Table 6 (cont.)

Case	No. of positive faeces	Known duration of excretion of <i>Salm. typhi</i>	Serum agglutinin titres			
			Date	<i>typhi</i> H	<i>typhi</i> O	<i>typhi</i> Vi
13. D.C.	2	15. x. 48-9. xi. 48 (25 days)	25. x. 48	1/25	1/50	.
			11. xi. 48	—	—	.
14. E.A.D.†	1	15. x. 48	21. x. 48	1/200	1/100	1/30
			3. xi. 48	1/200	—	.
15. A.B.*	1	18. x. 48	25. x. 48	—	—	.
			11. xi. 48	—	—	.
16. Mrs B.	1	4. xi. 48	8. xi. 48	—	—	.
17. J.Fa.*	1	18. x. 48	25. x. 48	1/25	—	.
			11. xi. 48	1/25	—	.
18. M.M.	1	5. x. 48	4. x. 48	—	—	.
			12. x. 48	—	—	.
			21. x. 48	—	—	.
			1. xi. 48	—	—	.
19. M.E.R.	1	28. ix. 48	28. ix. 48	—	—	.
			11. x. 48	—	—	.
			21. x. 48	—	1/100	.

* Previous T.A.B.

† Previous attack of typhoid fever (1921).

were taken to avoid contamination of specimens of faeces during collection, and the sterilization of bed-pans was closely supervised. As soon as *Salm. typhi* was isolated from a symptomless person the suspected excreter was isolated and further specimens of faeces were examined daily. In six of the cases recorded in Table 6 *Salm. typhi* was isolated from only one specimen of faeces in each case, and it may be contended that failure to repeat the finding indicated possible contamination during collection of specimens in spite of precautions taken. It is equally possible that failure to confirm the finding may have been due to cessation of excretion of the organism by the individual shortly after collection of the first specimen.

It is highly unlikely that contamination occurred in the course of laboratory handling of specimens and cultures. At the time of the outbreak numerous specimens were received from sources other than the Orthopaedic Hospital and there was no evidence of contamination of these specimens, although they were examined concurrently with those from typhoid cases and carriers. Furthermore, *Salm. typhi* was not isolated from the urine of any symptomless person, although these specimens of urine were transported and handled together with specimens from which the organism was isolated.

In an attempt to obtain confirmation of infection by *Salm. typhi* repeated serological examinations were carried out on some of the symptomless excreters quarantined in the Shrewsbury area. In Table 6 it may be seen that serological results were negative in cases 2, 9 and 10, although *Salm. typhi* was isolated repeatedly from the faeces. Cases 1, 4-6 and 12, however, showed a serum agglutinin response which would normally be regarded as diagnostic of typhoid infection.

Salm. typhi was isolated from the faeces of case 13 on 15 October 1948, but attempts to repeat the finding were only successful in the thirteenth subsequent specimen received on 9 November 1948. Thirteen further specimens collected over a period of 3 weeks were all negative. The serological results in this case are also suggestive of a mild typhoid infection.

VIII. EXCLUSION OF A CHRONIC TYPHOID CARRIER AS A CAUSE OF THE OUTBREAK

Investigation of the milk supply to the hospital had shown that the piped water supply to the cowsheds of one of the two farms concerned was obtained partly from a roadside stream which might easily have been subject to chance pollution by wayfarers, and partly from a stream which proved to be heavily polluted by human sewage (Bradley *et al.* 1951). Persons living at the point of origin of this sewage were questioned and a man (R.J.T.) stated that he had suffered a severe typhoid infection in the Donnington Camp (Shropshire) outbreak of 1940. He and his family had come to live in the district in November 1947 (9 months before the onset of the epidemic). A heavy growth of *Salm. typhi* was obtained from each of three specimens of his faeces. Agglutinins against *Salm. typhi* were found to the following titres: H, 1/200+; O, 1/50; Vi, 1/15. Biochemical reactions of this strain of *Salm. typhi* differed from the Oswestry strain in that it failed to ferment xylose. Cultures were sent to the Central Enteric Reference Laboratory for detailed examination and were found not to be identical with the Oswestry strain (Felix & Anderson, 1951).

