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Short reports

Can acetate replace bicarbonate in oral rehydration solution for infantile diarrhoea?

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SUMMARY In a double-blind trial two groups of 20 infants and young children suffering from diarrhoeal dehydration and acidosis were successfully treated with an acetate and a bicarbonate containing oral rehydration solution. The former was found to be as effective as the latter and was equally acceptable to the patient.

Most oral rehydration programmes use a single glucose electrolyte solution to treat diarrhoea.[1] Sodium bicarbonate is a useful constituent of oral rehydration solution for correcting acidosis. However, bicarbonate reacts with glucose to form furfural compounds leading to brown discoloration and a short shelf life in prepackaged oral rehydration formulations. This is a serious constraint so it is important to find an alternative.[2]

Patients and methods

Forty young children, 20 in each group, with history of acute watery diarrhoea and signs of moderate to severe dehydration[3] were studied in a double-blind trial. Each child was weighed and randomly assigned to one of two treatment groups. Clinical features and nutritional status are given in Table 1. Although the children in the control group were younger, had been vomiting for longer, and had had diarrhoea for longer, these differences were not statistically significant.

Children in the acetate group were given an oral solution containing sodium 90 mmol/l, potassium 20 mmol/l, chloride 80 mmol/l, acetate 30 mmol/l, and glucose 111 mmol/l. Children in the control group were given the formula recommended by the WHO which contains 30 mmol/l bicarbonate and no acetate; in all other respects it was the same. Patients presenting with signs of hypovolaemic shock (5 in acetate group and 9 in control) were given intravenous Ringer's lactate for between 1 and 2 hours (average dose 42 and 39 ml/kg respectively). Each patient was given oral tetracycline.[4]

Table 1 *Presenting feature, nutritional status, and aetiological agents*

	Acetate group (n=20)	*Control group (n=20)*
Age (month)	21·0	14·5
Median and range	(4·5–60)	(2·5–72)
Sex M:F	11:9	12:8
Mean duration (hours) of diarrhoea before admission (±SE)	17·8±3·6	26·5±5·5
Mean duration of vomiting (hours) ± SE	11·5±2·9	14·2±2·9
History of anuria (>6 hours)	9	11
Weight on recovery for age as % of Harvard median		
⩾80% (normal)	1	4
70–79%	3	4
60–69%	9	7
<60%	7	5
Enteropathogens		
V. cholerae	4	5
Enterotoxigenic *E. coli*	12	10
Heat labile and heat stable	6	6
Heat labile	3	1
Heat stable	3	3
Rotavirus	1	6
Combined infections		
V. cholerae + enterotoxigenic *E. coli*	2	1
Rotavirus+enterotoxigenic *E. coli*	1	3
Total number in whom one or more pathogen detected	14 (70%)	17 (85%)

Mothers were encouraged to feed the rehydration solution *ad lib* by cup and spoon or directly from a cup. Rehydration solution intake and stool output were measured. Breast feeding (47% were breast fed) was allowed from the beginning and both bottle- and breast-fed babies were offered dilute milk with added rice cereal while older children were offered solids within 8 to 24 hours of starting treatment. Plain water was offered with the food.

Laboratory investigations. Blood samples were analysed for haematocrit, plasma specific gravity, serum sodium, and potassium (by methods previously

described),[4] plasma CO_2 (by Natelson's micro gasometer), and chloride (by Buchler-Cotlove chloridometer), at the time of admission and at 6, 24, and 72 hours. Stool samples were cultured for *Vibrio cholerae*, enterotoxigenic *Escherichia coli*, *Salmonella* sp. and *Shigella* sp. *E. coli* colonies were tested for heat-labile and heat-stable enterotoxins.[5 6] Rotavirus antigen was detected in the stool by ELISA technique.[7] Statistical analysis was done using Student's *t* test.

Results

Enteropathogens could be isolated from nearly 78% of the patients (Table 1). All patients were successfully rehydrated orally (Tables 2 and 3). With the exception of one, all the patients were clinically cured as judged by passage of formed stool by 72 hours, the mean (± SE) duration of diarrhoea being 42·7 (± 3·4) hours in the acetate and 45·3 (± 6·2) hours in the control group. The only child (heat-labile enterotoxigenic *E. coli*) who still had diarrhoea on day 5 was in the control group. The oral rehydration solution intake and stool output were comparable (Table 2). Early feeding was well accepted and tolerated.[8]

Two patients in the control group and 5 in the acetate group developed puffiness of eye lids, some with swelling of the legs. But none had hypernatraemia and all recovered. There was no significant difference between the two groups in the rise of plasma CO_2 at 6, 24, 72 hours (Table 3). The serum potassium and chloride values were normal.

