A BANTU 'TYPHOID MARY'

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From 18 September 1965 cases suspiciously like typhoid fever began to reach hospital from a Bantu school in the North-Eastern Transvaal. The *final* number of cases from this school was 53.

Typhoid fever is endemic in the district and, though local investigations commenced, the occurrence of the school cases was not reported to the regional office until 29 September. On 30 September a meeting of interested personnel was held at the school and I attended this meeting.

Relevant Data

The school accommodates 166 boarders and 144 day scholars. It is a modern institution, lavishly built and equipped: the classrooms, dining hall and dormitories are large, well lit, and well ventilated; toilets, basins, showers and baths are in a well-designed tiled annexe; sewage disposal is by means of a septic tank and French drains—some hundreds of yards from the source of water; kitchen, sculleries, and stores are modern, spacious and clean.

Water is pumped into gravity tanks from a well some 20 ft. deep and about 25 ft. from a clean, sandy, river bed. The well is protected by a concrete lining and cover and surface wash is diverted by an intercepting ditch. There was no evidence of human contamination of the surrounding ground, but some dried cattle manure was seen nearby. There is no human habitation in the vicinity—the attendant only visits the installation to start and stop the engine. In general it was quite obvious that this splendid school was both well organized and managed.

EPIDEMIOLOGY

Preliminary enquiries were made and it was stated that none of the food-handling staff had been in hospital or absent through sickness during the year, and that day scholars as well as boarders were infected. This indicated that the drinking water, used by day scholars and boarders alike, was contaminated. A fairly high *E.coli* (presumptive) count seemed to support this, and, *faute de mieux* the water supply was blamed.

In spite of the evidence I found it difficult to believe that the water was the source of infection and I resolved to return during the afternoon and examine the food handlers. The third one examined, a stout female cook, had 'boarding' of the right upper segment of the rectus muscle and intense deep tenderness in the gallbladder region. The following history was then obtained:

Cook T.M. was engaged at the school as a temporary cook in January 1965. In June she felt ill for 3 days and went to hospital from 10 June to 15 June. She felt 'hot all over', with a bad headache and abdominal pains. She was given capsules in hospital, felt better and was discharged after 5 days with some capsules to take at home: 'at home' being the school kitchen.

The hospital confirmed that she was admitted with presumptive enteric fever, treated with chloramphenicol, and, when her temperature returned to normal, she was discharged with some capsules and a 'final diagnosis' of enteric fever.

The hospital to which the typhoid schoolchildren were sent was next visited (not the hospital that attended to cook T.M.). It was then found that the statement that day scholars as well as boarders were infected was incorrect. A few day scholars had been sent to the hospital with headaches but they did not look like cases of typhoid, were not even ill, and, after an examination and finding them afebrile and symptomless for 2 days, they were discharged.

The true facts were that a key member of the hostel kitchen staff had definite clinical typhoid fever in June and a tender (presumably infected) gallbladder in September, and that, although the whole school drank from the untreated water supply, only boarders who ate at the hostel contracted typhoid fever.

THE OUTBREAK

First Wave

Date (September):	18	19	20	21	22	23	24	25	26	27	28
No. of cases	4	2	8	1	3	1	18	1	—	2	1

There were 4 hospital admissions on 18 September 1965 and the peak number of 18 admissions was reached on 24 September. After a 3-day lull the second wave occurred but was not serious.

Second Wave

Date (Sept.-Oct.):	29	30	1	2	3	4	5	6	7	8	9	10	11	12
No. of cases	—	—	—	2	—	2	—	—	3	—	1	—	—	1

Three other cases were admitted from the school on 25 October, 30 October and 7 November.

As will be referred to later, cook T.M. was removed from the school on 30 September so that all cases up to 12 October could have been infected by her. The 3 later cases could have acquired the infection from cured patients returned to school in an undetected convalescent carrier state, or from some undiscovered source of infection left behind by cook T.M. or from some other unexplained source.

At the time of writing, 5 weeks after the November case, no further cases had been reported.

Treatment of Cook T.M.