IX. EXCLUSION OF CONNEXION WITH TYPHOID CASES AT ANOTHER HOSPITAL

The only other cases of typhoid fever reported in the area served by the Shrewsbury Public Health Laboratory for at least two years occurred in the Welshpool district, approximately 20 miles from the Orthopaedic Hospital. The first case (Mrs F.W.) developed typhoid fever shortly after admission to the Welshpool Memorial Hospital where she underwent an operation for strangulated femoral hernia in April 1948. It was not clear whether infection had taken place at the hospital or before admission. *Salm. typhi* isolated from this patient proved to be Vi-phage type A.

Four months later (5 weeks before the onset of the Oswestry epidemic) Mrs S.L. was admitted to Welshpool Memorial Hospital, her illness being diagnosed as typhoid fever, again due to *Salm. typhi* Vi-phage type A. Mrs S.L.'s son (R.L.) had been discharged from the Welshpool Memorial Hospital where he had been a patient for 18 months as a result of a complicated fracture of the pelvis. He had been a patient throughout Mrs F.W.'s stay at the hospital and had, from time to time, suffered pyrexia which was attributed to the various complications of his fractured pelvis. Two specimens of R.L.'s serum, taken at an interval of about

5 months, were examined at the Central Enteric Reference Laboratory by Felix's technique and gave the following results:

	Titre of agglutination		
	<i>typhi</i> H	<i>typhi</i> O	<i>typhi</i> Vi
7. x. 48	1/2000	1/5000	1/2500 (standard)
18. iii. 49	1/500	1/2000	1/800 (standard)

These results were interpreted as indicating that the boy was not a chronic carrier. *Salm. typhi* was not isolated from this person in spite of repeated examination of his excreta. Attempts to obtain specimens of duodenal juice were unsuccessful. It was difficult to determine the exact part he played in this typhoid incident. It was possible, for example, that he had suffered an undiagnosed enteric fever at the Welshpool Memorial Hospital; that he was the source of Mrs F.W.'s infection; and after discharge from the hospital had infected his mother (Mrs S.L.).

It was felt that there might be a connexion between the Welshpool Hospital and the Orthopaedic Hospital outbreaks, especially as the Oswestry strain had proved to be of Vi-phage type A. There was occasional transfer of patients between the two hospitals but there had been no exchange of nursing or domestic staffs. Examination of blood and excreta of the staff at Welshpool revealed no suspicion of typhoid infection.

Towards the end of September 1948, another of Mrs S.L.'s sons (G.L.) developed typhoid fever, and cultures of *Salm. typhi* from this patient were sent to the Central Enteric Reference Laboratory for detailed examination. The cultures were of Vi-phage type A but were, nevertheless, found not to be identical with the Oswestry strain (Felix & Anderson, 1951).

X. SECONDARY INFECTION OF CONTACTS

The difficulty of determining the main vehicle of infection within the hospital, and the knowledge that at least one of the cases developed fever 30 days or more after infection, made it impossible to distinguish between primary and secondary cases. It is of interest that no secondary cases occurred at the homes of the 21 cases (16 female and 5 male) among the non-resident staff. Neither were any cases reported among home contacts of others who developed typhoid whilst on holiday or after discharge from the hospital.

An office worker (O.P.) who had not been away from Shrewsbury for several months developed typhoid fever on 10 November 1948. Inquiries made by Dr A. D. Symons, the Medical Officer of Health, showed that all her meals had been shared with four other members of her family with the exception of mid-morning refreshment, which included buttered buns, prepared by a member of the office staff. It was discovered that the person (J.R.) preparing this refreshment during the period 16 September to 4 October had been an orthopaedic patient at Oswestry and had been discharged from the hospital on 3 September 1948—the date of onset of typhoid in the earliest cases at Oswestry. Furthermore, this

