

Diarrhoea Dialogue



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CAUSE — AND EFFECT

Acute diarrhoea can cause life-threatening dehydration very quickly. Rehydration must start at once and primary health care workers need to make decisions about treatment based on signs, symptoms and their own experience. Long before the actual cause of the diarrhoea can be identified, the patient may either be getting better — or be dead.



Making the best guess possible on the basis of signs, symptoms and past experience. A voluntary health auxiliary brings primary health care to a family near Lake Titicaca in Peru. *WHO photograph by D. Henrioud*

Many health workers do not have access to laboratory facilities to discover which infectious organisms in the gut are causing diarrhoea. They can, nevertheless, still save lives because rehydration therapy — particularly oral rehydration therapy — is the key factor in the management of acute diarrhoea, whatever the cause.

However, identifying the cause is important when decisions need to be made about treating people with drugs — or about control measures in the family or community, such as improved food handling, encouragement of breast-

feeding, cleaner water supplies and better sanitation.

This issue of *Diarrhoea Dialogue* looks at the various causes of diarrhoeal disease because, eventually, improved knowledge of this area will bring more effective cure and more appropriate prevention. The information in this issue should also assist primary health care workers in remote areas to make the best guess possible about causes so that standard treatments can be modified to give quicker results.

K.M.E. and W.A.M.C.

In this issue ...

- David Candy looks at the causes of childhood diarrhoea
- Cholera up-date
- Clinician's guide to aetiology

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Publications

Paediatricians in developing countries often have limited access to libraries and have difficulty in keeping up-to-date with current developments in their field. Two recent publications should help to fill this gap. Both are expensive but, if affordable, merit their cost as they provide excellent 'state-of-the-art' reviews and reference sources.

● *Secretary Diarrhoea* (Edited by Michael Field) 1980 237pp. Price \$30.00. Available from: Williams & Wilkins Company, 68 Upper Richmond Road, Putney, London SW15 2SF, UK.

● *Paediatrics in the Tropics* (Edited by R. G. Hendrickse) 1981 392pp. Price £20.00. Available from: Oxford University Press, Oxford OX2 6DP, UK.

Also available, free of charge, is a report recently published by the Committee on International Nutrition Programs — *Management of the Diarrhoeal Diseases at Community Level* (Edited by Richard Cash). The report focuses on oral rehydration and continued feeding in the management of acute diarrhoeal diseases. To obtain a copy, write to: National Academy Press, 2101 Constitution Avenue, Washington DC 20418, USA.

Diarrhoea agents in the environment

All the diarrhoeas discussed in this issue that are caused by intestinal pathogens are transmitted from the anus of the infected person to the mouth of a susceptible person. This faecal-oral transmission can take place directly (on hands for instance) or via contaminated food or water. To control diarrhoea transmission it is necessary to have information on the occurrence and survival of the various pathogens in the environment and to understand the effects of water and sewage treatment processes upon them.

We know a great deal about the environmental properties of the "classical" diarrhoea agents. This is especially true for Salmonellae and, to a lesser extent, for Shigellae, vibrios and *Entamoeba histolytica*. We also know much about the behaviour of commensal *E. coli* in the environment, although we do not yet know whether



Breastfeeding in Papua New Guinea.
Photograph by Dr Michael Gracey

Bottles on prescription

In *Diarrhoea Dialogue 3*, we mentioned that since 1977, feeding bottles have only been available on prescription in Papua New Guinea. Recent information from Port Moresby General Hospital indicates that this policy is having a significant impact. The number of shops selling feeding bottles has greatly reduced; breast-

feeding has increased; and general awareness of the dangers of bottle-feeding without adequate safeguards has grown. All these factors have played an important role in reducing the number of gastroenteritis admissions to the hospital and associated deaths in infants less than six months of age.⁽¹⁾

⁽¹⁾*Impact of Sales Control of Feeding Bottles 1981 Weekly Epidemiological Record No. 16 April 24: 126.*

the toxigenic and invasive strains of *E. coli* are similar to the non-pathogenic strains in their environmental properties.



Rotavirus in a faecal sample from a child with diarrhoea.

Electronmicrograph provided by I.L. Chrystie

We know very little about the environmental properties of the more recently recognized diarrhoea agents. Work on rotavirus, and other viruses, is

only just beginning and there are still enormous technical problems in isolating these organisms from the environment because they do not grow well in cell cultures. Environmental information about *Campylobacter*, ETEC and *Yersinia* is now being collected. The laboratory difficulties have been, or soon will be, overcome and we can expect an explosion of literature on these and other pathogenic enteric bacteria in the next few years. The same is true for the parasite *Giardia lamblia*.

