Gastroenteritis in Salisbury European Children— a Five-year Study*

"To everything there is a season."
—Ecclesiastes, 3, 1.

BY

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Vomiting and diarrhoea, which are the presenting symptoms of this disorder, are of course common symptoms in infancy and childhood occurring as features in a wide variety of illnesses, e.g., infection either within or without the gastrointestinal tract, dietetic disturbances, metabolic derangements, allergic states, poisoning and feeding difficulties. When employing the term gastroenteritis one refers to vomiting and diarrhoea occurring on a basis of an intestinal infection, even though the exact causal agent is often not identified. The occurrence of outbreaks and its mode of spread strongly suggest an infective cause, even though this is not always demonstrated.

Gastroenteritis remains a serious illness of infancy and childhood and an important cause of infant and childhood mortality, though mortality has dropped tremendously, especially among the economically privileged population groups. The disease is more serious in infancy and early childhood than later. This is partly due to the infant and young child's greater susceptibility to infections and their spread, but mainly due to the more rapid and serious water and electrolyte disturbances which ensue in this age group. Traditionally one thinks of it as a summer disease, its spread being aided by flies and the easier growth of bacteria on warm media, hence the name "filth diarrhoea." In all sophisticated areas this is no longer the case, and one finds that the disease is now as common in the winter months.

Etiology

The infective agents are bacterial, viral, protozoal and fungal. Among the bacteria are *Shigella, Salmonella, E. coli, Staphylococci, Proteus* and *Paracolons.* In staphylococcal infections one has to think of food poisoning both due to the organism itself and due to preformed toxins, primary staphylococcal infection which is mainly a disease of the newborn, the disease complicating abdominal surgery and the diarrhoea which results from a disturbed intestinal flora particularly following the use of antibiotics. Amongst the viruses are the Echo, Adenovirus and the Coxsackie groups. These viruses have not been isolated with any degree of regularity. Among the protozoa are amoebiasis and giardiasis. Giardia, though usually producing a chronic form of diarrhoea on occasion, can produce the acute form. Amongst the fungi the important one is monilia, which again occurs mainly as a complication of antibiotic therapy.

Clinical Picture

The clinical picture is too well known to describe in detail, but several points deserve stressing. A feature one attempts to recognise early is the development of dehydration. It is well to remember that not all cases present with the classical vomiting and diarrhoea. Some are ushered in by a convulsion or unexplained fever. The convulsion may be the simple febrile type, but may occur on the basis of encephalopathy, cerebral thrombophlebitis or tetany when there has been much vomiting. A rectal examination often may provide in such cases an early diagnosis. Excessive irritability at first gives a clue to dehydration in some children before the classical features of sunken eyes, loss of tissue turgor and poor pulse volume are manifest. In some children a striking thirst develops as an early sign of dehydration, and others become reluctant to feed early on in their illness. These latter children tend to become dehydrated earlier. Mothers may notice oliguria early, but they may mistake the nappies stained with watery stools as evidence of satisfactory urinary output. This is a trap in hospital practice as well, and unless the observer thinks of this possibility he may easily miss severe watery diarrhoea. Other children show the over-breathing of acidosis as a feature early on and out of proportion to dehydration. This in itself causes the loss of large amounts of water by transpiration and calls for early intravenous treatment. Another ominous sign one often sees in practice is the greyish skin colour suggestive of poor circulation. This may occur without one having seen the earlier cherry-red appearance of the lips so often described.

Management of the disease consists of isolation, identification of the causative organism where possible, exhibition of the appropriate antibiotic, ensuring adequate hydration, prevention of metabolic disturbances, recognising these and treating them when they arise.

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It might be informative to analyse the experiences of European gastroenteritis cases admitted to the Wilkins' Infectious Diseases Hospital over the last five years and continue our discussion using these figures as the basis.

Table I illustrates the numbers of cases admitted in each month over the five-year period. It gives an idea of the size of the problem among European children in the Salisbury area. It will be seen that there are two peaks of admissions occurring over May, June and July and another smaller peak during the hotter months. This pattern is also shown graphically in Fig. 1. In 1964-65 there was also a large number of cases admitted in March and April. (The Salisbury municipal figures are taken from July to June for each year.)