person (J.R.), a cousin of the patient (O.P.), had been ill from 19 September until the middle of October, but had remained at work until 4 October. Her employers noticed that she was ill and persuaded her to consult her doctor, who later advised her to rest in bed for a few days. No investigations were carried out at the time and her temperature was not taken. The main features of her illness were persistent headache, drowsiness, backache, and hot and cold 'flushes'. Meanwhile, *Salm. typhi* isolated from the Shrewsbury case (O.P.) had proved to be identical with the Oswestry strain by means of the tests described by Felix & Anderson (1951). Repeated specimens of excreta and blood were examined from the possible carrier (J.R.). *Salm. typhi* was not isolated, and no serum agglutinins were present against *typhi* H, O or Vi. In spite of these negative findings the circumstantial evidence suggesting that J.R. infected the Shrewsbury patient (O.P.) is very strong. If this probability is accepted, it illustrates how infection may be transmitted by an undiagnosed abortive case even though laboratory examinations fail to confirm recent infection by *Salm. typhi* in such a case.

XI. A TYPHOID INCIDENT INVESTIGATED BY MOORE'S SEWAGE-PAD TECHNIQUE

Fourteen months after the end of the epidemic a non-resident employee of the hospital developed an illness which was eventually diagnosed as typhoid fever. From 3 December 1949, he complained of shivering attacks but remained at work for 7 days. He then complained of forgetfulness and of inability to concentrate on his work. Unaware of the cause of his mental confusion he decided, in view of his incompetence, to resign his post. For the next 9 days he suffered diarrhoea and abdominal pain and on the 16th day of illness called in a general practitioner, who sent blood to the laboratory for examination. Serum agglutinins against *Salm. typhi* were found to the following titres: *typhi* H, 1/1600; *typhi* O, 1/200.

The patient was transferred to an isolation hospital, where he was examined by the medical superintendent who found him to be apyrexial. His symptoms had abated, and clinical examination did not suggest an enteric infection. On the next day, however, pyrexia reappeared and his subsequent illness was typical of typhoid fever and was confirmed by the isolation of *Salm. typhi* from a rectal swab.

Inquiries were now made in conjunction with Dr Wilson Evans, Medical Officer of Health, of Oswestry. It was found that the patient was a member of the hospital engineer's staff and that one of his daily tasks was to enter the main hospital sewer to rake a grid. As he did not feed at the hospital it was felt that he might have acquired his infection in the course of his work in the sewer. Attempts were therefore made to isolate *Salm. typhi* from sewage by the gauze-pad technique (Moore, 1948).

Pads were placed in the main sewer for 48 hr. periods and then sent to the laboratory for examination. It will be seen in Table 7 that *Salm. typhi* was isolated from the second and third pads examined. It was considered probable that the patient had been infected from the sewage. An attempt was now made to find a carrier within the hospital. Pads were placed in six manholes (1-6) thus subdividing the hospital's sewage system into six sections. *Salm. typhi* was not

isolated from the first series of samples but the organism was recovered from trap 4 in the second series. Five days before the sewage pad from trap 4 had been sent to the laboratory *Salm. typhi* was isolated from the faeces of a female patient in the course of periodic testing of old typhoid cases at the hospital. Although this patient's faeces and urine had given negative results repeatedly during the previous 12 months, serological examination had shown a *typhi* Vi agglutinin titre of 1/20, and she had therefore been regarded as a person who might still be harbouring the organism. After this finding had been confirmed in a second specimen of faeces, she was sent to an Isolation Hospital as an intermittent excreter. Sewage from this carrier's ward passed through trap 4, and it is shown in Table 7 that all attempts to isolate the typhoid bacillus from sewage were negative after the removal of this patient from the hospital site. In this instance, therefore, the finding of a carrier in the hospital by routine faeces examinations had forestalled probable detection of the same carrier by the gauze-pad method of sewage examination. Nevertheless, the findings illustrate the potential value of this method in tracing the source of infection in certain epidemics.