Recent reviews of enteroviruses (which, in the absence of laboratory techniques provide the best approximation of rotavirus) and *Vibrio cholerae* in the environment have been published in *Tropical Diseases Bulletin*, Vol 78, pp 185-230 and pp 865-880.

In the next issue . . .

Diarrhoea Dialogue 8 will look at the role of drugs in diarrhoeal disease control.

The most feared of the diarrhoeal diseases

“Cholera remains an enigmatic disease and has by no means yielded up all its secrets. There is much to be done in both the epidemiology and the immunology of cholera; many questions remain unanswered”⁽¹⁾

Historically, cholera has always been the most feared of the diarrhoeal diseases because it can spread fast and kill its victims within a matter of hours if treatment is not readily available. It therefore continues to be one of the four diseases subject to the International Health Regulations⁽²⁾, under which member states are required to notify the presence of cases to WHO. Although the Regulations have not prevented the international spread of cholera, they have helped to keep WHO and public health authorities informed of the global epidemiological situation and alert to any possible risk.

Seventh pandemic

The seventh pandemic of cholera, which began in Indonesia in 1961 and is caused by the El Tor biotype of *Vibrio cholerae*, has now spread to more than 90 countries in Africa, Asia, Europe, North America and Oceania. Although there have been periods of respite, the pandemic still shows no signs of decreasing (Table 1). In many countries cholera has become endemic, creating a greater risk of spread to new areas. However, experience in developed countries has repeatedly shown that, when importations of cholera are detected promptly, subsequent spread within a country can be prevented.

In 1980, as in previous years, the great majority of reported cases occurred in Africa (17,675 cases in 14 countries) and Asia (19,108 cases in 15 countries)⁽³⁾. In addition, a total of 32

cases occurred in nine countries in America, Europe and Oceania, but 27 of these were imported and only five were indigenous cases. While no new countries were infected that year, and the total number of countries reporting the disease declined substantially, there were a number of countries where cholera reappeared, sometimes after an absence of several years. This is, however, a common pattern.

Resurgence in the Eastern Mediterranean

This year, a total of 20,346 cases have been reported up to late September and notifications are still far from complete. Twelve countries in Africa and 15 in Asia have notified the presence of cholera, and small numbers of cases have again been reported by the USA and Australia. No new countries have been infected. Of interest is the apparent resurgence of cholera in the Eastern Mediterranean area, where an outbreak of 869 cases occurred in Jordan in July/August. Small numbers of cases were also reported in the Gaza Strip and the West Bank, and in Iran there have been 3,030 cases reported altogether since mid-April.

Cholera control

Cholera is an alarming disease and continues to attract more attention from public health administrators and the general public than any other cause of diarrhoea. However, it should be

pointed out that most cases of cholera caused by the El Tor biotype of the vibrio are mild, and that hospitalized cases constitute no more than 5 to 10 per cent of all diarrhoea cases in endemic areas, except at times of epidemics. Even then, they are treated



Preventing cholera. Inoculation before a religious fair in India. Latrines and piped water were also provided for the pilgrims. WHO photograph by Homer Page

and controlled in the same way as other acute diarrhoeas. Experience has shown that specific strategies for cholera control are not effective, and consequently the Technical Advisory Group of the WHO Diarrhoeal Diseases Control Programme, at its first meeting in 1978, concluded that cholera control could best be achieved through national programmes for the control of all diarrhoeal diseases.

Guidelines for the control of cholera within the framework of national diarrhoeal disease control programmes are available from: The Manager, Diarrhoeal Diseases Control Programme, World Health Organization, 1211 Geneva 27, Switzerland.

Dhiman Barua and Anne Martinez, Diarrhoeal Diseases Control Programme, World Health Organization, Geneva, Switzerland.

⁽¹⁾Mackay D 1979 *Cholera research laboratory in Dacca, Bangladesh — a brief history*. *Tropical Doctor*, January pp 31–32.

⁽²⁾*International Health Regulations (1969) Second annotated edition*. Geneva, WHO 1974.

⁽³⁾WHO Weekly epidemiological record 1981 No 56: 97–98.

