

THE EPIDEMIOLOGY OF GASTRO-ENTERITIS IN INFANCY: PART III

I. W. F. SPENCER, M.B., B.CH., D.P.H., D.T.M. & H., M.R.S.H. AND M. E. E. COSTER, M.B., B.CH., *City Health Department, Johannesburg*

THE INTERRELATIONSHIP OF FIELD, LABORATORY AND OTHER DATA

Apart from ensuring that there would not be repetition of work previously done in this community, no review of the literature was undertaken until after the planning of the project, its conclusion and the analysis of data had been completed. It was felt that opinion would thus be reached independently and not be influenced by the published views of others. This discussion compares findings with those obtained elsewhere.

Occurrence

Jones²¹ states that, in the past, epidemics of gastro-enteritis in the late summer in England took a heavy toll of infant life, but that the physician there is no longer faced with this problem. He quotes the Registrar General's *Statistical Review of England and Wales* which showed that 397 deaths from gastro-enteritis in infants under one year of age occurred in 1963. Parry²⁶ contends that infant gastro-enteritis in terms of these figures is still a common and often fatal disease in England and Wales, especially when related to those families living in the overcrowded slum dwellings of northern industrial towns. Nevertheless, 397 deaths from gastro-enteritis in the whole of England and Wales in 1963 is almost equalled by the 300 deaths from this cause in 1963 which occurred among Bantu in Johannesburg alone. He further draws attention to gastro-enteritis not being a notifiable disease in England and Wales, so that no statistics are available as to its true incidence, which similar problem we described as existing in Johannesburg.

Walker *et al.*,²⁷ in discussing the bearing of African urbanization on infections, state that literate, prosperous

populations experience lower morbidity and mortality rates than primitive or technically retarded populations, which is supported by the mortality rates of gastro-enteritis of Whites and non-Whites shown in Table II. There is considerable evidence²⁸ that gastro-enteritis is increasing in incidence in many parts of the world, especially in newly urbanized areas where infant feeding is following the world trend away from the breast to the bottle. This trend is equally followed by the Bantu of Johannesburg and is worsened by pressure of circumstances on the mother to leave her infant at an early age to the care of others and seek employment to supplement the family income.

We have not obtained mortality rates of 'comparable' communities from various parts of South Africa in attempting to assess the effects of climate and season. Neither have we comparable rates in cold climates approaching the polar latitudes and in tropical areas, for the variables of socio-economic and developmental level and many others intrude. In these extremes of more constant climate a more equal distribution throughout the year would be anticipated. In England²² epidemics occurred in late summer. In a study of diarrhoea in White children in Johannesburg, Koornhof and his co-workers²⁹ showed a higher incidence in early summer which follows a common pattern in temperate zones. Bokkenheuser and Richardson,³⁰ studying salmonellae and shigellae in rural South African Bantu schoolchildren who were apparently healthy, found the highest incidence of salmonellosis in early summer (December), but shigellosis was distributed throughout the year. In our study among the Bantu of Johannesburg gastro-enteritis is a summer epidemic as shown in Fig. 2. It is assumed that this should be antici-

pated as in other primarily intestinal diseases, whether poliomyelitis or typhoid, and yet Bokkenheuser and Richardson²⁰ found this not to be the case in shigellosis.

What are the precise factors which alter in summer to make gastro-enteritis more prevalent? It is assumed that greater heat increases proliferation of organisms in media such as milk or offal, that opportunities of transmission by flies and other means give greater opportunity of contact with higher levels of bacterial and viral challenge. And yet in this study, and in many others, clinical cases often yielded no pathogens at all in their stools.

Gordon and others²¹ investigated epidemiological patterns in Guatemalan villages and found that in these communities the disease predominated in children aged 6 months to 2 years, namely the period of weaning commonly encountered in those parts. They found the disease relatively infrequent during the first 6 months when infants were wholly breast fed. When weaning was completed, usually in the third year, the incidence declined sharply. They found a progressive decrease with age thereafter, which is well recognized in less developed areas, but felt that what is not recognized is the concentration of cases during the period of weaning. This they consider to be largely due to the common statistical practice of considering all cases of the first year as a unit and then grouping together cases of the 2nd-5th years.

Incidence statistics could not be obtained in this study but a suitable breakdown into age-groups was obtained in regard to mortality in the Bantu (Tables III and IV). Gordon *et al.*²¹ found that death rates were highest in the second year of life and that after the third year a sharp drop occurred, while for schoolchildren the rates were a fraction of those for the earlier years of life. The greatest number of deaths in Bantu in Johannesburg occurred in children in the first year of life, being highest in the age-group 1-6 months, while in our study of sample cases the highest incidence was 3-17 months. The earlier occurrence among Bantu in Johannesburg is probably again related to socio-economic circumstances causing mothers to return to work soon after birth of the baby and initiation of partial or total artificial feeding. This may occur in communities of high economic circumstances, for different reasons, but in these communities absence of malnutrition and a better environment render weaning a relatively non-hazardous procedure.

