Waterborne infectious disease in Britain

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SUMMARY

The incidence of waterborne typhoid fever and other intestinal infections in Britain since 1937 is reviewed. In the light of the current very low incidence of indigenous typhoid fever, it is suggested that the examination of relevant waterworks staff and prospective employees for carriage of the agents of potentially waterborne disease should be done selectively by appropriate laboratory tests only when indications for them are revealed by medical assessment.

INTRODUCTION

In 1974 regional Water Authorities were set up in Britain to assume statutory responsibility for all stages of the water cycle – including potable supplies, rivers and sewerage, which were previously administered separately by different bodies. The National Water Council was established at the same time to co-ordinate the work of these Authorities. Increased use of water, and the consequent need for re-cycling with its attendant potential hazards of pollution, render it important not only to monitor the safety of supplies but also to keep the quality of the sources under surveillance. It is also important to educate and train personnel to observe uniform codes of safety and good hygienic practice to ensure the continued excellent quality of British water supplies. Since the new Authorities inherited a variety of recommendations previously observed by the former Water Companies and Boards, it is opportune to reconsider such advice and the effect it may have had on the prevention of waterborne infectious disease in Britain over the past 40 years, with particular reference to typhoid and paratyphoid fever.

TYPHOID FEVER IN BRITAIN

The last waterborne outbreak of typhoid fever where a major piped supply was contaminated occurred at Croydon in 1937 with 310 cases (Holden, 1939). In other countries comparable outbreaks occurred at Zermatt in 1963 with 437 cases

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(Bernard, 1965) and at Dade County, Florida, with 255 cases (Hughes *et al.* 1975). Other outbreaks in Britain in which water has been a direct or indirect vehicle have been due to a variety of circumstances.

Some outbreaks have been due to the consumption of untreated or inadequately treated well-water subject to pollution by chronic carriers. This was the cause of a series of cases among the guests of an hotel in Cornwall from 1941 onwards, culminating in five deaths in 1944 (Bradley, 1949). An outbreak of typhoid fever at Ballyreagh in Northern Ireland in 1959 was attributed to a polluted well at a caravan site where there was no sewage disposal system (Brewster, Donaldson & Logue, 1960).

Other incidents have been caused by the drinking of water from polluted streams or rivers. In the summer of 1948 an outbreak occurred at Kilcreggan in Scotland (Anderson, 1951). The persons involved were attending a Sunday School outing, and as the day was very hot, they had drunk water from a stream. Investigations eventually showed that the stream was polluted by sewage from a nearby cottage, in which a chronic carrier lived who had contracted typhoid fever in 1895. In a small outbreak at Bristol in 1949 the patients had drunk water from a stream subject to pollution by sewage from a private mental hospital where one of the inmates was a chronic typhoid carrier (Hutton, 1950). Three similar outbreaks occurred in Hampshire, Belfast and Edinburgh respectively. The outbreak in Hampshire in 1951 involved the River Wallington which was subject to pollution from the Purbrook sewage system after heavy rainfall (Lendon & McKenzie, 1951); a chronic carrier was eventually found living in a house about half a mile from the river. In the outbreak near Belfast, affecting nine persons between 1951 and 1954, the source of infection was a stream polluted by a chronic carrier (Murdock & Lawson, 1957). In the third outbreak a number of persons who developed typhoid fever between 1963 and 1970 had all drunk water from the 'Water of Leith' in Edinburgh. A sewer from a group of houses where a chronic carrier lived had been erroneously connected to a surface drain which discharged into the stream (Conn *et al.* 1972). In each of these outbreaks the ultimate source of infection was a chronic typhoid carrier, from whose home untreated sewage had gained access to the water causing heavy pollution, and the victims had drunk the river water intentionally.

Other outbreaks of typhoid fever have occurred in which polluted water was not directly involved as the vehicle of spread. In the large outbreak at Bournemouth and Poole in 1936, infected milk was the immediate source, but the water from a polluted stream which flowed through the dairy farm had been the means of introducing the infection from a carrier to the farm (Shaw, 1937). During 1950–8 several cases were associated with the consumption of oysters from West Mersea and it was subsequently discovered that the waters of the estuary from which the oysters were gathered and partly cleansed in pits were polluted by a chronic carrier (Pilsworth, 1960).