TABLE 1. Global cholera situation, 1975–1980

	1975	1976	1977	1978	1979	1980
Number of countries reporting cholera	29	27	35	40	43	32
Number of new countries infected	1	—	3	8	2	—
Number of cases reported	92,123	66,020	58,087	74,632	56,813	36,815

Finding the guilty organisms

David Candy looks at the key micro-organisms in childhood diarrhoea and highlights recent advances in our understanding of this area.

The last decade has been an exciting time for clinicians and scientists interested in acute childhood diarrhoea. In addition to well-known microbial causes of human diarrhoea (*V. cholerae*, *Salmonellae*, *Shigellae* and certain strains of *E. coli*) rotavirus, *Campylobacter* and *Yersinia* are now recognized and our knowledge of the mechanisms involved in *E. coli* diarrhoea has grown.

Epidemiological studies have highlighted the world-wide importance of rotavirus and *E. coli* diarrhoea and this review will concentrate on these organisms.

Rotavirus

Rotavirus causes perhaps 50 per cent of childhood diarrhoea increasing to 80 per cent in temperate climates during winter. It was first identified in children in 1973 but had previously been found in other young mammals.

In young patients, watery diarrhoea preceded by vomiting should suggest rotavirus infection. Dehydration occurs particularly rapidly because of vomiting and, although recovery is usually uneventful with proper rehydration treatment, deaths do sometimes occur. Although a proven cause of diarrhoea, rotavirus has been found in the stools of healthy, newborn infants.

Loss of electrolytes and water

Rotavirus is thought to cause diarrhoea by destruction of the cells lining the small intestine. The cells replacing those shed into the gut lumen are less able to absorb sugars. Unabsorbed sugar draws fluid from the rest of the body into the gut by osmosis. At the same time, the process of sodium and water transfer, which depends on sugar absorption in the upper gut, becomes less efficient. The overall effect is a huge loss of electrolytes and water from within the small

intestine which the large bowel is then unable to reabsorb. The result is diarrhoea and further dehydration.

E. coli

Only certain strains of *E. coli* cause diarrhoea in humans and these are classified into three groups:

1. Enterotoxigenic *E. coli* (ETEC)

These *E. coli* produce enterotoxins which stimulate the small intestine to secrete electrolytes and water. Two enterotoxins of *E. coli* are recognized: a high molecular weight protein readily destroyed by heating (heat-labile toxin (LT) and a low molecular weight heat-stable toxin (ST).

2. Enteroinvasive *E. coli* (EIEC)

EIEC invade the mucosa of the ileum and the colon, unlike ETEC which remain on the mucosal surface.

3. Enteropathogenic *E. coli* (EPEC)

EPEC do not produce either LT or ST, neither do they invade the gut, yet they certainly cause diarrhoea. They have been implicated by epidemiological means in outbreaks of diarrhoea in infants.

Serological classification

E. coli can also be classified serologically on the basis of a bacterial cell wall antigen ('O' antigen). At present, 164 distinct O serogroups are recognized. ETEC, EIEC and EPEC strains tend to have distinctive O serogroups (see Table 1). However, *E. coli* possessing these O serogroups are not always pathogenic and *E. coli* which have not been serotyped may also cause diarrhoea.

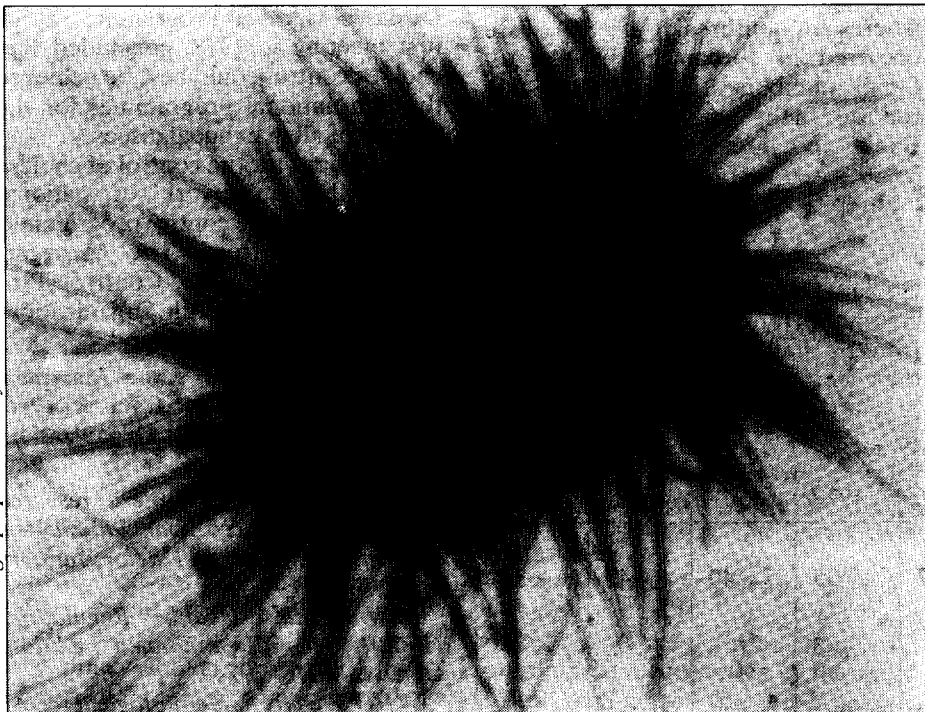
Nevertheless, the serotyping of *E. coli* is a useful epidemiological tool as other means of identifying potentially pathogenic *E. coli* are difficult, expensive and not widely available.