Table 7. *Bacteriological examination of sewage*

Date of removal of pads from sewer	Sites of sewers examined	Result and date	
28. xii. 49	Main sewer	Negative	5. i. 50
23. i. 50	Main sewer	<i>S. typhi</i> isolated	26. i. 50
27. i. 50	Main sewer	<i>S. typhi</i> isolated	31. i. 50
30. i. 50	Traps 1-6	Negative	2. ii. 50
6. ii. 50	Traps 2, 3, 5 and 6	Negative	9. ii. 50
	Trap 4	<i>S. typhi</i> isolated	9. ii. 50
7. ii. 50	Carrier sent to isolation hospital		
9. ii. 50	Main sewer	Negative	14. ii. 50
13. ii. 50	Traps 2-6	Negative	16. ii. 50
24. ii. 50	Traps 1-4 and 6	Negative	27. ii. 50
3. iii. 50	Traps 1-6	Negative	11. iii. 50
7. iii. 50	Traps 1-6	Negative	13. iii. 50
13. iii. 50	Traps 2-4 and 6	Negative	20. iii. 50

The typhoid cultures isolated from the sewer worker, from the intermittent carrier and from the sewage were all of Vi-phage type A and showed the characteristics of the Oswestry strain (Felix & Anderson, 1951).

It should be mentioned that since these investigations were carried out Moore now recommends a longer period of immersion of the pads in the sewage (Lendon & Mackenzie, 1951). The findings also indicate that this type of sewage examination might well be employed as a supplementary post-epidemic method of ensuring that an institution has been cleared of chronic carriers; even so, intermittent excretors might still escape detection. The importance of routine application of the Vi-agglutination test to every recovered typhoid patient is thus emphasized (Ministry of Health, 1945).

XII. DISCUSSION

In recent years, three major advances in the bacteriological investigation of typhoid epidemics have been developed. In the first place there has been a great improve-

ment in the media employed to isolate *Salm. typhi* from the faeces. Secondly, facilities for detailed typing of strains of the organism are now available. Apart from attempts to detect minor biochemical differences between strains of *Salm. typhi*, there were no means of proving that a carrier's strain was identical with the epidemic strain until Vi-phage typing was introduced (Craigie & Yen, 1938). Thirdly, the Vi-agglutination test serves as an aid to the detection of chronic carriers (Felix, 1938).

The use of highly selective techniques in the search for faecal excreters in future typhoid epidemics may well lead to the finding of a greater number of incubating cases and symptomless excreters than it has been the custom to expect in the past. The proportion of symptomless excreters will probably depend on the virulence of the strain of *Salm. typhi* responsible for the outbreak. Thus an epidemic due to a strain of relatively low virulence will be comparable to a paratyphoid-B epidemic where a high proportion of ambulant excreters, usually attributed to the relatively low virulence of *Salm. paratyphi B*, increases the difficulty of tracing the true source of infection, and also leads to a higher incidence of secondary cases.

Reports of successful investigation of typhoid epidemics in the past frequently described the finding of a single carrier who had had the opportunity of infecting milk, food or water. In the Oswestry outbreak the absence of strong evidence pointing to any one vehicle of infection led to a wide search for the carrier who originated the epidemic. As a result several food-handling symptomless excreters were discovered, none of whom could be regarded as chronic carriers. It may be reasonable to assume that a temporary symptomless excreter could, in certain circumstances, infect food, milk or water thereby initiating a major epidemic. On the other hand, had an excreter been discovered at the hospital who gave a history of previous typhoid infection it might have been difficult to decide whether such a person was a true chronic carrier or whether he had been reinfected by the epidemic strain.

When a true chronic carrier, who had no direct connexion with the hospital, was found to be living at a point where his excreta could have contaminated the hospital milk supply, it was hoped that the source of the epidemic had been traced, but detailed examination of this carrier's strain of *Salm. typhi* led to the conclusion that he had no connexion with the outbreak. It is a matter for speculation whether the same conclusion would have been reached a few decades ago when techniques for detailed typing were not available.

SUMMARY

1. An account is given of the finding of 19 symptomless excreters of *Salm. typhi* and of 15 incubating cases ('precocious carriers') in the course of repeated examination of the faeces of 435 staff and patients at the hospital.