The large outbreak at Aberdeen in 1964 associated with imported corned beef (Scottish Home and Health Department, 1964) was preceded by several similar but smaller outbreaks in which corned beef or other imported canned meats were
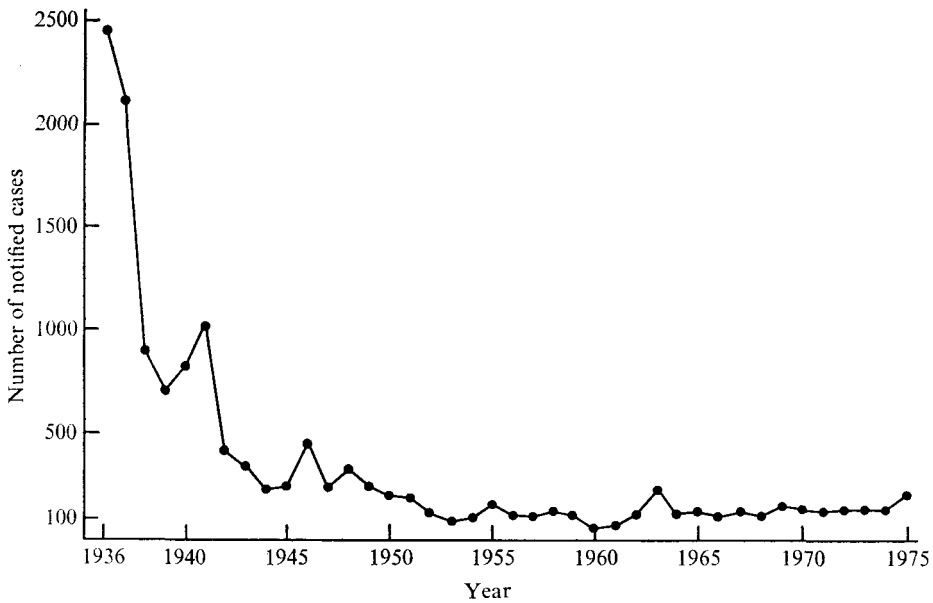
Fig. 1. Total yearly notifications of typhoid fever since 1936 in the U.K. excluding
Scotland (Registrar General's figures).

Table 1. *Cases of typhoid fever recorded in the Communicable Disease Reports**
of the P.H.L.S. in 1975–6

	1975	1976
Infected abroad	180	175
In contact with persons from abroad	4	2
Not recently abroad	35	23
Total	219	200

* The Communicable Disease Report covers England, Wales and Northern Ireland.

Table 2. *Sources of typhoid fever infections contracted abroad*

	1975	1976
Europe (excluding Britain)	32	25
Near East	12	8
Far East	6	6
Africa	16	17
Indian subcontinent	110	115
Other	4	4
Total	180	175

suspected as the vehicle of spread. These occurred at Crowthorne (Moore, 1950),
Pickering (Couper, Newell & Payne, 1956), Harlow (Ash *et al.* 1964) and others at
South Shields and Bedford. The source of the outbreak at Oswestry Orthopaedic
Hospital in 1948 was never conclusively proved at the time (Bradley, Evans &
Taylor, 1951) but after subsequent re-examination of the epidemiological evidence
and of the phage type of the organisms isolated, it is probable that infected

corned beef was also the cause of this outbreak (Anderson & Hobbs, 1973). Although the immediate vehicle of infection in these outbreaks was canned meat, the use of untreated or inadequately chlorinated river water to cool the cans in the processing plants in South America was ultimately responsible for introducing the infecting agent into the cans (Howie, 1968).

Apart from the Aberdeen outbreak, a notable feature in Britain for more than two decades has been the very low incidence of typhoid fever infections (Fig. 1). Furthermore, during 1975–6 for example, out of 419 patients with typhoid fever in England, Wales and Northern Ireland notified to the Epidemiological Research Laboratory of the Public Health Laboratory Service, 355 (85 %) were infected abroad, and a further 6 were contacts of persons from abroad. The details are given in Tables 1 and 2. In no instance, where it was possible to determine the source of infection in patients infected in Britain during this period, was water or water supplies incriminated. A similar survey for Scotland during 1967–74 (Sharp & Heymann, 1976) showed that of 64 patients with confirmed typhoid fever during that period, 35 acquired the infection outside Britain.