Infections

Infections with ETEC cause copious, watery diarrhoea and are an important cause of warm season diarrhoea in young children in developing countries.

EIEC produces fever, abdominal cramps, urgent and painful defaecation (tenesmus) and watery diarrhoea, followed by scanty discharges of blood and mucus (dysentery). Microscopic examination of methylene-blue stained faecal mucus shows pus and red cells.

EPEC can produce sudden cholera-like diarrhoea in adults whereas, in infants, the disease often tends to be



Electronmicrograph provided by Stuart Knutton

Fimbriae help *E. coli* stick to the lining of the intestine, thus preventing expulsion.

more prolonged with high mortality. It is possible that these organisms produce uncharacterized enterotoxins.

Another factor which may determine the ability of *E. coli* to cause diarrhoea is whether they can produce hair-like structures called fimbriae or pili⁽²⁾. These fimbriae anchor *E. coli* to the lining of the small intestine, overcoming the attempts of the bowel to expel them and allowing intestinal colonization.

V. cholerae

The vibrio associated with cholera was probably confined to the area around Calcutta until 1813 when a series of pandemics occurred. Improved sanitation in industrialized countries now keeps cholera at bay, but it is still endemic in parts of Asia (see page three). Diarrhoea caused by cholera looks like rice water and a litre or more of fluid can be lost every hour for several days. An enterotoxin almost identical to LT is the cause of this symptom.

Campylobacter

Campylobacter have been reported mainly from Europe, South Africa and North America where as much as 15 per cent of infant diarrhoea may be due to this organism. Abdominal pain, fever, diarrhoea and, occasionally, dysentery are the usual features. Pet dogs, poultry and milk are likely sources of infection.

Yersinia

Yersinia enterocolitica has been identified as a cause of gastroenteritis in children in Canada, Europe, Japan and South Africa. It produces pain severe enough to suggest a surgical emergency. *Yersinia* are invasive and also produce ST. Special bacteriological techniques are needed to grow *Yersinia* and *Campylobacter* from stools. During incubation, high temperatures favour *Campylobacter* and low temperatures encourage *Yersinia*.

Shigellae and Salmonellae

Shigellae are an important cause of

diarrhoea in infants aged six months to two years. Since shigellosis is spread by person-to-person contact, incidence is higher where environmental health and personal hygiene are poor⁽³⁾. As few as ten swallowed bacteria are enough to cause the disease.

Salmonellae are food-borne and contamination of animal carcasses in slaughterhouses is the usual source. Symptoms resemble those produced by *Campylobacter*.

Salmonellae and Shigellae are invasive and probably release toxins from inside the intestinal cells. These cause secretion of fluid in the upper gut and cell damage in the lower gut.

Protozoal infections

Giardia lamblia and *Entamoeba histolytica* are single cell micro-organisms (protozoa) which have been reported in most countries. *G. lamblia* grows in the small intestine and is thought to be a cause of both acute and chronic diarrhoea, by unknown mechanisms. *E. histolytica* prefers tropical zones and causes ulceration of the large intestine.

Other causes

Diarrhoea may be due to infections outside the gut, such as pneumonia, and this possibility must be considered in any child with diarrhoea.

Conclusions

At present, the mainstay of treatment for acute diarrhoea is replacement of water and electrolyte losses. Even in rotavirus infections, when sugar absorption is impaired, the gut has sufficient reserves to allow successful treatment of diarrhoea by oral rehydration with sugar and salt solutions.

