Role of Soy-Based, Lactose-free Formula During Treatment of Acute Diarrhea

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ABSTRACT. A controlled study was conducted comparing the standard method of treating hospitalized infants with acute diarrhea (limited starvation) with the initiation of "early feeding" using a soy-based, lactose-free formula in infants of an American Indian tribe 12 months of age or younger. Forty-three patients, randomly assigned to group A, were given a soy-based, lactose-free formula four hours after hospitalization, and 44 patients, randomly assigned to group B, received standard therapy (food was withheld for the first 48 hours of hospitalization). After the first 48 hours, the same soy-based, lactose-free formula was given to the group B patients. Fluid intake and output of stool, urine, and vomitus were measured until the diarrhea resolved. Overall, group A patients showed less mean stool output (121 ± 129 (SD) mL/kg) than group B patients (299 ± 319 mL/kg) ($P < .001$). Furthermore, the duration of illness was significantly shorter in group A patients (54 ± 28 hours *v* 93 ± 56 hours) ($P < .001$). It was concluded that soy-based, lactose-free formulas can be safely used during the acute phase of diarrheal illness in infants and that their use shortens the duration of illness and decreases stool output in comparison with standard therapy. *Pediatrics* 1985;76: 292–298; *feeding, diarrhea, soy-based formula.*

Diarrhea continues to be a leading cause of morbidity and mortality among children less than 5 years old in many parts of the world.[1] The standard practice for managing hospitalized infants with diarrhea is to rehydrate them orally or intravenously and to withhold feeding during the first 24 to 48 hours of therapy. The rationale given for withholding food is that secondary disaccharidase deficiency (such as sucrase and lactase) is often seen in children during diarrheal illness and that the introduction of food during diarrheal illness may aggravate the diarrhea. Other investigators have argued that, although stool output may increase by feeding infants with diarrhea, there is a net increase in absorption of nutrients when infants are fed.[2] Despite those statements there are limited data comparing different forms of feeding during the acute phase of diarrheal illness.[2]

In a previous pilot study (unpublished data), we evaluated the safety and efficacy of using a lactose-containing formula during the acute phase of diarrheal illness. This regimen was compared with one of two other treatment schedules: (1) withholding food during the acute phase of diarrheal illness or (2) introducing a soy-based, lactose-free formula. We found that two of the 21 patients given the lactose-containing formula experienced explosive diarrhea during the first 24 hours of the therapy. Similarly, Torres-Pinedo et al[3] also demonstrated that stool output increased when infants were given a lactose-containing formula.

Based on these findings, we did not use the lactose-containing formula in our present study. Instead, a soy-based, lactose-free formula was fed to infants during the acute phase of their diarrheal illness.

MATERIALS AND METHODS

Patient Population

Patients aged 0 to 12 months with acute (less than seven days duration) watery diarrhea (at least

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five watery stools per day) hospitalized at the Indian Health Service Hospital, Whiteriver, Arizona, were randomly assigned (by using block randomization with groups of four) to one of two groups (A or B) after informed consent had been obtained from their parents. The decision to hospitalize patients was made by the primary care physicians and was independent of the investigators' judgments. The study extended from September 1982 to Dec 31, 1983.

Rehydration

After obtaining a standard history and performing a physical examination, we made a clinical assessment of the patient's degree of dehydration using standard criteria.[4] Severely dehydrated (>9% of body weight) patients were given Ringer's lactate (40 mL/kg/h) until blood pressure and pulse rate returned to normal. Following this, rehydration was completed within four hours by administering the oral rehydration solution recommended by the World Health Organization (WHO-ORS). If patients were minimally, mildly, or moderately dehydrated, only WHO-ORS was administered for a four-hour period. In these patients, the calculated deficit was replaced by giving the following volumes of ORS: minimal dehydration (<5%), 25 mL/kg; mild dehydration (5% to 6%), 60 mL/kg; and moderate dehydration (7% to 9%), 80 mL/kg.

Feeding Regimen

After the first four hours of hospitalization, patients were fed according to the feeding protocol of their assigned group.

Group A patients were given full-strength Isomil (150 mL/kg/24 h), a soy-based, lactose-free infant formula produced by Ross Laboratories, until their discharge from the hospital. The caloric content of Isomil is 67 calories per deciliter, and its carbohydrate source is sucrose and corn syrup.

Group B patients received WHO-ORS and water (each at 75 mL/kg/24 h) for the first 48 hours of hospitalization. After the initial 48-hour period, half-strength Isomil was given for an additional 24 hours. After this period, full-strength Isomil was given until the patients were discharged from the hospital.

In both groups, ongoing stool loss for each patient was replaced on a 1:1 basis with the WHO-ORS. The ORS was discontinued as soon as diarrhea stopped (no watery stools during a continuous 16-hour period). Duration of diarrhea was defined as the duration in hours from time of admission to the last diarrheal stool.

Intake and Output Measurements

All oral and intravenous intakes were measured and recorded until the diarrhea resolved. Stool output was measured using wet and dry diaper weights. Urine output was measured separately from stools in male patients using urine bags. Volume of vomitus was estimated using wet and dry linen weights.