PARATYPHOID FEVER IN BRITAIN

Enteric fever caused by *Salmonella paratyphi B* is more often food-borne than waterborne, but polluted water may occasionally be involved directly or indirectly. Of the 40 outbreaks reviewed by Savage (1942), only one was definitely waterborne. This occurred in 1941 at Brixworth among the inhabitants of a group of condemned cottages. Their only water supply was from a well subject to pollution from defective drains (Jones, Gell & Knox, 1942). At Beccles during the years 1939–45 a series of cases due to a new phage type occurred among school children who had used a swimming pool on the River Waveney during the summer holidays. It was discovered that crude sewage was being discharged directly into the river from a group of nearby bungalows where two healthy excretors of the same phage type of organism were living (Martin, 1947). Sloan, Wilson & Wright (1960) described a similar outbreak associated with a particular stream near Edinburgh. The investigation of the source of the contamination was complicated by the fact that several phage types of *Salmonella paratyphi B* were involved, but a chronic carrier excreting at least three of the phage types simultaneously was eventually discovered (Bernstein, 1960). In 1946 at Wootton, Isle of Wight, an outbreak of paratyphoid fever occurred in circumstances very similar to those of the Bournemouth typhoid outbreak (Wallace & McKenzie, 1947). Infected milk was the immediate vehicle of infection but a stream running through the dairy farm concerned was found to be subject to pollution by sewage effluent from a cesspool at a house where two chronic excretors were subsequently found. Similar water- and milk-borne outbreaks occured in Gowrie (Kelman, 1944) and in Worcestershire (Parry, 1942). More recently in North Yorkshire a waterborne outbreak of paratyphoid fever occurred in a group of villages at the same time as an outbreak of infection with the same phage type in cattle and in human beings at a farm several miles away (George *et al.* 1972; Harbourne *et al.* 1972). The water supply to

the villages was derived from several springs in a valley and, although chlorinated, the water was subject to pollution from a fractured drain from a septic tank at a cottage occupied by workers at the infected farm. The infections at the farm originated from a stream which received effluent from a sewage-disposal works serving a village where a chronic carrier was detected.

OTHER WATERBORNE INFECTIONS

Sewage polluted water may also be responsible for outbreaks of dysentery and gastro-enteritis. Thus, an outbreak in a Somerset town was due to inadequately chlorinated well-water, subject to pollution from the cesspits of nearby houses (Green & Macleod, 1943). A similar outbreak occurred in a village in Scotland where untreated well-water was used (Murchison, 1966).

In Montrose (Green *et al.* 1968) a large explosive outbreak of Sonne dysentery and gastroenteritis was considered to be due to failure in the chlorination of the town's water supply which was derived from a sewage-polluted river.

Cross-connexions between raw river water and the main potable supply have been responsible for other outbreaks of dysentery or gastroenteritis as in Leicester (Ross & Gillespie, 1952) and at Rochdale in 1974 (Watkinson, personal communication). In contrast, despite their occurrence in many other countries, there have been no reports of waterborne outbreaks of viral hepatitis or parasitic disease such as giardiasis although the incidence of viral hepatitis in Britain is very much greater than that of typhoid fever.

DISCUSSION

In the United States the incidence and causes of waterborne disease are reviewed periodically. Craun & McCabe (1973) described 358 outbreaks which occurred between 1946 and 1970, and affected 72358 persons. Outbreaks involving private supplies were nearly all associated with untreated ground-water. In public piped supplies, the majority of outbreaks were caused by contamination in the distribution system via cross-connexions and back siphonage, although a few were associated either with contamination at the source or breakdown in treatment. Similar reviews (Merson *et al.* 1974) for the years 1971–2 record 47 outbreaks affecting 6817 persons and in 1973, 24 outbreaks with 1720 cases (Hughes *et al.* 1975). In contrast to this, it is remarkable that in the 40 years since the Croydon episode there have been so few outbreaks of waterborne infectious disease in Britain. While improvements in sewage treatment and disposal and the adoption of efficient methods of purification for all but the smallest piped supplies of drinking water have no doubt played their part, the risk of waterborne typhoid fever in particular has been greatly reduced by the approximately tenfold decrease in the incidence of this disease in Britain since 1937 (Fig. 1), with a resulting proportional decrease in the number of chronic carriers in the community. Furthermore, since most of the new infections notified each year have been acquired abroad, the population among whom chronic carriers are most likely to be found is becoming more clearly defined. Also noteworthy is the virtual absence of

secondary spread from person to person – the communicability of typhoid fever is frequently overemphasized. A further safeguard is the present system of central notification of new cases, and the referral of all strains of *S. typhi* isolated to the Enteric Reference Laboratory of the Public Health Laboratory Service for phage typing. These enable efficient surveillance of cases and carriers to be maintained.

