

CROSS-INFECTION IN HOSPITAL DUE TO *SALMONELLA DERBY*

By SYDNEY D. RUBBO

From the School of Bacteriology, University of Melbourne

(With Plates 5 and 6 and 3 Figures in the Text)

Cross-infection in hospital wards has been the subject of much discussion in recent years. The principles underlying the control of gastro-enteritis, particularly in children's wards, have been described by Sauer (1935), Harries (1935), Field, MacCarthy & Wyllie (1943), by the Medical Research Council War Memorandum No. 11 (1944) and by the Ad Hoc Committee (1946).

The purpose of the present communication is to focus attention once more on the seriousness of cross-infections by describing some features in the natural history of an outbreak of gastro-enteritis among infants. As far as can be discovered from a survey of the literature, studies on hospital cross-infections have been concerned mainly with the spread of haemolytic streptococci (Allison & Brown, 1937; Cruickshank & Godber, 1939), diphtheria bacilli (Glass & Wright, 1938; Crosbie & Wright, 1941) and other upper respiratory tract pathogens. However, the spread of gastro-intestinal infections has not been subjected to intensive study mainly because of the frequent difficulty in establishing the aetiology in many of these outbreaks (Rice, Best, Frant & Abramson, 1937; Crowley, Downie, Fulton & Wilson, 1941). In the present instance we were fortunate in dealing with *Salmonella derby*, a pathogen of apparent rarity in Australia. This fact greatly simplified our search for its distribution in the environment of the patients.

One of the most interesting features of this outbreak, the bacteriological aspects of which are fully described by Mushin (1948), was the recovery of *Salm. derby* from five samples of ward dust. This finding somewhat reorientated our previous views on the spread, and hence control, of gastro-intestinal infections. We have failed to discover any reports on gastro-enteritis among infants in which the dust factor of spread was demonstrated bacteriologically or seriously considered. For this reason it is hoped that the present findings may offer some small contribution towards the recurring problem of institutional enteritis.

The second interesting feature of this particular outbreak was the finding of *Salm. derby* as the causative agent (Mushin, 1948). This organism has