In Guatemala, diarrhoeal disorders are the first cause of death and their importance is appreciated by comparing figures of death from that cause with those prevailing in societies of better socio-economic status. For infants under one year in Guatemala the death rate was 25 times that for infants in the USA, which tallies exactly with the figure previously quoted from Hansen,² that the death rate for non-Whites was 25 times as high as that for Whites at the Red Cross Hospital in Cape Town in 1966, while the figures of the present study showed a death rate 32.2 times as high for Bantu as for Whites. In the present investigation a sharp decline was also found after 3 years of age.

It would be reasonable to assume that the intestinal bacterial and viral populations in a community, such as in

the Eantu areas of Johannesburg, remain relatively constant, except in case of any epidemic outbreak, and presumably exposure thereto. A decline of occurrence with increasing age would suggest that symptomatic or asymptomatic infection produces immunity to this population of infective agents. The first exposure may be either entry by birth into the area or as an immigrant from some other area. It is likely that the seriousness of the first attack depends on the challenge of organisms and the resistance of the individual—which would be low in a malnourished infant—and the standard of medical care available. Presumably the neonate either succumbs after exposure or survives with reasonable immunity to the organism or group of organisms involved. Subsequent exposure to the remaining intestinal organisms of the area may permit the infant to take his place as an intermittent asymptomatic carrier with satisfactory immunity to the average local hazards of intestinal infection. An investigation of asymptomatic excretion rates among communities of generally higher economic status such as the Whites in Johannesburg would be interesting and might reveal that even they recurrently excrete these organisms without illness. These factors would similarly apply to older children and adults who immigrate into the area. Presumably such persons bring with them immunity already developed to a large segment of the local population of intestinal organisms due to exposure to many of these agents elsewhere. They would become infected, with or without symptoms, depending on factors such as waning immunity, infection with agents to which they had not been previously exposed, with no resultant immunity, and nutrition. Similar circumstances probably obtain when the holiday-maker goes to another area for his vacation or the world traveller reaches another city, when the occasion is frequently marred by a bout of diarrhoea.

Walker *et al.*¹⁷ studied the presence of shigellae and salmonellae in Bantu children in rural, peri-urban and urban schools. Health services for the peri-urban and rural schoolchildren were not of the high standard of the urban areas, and yet serial studies in all 3 groups of children showed that nearly every child had one or more isolations of these organisms during a period of a year. In terms of previous reasoning, if there is such a general prevalence of these infections without illness, are isolations necessarily directly related to morbidity and mortality? Prevalence of infective agents may make a very significant contribution to deaths from kwashiorkor.

Gordon *et al.*²¹ considered a case a new or primary case when the interval between cases was longer than the incubation period which they accepted as 1-7 days. They calculated the over-all secondary attack rate in their Guatemalan study for 390 family outbreaks as only 1.4%. In another study on 450 cases in families they found 86.7 new primary cases and only 7.5% were secondary infections. The low secondary attack rate was apparent in our investigation.

This consideration of occurrence is confined to the situation encountered in the domiciliary areas investigated, and is not concerned with epidemic outbreaks of gastro-enteritis in hospitals or other institutions where well-defined single aetiological agents such as entero-

pathogenic *Escherichia coli* are incriminated, and control is achieved by treatment and interruption of a far less complicated environmental anus-to-mouth transmission than occurs in a population group such as the Bantu of Johannesburg.

Presentation and Symptomatology

Clinical features of gastro-enteritis, which are not a primary concern of this study, have been fully reviewed by authors here and elsewhere.^{2,12,22}

The range of presentation and symptomatology of gastro-enteritis was defined in the criteria of what constituted the condition under investigation, namely primary, significant diarrhoea during the first 3 years of life. The clinical examination of 380 sample cases with primary significant diarrhoea, which at present causes the death of over 400 Bantu infants in the first year of life in Johannesburg, revealed that 290 (76%) were not seriously ill, and with the available medical care would not normally have undergone clinical deterioration. In the absence of figures of incidence, these figures, coupled with the large number of 404 Bantu deaths in the first year of life in 1966, suggest that there must be a high incidence to give rise to so many deaths, and that there is a large background of less serious manifestation. It is emphasized again that case mortality should not be assessed on the seriously ill patients admitted to hospitals and their drip units. The fact that so many do become critically ill is an indication of individually poor resistance to a widespread infection which the normal infant should meet with near equanimity. That many do not is an indictment of poor resistance occasioned by socio-economic and environmental inadequacy.

Aetiology

The hope that this study might reveal some causative agent other than those classically implicated and not hitherto isolated was naïve. So was the view that transmission was simply ano-oral spread of this agent largely facilitated by misused unhygienic feeding bottles. The epidemiological pattern is complex and a challenge.