The immediate source of the Croydon outbreak in 1937 was thought to be a chronic typhoid carrier employed by the water undertaking for building work in an adit to a well (Ministry of Health, 1938). However, the exact way in which the contamination occurred was never fully established, but owing to an unfortunate series of circumstances in relation to the work the water was distributed without chlorination. With a view to preventing the recurrence of such an incident, the Ministry of Health (1939) issued an advisory Report (Memorandum No. 221) on the precautions and safeguards to be taken in the day to day management of water works. One recommendation was that 'every man who was to work on any part of the works where there was a risk of his contaminating the supply, should be examined by means of a Widal test of his blood to ascertain whether or not he is likely to be a typhoid carrier'. In 1961 a Working Party of the Public Health Laboratory Service studied the problem of detection of the typhoid carrier state, with particular reference to the Vi agglutination test. It suggested rewording the relevant passage in Memorandum No. 221, together with the addition of notes on the agglutination tests to be performed, and on the significance of the results obtained (PHLS, 1961). These modifications were later incorporated in a revised edition of the Memorandum (Ministry of Housing and Local Government, 1967). We are not aware of any similar recommendations officially issued in any other countries, but in a recent publication (W.H.O. 1976) on the surveillance of drinking water quality, the relevant part of the British memorandum is quoted verbatim.

It is tempting to attribute the complete absence of any waterborne typhoid outbreaks caused by chronic carriers working for water undertakings since 1937 to the serological screening of water works employees. In fact, despite the many thousands of tests on staff and prospective employees which have been done each year, in Britain there is not a single report of a carrier ever having been detected by this or by any other method. Furthermore, as the number of carriers in the population decreases each year, the chances of finding one by this means become less and less. The same reasons apply also to paratyphoid, although there is one report of a water works employee found to be a carrier of *S. paratyphi B* (Sharp, 1965). This discovery was the result of serological and bacteriological screening of 69 employees of the Edinburgh City Waterworks, undertaken as a special project after an outbreak of paratyphoid fever in the city spread by contaminated cream cakes. Although in this paper the need to screen water works employees by serological and cultural tests is reaffirmed, there was no evidence that the man ever infected anyone.

The effectiveness of the Vi agglutination test as a screening procedure has been challenged (Bokkenheuser, 1964; Bokkenheuser, Smith & Richardson, 1964). In countries where the incidence of typhoid fever is high there are certainly people with Vi antibodies who are not carriers, as in Malaya (Ponnampalan, 1967) and

Hong Kong (Forrest *et al.* 1967). The test should theoretically be more specific in countries where the incidence of typhoid fever is low, but in practice the disease could become so rare that screening of employees would no longer be justified or indeed necessary.

In the search for potential carriers of *S. typhi* the use of the Widal and Vi agglutination tests has certain inherent drawbacks. Thus, the presence of agglutinins does not itself indicate the carrier state but could mean that the person (i) has had enteric fever, (ii) has been inoculated with TAB or typhoid vaccine or (iii) has been infected by salmonellas or other organisms which share antigens identical with or similar to those of the enteric group. Serological screening can therefore do no more than indicate which persons require further testing by cultural methods. Furthermore, some chronic carriers of *S. typhi* have no relevant demonstrable antibodies and such persons could not therefore be detected by serological tests alone. Attempts to relate the presence of Vi agglutinins with carriage of *S. typhi* have usually been based on studies of groups of known chronic carriers in which the proportion with Vi agglutinins was compared with that in control groups from the normal population. Although some persons among the control group may have had TAB vaccine, few if any are likely to have had typhoid fever. One survey of this kind, carried out by a working party of the Public Health Laboratory Service, showed that Vi agglutinins were present in 48 of 67 known carriers (72 %), but in only 4 of 343 persons (1·2 %) in the normal control group (PHLS, 1961). In a similar survey in America, in which various methods of detecting Vi antibodies were studied, the standard Vi tube agglutination test was positive in 115 of 172 (66 %) chronic carriers, and in 22 of 200 (11 %) control persons (Schubert, Edwards & Ramsey, 1959). If there is in fact a close association between the presence of Vi agglutinins and chronic carriage of *S. typhi*, it should be possible, after a major outbreak of typhoid fever, to show that Vi antibodies persist in those patients who become chronic carriers, but disappear in others – from whom the organism is presumably eradicated. The Aberdeen outbreak in 1964 provided such an opportunity, although only 6 of 507 persons infected (1·18 %) became chronic carriers (Brodie, 1977a). In patients followed up by serial tests for 2 years, there was no difference between the carriers and non-carriers as regards persistence of Vi antibodies. Brodie also found a high incidence of Vi antibodies in healthy individuals whether or not inoculated with TAB vaccine, and he therefore concluded that the Vi agglutination test is of little value in screening for typhoid carriage; he also considered that it was of limited value in diagnosis. The continued use of a screening test which will fail to detect one out of three, or at the most four, typhoid carriers, and by which up to 90 % of 'positive' reactions may be given by non-carriers – as for example in South Africa – thus requires considerable justification (Bokkenheuser *et al.* 1964). The Vi agglutination test also has the added disadvantage of poor reproducibility in practice. For example, in a recent Quality Control exercise organized by the Public Health Laboratory Service six sera were examined for Vi agglutinins by more than 80 laboratories: taking titres of $\geq 1/5$ as positive, false results were reported in 211 of 498 (42 %) test results (Table 3).