In future it may be possible to offer specific preventive measures or treatment for specific causes of diarrhoea. Simple, low-cost methods for detecting the guilty organisms will then be of great importance. Rotavirus can already be detected in stools by a test relying on antibodies against the virus (enzyme-linked immunoabsorbent

assay — ELISA) which can be carried out without expensive equipment such as electron microscopes.



Replacing water and electrolyte losses.

Photograph by Michael McQuestion

The use of simpler tests in the field will mean that specific therapy is only given when necessary (see table on page six) and the use of antibiotics prevented when contraindicated (e.g. rotavirus) or where such drugs may actually prolong the illness (e.g. Salmonellae).

Realizing these aims will depend on health workers being able to recognize the causative organism early on in the disease.

David Candy, Institute of Child Health, University of Birmingham.

⁽¹⁾Rohde J E and Northrup R S 1975 *Taking science where the diarrhoea is. Acute Diarrhoea in Childhood (Ciba Foundation Symposium 42): 339-366.*

⁽²⁾Stickiness and sickness 1980. *Diarrhoea Dialogue 2:3.*

⁽³⁾Khan M U 1980 *Soap, water and shigellosis. Diarrhoea Dialogue 2:3.*

Further reading:

The most up to date review on all aspects of causes of acute diarrhoea is: *Infections of the G.I. tract, Clinic in Gastroenterology, Vol 8 No 3 (1979). Ed. H P Lambert. W B Saunderson Co. Ltd., Eastbourne, UK. Especially chapters 1, 2, 3, 4, 5, 8 & 9.*

For readers who would like further information David Candy has prepared a reading list which is available free from AHRTAG. Please address your letter to the Executive Editor, *Diarrhoea Dialogue*, AHRTAG, 85 Marylebone High Street, London W1M 3DE, UK.

Table 1	O Serogroups in which ETEC, EIEC and EPEC commonly occur
ETEC	06, 08, 015, 020, 025, 078, 0115, 0148, 0159
EIEC	028, 0112, 0115, 0124, 0136, 0143, 0144, 0147, 0152
EPEC	055, 086, 0111, 0127, 0128, 0142

Practical advice series

CLINICIAN'S GUIDE TO AETIOLOGY

This table gives the information that will help to identify, on clinical grounds alone, the most common agents of diarrhoea. It is greatly simplified. For example, some agents produce a variety of clinical features. Only agents of major importance world-wide have been included. In certain areas, at certain times, the picture may be quite different.

Try and find out what the important causes of diarrhoea are in your area.

Caution: There are a number of other conditions associated with diarrhoea such as infections outside the gut (e.g. measles and malaria), malnutrition, food intolerance etc. Remember to look for these and give specific treatment where appropriate.

If readers find this table useful, we may present other information in the same way in future issues of *Diarrhoea Dialogue*. Please send us your comments on this clinician's guide.