Fig. 2 shows the results of bacteriological examinations carried out on specimens of stools of the patients. In Fig. 2a the analysis of the peak month May, 1965, is shown. Where more than one organism was isolated, only the one considered to be the dominant one is recorded. No pathogens were isolated in the vast majority of cases, sterile cultures being obtained from 106 of the 230 cases. Strains of *Shigella* were found in 22 cases, *Salmonella* in 16, *Staphylococci* in 12, Monilia in 7, Giardia in 4 and enteropathic *E. coli* in 4. No specimens were submitted for examination in five cases. Many children had received antibiotics prior to admission and this may have obscured the picture, but one's impression is that this has not been a very important factor in our failure to isolate the infective agent in the majority of cases. In a three months' period in 1960 before the start of this series, specimens from 31 consecutive cases were submitted for bacteriological examination prior to the start of any treatment. Of these, 15 grew *Staph. aureus,* two grew enteropathic strains of *E. coli,* while 14 were sterile. The significance of the isolation of *Staphylococci* in such cases is doubtful. Over the last five years investigations of children not admitted to hospital prior to starting treatment have yielded essentially the same negative results. The excretion of the affecting organism may be intermittent and the percentage of positive cultures might be a little higher if repeated specimens were examined. This is especially so in the case of *Salmonella* infections.

The explosive outbreaks occurring in the winter months suggest a viral cause, but this has not been shown in our cases investigated. In 1960 seven stools were submitted to the South African Institute for Medical Research for virus studies with negative results. In the period 1962-63, 12 specimens were submitted with negative results, and in the 1963-64 period, out of another 12 specimens, Echo virus was grown from one specimen only. One child, not included in the series, died before reaching hospital. Specimens taken from various organs were submitted for virus studies with negative results. The case for a virus cause of gastroenteritis so far is not a strong one. In an investigation carried out during the winter months in the Baragwanath hospital, Johannesburg, entero viruses were recovered from 18 per cent. of children with diarrhoea and 13.5 per cent. of

Table I

GASTROENTERITIS OVER A FIVE-YEAR PERIOD

	1960-1961	1961-1962	1962-1963	1963-1964	1964-1965
July	9	13	22	13	11
August	8	6	9	11	9
September	9	7	8	9	10
October	2	12	12	13	17
November	8	8	12	9	11
December	13	18	12	9	14
January	4	11	7	15	15
February	6	3	2	9	19
March	5	4	6	5	27
April	2	9	3	10	23
May	11	30	25	19	51
June	18	34	31	22	23
TOTAL	95	155	149	144	230

