THE PROBLEM OF DIARRHOEA IN INFANTS AND CHILDREN

CAROL J. JACCARINI
M.D., M.R.C.P., D.C.H.

Diarrhoea is a common problem in paediatric practice and is due to causes ranging from unwise eating in older children leading to trivial brief episodes to those producing severe and potentially fatal gastroenteritis in infants. Diagnosis and treatment are not usually difficult, but at times both can be extremely so necessitating the admission of the child to hospital. In practice, however, the commonest reason for admitting a child with diarrhoea is the necessity of giving intravenous fluid and providing electrolyte replacement.

The fully breast-fed baby normally has loose, golden-yellow, sometimes green, frequent stools and mothers often complain that their baby has diarrhoea, when in fact the stools are quite normal. One should therefore always have a look at the stools oneself and it is wise to enquire if the mother is taking purgatives, as some of these are secreted in the milk. It is also important to bear in mind that the fully breast-fed infant hardly ever suffers from gastroenteritis.

Gross underfeeding of a young infant produces small, green, loose stools. This may be mistaken for diarrhoea leading to more withdrawal of food by the mother. It is vital to stress to the mother that, in contrast to underfeeding, overfeeding is virtually never the cause of diarrhoea, for a full-term infant knows when to stop. In such a case, if the food intake is increased, the stools will rapidly revert to normal. In the bottle-fed infant looseness of the stools may be produced by the excessive addition of sugar in milk feeds or else by the administration of orange juice. Teething is commonly said to cause many symptoms in infants and it is important to remember that diarrhoea is never due to teething. Another commonly supposed cause is milk intolerance or allergy. This is actually extremely rare and one is hardly ever justified in changing from one brand of milk to another for this or indeed for any other reason.

Acute gastroenteritis is the commonest problem in practice and usually occurs in the summer months though cases are seen sporadically throughout the year. Vomiting usually precedes diarrhoea but may be completely absent. An extremely common mistake is for the mother to continue feeding the baby as usual, often adding more solids in an attempt to "keep the feeds down". Often, though the baby's feeds are rightly replaced by 'clear fluids', these are constituted badly. Too much salt produces hypernatraemia with its many possible harmful effects especially on the central nervous system; excess sugar often produces more intestinal hurry through an osmotic effect and thus further diarrhoea. Oral or intramuscular antibiotics, commonly of the broad spectrum type, are often prescribed. These do not help and may be positively harmful. The child's condition, especially in infancy, can deteriorate at an alarmingly rapid rate because of fluid and electrolyte loss and if there is little or no response to home treatment, there should be no delay in referring the child to hospital. It is particularly easy to miss dehydration in an obese infant. In particular the skin and subcutaneous tissue will show no obvious change and the important signs are sunken eyes, depressed fontanelles and dry mucosae. It is vital not to mistake dehydration and rapid acidotic breathing in an obese baby for a respiratory infection with parenteral diarrhoea. The latter is common, especially with otitis media and urinary tract infection, and antibiotics should be used for the primary condition.

Not uncommonly, after an apparently

complete recovery from an attack of gastroenteritis, there is a relapse. On re-introducing full-strength milk feeds, the loose stools return, often causing marked soreness of the baby's buttocks. In most cases, this is due to *lactose intolerance* which results from damage to the superficial part of the small intestine mucosal surface and a decrease in the sugar-splitting enzyme lactase, which is essential for the absorption of lactose. On pH testing, the stools are found to be acid and reducing substances are detected in the stools by the usual tests for sugar. Typically, Clinitest (which detects any reducing agent) is positive, while Clinistix (which is specific for glucose) is negative. All milk and milk-products must be excluded from the infant's diet for a variable period of time till the intestinal mucosa recovers fully its absorptive functions.