COMPLAINT	ASSOCIATED CLINICAL FEATURES		INCUBATION PERIOD	EPIDEMIOLOGICAL FEATURES	ORGANISMS	FIRST LINE TREATMENT
	COMMON	OTHERS				
ACUTE WATERY DIARRHOEA ↓ The stool takes the shape of the container	<ul style="list-style-type: none"> • Vomiting • Fever 	<ul style="list-style-type: none"> • Severe dehydration in some 	24-72 hours	<ul style="list-style-type: none"> • Infants and young children • Common world-wide in all socio-economic groups • Peak in colder seasons in temperate climates 	Rotavirus	<ul style="list-style-type: none"> • Rehydration therapy
	<ul style="list-style-type: none"> • Nausea • Vomiting • Abdominal pain 	<ul style="list-style-type: none"> • Fever • Malaise • Severe dehydration 	6-72 hours	<ul style="list-style-type: none"> • Infants and young children in developing countries • Travellers diarrhoea in adults 	Enterotoxigenic <i>Escherichia coli</i> (ETEC)	<ul style="list-style-type: none"> • Rehydration therapy
	<ul style="list-style-type: none"> • Nausea • Vomiting • Fever • Chills • Abdominal pain 	<ul style="list-style-type: none"> • Malaise 	8-36 hours	<ul style="list-style-type: none"> • Children • Common world-wide • Food-borne outbreaks (animal products) • Warmer seasons 	Non-typhoid Salmonellae	<ul style="list-style-type: none"> • Rehydration therapy
	<ul style="list-style-type: none"> • Abdominal pain • Fever • Malaise 	<ul style="list-style-type: none"> • Chills • Blood and pus in the stools 	3-5 days	<ul style="list-style-type: none"> • World-wide distribution • In developed countries may be food-borne (animal products) or transmitted by handling of animals 	<i>Campylobacter</i>	<ul style="list-style-type: none"> • Rehydration therapy • Erythromycin in severe cases
	<ul style="list-style-type: none"> • Vomiting • Abdominal pain 	<ul style="list-style-type: none"> • Severe dehydration • Circulatory collapse, 'shock' 	1-3 days	<ul style="list-style-type: none"> • Children in endemic areas • Adults in newly affected areas • Not found in Latin America 	<i>Vibrio cholerae</i>	<ul style="list-style-type: none"> • Rehydration therapy • Tetracycline
	<ul style="list-style-type: none"> • Nausea • Vomiting 	<ul style="list-style-type: none"> • Fever 	6-72 hours	<ul style="list-style-type: none"> • Nursery outbreaks in developed countries • Uncertain in developing countries 	Enteropathogenic <i>Escherichia coli</i> (EPEC)	<ul style="list-style-type: none"> • Rehydration therapy
DYSENTERY ↓ The stool is soft and watery with blood and/or pus	<ul style="list-style-type: none"> • Fever • Abdominal pain 	<ul style="list-style-type: none"> • Malaise • Vomiting • Urgency to defaecate • Painful spasm on defaecation 	36-72 hours	<ul style="list-style-type: none"> • Children • Poor hygiene • Malnutrition • Institutions • Warmer seasons 	Shigellae	<ul style="list-style-type: none"> • Rehydration therapy • Ampicillin or Trimethoprim-Sulfamethoxazole
PROLONGED DIARRHOEA (OR DYSENTERY) ↓ For at least 7 days, stools have been more frequent or of softer consistency (with or without blood or pus)	<ul style="list-style-type: none"> • Abdominal discomfort 		2-6 weeks	<ul style="list-style-type: none"> • All age groups • World-wide distribution 	<i>Entamoeba* histolytica</i>	<ul style="list-style-type: none"> • Metronidazole
	<ul style="list-style-type: none"> • Abdominal distension • Flatulence 	<ul style="list-style-type: none"> • Anorexia • Nausea • Malabsorption • Frothy stools 	1-3 weeks	<ul style="list-style-type: none"> • Young children • Some travellers • Poor hygiene • World-wide distribution 	<i>Giardia* lamblia</i>	<ul style="list-style-type: none"> • Metronidazole

*Can be identified on examination of the stools with a light microscope. Blood and pus from Shigellae and *Campylobacter* can also be identified.

Produced in collaboration with the Ross Institute of the London School of Hygiene and Tropical Medicine and The Save the Children Fund.

Teaching by example

The highly successful Costa Rican experience with oral rehydration therapy has been shared with several other Latin American countries.

Since a trial oral rehydration (OR) project was begun in Costa Rica at the beginning of 1978, ORT has proved an effective life-saver in both bacterial and rotaviral infant diarrhoea, including neonates^(1 & 2). Routine implementation of OR in the National Children's Hospital has resulted in more than an 80 per cent reduction in mortality. The technique is easily understood both by health personnel and mothers visiting the emergency unit at the hospital⁽³⁾. In addition, health centres in both urban areas and the countryside have also been able to introduce oral rehydration therapy.

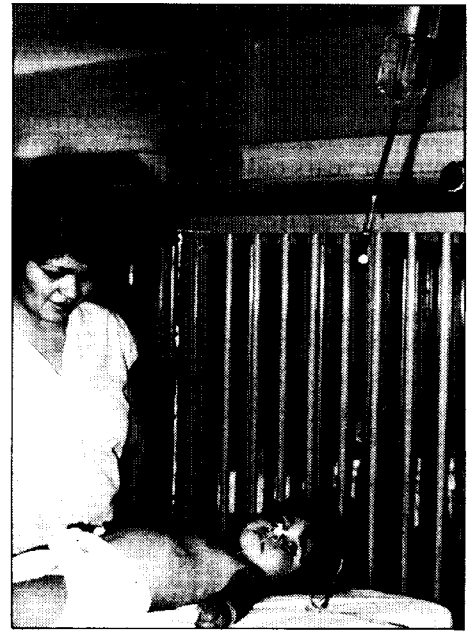
Field project

A field project to monitor oral rehydration therapy given by mothers to their children in rural areas was started by the Instituto de Investigaciones en Salud (INISA) in 1980. Mothers soon learned the technique

and treated children successfully. At the same time, comprehensive teaching material for health personnel was prepared by the state welfare system, while the Ministry of Health established a national programme of diarrhoeal disease control with technical assistance from the Pan American Health Organization (PAHO).