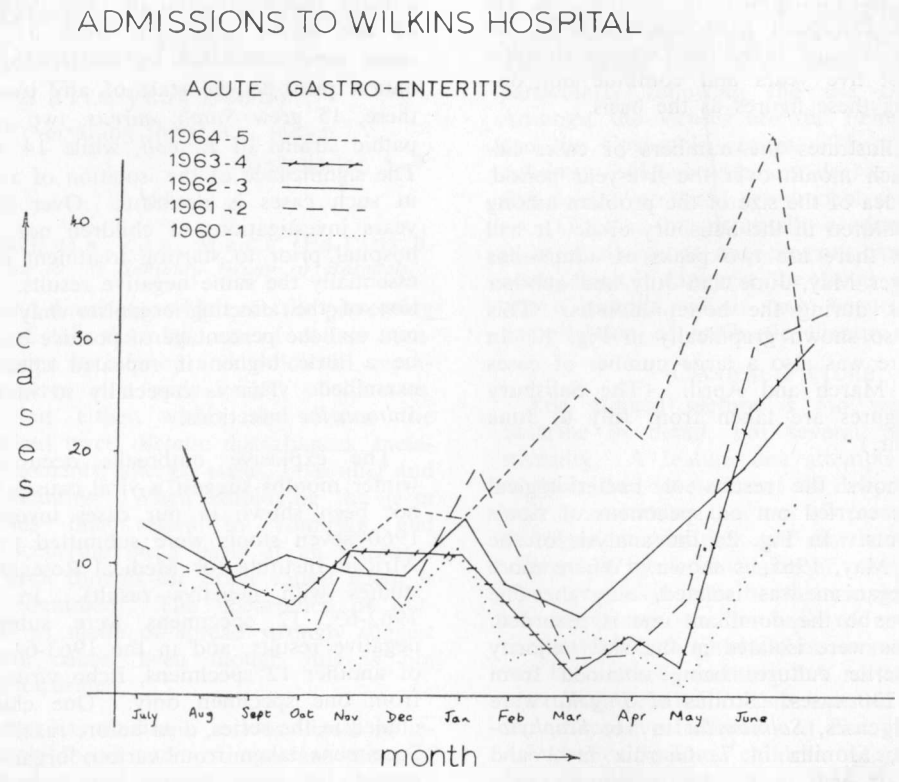

FIG. 1

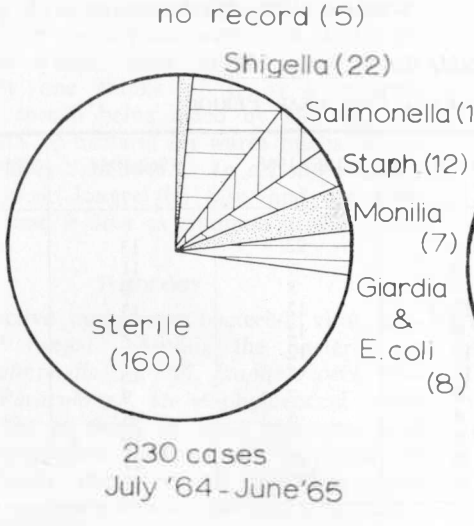

FIG. 2.

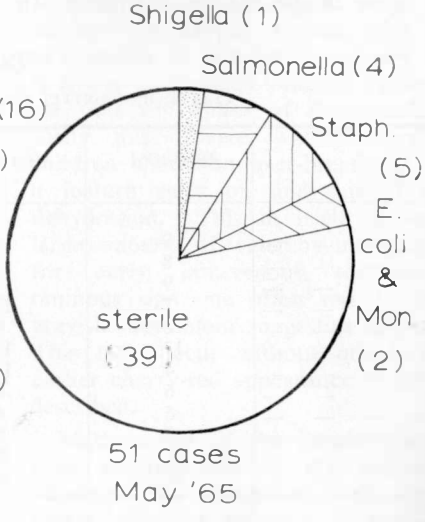

FIG. 2A.

controls (Roux *et al.*, 1963). In a large investigation carried out on diarrhoeal diseases among the indigent rural population of Guatemala, 439 out of 578 specimens of faeces from patients with diarrhoea yielded no bacteria or viruses recognised as pathogenic, yet the disease was always severe and often fatal (Gordon *et al.*, 1964). Commenting on this, an editorial in the *Lancet* (1965) states, "Many new entero viruses have been identified, and on the analogy of the entero bacteria some have been taken to be the cause of diarrhoea. In fact, the evidence that they cause or result from diarrhoea is about equal: that they are there by chance is more probable."

So far our understanding of the etiology of local gastroenteritis, particularly the cases occurring in winter, is very incomplete and it suggests that apart from perhaps improving culture media, non-infective causes have to be investigated.

RESULTS

Over the five-year period under discussion the total number of children admitted to the hospital was 773. No deaths occurred in this series.

Further analysis of the last year under discussion shows that out of the total 230 cases, 83 children required intravenous infusions, and in the peak month of May, 23 did so out of a total of 39. These figures do not take into account the fact that frequently prolonged and repeated infusions were necessary. These figures indicate that the disease as seen in the Wilkins' hospital is a severe one.

Ninety-seven of the 230 cases (1964-65) were pyrexial on admission, i.e., rectal temperature above 100, but this does not necessarily indicate infection, but in part may be due to dehydration.

One hundred and sixteen cases—that is, one-half of the number—were aged less than one year, again showing that gastroenteritis in its severe form is mainly a disease of the very young age group.

TREATMENT

Reflection on the high proportion of sterile stool cultures immediately indicates the difficulty in advising on specific antibiotic treatment. Nevertheless, it is logical to culture specimens, as in some cases appropriate antibiotics can be selected, and if the specimen proves sterile it may avoid the unnecessary administration of a succession of potent antibiotics, each of which carries a certain risk, when the agent first prescribed does not seem to control the disease. The bacillary dysenteries respond well to sulphonamides and tetracyclines. The management of *Salmonella* infections is more difficult, and here the carrier state is another problem. Treatment generally has to be prolonged, but this is very worrying in the case of Chloramphenicol. The local results with Ampicillin have not been as good as one had hoped for and it may be that much higher dosages than we have used until now may bring better results. Though the results of *in vitro* tests for antibiotic sensitivities are notoriously misleading, one does occasionally choose the correct drug as the result of this investigation. The isolation of monilia is useful in the case of continuing diarrhoea where one would use an antifungal agent rather than continue the potentially harmful broad spectrum antibiotics.