Table 3. *Reports from laboratories on each of six quality control sera prepared by the Standards Laboratory for Serological Reagents*

Serum	Standards laboratory titre	Number of laboratories reporting titres of		False results (%)
		< 5 (−ve)	⩾ 5 (+ve)	
A	20 (+ve)	31	54	36·5
B	< 5 (−ve)	11	74	87
C	< 5 (−ve)	37	48	56·5
D	1280 (+ve)	10	74	12
E	320 (+ve)	5	68	6·8
F	20 (+ve)	43	43	50

False 'positives' (Sera B, C)	122 out of 170 tests (71·76%)
False 'negatives' (Sera A, F)	74 out of 171 tests (43·27%)
False 'negatives' (Sera D, E)	15 out of 157 tests (9·55%)
Total false results	211 out of 498 tests (42·37%)

Because of these limitations, the application of other methods for detecting Vi antibodies such as haemagglutination with Vi-sensitized sheep red cells (Schubert *et al.* 1959), complement fixation, Coomb's anti-human globulin (Brodie, 1977*b*) and other tests have been tried, but with limited success. Indeed from their work Chau & Chan (1976) concluded that a fluorescent Vi antibody test was superior to other methods for detecting typhoid carriers. In Britain, however, the direct agglutination test has so far remained the standard method, and it is doubtful in the present circumstances whether the use of more elaborate or sensitive techniques is justifiable or necessary in routine laboratories.

If a single test were to be recommended for the detection of typhoid carriers in the U.K. today, then, in the light of the current very low incidence of indigenous typhoid fever, the most efficient means would be culture of a specimen of faeces. This test could be applied in the examination of water works employees to all persons with a medical history suggestive of past typhoid fever, without any preliminary serological tests. In persons with such a history, more than one specimen of faeces as well as of urine would however usually be examined; if serological tests were also done, the results might well be useful in assessing how many further faecal specimens, if any, need be examined. Because excretion of *S. typhi* may be intermittent, persons convalescing from recent typhoid fever should be kept under bacteriological surveillance for at least 12 months before it can be established with any certainty whether or not they have become chronic carriers. In screening programmes the number of negative specimens stipulated must therefore be a compromise between what is theoretically desirable and what is practicable in the light of prevailing circumstances.

If the screening of prospective water works employees in Britain is to continue, it should be applied selectively – as is recommended by the Department of Health and Social Security (Report, 1974) for catering workers – to those persons in whom there are definite indications as revealed by question and medical assessment. At present, workmen may be referred for blood tests direct to laboratories by non-medical persons such as administrators, managers and engineers. It is

essential, therefore, that Water Authorities should have the services of medical advisers, preferably with some experience of laboratory medicine and epidemiology, who will be responsible for assessing the need for laboratory tests in consultation with medical microbiologists. Indications for such tests would include, for example, a past history suggestive of typhoid fever; recent visits to or residence in a country where the incidence of typhoid fever is high; a history of intestinal upset or prolonged pyrexial illness during or after visits abroad; or close contact with any outbreak of infectious intestinal disease, as at home. It must be emphasized that typhoid fever is not the only potentially waterborne infection, and the choice of laboratory tests to be done will vary according to circumstances. Appropriate tests may be serological or may involve microscopy and/or culture of specimens of faeces and urine.

It is illogical that in the catering and food trades, where many more persons including immigrants are employed and where direct contact with food provides numerous opportunities for spreading infection, screening tests are recommended only for those staff in whom definite indications exist, while in the water industry prospective employees are currently screened for typhoid carriage whether or not they have a suggestive history or any other indications. Laboratory facilities are limited and the annual cost of the tests at present done must be considerable. The education of all employees in the principles of hygiene and safety by clear codes of practice would not only be more economical but would in our opinion be more likely to prevent the recurrence of an outbreak such as that at Croydon than the continued use of a mass screening test of questionable value (Rahman & Cowan, 1974). There is, however, a clear need for more stringent medical assessment as opposed to laboratory screening of relevant personnel.

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