Sharing experiences

During the past three years, the Costa Rican experience has been shared with several Latin American countries. Health personnel from Bolivia, El Salvador, Guatemala, Honduras, Panama, Paraguay and Venezuela visited Costa Rica for a first-hand view of the OR programme. Visiting doctors spent a week in the emergency unit of the National Children's Hospital, INISA's rural programme in Puriscal, the rural hospital in Grecia and the Department of Maternal and Child Health at the health ministry.



Sometimes naso-gastric rehydration is necessary.

Photographs by Dr Daniel Pizarro

Mortality rates drop

Since 1978, about 15,000 dehydrated children (including 160 neonates) have been rehydrated in the emergency unit of the National Children's Hospital. Mothers have been taught about the causes, transmission and management of diarrhoea as well as techniques to rehydrate and prevent dehydration among infants.

All these efforts have had a considerable impact on both hospital mortality rates and overall diarrhoeal disease mortality in Costa Rica⁽⁴⁾.

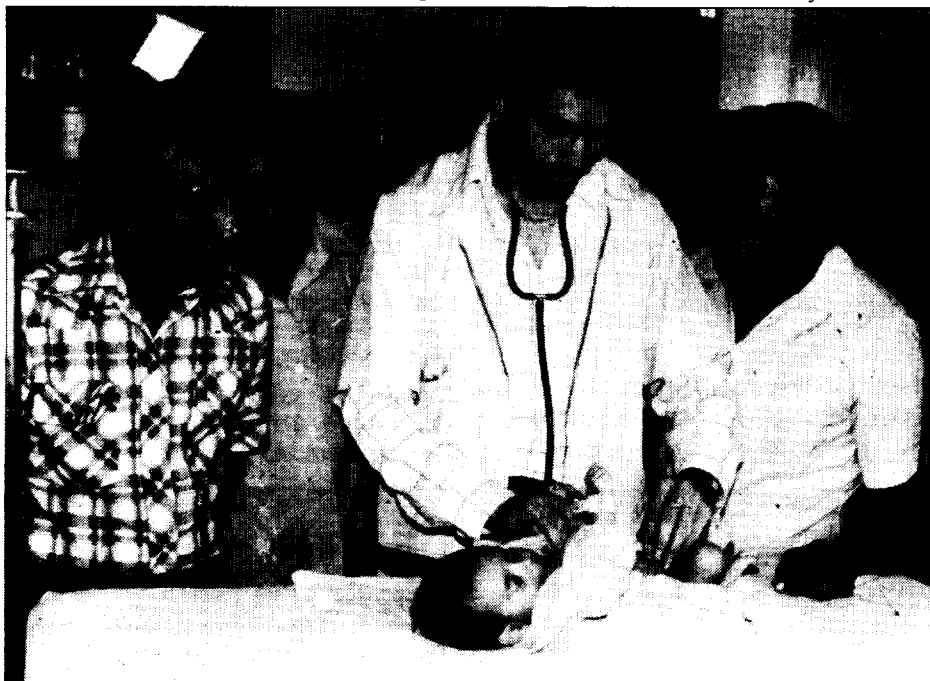
Daniel Pizarro, Emergency Service, National Children's Hospital, San Jose, Costa Rica.

⁽¹⁾Nalin D R et al 1979 Oral rehydration and maintenance of children with rotavirus and bacterial diarrhoeas. *Bulletin of the World Health Organization* vol 57: 453-459.

⁽²⁾Pizarro D et al 1979 Evaluation of oral therapy for infant diarrhoea in an emergency room setting; the acute episode as an opportunity for instructing mothers in home treatment. *Bulletin of the World Health Organization* vol 57: 983-986.

⁽³⁾Pizarro D et al 1979 Oral rehydration of neonates with dehydrating diarrhoeas. *The Lancet*, vol 2: 1209-1210.

⁽⁴⁾Mata L J 1981 Diarrhoeal diseases. How Costa Rica won. *World Health Forum*, 2: 141-142.



Health auxiliaries from Panama visit Costa Rica to learn about the oral rehydration programme.

Ethiopia: too few medical workers

I am the Director of an orphanage in a rural part of Ethiopia. In the orphanage there are children between seven to fourteen years old. They are from the drought stricken area. I have found your issues of *Diarrhoea Dialogue* very helpful since I am not medically trained and there is also a scarcity of medical workers in my country. I am interested in receiving future issues.