Table 2 *Clinical course, weight gain, oral rehydration solution intake, and stool output during therapy*

	Acetate group (n=20)	*Control group (n=20)*
Vomiting after admission		
Total number vomited	15	8
First urine voided within 24 hours	20	20
Mean weight gain (% over recovery weight ± SE)		
6 hours	4·7±0·6	4·2±0·5
24 hours	8·3±0·8	6·8±0·6
48 hours	9·6±0·9	7·6±0·6
72 hours	10·6±0·9	8·7±0·6
Purging rate ml/kg (mean±SE)		
First 24 hours	142±17	171±24
Total	223±55	285±78
Oral rehydration solution intake ml/kg (mean±SE)		
First 6 hours	138±10	141±14
First 24 hours	296±28	293±28
Total	401±50	474±78

Discussion

This study shows that acetate is as effective as bicarbonate in the correction of dehydration and acidosis caused by acute diarrhoea. Oral rehydration solution containing acetate is well tolerated and the child accepted it as willingly as the solution containing bicarbonate. The increase in cost of packets for replacing bicarbonate with acetate is slight and the longer shelf life, ease of packaging and tablet making should more than compensate for it. It is thought that brown discoloration and the formation of furfural compounds in oral rehydration containing bicarbonate salt packets do not substantially compromise efficacy but the patient is less willing to take it.

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Table 3 *Biochemical parameters (mean ± SE) during the study period*

	On admission (0 hour)	*6 hour*	*24 hour*	*72 hour*
Plasma specific gravity				
Acetate	1·028±0·001	1·024±0·001	1·022±0·001	1·021±0·001
Control	1·027±0·001	1·024±0·001	1·022±0·001	1·021±0·001
Haematocrit (%)				
Acetate	39·7±1·4	34·3±1·3	31·6±1·1	31·3±0·8
Control	41·8±1·4	35·5±1·3	33·5±0·7	33·2±1·1
Plasma CO_2 (mmol/l)				
Acetate	15·4±1·0	17·5±1·1	20·7±0·9	24·1±1·2
Control	14·0±0·8	18·4±1·3	20·8±0·8	24·0±1·9
Serum sodium (mmol/l)				
Acetate	126·8±1·6	126·8±1·1	127·7±1·7	127·8±1·7
Control	126·9±1·5	127·3±2·5	128·3±1·7	129·2±2·4

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Amniocentesis and fetal lung development

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SUMMARY The crying vital capacity was measured in 10 healthy, term babies after amniocentesis during pregnancy, and in 10 control babies after normal pregnancies. The mean ratio of crying vital capacity to body weight was significantly lower after amniocentesis. This may be caused by the alteration in the lung fluid volume and the diminished intrauterine respiratory movements.

Oligohydramnios secondary to lack of urine production is thought to be responsible for lung hypoplasia in Potter's syndrome. Perlman *et al.*[1] however, have reported similar effects on lung growth resulting from chronic leakage of amniotic fluid, not related to renal agenesis. The use of amniocentesis performed for antenatal diagnosis of genetic or neural tube defects has become common. This is generally carried out in the midtrimester. The volume of amniotic fluid at 16 weeks is estimated to be between 130 and 170 ml. No ill effects on the fetus have been observed with removal of between 20 and 45 ml of amniotic fluid at 16 weeks, but the procedure produces a pronounced alteration in the fetal movements and a significant decline in the fetal breathing at 24 and 48 hours after amniocentesis.[2] Studies on animals have shown that amniocentesis performed in the early fetal period can cause lung hypoplasia.[3] The British study in 1978[4] showed a 1% increase in severe, unexplained, respiratory distress at birth after amniocentesis, being particularly pronounced between 34 and 37 weeks' gestation. The corresponding American study also revealed this, although the figures were not statistically significant. We therefore decided to study healthy, term neonates delivered to mothers subjected to amniocentesis to assess its effect on lung growth.

Method

A face mask attached to a pneumotachograph was used for measuring the crying vital capacity. The rim of the rubber mask was coated with soft paraffin to prevent leaks. It was placed over the baby's face enclosing the nose and the mouth. The baby was then stimulated to cry, into the face mask, by flicking the soles of its feet.

The flow signals were transmitted to a differential transducer and recorded on to a 4-channel FM recorder. A total of four to five bursts of crying was recorded. Volume was obtained by electronic integration of flow signals. At the end of the investigation volume was calibrated by injecting a known volume of air across the pneumotachograph, using a syringe. The output of the tape recorder was fed to an oscillograph and the record was obtained on ultraviolet-sensitive paper. The whole record was examined for the maximal deflection in a single cry and this was designated the crying vital capacity. Chest circumference was measured at the level of the nipple at the end of quiet expiration.

Subjects

We studied 10 healthy, term babies whose mothers had been subjected to amniocentesis. Six mothers had had amniocentesis carried out because of raised maternal serum alpha-fetoprotein levels noted on routine screening. The remaining 4 had been offered amniocentesis because of their age, 35 years or older.