Cook T.M. was sent to hospital on 30 September. She was given a course of Ampicillin and Pantofenicol. It was probably taken for granted that this special case had had laboratory diagnosis carried out before admission because the usual blood culture was not carried out before treatment. To rectify this, treatment was stopped on the 6th, 7th and 8th days and blood for culture and Widal tests was taken as well as a stool specimen for culture. Blood and stool cultures were both negative; but the Widal test was positive in a titre of 1/400 for *S.typhi* 'H', and in a titre of 1/100 for *S.paratyphi* C 'H'. T.M. had never received prophylactic immunization.

It is suggested that the *S.paratyphi* C 'H' 1/100 positive is not significant, possibly an anamnestic reaction.

On 23 October—i.e. after 24 days in hospital and after 4 days without treatment—stool, blood and urine cultures were all negative. The Widal test was unfortunately not repeated. Cook T.M.'s brother, who is a Bantu minister of religion, undertook to look after her and ensure that she would handle no food except her own and would attend hospital regularly for tests. She was therefore discharged to his care after 34 days in hospital.

THE COURSE OF THE DISEASE

There were no deaths out of 53 cases and the disease was fairly mild in all but 1 of the cases. The patient who suffered severely was the only one who was not immunized with 2 injections of typhoid-paratyphoid A and B endotoxoid in May-June. The temperature and pulse charts, and general pattern of the disease were similar in all the cases, but the non-immunized boy was notably more seriously ill than the others. It seems probable that, though the immunization did not prevent the infection, it modified the severity of the disease—especially as there were no deaths.

Fifteen of the patients had 1 relapse, i.e. about 30%, and these cleared up after a second course of chloromycetin.

The Causative Organism

Blood culture was positive in 27 of the 53 cases (51%) and all were Vi-phage type A. Two stool cultures were positive and 8 Widal tests were significantly positive.

Treatment

The specific treatment was Chloromycetin, 250 mg. 6-hourly for 12-15 days, but 13 cases required from 22 to 26 days treatment because of relapses and positive stools.

In the case of cook T.M., her gallbladder pain was relieved after a few days treatment with ampicillin and Pantofenicol and did not recur.

CONCLUSION

It is beyond reasonable doubt that one Bantu school cook suffering from typhoid fever, and released from a hospital prematurely, infected at least 53 schoolchildren, that is, one-third of the school boarders. This episode is described because it illustrates clearly a tendency when investigating typhoid outbreaks to make superficial enquiries, take a water sample and blame the water supply if the presumptive *E.coli* content is above some arbitrary standard—even though the *E.coli* content could be of avian, bovine or wild animal origin.

It has been my experience in dealing with typhoid outbreaks that deep palpation of the upper abdomen will usually incriminate a person, among contacts, with a tender gallbladder who gives a history of recurring diarrhoea and is possibly responsible for the outbreak.

I have come across numerous reports on the investigation of cases of typhoid fever, which all state (to my mind without justification) that the source of infection was undoubtedly the poor water supply—even though cases occurred in only 1 family or 2 adjacent kraals out of hundreds of users of the supply. In one case, the only available water was from a deep borehole.

These reports are all signed by health inspectors, and it is suggested that such investigations would be more useful if medical personnel took part in them.

The clearing up of gallbladder symptoms after a few days of ampicillin and Pantofenicol in a person who had typhoid fever treated with chloramphenicol in June and gallbladder tenderness in September, suggests that ampicillin was effective after a comparative failure with chloramphenicol.

SUMMARY

A cook, a convalescent carrier of typhoid fever, caused 53 cases in a Bantu school. The carrier was discovered when palpation of the right upper abdomen indicated a cholecystitis. Misleading information had at first wrongly suggested that the water supply was the source. All cases recovered with chloramphenicol treatment though 15 (30%) had relapses and needed 2 courses of treatment.

The importance of a medical examination of contacts of typhoid fever cases is emphasized, and water should not be too readily blamed. The danger of discharging infective typhoid patients from hospital and the vitally important part played by food handlers in spreading the disease are also accentuated by this occurrence.

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