Total body weights were obtained at admission, eight hours after admission, 24 hours after admission, and every 24 hours thereafter until discharge. Total body weight was also obtained 2 weeks after the patients were discharged from the hospital.

Antibiotic Therapy

Antibiotic therapy was used during hospitalization if the patients were suspected of having a complicating bacterial illness in addition to diarrhea. Antibiotic therapy was discontinued if bacterial cultures were subsequently negative. Also, if the patients' stool cultures confirmed the presence of *Shigella* organism, appropriate antibiotics were administered.

Laboratory Studies

At the time of hospitalization, specimens were obtained from each patient as follows:

1. Stool was aspirated using a rectal catheter. One aliquot of stool was used to identify bacterial pathogens by standard laboratory rechniques.[5] The presence of enterotoxigenic *Escherichia coli* was confirmed by using the DNA hybridization technique.[6] A second aliquot was stored in 10% phosphate-buffered saline for identification by enzyme-linked immunosorbent assay of rotavirus antigen,[7] enteric-type adenovirus antigen,[8] and *Clostridium difficile* toxin.[9] A third aliquot was used for parasitologic examination, a fourth aliquot to identify reducing substances in the stool (using Clinitest tablets), and a fifth aliquot to measure the pH using a pH meter.
2. Blood was obtained for determination of hematocrit, total WBC count with differential, and serum concentrations of sodium, potassium, chloride, bicarbonate, and total protein.

Twenty-four hours after admission and at discharge, hematocrit determinations and serum concentrations of total protein, sodium, potassium, chloride, bicarbonate, and BUN were repeated.

Stool pH and reducing substance tests were repeated every eight hours until diarrhea stopped.

Treatment Failures

A patient was considered to be a treatment failure if his or her stool output exceeded 80 mL/kg of

body weight in any eight-hour period after the first eight hours. A patient was also considered to be a treatment failure if he or she had persistent vomiting (more than three times in an eight-hour period), thus necessitating intravenous therapy, or if his or her diarrheal illness continued for more than seven days. Patients who were considered treatment failures were removed from the study and the primary physician then managed the treatment according to his or her clinical judgment.

Statistical Methods

χ^2 analysis was used on nominal data except that for which Fisher's exact analysis was indicated (the exact number in any cell was less than five). A two-tailed t-test was performed on interval data with normal distributions and equal variances and modified appropriately for unequal variances. The Wilcoxon non-parametric test was used when the distribution was not normal.

RESULTS

Responses to Treatment

Eighty-nine patients were initially enrolled into the study with 44 randomly assigned to group A and 45 to group B. However, one person in each group was eliminated from the study because food other than that allowed in the study protocol had been given to them. The admission characteristics of the remaining 43 patients in group A and the 44 patients in group B are shown in Table 1. None of the differences between the groups was statistically significant. All patients were determined to be well nourished; all had weight and height above the third percentile according to the standards published by

TABLE 1. Admission Characteristics of Patients with Diarrhea Receiving "Early Feeding" with Soy Formula (Group A) and Patients Receiving "Standard Therapy" (Group B)*

	Group A (N = 43)	Group B (N = 44)
Sex (M/F)	24/19 (56%/44%)	26/18 (59%/41%)
Age (mo)	5.0 ± 3.3	5.4 ± 3.4
No. receiving formula feeding only	34 (79%)	35 (80%)
No. receiving formula feeding and baby food	9 (21%)	9 (20%)
No. of days of diarrhea before admission	3.1 ± 2.2	2.5 ± 1.5
No. with history of vomiting prior to admission	20 (47%)	18 (41%)
Temperature (°C) on admission	38.3 ± 1.6	38.3 ± 0.8
No. receiving antibiotics prior to therapy	9 (21%)	8 (18%)
Hematocrit (%)	39.3 ± 5.2	36.6 ± 5.4
Total protein (g)	7.0 ± 0.7	6.5 ± 1.2
Glucose (mg/dL)	81 ± 12	80 ± 23
BUN (mg/dL)	13 ± 6	12 ± 8
Potassium (mmol/L)	4.2 ± 0.7	4.2 ± 1.0
Sodium (mmol/L)	136 ± 5	137 ± 6
Chloride (mmol/L)	109 ± 7	106 ± 7
Bicarbonate (mmol/L)	15 ± 3	17 ± 5
Admission weight (kg)	6.4 ± 2.0	6.1 ± 1.8
Estimated degree of dehydration		
Minimal (<5%)	28 (65%)	26 (59%)
Mild (5%–6%)	8 (19%)	8 (18%)
Moderate (7%–9%)	5 (11%)	7 (16%)
Severe (<9%)	2 (5%)	3 (7%)
Pathogens† identified in stools		
Rotavirus	9/32‡ (28%)	3/26 (12%)
Enteric adenovirus	1/32 (3%)	3/26 (12%)
Shigella	1/40 (3%)	1/37 (3%)
Campylobacter	0	1/37 (3%)
Salmonella	0	0
Enterotoxigenic *Escherichia coli* (ETEC)	7/26 (27%)	5/27 (19%)
Heat-stable enterotoxin	2	3
Heat-labile enterotoxin	3	1
Both enterotoxins	2	1

* Values are means ± SD. Differences were not statistically significant.
† Bacterial cultures were performed for 77 patients (89%); findings were positive for pathogen in 15 (20%). Enzyme-linked immunosorbent assay (ELISA) for rotavirus and adenovirus was performed for 58 patients (67%); findings were positive for 16 (28%).
‡ Values are number positive/number tested.

the US National Center for Health Statistics, 1976. The amounts of fluid intake for both groups during illness are shown in Table 2. Group A patients drank significantly less ORS than group B patients during the first 24 hours and 48 hours after admission as well as by the time illness resolved. The total fluid intake was also significantly less in group A than in group B.