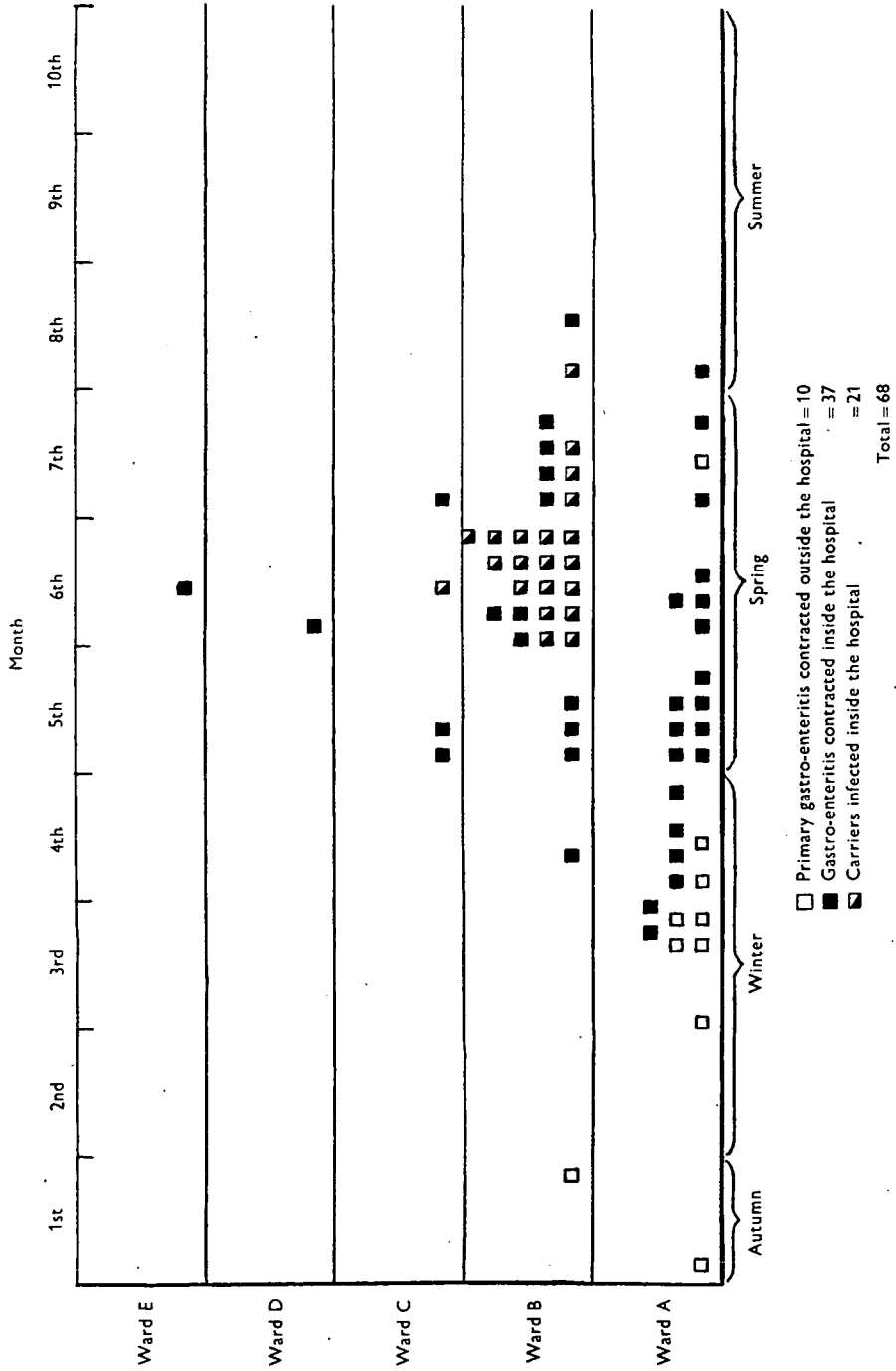
seldom been encountered in human enteric infections in Australia, only seven sporadic cases having been recorded previously (Atkinson & Woodroffe, 1944; Atkinson, Woodroffe & Macbeth, 1947). Mushin in her review of the incidence of *Salm. derby* in man, concluded that this organism is an infrequent cause of salmonellosis in the United Kingdom, but is more frequently encountered in America. It is noteworthy that the epidemic described herein was the largest outbreak of *Salm. derby* yet recorded in man. This was due largely to the circumstances under which it occurred.

CLINICAL ASPECTS

Very briefly, the outbreak included forty-seven cases of gastro-enteritis due to *Salm. derby*, of which thirty-seven were contracted in hospital over a period of eight calendar months, commencing in the autumn of 1946 and extending throughout the winter and spring and terminating at the commencement of the summer. In addition, twenty-one carriers were detected, all of whom were infected while in-patients at the hospital.

The clinical signs and symptoms were typical of acute inflammation of the alimentary tract being characterized by an acute onset of vomiting, diarrhoea (sometimes with blood and mucus), high temperature and dehydration. Treatment was chiefly concerned with maintaining an adequate fluid balance by saline transfusions, while sulphaguanidine therapy was used in attempt to control the infectious element, but without much success.

Ten of the patients were admitted with the primary diagnosis of gastro-enteritis from which *Salm. derby* was recovered by culture of the stools. These cases were not included in the cross-infected group (Text-fig. 1) but it should be emphasized that intervals up to a week elapsed between the time of admission and the performance of the bacteriological examination. In view of the evidence to be presented later it is not inconceivable that some of these primary cases of gastro-enteritis were, in fact, not due to *Salm. derby* but were secondarily infected with *Salm. derby* whilst in hospital.



Text-fig. 1. The distribution of *Salmonella derby* infections in hospital.

Of the cases which were admitted for conditions other than gastro-enteritis, twelve were suffering or recovering from broncho-pneumonia, five were dietetic problems, three upper respiratory tract infections, two were cases of bronchiolitis, two suffered from pyloric stenosis, two from meningitis and two from hare lip. Other cases developing gastro-enteritis in hospital suffered from prematurity, tonsillitis, disseminated tuberculosis, eczema, burns, congenital syphilis, etc. The great majority (fifty-six cases), which were brought to our notice on account of superimposed gastro-enteritis, were infants under 1 year of age.