Diarrhoea brought on by *drugs*, usually broad-spectrum antibiotics, is also fairly common. The indication for antibiotic therapy is very often dubious and the child would no doubt have been better off if left 'untreated'. Most childhood infections are viral and if the child is on antibiotics his diarrhoea usually clears on stopping chemotherapy. In gastroenteritis, the indiscriminate use of antibiotics is common practice. As mentioned above, the treatment of this condition is basically that of fluid and solute replacement and perhaps, oral intestinal antibiotics should be reserved for cases from the faeces of which a pathogen has been cultivated. It has been shown conclusively by several workers that antibiotics do not improve the child's symptoms, often do not eradicate the bowel infection and may indeed prolong the carrier state. However, in the critically ill child, especially one that remains so after a period of adequate rehydration, it is wrong to withold systemic antibiotics, for in such cases septicaemia is a real possibility.

The child with pale, loose, greasy, bulky and offensive stools has steatorrhea, which is the cardinal feature of the *'malabsorption syndrome'*. The motions typically stick to the nappy and are difficult to flush down. In some cases, however, the stools may not look abnormal at all. The commonest cause in Malta is *coeliac disease* (gluten-sensitive enteropathy) while *Cystic fibrosis* which is common in the United Kingdom seems to be rare. Coeliac disease nowadays presents around the age of six months because of the current practice of introducing cereals early into the infant's diet. It responds dramatically to a gluten-free diet, but before starting treatment it is important to confirm the diagnosis by special investigations in hospital. This is essential because the diet, which is not an easy one to stick to, will need to be continued to well beyond puberty.

In the older child, *spurious diarrhoea due to constipation and faecal soiling* is another commonly encountered problem. The diarrhoea is produced by the fluid part of the stool above a hard faecal mass in the rectum leaking through out of the anal canal i.e.: encopresis. It is important to make a proper diagnosis for in such cases anti-diarrhoea measures are quite obviously useless. Inspection of the anal orifice for the presence of a fissure, especially common in infants, and a rectal examination are essential. Treatment is that of constipation and its cause. One must remember that Hirschsprung's disease (aganglionic megacolon) can also present in a similar way with diarrhoea rather than constipation.

In the preadolescent child, *emotional stress* often causes mild diarrhoea. This is ascribed to intestinal hurry and is usually brought on by stressful situations like those caused by overdemanding parents, imminent examinations, and worries about puberty. Similar to this 'nervous diarrhoea', but less well understood, is the so-called *'irritable colon syndrome'*. This typically occurs in the pre-school child who is otherwise healthy and who presents with chronic or recurrent mild bouts of loose stools, often offensive and blood-streaked, with three or four motions a day. There may be periods of constipation in between. It is difficult to make a proper diagnosis in these cases and most of them are probably due to dietary indiscretion. The condition usually returns to normal

when fried foods, fruit and sweets are excluded from the child's diet. Some cases may be due to a low grade infection with an enteropathogen.

Lastly, there are occasional cases of diarrhoea which are extremely difficult to diagnose or treat in the home and which need to be managed in hospital. Two such cases were seen recently: Case I — Coeliac disease with secondary lactose intolerance; and Case II — Primary Sucrase-Isomaltase deficiency with a complicating lactase deficiency following gastroenteritis.

Case I

M.S. (date of birth: 6.3.69).

This child was first seen at 14 months of age with a history of failure to thrive. She was born of unrelated parents, after a normal pregnancy and delivery. Birth weight was 6 lbs 9 oz. She took milk feeds well up to the age of 5 months when she weighed 17 lbs. At this time cereals and other solids were introduced and from then on the child fed poorly, vomited frequently and had bouts of bulky, greasy, offensive stools, about four times a day. There were two healthy older sibs in the family. Examination showed a pale, miserable and extremely irritable child who was markedly underweight at 15 lbs. She had marked wasting of the subcutaneous tissue and loose folds on the buttocks. The abdomen was distended. She was tentatively put for a period on a milk-free and lactose-free diet but made no improvement. She was then started on a gluten-free diet and there was an immediate improvement both in the child's behaviour and in the nature of the stools. However, the mother found it rather difficult to keep up this strict gluten-free lactose-free diet and on 7.6.70 the child was admitted to hospital because of frequent, watery stools. Her weight on admission was 13 lbs 4 oz and the subsequent weights during her 10 week stay in hospital are shown in the figure. It should be noted that once more there was no improvement on low-lactose milk and during this period she had several relapses of severe diarrhoea needing intravenous fluid therapy. When a strict gluten-free diet was re-introduced the diarrhoea soon settled and the child's weight continued to increase steadily. The child was discharged home well on 22.8.70 weighing 17 lbs 8 oz and has remained well since. She can now tolerate milk and milk-products but has to continue on a gluten-free diet.