Causative agent. The extensive literature on numerous organisms, varying from salmonellae, shigellae, enteropathogenic *Escherichia coli* and others to listeria, and a wide range of viruses isolated from stools and incriminated in the causation of gastro-enteritis in South Africa^{19,23-30} and in other countries^{1,12,22,34-55} bears testimony to an unsolved dilemma. At this stage no single constant causative agent has been isolated, nor was this achieved in the present investigation. A unitarian concept of this kind is unlikely to materialize even though it might be reasoned that such a well-defined clinical syndrome would likely be caused by a specific organism or group of organisms. The finding in the present investigation of a relatively large number of infants with gastro-enteritis from whose stools no bacteria or viruses could be isolated is supported by similar experience here and elsewhere. So is the fact that organisms isolated from the stools of children with gastro-enteritis could be recovered from the stools of large numbers of infants who were completely well. Similarly, our experience that a variety of bacterial and viral agents were isolated from sample and control

cases is common experience. Clearly, there must be more epidemiological factors than an infective agent and its transmission from one susceptible infant to another. There were sample group infants with gastro-enteritis where infection seemed to play no part, and such diarrhoea may have been initiated by nutritional or dietary causes, or by the abuse of medications and enemata by Bantu. Concurrently existing changes in the intestinal mucosa may have exacerbated the situation. The role of bacterial overgrowth and secondary invaders was hard to assess.

Nutrition. The word 'malnutrition' is in unsatisfactory usage and is consistently related to undernourishment, whereas the grossly obese are equally as malnourished as the starving. The importance of undernutrition in relation to gastro-enteritis is well established^{2-4,21,26,30,56} and in this investigation it was 5 times more frequent among infants with gastro-enteritis than in the control group of well babies. Malnutrition, in the meaning of inadequate nutrition, may itself give rise to symptomatic diarrhoea without a primary infective cause. Diarrhoea may be a symptom associated with kwashiorkor. Malnutrition may initiate gastro-enteritis, and gastro-enteritis of infective origin may precipitate kwashiorkor. In this complex inter-relationship it is not surprising that there is often confusion in public reporting in understanding that not all infantile diarrhoea in less advanced communities is due to kwashiorkor, and that gastro-enteritis and kwashiorkor are not synonymous. Certainly, it is the child with malnutrition in whom gastro-enteritis becomes a serious and often fatal disease. A clinical orientation to malnutrition is fundamental to rectify deficiency when it is encountered.

There is further satisfaction when to this is added a preventive approach to combat nutritional, in this case particularly protein, deficiency by the mass distribution of powdered milk to infants of communities in need. Before the inception of the highly laudable subsidized milk scheme introduced in this country by the South African Government, mass free issues of powdered milk had been given by the clinic services of the Johannesburg City Council to Bantu infants since 1947. As an example of the extent of this issue, 2,600,000 lb. of powdered milk was distributed during the period 1947-1963. This undoubtedly contributed largely to the progressive lowering of mortality rates from gastro-enteritis in Bantu in Johannesburg, but, as reflected in present mortality rates, this preventive approach is not enough. Modern epidemiology demands that public health services should earn the designation of a preventive and a promotive service. They should promote not only by adjuncts such as health education, but go even further towards establishing a climate in which preventive measures can be effective, such as urging raising of wages in lower socio-economic groups to a minimal level less than which no worker may receive. Apart from purely medical aspects, hospitalization and other medical service costs for diseases like gastro-enteritis and tuberculosis would be tremendously lowered. Though most important, better treatment and environmental control will have relatively insignificant benefit unless socio-economic and nutritional status is elevated.

Transmission. The method of spread of gastro-enteritis

in instances where it is an infective condition must basically be that of typhoid, dysentery or parallel intestinal infection but rendered more complex by various epidemiological factors. Richardson *et al.*⁵⁷ showed that improvement of the water supply had no effect on the prevalence of salmonellae, shigellae and helminths among rural Bantu children. They subsequently showed that good quality potable water supplied to the Bantu areas of Johannesburg was not enough to lower the incidence of intestinal salmonella and shigella infections.