Among the forty-seven cases there were ten deaths. The autopsy findings (five cases) consistently revealed enlargement and hyperaemia of the lymphoid follicles of the ileum and colon and, in one instance only, ulceration of the lower bowel. These findings suggest only slight damage to the alimentary canal in terms of morbid anatomy and it was apparent that the morbid physiological effects of fluid loss had a more profound influence in contributing towards a fatal result. No attempt is made here to interpret the pathogenicity of *Salm. derby* in infants because, in the majority of our cases, this infection was superimposed on other debilitating illnesses. The healthy infant may suffer no more than a mild diarrhoea with slight vomiting or even be symptomless, whereas the sick child may present a far more serious syndrome, not infrequently terminating in death.

EPIDEMIOLOGICAL ASPECTS

The initial cases of gastro-enteritis in the first 3 months were admitted under this diagnosis and were due to *Salm. derby* infection. They appeared to be quite unrelated to one another in point of time and they arose in widely separated geographical areas. In two cases at least it seemed probable that infection was contracted outside Australia, as the patients were admitted to the hospital immediately on arrival in this country. It is difficult, however, to explain the appearance of the causal agent in the other seven cases particularly in the light of Mushin's experience over the past 2 years. During this time no case of *Salm. derby* had been detected (Mushin, unpublished). It is not improbable, as already suggested, that these unrelated cases were infected in hospital or, alternatively, the stools were contaminated adventitiously with *Salm. derby* during collection. The remaining twenty-seven cases and twenty-one carriers were infected with *Salm. derby* during hospitalization for conditions other than gastro-enteritis.

One curious feature in fourteen cases was the development of gastro-intestinal symptoms in ap-

parently healthy infants 2-5 days after discharge. This delayed onset was attributed to contraction of infection while in hospital and its manifestation at the clinical level when removed from the attention and rest characteristic of the hospital environment. This supposition was strengthened by detection of infant carriers of *Salm. derby* in the wards who, subsequent to the bacteriological diagnosis, developed signs and symptoms of gastro-enteritis. Further, as there were no other admissions of *Salm. derby* infections from sources outside the hospital, it seemed unlikely that the fourteen cases mentioned above contracted infection after their discharge. Accordingly, in the present analysis, all cases developing clinical gastro-enteritis in hospital or within several days of discharge were classed as cross-infections.

In Text-fig. 1 the smouldering nature of the outbreak is shown, together with its distribution in the wards and the number of cases considered to have arisen as a result of cross-infection.

It will be noted that the onset of the outbreak was not characterized by an explosive suddenness involving many cases scattered throughout the hospital as would be expected if the contamination of food prepared in the central kitchen was responsible. In contrast, there was a slow increase in morbidity reaching a maximum in the fifth and sixth months of the outbreak (September and October), then a fairly rapid decline as improved methods of ward hygiene were introduced. In view of the nature of the incidence rate and the restricted distribution in the hospital (confined mainly to wards A and B) it was concluded that the cases arose from intermittent contamination of isolated feeds in the wards. This conclusion was strongly supported by the bacteriological findings and, in consequence, formed the basis upon which methods of control were recommended.

In ward A the primary cases of gastro-enteritis (Text-fig. 1 □ squares) were sufficient to provide the initial seeding of the infection which, once established in the ward, was self-perpetuating. However, in ward B only one case was admitted in the first month and the second case, attributed to cross-infection, developed after an interval of 4-5 weeks. The third, fourth and fifth cases in the same ward B developed 3 or 4 weeks later and in the sixth month the incidence of cross-infection rose acutely and rapidly in this ward. These facts suggested that cross-infection first commenced in ward A following the admission of the primary cases to that ward and probably spread to ward B on the hands of a nurse or through the agency of a carrier nurse or nurses working in both wards. Once established the continuation of the epidemic in both wards was undoubtedly achieved by the various pathways shown in Text-fig. 2.