Birhanu T Michael, P.O. Box 545, Dessi/Wollo, Ethiopia.

Lack of one litre bottles

In the nutrition centres in the Salvadorean refugee camps in Honduras we started oral rehydration programmes for children with diarrhoea. The oral rehydration solution is prepared daily for those malnourished children with diarrhoea. In addition to using the available packets of oral rehydration salts, the nutrition workers teach the mothers how to prepare the home-made solution. When the programme started in August, other mothers in the camps began bringing their children who had diarrhoea to the nutrition centres to receive the oral rehydration solution even though they receive the packets at the health centres.

The mothers told us that the problem with the packets is that they do not have containers in which to measure or store one litre of rehydration solution. Since we prepared the solution in the nutrition centre and the mothers would bring whatever containers they had, such as ½ litre bottles or ¼ litre glasses which we would refill as needed, children with diarrhoea began receiving the oral rehydration solution.

The shortage of one litre containers appears to be very common and one idea to help resolve it would be to make the oral rehydration packets with four sub-sections, each containing the correct amount of salts for a ¼ litre



Giving oral rehydration at home: often, containers are a problem.

WHO photograph by J. Abcede

glass — a container most mothers have in their homes. I am interested in learning whether such packets have been manufactured and at what cost in comparison to the others. I would also recommend that UNICEF consider developing such packets so that more mothers could prepare the oral rehydration solution in their homes for their children with diarrhoea.

Solange T Muller, Apartado A-51, Managua, Nicaragua.

Editors' note:

A number of readers from as far apart as Kiribati in the Pacific to the Ivory Coast in West Africa, have commented on the value of naso-gastric feeding for rehydration in diarrhoea. In some cultures, this procedure is associated with feeding unconscious patients, and parents have become worried because they associate the technique with impending death. However, in cases of severe diarrhoea, where the child is vomiting, naso-gastric feeding can be a life-saver and an excellent means of showing how effective the techniques can be.

Naso-gastric feeding in Kiribati . . .

Ms J. Aitken's letter (*Diarrhoea*

Dialogue 6) prompts us to write concerning our experience with naso-gastric administration of oral rehydration (OR) fluids in childhood diarrhoea. Like her, we find this an extremely useful method in children whose diarrhoea is accompanied by vomiting, where frequently the mother is reluctant to give fluids by mouth because it provokes vomiting.

Supplies to these small coral atolls are precarious and frequently sugar, salt and OR packets are unavailable. So we use water from unripe (drinking) coconuts with an added tablespoon (for each nut) of sugar or honey, if available, or a local syrup (kamaimai) prepared from the sap of the coconut blossom. This mixture is equally effective by spoon or naso-gastric drip, but in the latter case it needs to be filtered before use since fragments of the unripe coconut flesh tend to block the tube.

For those unfamiliar with techniques of naso-gastric rehydration, details are given in Dr Maurice King's book *Primary Child Care: a manual for health workers* Vol 1 1980 (reprinted), available from Oxford University Press, Oxford OX2 6DP, UK.

Dr Antony Franks (District Medical Officer) and Miss Baane Timi (Public Health Nurse), Abemama Atoll, Kiribati, Central Pacific.

. . . and Afghanistan

The naso-gastric method has been used successfully for the rehydration of babies, who refuse to drink fluids or in whom even small amounts cause vomiting, in one of the MCH centres (Shewaki) near Kabul for over three years. Health workers are taught how to insert a naso-gastric tube and how to make sure it is in the stomach before starting the drip. This method has also been included in the training material for health workers in Afghanistan.

Dr Shanti Ghosh, WHO Medical Officer on Family Health Services Development, Afghanistan.

Editors: Dr William Cutting (U.K.) and Dr Katherine Elliott (U.K.)

Editorial advisers: Dr David Candy (U.K.), Dr I. Dogramaci (Turkey), Dr Richard Feachem (U.K.), Dr Michael Gracey (Australia), Dr N. Hirschhorn (U.S.A.), Dr D. Mahalanabis (India), Dr Leonardo Mata (Costa Rica), Dr Mujibur Rahman (Bangladesh), Dr Jon Rohde (Rockefeller Foundation), Dr E. O. Sullesta (Philippines), Dr Paul Vesin (France), Dr M. K. Were (Kenya).

Executive editor: Denise Ayres

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