Richardson *et al.*⁵⁷ recently completed a meticulous bacteriological assessment of abattoir meat, meat and milk, and fowl and dog faeces in the Bantu residential areas of Johannesburg as a possible source of human infections. Their results showed that reasonable care was taken to ensure that slaughtered animals were processed in a satisfactory manner, but the reverse held in respect of offal. Tripe and intestines were distributed by an offal pool and it was clear that they were not adequately treated after leaving the abattoir to render them comparatively free from faecal contamination. These authors conceded that this commodity forms an important source of protein in the staple diet of the community, as was shown in our study, but that with lack of knowledge of modern hygienic practices the risk of infection, and particularly salmonellosis, from this source was very real. They isolated more *Salmonella typhimurium* from samples tested than other strains, and it is well known that this salmonella commonly causes diarrhoea. They further collected specimens of sour milk at random from street pedlars. No salmonellae were isolated from these samples, but 24 out of 28 (85.6%) yielded faecal *E. coli*. Illegal meat, offal and milk supplies emerged as points of comment in the analysis of field findings in our study. Richardson and his co-workers⁵⁷ further isolated salmonellae from 21.4% of specimens of fresh canine faeces collected from pavements near offal stalls, and from 14.3% of specimens of fowl faeces. They considered that transient human carriers among Bantu in Johannesburg townships may contribute an important reservoir of salmonellae responsible for diarrhoea; that infected offal may well be directly responsible for this high incidence; that improvement in handling, distributing and selling of offal is an obvious necessity; and that a concentrated health education drive is indicated. While agreeing that these things should be done, it might well be that in such a vast sea of salmonellae in humans, dogs and fowls, with little discomfort to the host in many instances, cleaner offal at a consequently raised price, which the community will likely in large part not be able to afford, may have no more effect than the previous experience of improving water supplies in regard to salmonellosis, but may have considerable effect in the loss of a cheap source of protein.

Salmonellae have not been shown in our study or elsewhere to be the primary, the only or even a necessary infective agent in gastro-enteritis. In the present peculiar circumstances of this area nutrition may be more important than lessened hazards of infection. Many Bantu have a traditional liking for offal and in some groups it becomes more delectable the lower the gut level. Considerable educative effort will be necessary. Adults and not infants susceptible to gastro-enteritis are the greatest con-

sumers of offal, and no outbreak of diarrhoea among adults has been traced to this source. It would seem reasonable, in view of the needs of these communities and the circumstances apparent, that the offal problem should be left broadly as it is, with improvement in handling by abattoirs and in distribution, but with maintenance of a price which can be afforded by the Bantu. It is unlikely that all human food was intended to verge on sterility. There is an acid barrier in the stomach. Other alternatives would be to fully cleanse and treat intestines, which could be uneconomical, or to discontinue the supply of offal from abattoirs and use intestines for sausage skins and processing to fertilizer. Many workers have shown the hazard of abattoir processing of meats as a cause of human infection,^{58,59} and these decisions regarding the disposal of offal should be taken for the whole country at Government level.

A somewhat similar problem was met by Mackenzie and Livingstone⁶⁰ in Durban, who described the successful use of tilapia species (*kurper*) in the biological control of mosquitoes in natural and man-made ponds which was followed by the weekly netting of several tons of these very edible fish from effluent ponds and their distribution to the poor and needy. These ponds yielded 2-3 tons of fish per acre per year. However, their studies showed that the flesh of these tilapia nurtured in sewage-effluent ponds included numbers and varieties of salmonellae, and they concluded that this cheap source of protein to the underprivileged was now inaccessible. Here, too, the question arises whether the potential threat of salmonellosis or the deprivation of a cheap protein source to undernourished communities is the most important. Mackenzie and Livingstone describe no outbreak of diarrhoeal disease among the recipients of the fish, who presumably cooked them before ingestion. In the floating populations of the 'boat cities' of the East, such as Hong Kong, where human excrement freely pollutes the water, it is believed that kwashiorkor and diarrhoea are minimal because of high protein diets derived from the fish recovered by the boat dwellers from these waters.

While not advocating the consumption of salmonella-contaminated food, an attempt is made to weigh the critical need of cheap protein in combating the malignant effects of malnutrition in regard to kwashiorkor, gastro-enteritis, tuberculosis and other conditions. Numerous epidemiological facts point to transmission from case or carrier to susceptible infant in gastro-enteritis; faeces to mouth via devious channels of the environment, meat, milk, unhygienic utensils and others, with the role of an infective agent variable, sometimes specific, in others obscure where pathogens become virtual commensals and non-pathogens become virulent, and in others playing no role at all. There is no indication of a common source origin or uncomplicated spread, except in institutional outbreaks of infantile gastro-enteritis. In the general community transmission is grossly complicated by the intrusion of socio-economic factors with malnutrition initiating or exacerbating clinical disease after infection.

An epidemiological hypothesis of infection. It may well be that in various human communities there are populations of intestinal organisms broadly peculiar to each, though with several types of organisms common to many.

The extent and virulence achieved by the bacterial and viral population would likely be dependent upon standards of environmental hygiene and of resistance to infection. New entrants, either by birth or immigration, as suggested, are exposed to these bacterial and viral populations and contract infections by those organisms to which they have no immunity from previous exposure, and may or may not have associated diarrhoea or constitutional disturbance, the latter being largely dependent on the state of nutrition. The process of exposure to infection by organisms to which no immunity has been developed may be repeated, if initial attacks are survived, until the new entrant attains a stage of virtual symbiotic host-organism equilibrium with the environment. In these circumstances many individuals will be recurrent asymptomatic excretors of various organisms. Apart from secondary diarrhoeas, infective gastro-enteritis in well-nourished and properly cared for infants should be a relatively minor episode.