Case II

J.N. (date of birth: 14.4.71)

This infant was born at 37 weeks gestation following a normal pregnancy and delivery. Birth weight was 6 lbs. 11 oz. and there were no immediate neonatal problems. During the first 2 months she was a poor feeder, had infantile colics and her buttocks were constantly sore. In August 1971 (at four months of age), she had an episode of diarrhoea and very sore buttocks. This recurred in September when the family was on holiday in Malta. Seen at this time a diagnosis of "probably lactose intollerance following gastroenteritis" was made and the child started on a lactose-free milk formula ('Nutramigen'). The diarrhoea soon cleared up. Back in London, she was seen by Dr. A.P. Norman and the mother was to introduce the usual milk feeds slowly.

In October the child was given a full-strength milk feed by mistake and immediately she started vomiting and developed diarrhoea and sore buttocks. On 16.10.71 she was admitted to Great Ormond Street Hospital under the care of Dr. Norman and the diagnosis made was again one of secondary lactose intolerance. Investigations done were: Hb and Blood film Urea, Electrolytes, Urinalysis, X-Ray Chest — all normal; urine culture-sterile. Sweat Na-17 and 15mEq/lit. Swab from buttocks grew coagulase negative Staphylococcus. Stools for sugars contained 300 mg lactose and 100 mg galactose per 100 grams. 24-hour urine contained less than 5 mg/100 ml of sugar and the amino-acid pattern was normal. The stools were shown to be persistently acid on pH testing. The child was put on lactose-free milk 'Galactomin' and her diarrhoea settled soon after. She was sent home on 20.10.71, and remained well till April 1972 when during at attempt at restarting cow's milk and sugar her diarrhoea and sore buttocks recurred. On the same day, the child had also been given 'junior' aspirin' in syrup form. The diar-