PATHWAYS OF INFECTION

The problem of determining possible pathways of infection usually demands correlation of epidemiological data, environmental factors, clinical and bacteriological findings. In the present study such a correlation was possible and formed the basis upon which Text-fig. 2 was constructed.

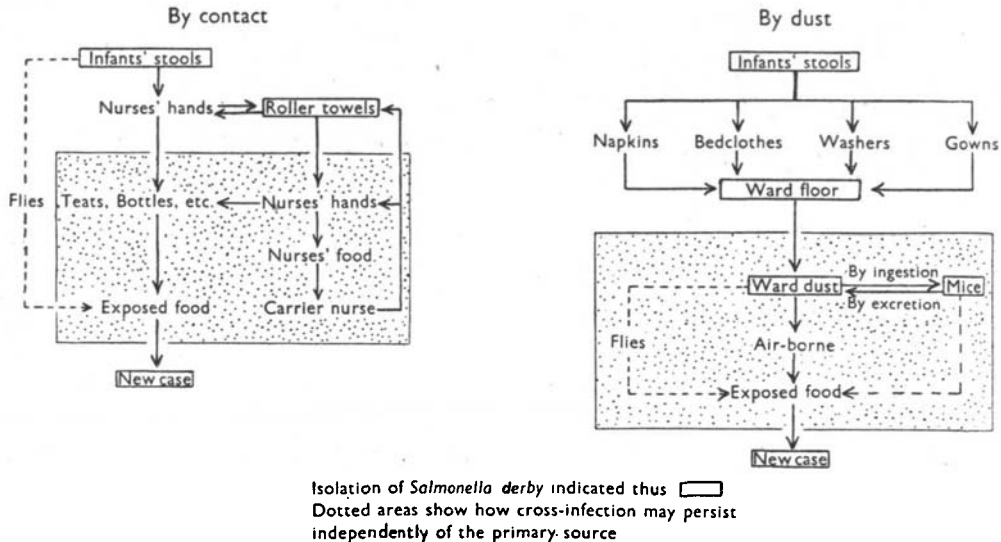
As would be expected, emphasis is placed on the contamination of exposed food. The novel feature in this diagram, however, is the supposition that food contamination arose, not only through the accepted mechanism of contact, but also through the agency of dust.

the height of the outbreak. From one *Salm. derby* was isolated, from the other a heavy coliform flora.

The broken lines in Text-fig. 2 postulating fly-borne infection of food and direct contamination by mice were included for theoretical completeness. There was no evidence that either of these mechanisms operated here.

CONTROL OF INFECTION

This resolved itself into a problem of introducing well-established techniques designed to eliminate contact and dust contamination of exposed surfaces.



Text-fig. 2. Probable pathways of infection due to *Salmonella derby*.

The isolation of *Salm. derby* on several occasions from the dust of the ward floors, from dust lying on top of a linen cupboard, and the recovery of the organism from a mouse provided strong circumstantial evidence on this point. This evidence, when considered with the method of handling feeds in the wards, strengthened our contention that some of the cases were due to air-borne dust contamination of food or food utensils (viz. teats). The following photographs (Pl. 5, 6, figs. 1, 4, 10) taken in the ward at the time of feeding show how readily this could happen. Further, Pl. 5, fig. 5 shows how teats may be contaminated even by the most skilful nurse.

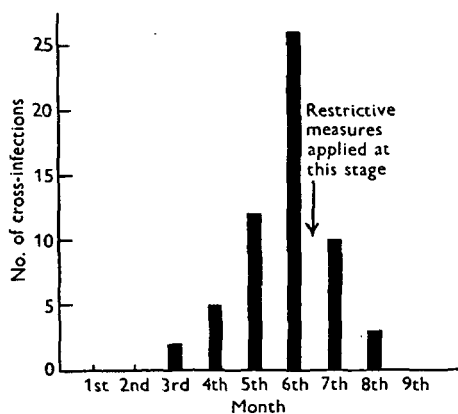
The communal roller towel has been strongly condemned by all authorities on hospital cross-infection, but seldom is it possible to recover the specific gastro-intestinal pathogen from these articles. We were singularly fortunate in our examination of two ward towels taken at random during