In the Bantu residential areas of Johannesburg it is unlikely that marked change will occur in the population of existent bacteria and viruses, even though environmental and other control measures be intensified. The endeavour should mainly be to assist infants to pass rapidly and safely through the infector pool of organisms to attain the requisite state of host equilibrium to the endemic intestinal organisms of the area. Unwise antibiotic assault on the endemic bacterial populations as a vaguely applied control measure could have serious untoward effect in the development of resistant strains and, in addition, serious parenteral complications in adults with salmonella, *E. coli* or other intestinal invasion.

Finally, in the interrelationship of data, Gordon,^{21,22,34,50,56} who with his various co-workers has completed major and exemplary work on diarrhoeal disease, has said that investigations have usually been in clinic and laboratory, but field epidemiological research should be combined with these. This was the attempt of the present study.

TREATMENT

This investigation was not majorly involved with treatment, but therapy was assessed to determine its position as one of the multiple facets of epidemiological control and thus to permit a basis of sound health educative persuasion to mothers.

Treatment of acute gastro-enteritis aims at recovery and at reduction of the number of deaths, most of which are due to dehydration contributed to by unnecessarily prolonged dietary restriction during therapy. Rehydration and restoration of electrolyte balance is critically important, and effective, but less effective in the presence of malnutrition. Hansen² advises that steps be taken to ensure that mothers understand the dangers arising from dehydration and know how to recognize it, how to prevent it and how to control it in the early stages. Feeding should not be stopped for more than 12 hours. In hospitals rehydration and replacement of salt lost is usually with half-strength normal saline. Health authorities and mothers, he recommends, should know that any infant with diarrhoea for even a day should forthwith go onto a mixture of one pint of boiled water and half a teaspoon

of salt with 2 tablespoons of sugar. Milk and food should be stopped for 12 hours but the child should be given as much of the saline mixture as it will take. The infant must not be starved for more than 12 hours and should then go back onto undiluted milk, according to Hansen.² He considers that if the milk is diluted, the infant will be deprived of valuable protein. If these things be done by a mother in the early stages of her child's illness, then the urgent hazards of dehydration, electrolyte imbalance and starvation will be avoided.

A consensus of opinion^{2,18,50,61-68} is that antibiotics are of questionable value in the treatment of gastro-enteritis and should not be used in uncomplicated cases. Some² rely on sulphadiazine if a drug is necessary. The control of infection does not mean that diarrhoea is necessarily controlled. Administration of antibiotics to cases of salmonella gastro-enteritis and carriers prolongs the excretion of these organisms by inducing changes in the intestinal flora with increased susceptibility to bacterial invasion.

Rosenstein⁶¹ expresses the interesting view, in support of hypotheses in this paper, that symptomless salmonella excretors are not particularly dangerous to their contacts. Antibiotics are not harmless and should only be used under special circumstances of systemic complications such as abscess, meningitis, osteomyelitis or endocarditis. The potential dangers of transferable drug resistance are receiving increasing attention,^{62,64} and organisms such as *Salmonella johannesburg*, commonly associated with recent self-limiting occurrence in the Bantu of Johannesburg, are exhibiting resistance to antibiotics.

CONTROL

Basic epidemiological procedures, whether applied to yellow-fever, malaria or bilharzia, or modern epidemics such as road accidents, coronary thrombosis or smoking and lung cancer, require that the cycle of causation of any condition to be brought towards better or ultimate control should first be established, and that methods to interrupt the causative cycle at as many points as possible should then be devised. These methods must be applied concurrently and their priority in regard to extent of application modified in terms of the circumstance of the community to which they are applied. These principles and their applications have been described in previous communications in respect of tuberculosis⁷ and modern stress phenomena.⁶⁵

In this study an epidemiological cycle of interrelated causative factors in regard to primary gastro-enteritis of infancy in the Bantu of Johannesburg has emerged. Again, in terms of fundamental epidemiological principle, it is based on interaction between host and causative agent, with the environment contributing many factors, and further complicated in that one of the factors associated with the environment and host reaction, namely malnutrition, may itself give rise to gastro-enteritis without the apparent intrusion of a primary infective agent, and that, where infective agents are involved, no single specific organism but many bacteria and viruses are implicated.

The application of methods of concurrent multifactorial interruption of this complex cycle is represented in Fig. 3.

Gastro-enteritis is a relatively minor problem in communities of advanced socio-economic status. In regard to the Bantu of Johannesburg the primary endeavour therefore is to advance existing socio-economic status, which, though considerable strides have been made, is a long-term procedure. Pending this achievement, endeavour should be directed to assisting infants of this community towards rapid and safe passage through their inevitable exposures to intestinal infective agents, as occurs in infants of communities of higher socio-economic level, and thus towards acquisition of resistance to the endemic intestinal organisms of the area.