2. In a comparison of the frequency of isolation of *Salm. typhi* from the faeces it was found that the selenite F enrichment method was more efficient than enrichment in Kauffmann's tetrathionate broth, and that both methods were superior to direct plating on desoxycholate citrate agar.

3. Examination of sera from cases of typhoid fever showed that, in some cases, H- and O-agglutinin titres which would normally be regarded as 'diagnostic' may be reached well within the first week of illness.

4. Measures are described which were taken to exclude connexion between this outbreak and (a) a chronic carrier found during investigation of the hospital milk supply, and (b) the incidence of typhoid fever at another hospital.

5. A case of typhoid fever at Shrewsbury is described which was probably infected by an abortive missed case. Attempts to make a retrospective bacteriological or serological diagnosis of the latter were unsuccessful.

6. An account is given of the use of the sewage-pad method in an attempt to trace the source of infection in the case of a sewer worker who developed typhoid fever 14 months after the main epidemic and was probably infected by an intermittent carrier at the hospital.

I wish to acknowledge the help I received in many ways from several colleagues in the Public Health Laboratory Service; in particular Dr A. Felix, F.R.S., for his guidance in the investigation of the epidemic and his interest in the preparation of this paper. I wish to record my appreciation of the efficient and unstinting work of Mr P. H. Everall, F.I.M.L.T., and the technical and clerical staff of the Shrewsbury Public Health Laboratory. My thanks are also due to Dr G. H. Grant, Pathologist to the Royal Salop Infirmary, and his staff for their willing co-operation.

REFERENCES

- BRADLEY, W. H., EVANS, L. W. & TAYLOR, I. (1951). *J. Hyg., Camb.*, **49**, 324.
 CONRADI, H. (1907). *Klin. Jb.* **17**, 297.
 CRAIGIE, J. & YEN, O. H. (1938). *Canad. Publ. Hlth J.* **29**, 448, 484.
 FELIX, A. (1924). *J. Immunol.* **9**, 115.
 FELIX, A. (1930). *Lancet*, **1**, 505.
 FELIX, A. (1938). *Lancet*, **2**, 738.
 FELIX, A. & ANDERSON, E. S. (1951). *J. Hyg., Camb.*, **49**, 349.
 FRY, R. M. (1944). *Mon. Bull. Minist. Hlth & Emerg. Publ. Hlth Lab. Serv., Lond.*, **3**, 103-4.
 HOBBS, B. C. & ALLISON, V. D. (1945). *Mon. Bull. Minist. Hlth & Emerg. Publ. Hlth Lab. Serv., Lond.*, **4**, 12-19.
 KLINGER, P. (1909). *Arb. Reichgesundh.Amt.* **30**, 584.
 KNOX, R., GELL, P. H. G. & POLLOCK, M. R. (1943). *J. Hyg., Camb.*, **43**, 147.
 LEDINGHAM, J. C. G. & ARKWRIGHT, J. A. (1912). *The Carrier Problem in Infectious Diseases*. London.
 LENDON, N. C. & MACKENZIE, R. D. (1951). *Mon. Bull. Minist. Hlth & Emerg. Publ. Hlth Lab. Serv., Lond.*, **10**, 25.
 MAYER, G. (1910). *Zbl. Bakt.* **53**, 234.
 MINISTRY OF HEALTH (1945). *Mon. Bull. Minist. Hlth & Emerg. Publ. Hlth Lab. Serv., Lond.*, **4**, 224.
 MOORE, B. (1948). *Mon. Bull. Minist. Hlth & Emerg. Publ. Hlth Lab. Serv., Lond.*, **7**, 241.
 PRIGGE, R. (1909). *Klin. Jb.* **22**, 245.
 SCOTT, W. M. (1941). *Lancet*, **1**, 389.
 SEMPLE, D. & GREIG, E. D. W. (1908). *Sci. Mem. med. sanit. Dep. India*, no. 32.
 TSUZUKI, J. (1910). *Arch. Schiffs- u. Tropenhyg.* **14**, 147.
 WILSON, W. J. (1938). *J. Hyg., Camb.*, **38**, no. 4, 507.