In the present instance our attention was centred on food surfaces and articles entering the infant's mouth. It is not within the scope of the present article to enter into a detailed description of our recommendations. Details of procedure in nursing practice and hospital management depend entirely on local conditions, such as the availability of staff, the construction of wards, the disposition of cases in the wards, the methods of handling food, etc. The following photographs (Pl. 5, figs. 2, 3, 6 and Pl. 6, figs. 10, 11) show very briefly some of the mechanical methods adopted for preventing contamination by contact and by dust.

One minor point illustrated in Pl. 6, figs. 7, 8 is worthy of comment. These photographs show how the disinfection of hands may be ineffective not because of an inadequate disinfectant but because of incomplete filling of bowls and consequent imperfect immersion of the hands.

In addition to revising nursing methods in order to lessen the risk of contact contamination of food, and recommending the oiling of floors and bed linen, ultra-violet lamps were installed in one ward. The object here was to prevent the accumulation in the ward dust of any recently shed *Salm. derby* of human or rodent origin.

It is freely admitted that not all our recommendations were acted upon for reasons outside our control, but those that were introduced appeared to contribute towards the waning of the outbreak. This is well shown in Text-fig. 3 below.



Text-fig. 3. Number of infants contracting infection in hospital.

DISCUSSION

In this particular outbreak of gastro-enteritis we were fortunate in dealing with a pathogen, *Salmonella derby*, which, up to the present outbreak, had not previously been isolated in Melbourne. For two years prior to this Mushin had examined stools of all cases of gastro-enteritis admitted to this hospital, approximately 260 faecal specimens, without once isolating this pathogen. More recently, May 1947, further cases due to *Salm. derby* have been admitted. In view of these facts and on the evidence that two of the primary cases described herein contracted infection outside Australia it is not improbable that *Salm. derby* was first introduced into Victoria, Australia, during the latter half of 1945 or early in 1946. This contention is further supported by our knowledge from actual experience that the carrier state may continue as long as 12 months without showing any abnormal gastro-intestinal symptoms. As *Salm. derby* has been responsible for a large percentage of recent cases and outbreaks in other institutions we have come to regard it as one of the

most important causes of gastro-enteritis in infants in this city (Melbourne).

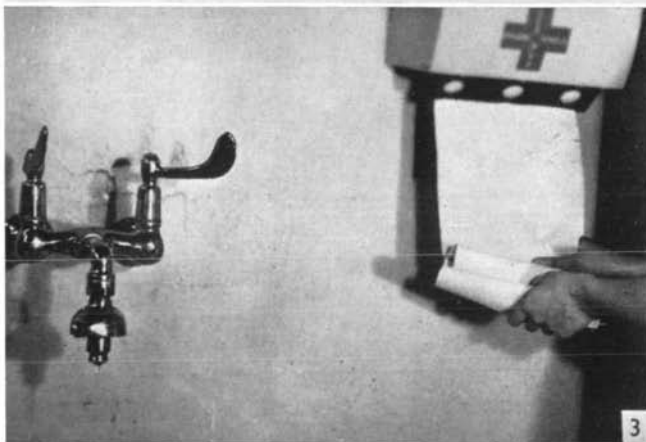
Apart from the interest we hope to stimulate in this organism we would wish to place equal emphasis on its mode of transmission. As already stated, the literature on cross-infection in hospital is wanting in examples wherein a specific gastro-intestinal pathogen has been recovered from ward dust. This gap in our knowledge is now closed, for it was only too evident in this particular outbreak that *Salm. derby* could be recovered almost as consistently from ward dust as from an actual case. This fact, combined with the practice of delivering milk feeds in open containers to the wards, was undoubtedly responsible for many of our cross-infections. Accordingly, in addition to measures designed to control dust and improve ward hygiene we strongly urge as a general principle that all foods for infants under 1 year of age be protected by sterilized close-fitting covers, preferably of glass, which are assembled in a central food kitchen and removed aseptically only at the moment of feeding. The bottle shown in Pl. 6, fig. 9, is an ideal one for this purpose.