Measures directed towards this objective may be divided into those which prepare the infant for safe and minor episodes on exposure to infection (I in Fig. 3), and those which ensure rapid and safe recovery from gastro-enteritis (II in Fig. 3).

Measures Directed Towards the Infant

1. Education of the mother and others involved in care of the infant should cover the broad causation of gastro-enteritis, importance of breast feeding, hazards of artificial feeding, principles of cheap but adequate nutrition, dangers of the weaning period, the abuse of medications and enemata, preparation and storage of infant food, and methods of cleansing utensils and feeding receptacles. The use of hypochlorite solutions for this purpose has been advocated^{60,67} and was found satisfactory for sterilization of feeding bottles when submitted to laboratory testing⁶⁸ in our services.

2. Nutritional assistance can be given by issues of supplementary powdered milk or other commodities.

3. Intensified home visiting by health visitors when infants are 3-17 months of age or during any subsequent period of weaning should be instituted.

4. Greater inducement to attend well-baby child health clinics is important. Contact tends to be lost on completion of routine free immunization procedures against diphtheria, whooping cough, tetanus, smallpox, poliomyelitis and tuberculosis.

5. Though many preschool institutions exist in the Bantu areas, additional provision should be made for places of day care for young babies of working mothers.

Measures aimed at the socio-economic and sanitary environment should receive increased attention in regard to the following:

- (a) The urging of legislation to set minimal wage levels less than which no employer may pay a full-time employee.

- (b) Health education on family planning, budgeting and the proper direction of income to priorities, personal and general hygiene, health practices, and fly control.

- (c) Environmental hygiene and general sanitation should receive special orientation towards control of over-

Hospital Services.

Clinic and Domiciliary Services.

Information to Medical Officers regarding use of Antibiotics.
Issue of Tablets to all Mothers for constituting Saline Mixture should their Infants develop Diarrhoea.
Health Education of Mothers regarding early Diagnosis and preliminary treatment.

Nutritional Assistance.

Intensified Home Visiting by Health Visitors at 3-17 months of age of Infants. Health Education of Mothers. Greater Inducement to attend Child Health Clinics.

Places of Day Care for Infants of Working Mothers.

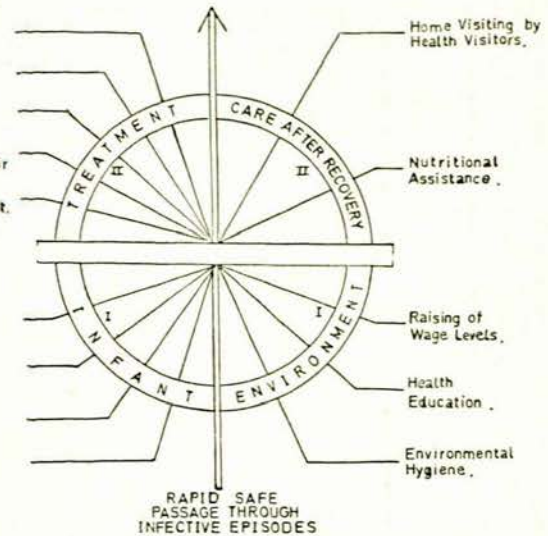


Fig. 3. Epidemiological control of gastro-enteritis in a Bantu community.

crowding, fly breeding, illegal hawking of illicit meat, offal and milk supplies, and improved processing and distribution of offal.

Prompt Treatment of Infants

Those infants who contract clinical illness on exposure to infective agents (or develop nutritional diarrhoea) require prompt and effective treatment.

Health education services must inform mothers and families of the dangers of dehydration, salt loss and unnecessarily prolonged starvation periods in infants with diarrhoea, even if of only a day's duration, of the simple methods of withholding food for 12 hours and the administration of a saline mixture to correct these conditions and initiate early life-saving treatment before these cases finally come to the attention of the medical services.

Suitable tablets⁶⁴ should be issued to mothers to facilitate the preparation of the necessary saline mixture by dissolving the tablet in boiled water for use in the event of her baby contracting diarrhoea.

Attention of medical personnel should be drawn to the inadvisability of prescribing antibiotics in uncomplicated cases and for carriers of infective agents associated with gastro-enteritis.

Existing clinic, domiciliary and hospital services will continue to provide medical cover.

Care after recovery would require intensified health visiting to such cases for advisory and educative support to mothers and families, to make provision for supplementary food to the infant when considered necessary, and to ensure that the infant and not other members of the family partakes of this sustenance.

CONCLUSIONS

Gastro-enteritis in Bantu infants in Johannesburg, though associated with infective processes, is largely a socio-economic disease.