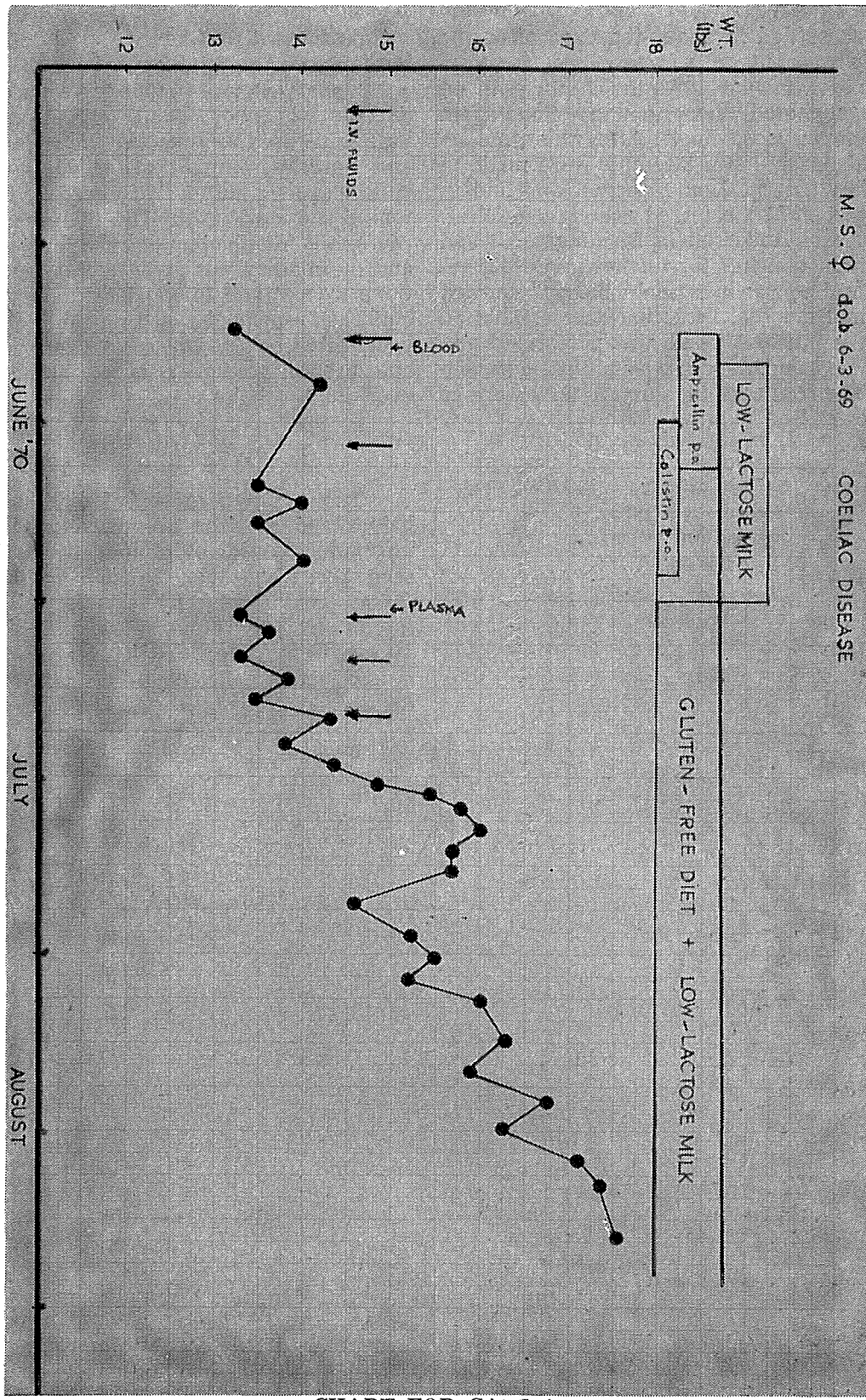

CHART FOR CASE 1

rhoea settled in about ten days after the baby was put on a lactose-free, fructose-free diet. In June 1972, the child was given a sugar-coated 'Phenergan' tablet and again developed loose stools. After this time several unsuccessful attempts were made to restart the child on a normal diet and in August 1972 I referred the child to Prof. Charlotte Anderson in Birmingham, and she was admitted for further investigation. Peroral upper intestinal biopsy showed normal mucosa. Disaccharidase estimation on the mucosal tissue was performed and this revealed a sucrase-isomaltase deficiency. The results of the enzyme tests were as follows:

Enzyme	*Units activity (u/mole substrate split/g wet weight mucosa)*	*B.C.H.lab range*
Lactase	0.9	(1.0 — 5.7)
Sucrase	0.3	(4.0 — 8.6)
Maltase	11	(14.6 — 33.0)
Palatinase	0.3	(1.2 — 6.8)

This showed that lactase was on the borderline of normal, probably indicating a secondary lactase deficiency earlier on. Sucrase was almost completely absent. Palatinase is used as a marker for isomaltase and there was only a trace of activity. The child was first put on half and half 'Galactomin' and cow's milk and then just on cow's milk. She had no diarrhoea after this and the mother was given dietary instructions to continue to avoid sucrose. The child has remained well since and it is expected that as she grows older she will be able to tolerate more sucrose than she ever did as a baby.

Summary

The common causes of diarrhoea in babies and children are discussed. Two uncommon causes of prolonged diarrhoea seen recently — one a case of coeliac disease complicated by secondary lactose intolerance, the other a case of primary sucrase-isomaltase deficiency with acquired lactase deficiency — are reported in detail.