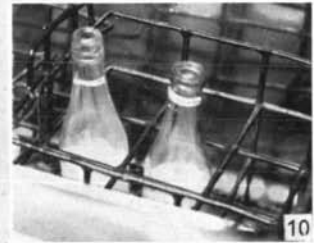
In conclusion, it is hoped that this paper may re-emphasize the tragic reality of hospital cross-infections of the gastro-intestinal type. We have made no attempt to detail methods of control because these are matters of internal organization. Our main purpose has been to indicate in general terms what methods must be considered. These may be summarized thus: (1) the detection of carriers; (2) the control of contact with food by aseptic nursing technique; (3) the control of dust contamination of food.

SUMMARY

1. An outbreak of gastro-enteritis in hospital due to *Salmonella derby* is described.
2. The probable pathways of infection are indicated diagrammatically (Text-fig. 2).
3. Attention is drawn to the importance of dust as an environmental source of infection in gastro-enteritis in a closed community.
4. Methods of control including the detection of carriers, the avoidance of contact with anything that goes into an infant's mouth and the protection of exposed food from dust contamination are stressed.

My thanks are due to my colleague, Rose Mushin, M.Sc., for undertaking the bacteriological work, to the Superintendent for his co-operation, to Miss Hanson and the Visual Aid Centre of the Melbourne University for photographic work and to the Board of the Hospital for permission to publish this paper.





REFERENCES

- AD HOC COMMITTEE (1946). Cross-infection in children's wards. *Brit. med. J.* **1**, 673.
- ALLISON, V. D. & BROWN, W. A. (1937). Reinfection as a cause of complications and relapses in scarlet-fever wards. *J. Hyg., Camb.*, **37**, 153.
- ATKINSON, N. & WOODROOFE, G. M. (1944). The occurrence of *Salmonella* types in Australia. *Aust. J. exp. Biol. med. Sci.* **22**, 51.
- ATKINSON, N., WOODROOFE, G. M. & MACBETH, A. M. (1947). The occurrence of *Salmonella* types in Australia, 3. *Aust. J. exp. Biol. med. Sci.* **25**, 25.
- CROSBIE, W. E. & WRIGHT, H. D. (1941). Diphtheria bacilli in floor dust. *Lancet*, **1**, 656.
- CROWLEY, N., DOWNIE, A. W., FULTON, F. & WILSON, G. S. (1941). Epidemic neonatal diarrhoea in maternity hospitals. *Lancet*, **2**, 590.
- CRUICKSHANK, R. & GODBER, G. E. (1939). The aerial spread of streptococcal infections. *Lancet*, **1**, 741.
- FIELD, C. E., MACCARTHY, D. & WYLLIE, W. G. (1943). A standardized plan of treatment for gastro-enteritis in infants. *Brit. med. J.* **1**, 371.
- GLASS, V. & WRIGHT, H. D. (1938). Cross-infection in diphtheria wards. *J. Hyg., Camb.*, **38**, 248.
- HARRIES, E. H. R. (1935). Infection and its control in children's wards. *Lancet*, **2**, 173 and 233.
- MED. RES. COUN. LOND. WAR MEM. (1944). The control of cross-infection in hospitals. No. 11, 34 pp.
- MUSHIN, R. (1948). An outbreak of gastro-enteritis due to *Salmonella derby*. *J. Hyg., Camb.*, **46**, 151.
- RICE, J. L., BEST, W. H., FRANT, S. & ABRAMSON, H. (1937). Epidemic diarrhoea of the new-born. *J. Amer. med. Ass.* **109**, 475.
- SAUER, L. (1935). Enteritis in infants: Prevention of its spread. *J. Pediat.* **6**, 753.

LEGENDS TO PLATES 5 AND 6

For explanation of these photographs see p. 161. Fig. 2 shows a ward roller towel used before this outbreak and Fig. 3 ward tissue towelling now in use.

(MS. received for publication 13. II. 48.—Ed.)