Endeavour should be directed to assisting infants to pass rapidly and safely through inevitable exposures to

infective agents, thus acquiring a status of host equilibrium and resistance to the endemic intestinal organisms of the area. This should be achieved by application of the methods described in relation to the epidemiological instrument devised for this purpose.*

*A parallel longitudinal cohort study of newborn infants in regard to viral isolations and antibody levels was carried out by Prof. J. H. S. Gear and his co-workers, and will be submitted for independent publication.⁶⁹

SUMMARY

An investigation is described into the epidemiology of gastro-enteritis in infancy in Johannesburg. The project involved 770 infants under 3 years of age, of whom 380 had primary gastro-enteritis and 390 were well babies who constituted a control group.

Clinical, laboratory and epidemiological field procedures were undertaken. An epidemiological cycle of interrelated causative factors is advanced and methods of interruption of this cycle, with the purpose of decreasing incidence and mortality, are described.

We wish to thank the Johannesburg City Council who gave authority for this undertaking and bore the cost of the bacterial investigations; the South African Institute for Medical Research which carried out the bacteriological investigations; the Poliomyelitis Research Foundation which conducted viral studies; the staff of the Johannesburg City Health Department who unstintingly gave of their time and effort; and Prof. A. H. Smith, Medical Officer of Health, Johannesburg, for permission to publish.