Prevention and early treatment of dehydration are the mainstay of treatment, and in the Wilkins' hospital the trend has been to institute intravenous therapy before gross signs of dehydration develop. It is felt that the results have justified this approach. Dehydration can often be anticipated in the cases where severe and prolonged vomiting and diarrhoea are present. The commonest used fluid has been half strength Darrow's solution. Like the Cape Town workers, we have found this a simple and effective solution (Ranier-Pope, 1962). In cases of severe acidosis, M/6 sodium lactate has also been used. To prevent the possibility of tetany developing, calcium gluconate has usually been added to the fluid, 5 cc. 10 cent. solution to 300 cc. of the intravenous fluid.

Initially the children are fed on half strength Darrow's solution and, as their condition improves, skimmed milk is introduced prior to the usual milk feed being given.

Sodium Gardenal by injection is a safe antiemetic. One has seen several severe reactions to the Phenothiazine derivatives used for this purpose, and this seems especially likely to occur in the presence of dehydration.

CAUSES OF CONTINUING DIARRHOEA

Continuing diarrhoea may indicate persistent infection, and this is particularly a possibility in *Salmonella* enteritis. Repeated stool cultures are indicated where symptoms persist to exclude the presence of the original infection or a superadded one, like moniliasis.

Disturbance of the normal intestinal bacterial flora is a possibility, but difficult to prove. The use of *Lactobacillus acidophilus* preparations

which have been rendered antibiotic resistant may correct such states.

It has been known for a long time that after an attack of gastroenteritis some children, when they become intolerant of fat, can be maintained on skimmed milk for a variable period of time.

Others seem not to be able to tolerate cow's milk protein, and several such children have been seen in this series who have done well on substitutes like soya bean preparations or amino acid preparations like Nutramigen. These cases have been interpreted as temporary milk intolerance or allergy triggered off by infection.

Temporary disaccharide intolerance following gastroenteritis has been described (Sunshine and Kretchmer, 1964). In these cases the unhydrolysed sugars are acted on by bacteria resulting in fermentative diarrhoea. The stools contain an excess of lactic acid and the patient fails to show a significant rise in serum glucose after oral challenge with the offending disaccharide. We have not investigated our cases in this way, but one child's persistent diarrhoea ceased after feeding with a mixture of cream, egg, Casilan and water, in this way excluding disaccharides.

Combinations of milk allergy and dissaccharide intolerance are possible. It may be that mucosal oedema resulting from gastrointestinal milk allergy may also affect the enzymes present in intestinal epithelial cells responsible for the hydrolysis of the sugars.

SUMMARY AND CONCLUSIONS

An attempt has been made to illustrate various aspects of gastroenteritis by reference to experience in Salisbury over a five-year period, when 773 cases were treated without loss.

A fairly constant pattern is demonstrable, showing that each year a severe outbreak may be anticipated in the winter months. This finding may be of value in planning hospital staffing and hospital accommodation.

A disappointing finding has been the high failure rate in identifying a causal agent, particularly as at the start of the talk gastroenteritis was defined as a disease with an infective cause. Charles Creighton (1965), the author of *A History of Epidemics in Britain*, was largely discredited by his medical contemporaries because in the *Encyclopaedia Brittannica* he expressed the view that bacteria and viruses were incidental to the phenomena of infectious disease. He was the last of the humoral pathologists. It does seem as if our findings lend strength to the argument of this medical scholar who lived from 1847 to 1927.

Yet the sharp outbreak of the disease in winter is striking and indicates some recurring factor, infective, physical or chemical. One is tempted to turn one's attention to possible common sources of trouble, e.g., food or water supplies. Over the recent years the presence of large quantities of algae in Lake McIlwaine after the rains has become a problem. It has been suggested that toxins are released when algae die (Taylor, 1958). They tend to die in the cold months, and it is tempting to postulate that this may be related to the high incidence of the disease at this time of the year. The disease does not seem to be so prevalent amongst the Salisbury Coloured population, who live mainly in Arcadia suburb and whose water supply is not Lake McIlwaine. Bulawayo (Kibel) has not had this winter experience nor do they have an algal problem. This is an avenue of investigation which, I think, merits attention. Though I do not expect it to prove the final answer, it may be that alteration in vegetation in and around water supplies may be playing some role.

The outstanding fact which emerges from this study is the well-known one that one should not over-emphasise the antibiotic treatment of gastroenteritis, but that dehydration must be forestalled and intensively treated when it occurs.

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