Epidemic Infantile Gastro-enteritis Today

'But for the grace of God . . . ' the episode described by Jacobs *et al.* on p. 656 of this issue would have occurred in nearly any paediatric unit in this country, as they were not designed to prevent epidemics of *Escherichia coli* enteritis. Such epidemics will most frequently affect hospitals like Booth Hall where the consultants have special skills, so that babies are transferred for their attention from other paediatric centres, where they may have become carriers of an enteropathogenic serotype of *Esch. coli* (Rogers, 1963).

Since the Aberdeen epidemic when many babies were infected in their homes (Giles, Sangster, and Smith, 1949), epidemics have most frequently been started by the admission of a carrier, or a baby with enteritis, who became infected when in a different community of babies. The Manchester epidemic was difficult to control because as in Aberdeen there was an endemic infection within the community; paediatric units serving the area thus came to admit babies excreting the same enteropathogenic *Esch. coli*, which helps to explain why so many hospitals in Manchester became involved in the epidemic.

Epidemics spreading into neighbouring paediatric units have been described (Rogers and Kogler, 1951, and Thomson, Watkins, and Gray, 1956). By 1952 in Birmingham this form of enteritis created serious problems, and to prevent babies from an infected environment being admitted to clean wards, the city's bacteriologists informed the Public Health Department of the presence of enteropathogenic types of *Esch. coli* in their units, and the Medical Officer of Health circulated to the city's paediatricians and bacteriologists a weekly summary naming the hospital wards in which babies were excreting these specific types of *Esch. coli* (Rogers, 1956). When epidemics became less frequent and serious, this scheme was discontinued. In the recent Teeside and Manchester epidemics a similar system was introduced, but only when the epidemics had caused serious disease: these two epidemics should remind everyone of the need to

prevent an epidemic spreading into neighbouring hospitals, and this would be helped if local notification schemes were always used in the larger conurbations.

Marshall (1969) stated that cross-infection had been prevented by a specially designed 20-bedded gastro-enteritis unit at Monsall Hospital, but he presents a picture typical of ward cross-infection, with a pattern of disease the same as that recorded between 1948 and 1956. He noted that four of the fatal cases were admitted with an attack of non-specific gastro-enteritis, from which they apparently recovered 5 to 10 days before they were readmitted with a recurrence of diarrhoea and vomiting from which they died: other cases which eventually recovered had a similar history. He presented no data to show that these babies were excreting the same *Esch. coli* on their first and second admissions.

Nowadays few paediatric units have adequate isolation facilities. This is particularly dangerous when a baby has *Esch. coli* enteritis, as there is very rapid heavy contamination of its environment, and though a ward may be made of up cubicles, infection spreads through its length just as if no cubicle walls were present (Giles *et al.*, 1949; Rogers, 1951; Gibson and Mann, 1969): this may also occur when a baby has severe salmonella enteritis. The spread of infection in an isolation unit might be prevented if each cubicle became a miniature self-contained ward (Rogers, 1956): because of the expense, and the number of nurses required in such a unit, and because since 1956 epidemics as serious as that recently encountered in Manchester rarely occur, such strict isolation facilities are seldom required and no such unit has been built. Many hospitals try to prevent infants who are carriers being admitted to 'clean' wards and screen the faeces of newly admitted babies; Gibson and Mann (1969) take a rectal swab in the ambulance before a baby is admitted to their unit, but few hospitals could do this. Some hospitals have found rectal swabs unreliable, because the very loose mucosa of the anal canal prevents the cotton wool swab reaching

the rectum, so the cultures may be very unreliable as there is neither faeces nor even faecal staining on the swab. The lubricated sheath of the sleeve swab is easily passed through the anus and the swab can be pushed through this into the rectum to provide a better specimen (Rogers, 1954).

Jacobs' paper deals with one part of the epidemic described by Marshall (1969). Their reports record that several of the babies were vomiting on readmission. Because the vomit contains the enteropathogenic type of *Esch. coli* causing the diarrhoea (Rogers, 1951; Thomson, 1955), the examination of throat swabs can assist the screening programme to identify carriers.

As many experienced virologists have failed to prove that epidemics of severe diarrhoea have been caused by a virus, when an unexplained epidemic breaks out it would be better to spend time looking for a new or less well-known enteropathogenic sero-type of *Esch. coli* than for a virus (Taylor, 1970). Adam (1923) found that strains of *Esch. coli* from every child involved in an epidemic provided the same biochemical reactions: this identification of a common antibiogram will help to show that a single serotype of *Esch. coli* is involved in an epidemic. The Salmonellae-Escherichia Reference Laboratory of the Public Health Laboratory service always gives very generous assistance with this sort of problem. (All those who have had to investigate an epidemic of *Esch. coli* enteritis will miss its director, Joan Taylor, when she retires in the near future.) This laboratory can not only serotype any *Esch. coli* but also state whether it has been associated with infantile enteritis. This was illustrated by the epidemic described by Hughes, Greaves, and Bettelheim (1968).

Valman and Wilmers (1969) did not report any bacteriological relapses following oral gentamicin therapy. Jacobs and his colleagues are cautious in their reports on gentamicin therapy; they had evidence of reduced morbidity but they wondered if the prolonged excretion rates they observed were because the antibiotic was given systemically. We have given oral gentamicin therapy; the number of patients treated is too small for a significant analysis, but there has been a bacteriological relapse rate similar to those after treatment with other antibiotics, in the region of 20%.

Mann (1969) has suggested that chemoprophylaxis could encourage the emergence of resistant strains, but Datta (1968 and 1969) has shown that a resistant factor (R) is carried by many wild enterobacteria, and in natural circumstances in the bowel of man it can be transferred to sensitive strains, making them resistant to a wide range of antibiotics.

Moorhouse (1969) found that a high proportion of healthy babies out of hospital were excreting antibiotic resistant enterobacteria carrying R factors.

After Todd and Hall's controlled trial showed that chemoprophylaxis reduced the incidence of a gastro-enteritis when an enteropathogenic *Esch. coli* was in a unit (Todd and Hall, 1953), many paediatric units used chemoprophylaxis to protect infant contacts of a baby excreting recognized enteropathogenic *Esch. coli*. When chemoprophylaxis was widely practised in the period before 1960, there were no reports of resistance developing in an epidemic strain during this therapy. Induction of resistance in an epidemic strain due to R factor transfer is more likely than such resistance developing because of chemoprophylaxis.

Infants undergoing specialized investigations and surgery are in hospital longer than other babies, and as it is known that the cross-infection rate rises the longer the baby is in hospital (Anderson, Crockatt, and Ross, 1954), it is significant that the most serious part of the Booth Hall epidemic was in the infants' neuro-surgical unit.

The life of a baby with severe enteritis is saved by the treatment of the dehydration and by electrolyte replacement and not by antibiotic therapy which will, however, reduce the number of organisms excreted and therefore the environmental contamination. Jacobs *et al.* (1970) describe the elaborate efforts made to identify and correct the unusual metabolic upsets they encountered, in spite of which some babies died. Wide, and sometimes, adverse publicity was given to the Teeside epidemic and even more to the Manchester epidemic. Perhaps those who came near to pillorying the paediatricians and the Public Health Authorities for their control of that epidemic will read this journal and appreciate the extremely high standard of care and control exercised at Booth Hall Hospital in both the wards and the Laboratories, and the significance of the spread of the infection in the home.

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