1. Editorial (1968): *S. Afr. Med. J.*, **42**, 825.
2. Hansen, J. D. L. (1967): *Publ. Hlth (Johannesburg)*, **67**, 285.
3. Jelliffe, D. B. (1966): *Assessment of the Nutritional Status of the Community*. Geneva: WHO.
4. Wills, V. G. and Waterlow, J. C. (1958): *J. Trop. Pediat.*, **3**, 167.
5. Spencer, I. W. F. and Coster, M. E. E. (1962): *S. Afr. Med. J.*, **36**, 881.
6. Spencer, I. W. F., Coster, M. E. E., Richter, M. and Maher, M. (1964): *Ibid.*, **38**, 103.
7. Spencer, I. W. F., Coster, M. E. E., McPhail, A. V. and Richter, M. (1964): *Ibid.*, **38**, 223.
8. *Idem* (1965): *Ibid.*, **39**, 319.
9. Medical Superintendent, Baragwanath Hospital (1968): Personal communication.
10. *Idem* (1967): Personal communication.
11. Hammond, E. C., Irwin, J. and Garfinkel, L. (1967): *Amer. J. Publ. Hlth*, **57**, 1979.
12. Jones, H. E. (1966): *Practitioner*, **197**, 321.
13. Parks, W. P., Melnick, J. L., Queiroga, L. T. and Ali Khan, H. (1966): *Amer. J. Epidemiol.*, **84**, 382.
14. Scudder, H. I. (1947): *Publ. Hlth Rep. (Wash.)*, **62**, 681.
15. Kahn, E. and Freedman, M. L. (1959): *S. Afr. Med. J.*, **33**, 934.
16. Parry, W. H. (1966): *Nursing Times*, **62**, 1062.
17. Walker, A. R. P., Koornhof, H. J., Richardson, N. J. and Hayden-Smith, S. (1965): *Trans. Roy. Soc. Trop. Med. Hyg.*, **59**, 483.
18. Editorial comment (1966): *J. Pediat.*, **68**, 792.
19. Koornhof, H. J., Richardson, N. J., Politzer, W. M., Utian, H. L. and Malherbe, H. (1964): *S. Afr. Med. J.*, **38**, 821.
20. Bokkenheuser, V. and Richardson, N. J. (1960): *J. Hyg. (Lond.)*, **58**, 109.
21. Gordon, J. E., Guzman, M. A., Ascoli, W. and Scrimshaw, N. S. (1964): *Bull. Wld Hlth Org.*, **31**, 9.
22. Gordon, J. E., Behar, M. and Scrimshaw, N. S. (1964): *Ibid.*, **31**, 1.
23. Mitchell, E. E., Van den Ende, M., Grant, I., Rabkin, J. W., Selzer, G. and Parker, R. G. F. (1948): *Clin. Proc.*, **7**, 251.
24. Coetzee, J. N. and Pretorius, H. P. J. (1955): *S. Afr. J. Lab. Clin. Med.*, **1**, 188.
25. Kahn, E. and Robertson, G. H. (1952): *S. Afr. Med. J.*, **26**, 671.
26. Kahn, E. (1957): *Ibid.*, **31**, 47.
27. Roux, P., Kahn, E., Malherbe, H. and Cassel, R. (1963): *Ibid.*, **37**, 256.
28. Malherbe, H. and Roux, P. (1963): *Ibid.*, **37**, 259.
29. Richardson, N. J. and Bokkenheuser, V. (1963): *J. Hyg. (Lond.)*, **61**, 257.
30. Bokkenheuser, V. and Richardson, N. J. (1960): *Ibid.*, **58**, 109.
31. *Idem* (1959): *S. Afr. Med. J.*, **33**, 784.
32. Becker, N. (1968): *Ibid.*, **42**, 905.
33. Richardson, N. J., Hayden-Smith, S., Bokkenheuser, V. and Koornhof, H. J. (1968): *Ibid.*, **42**, 46.
34. Gordon, J. E., Chitkara, I. D. and Wyon, J. B. (1963): *Amer. J. Med. Sci.*, **245**, 345.
35. Ramas-Alvarez, M. and Sabin, A. B. (1958): *J. Amer. Med. Assoc.*, **167**, 147.
36. D'Alessandro, G. and Dardanoni, L. (1961): *Riv. Ist. sieroter. ital.*, **36**, 129.
37. Gardner, P. S., McGregor, C. B. and Dick, K. (1960): *Brit. Med. J.*, **1**, 91.
38. Barrie, D. (1966): *Ibid.*, **1**, 1574.
39. Parks, W. P., Melnick, J. L., Queiroga, L. T. and Ali Khan, H. (1966): *Amer. J. Epidemiol.*, **84**, 382.
40. Mohieldin, M. S., Gabr, M., El-Hefney, A., Mahmoud, S. S. and Abdallah, A. (1966): *J. Trop. Pediat.*, **11**, 88.
41. Olarte, J., Galindo, E., Filloy, L. and Joachin, A. (1965): *Bull. Hyg. (Lond.)*, **40**, 1278.
42. Ingram, V. G., Rights, F. L., Ali Khan, H., Hashimi, K. and Ansari, K. (1966): *Amer. J. Trop. Med. Hyg.*, **15**, 743.
43. Dammin, G. J. (1964): *Bull. Wld Hlth Org.*, **31**, 29.
44. White, W. D. (1967): *Brit. Med. J.*, **2**, 283.
45. Behbehani, A. M. and Wenner, H. A. (1966): *Amer. J. Dis. Child.*, **111**, 623.
46. Lie Kian, J., Rukmono, B., Sri Oemijati, Sahib, K., Newell, K. W., Sie Ting Hway and Widodo Talogo, R. (1966): *Bull. Wld Hlth Org.*, **2**, 197.
47. Rosner, R. (1966): *Amer. J. Clin. Path.*, **45**, 732.
48. Costin, I. D. (1966): *Path. et Microbiol. (Basel)*, **29**, 214.
49. Severs, F. P., Acres, S. and Davies, J. W. (1966): *Canad. Med. Assoc. J.*, **94**, 373.
50. Gordon, J. E., Ascoli, W., Pierce, V., Guzman, M. A. and Mata, L. J. (1965): *Amer. J. Trop. Med. Hyg.*, **14**, 404.
51. Cramblett, H. G. and Siewers, C. M. F. (1965): *Pediatrics*, **35**, 885.
52. Reber, H. (1964): *Practical Epidemiology of Microbial Infections*, p. 61. Basle: F. Hoffman-La Roche.
53. Anderson, E. S., Galbraith, N. S. and Taylor, C. E. D. (1961): *Lancet*, **1**, 854.
54. Leading Article (1968): *Brit. Med. J.*, **1**, 70.
55. Galbraith, N. S., Archer, J. F. and Tee, G. H. (1961): *J. Hyg. (Lond.)*, **59**, 133.
56. Gordon, J. E., Behar, M. and Scrimshaw, N. S. (1964): *Bull. Wld Hlth Org.*, **31**, 21.
57. Richardson, N. J., Burnett, G. M. and Koornhof, H. J. (1968): *J. Hyg. (Lond.)*, **66**, 365.
58. Working Party of the Public Health Laboratory Service (1964): *Ibid.*, **62**, 283.
59. Beasley, J., Hopkins, G. B., McNab, D. J. N., Rickards, A. G. and King, G. J. G. (1967): *Lancet*, **1**, 560.
60. Mackenzie, C. R. and Livingstone, D. J. (1968): *S. Afr. Med. J.*, **42**, 999.
61. Rosenstein, B. J. (1967): *J. Pediat.*, **70**, 1.
62. Schroeder, S. A., Terry, P. M. and Bennet, J. V. (1968): *J. Amer. Med. Assoc.*, **205**, 903.
63. Bowmer, E. J. (1964): *Amer. J. Med. Sci.*, **247**, 467.
64. Anderson, E. S. (1968): *Brit. Med. J.*, **1**, 293.
65. Spencer, I. W. F. (1968): *S. Afr. Med. J.*, **42**, 161.
66. Gatherer, A. and Wood, N. (1966): *Mth. Bull. Minist. Hlth Lab. Serv.*, **25**, 126.
67. Gatherer, A. D. (1966): *Nursing Times*, **62**, 665.
68. Osborn, D. W., Laboratory and Technical Service Branch, Johannesburg City Health Department (1966): Personal communication.
69. Gear, J. H. S., Director, SAIMR (1969): Personal communication.