The stool output at the end of 24 hours, 48 hours, and at resolution was noted to be significantly less (by Wilcoxon nonparametric analysis) in group A patients than in group B patients (P values of .049, .018, and .001, respectively). When stool output was analyzed for only the male patients (for whom U-bags facilitated separation of urine and stool output measurement), infants in group A had less stool output than infants in group B at 24 hours, 48 hours, and at resolution. However, these differences were only statistically significant at resolution (P = .049). The female patients in group A also had less stool output in all periods; this was significant at 48 hours and at resolution. Within each group there was no significant difference in stool output between male and female patients. The mean duration of diarrhea was considerably less in group A compared with group B (54 hours and 93 hours, respectively; $P < .001$). The mean duration of diarrhea of male patients in group A compared with those in group B was also significantly less (54 hours *v* 87 hours, respectively; $P < .02$). The percent of weight gained (Table 3) was similar in the two groups at the end of 24 and 48 hours, at resolution, and 2 weeks after discharge.

None of the patients was either hyponatremic (serum sodium < 130 mmol/L) or hypernatremic (serum sodium > 150 mmol/L) at admission, 24 hours after therapy, and at discharge. None remained acidotic (serum bicarbonate < 15 mmol/L) after the first 24 hours of therapy.

Antibiotic therapy was administered during hospitalization to 21 (49%) patients in group A and 24 (55%) patients in group B (χ^2 analysis: P = NS) for one of the following reasons: otitis media (11 patients in group A and 18 in group B); pneumonia (four patients in group A and two in group B) and suspected sepsis (five patients in group A and three in group B). In addition, one patient in group A and one in group B were treated with trimethoprim-sulfamethoxazole when their stool cultures revealed the presence of *Shigella* organism. Stool output, however, had already decreased considerably in both patients before they were given trimethoprim-sulfamethoxazole.

Treatment Failures

Three (7%) of the patients in group A and seven (16%) of the patients in group B were considered to be "treatment failures" (this difference was not statistically significant using Fisher's exact analysis).

The reasons for which patients were considered to be treatment failures were as follows:

1. Three patients required unscheduled intrave-

TABLE 2. Intake of Fluids During Illness*

	Group A (mL/kg)	Group B (mL/kg)	P Value‖
Oral rehydration solution (ORS) intake first 24 h	75 ± 33	148 ± 98	<.001
Water intake first 24 h	22 ± 18	55 ± 34	...
Full-strength soy formula intake first 24 h	90 ± 32	...	...
Total intake of fluids first 24 h†	179 ± 52	211 ± 95	.306
ORS intake first 48 h‡	241 ± 520	415 ± 394	<.001
Water intake first 48 h	...	148 ± 69	...
Full-strength soy formula intake first 48 h	211 ± 59	...	...
Total intake of fluids first 48 h	380 ± 103	500 ± 211	.010
Total intake of ORS§ during illness	159 ± 144	449 ± 343	<.001
Total intake of water during illness	...	174 ± 104	...
Total intake of full-strength soy formula during illness	241 ± 158	236 ± 297	.094
Total intake of half-strength soy formula during illness	...	114 ± 76	...
Total intake of all fluids during illness	422 ± 294	896 ± 580	<.001

* Values are means ± SE.
† Patients with severe dehydration were also given intravenous fluids during first four hours.
‡ Only patients requiring ORS after 24 hours of therapy included (24 patients in group A and 28 in group B).
§ All patients were included regardless of length of illness.
‖ Wilcoxon nonparametric test.

TABLE 3. Stool Output, Duration of Diarrhea, and Weight Gain*

	Group A	Group B	*P* Value
Stool output first 24 h (mL/kg)	45 ± 25	78 ± 90	.049†
Males	47 ± 28	81 ± 101	.240†
Females	36 ± 23	70 ± 59	.090‡
Stool output first 48 h (mL/kg)	85 ± 63	150 ± 167	.018†
Males	94 ± 73	153 ± 189	.150†
Females	71 ± 46	146 ± 106	.045‡
Total stool output during illness (mL/kg)	121 ± 129	299 ± 319	.001†
Males	142 ± 156	294 ± 355	.048†
Females	89 ± 67	310 ± 227	.009‡
% weight gain§			
24 h after therapy	1.8 ± 3.6	2.4 ± 3.2	.559†
48 h after therapy	1.3 ± 4.5	2.8 ± 3.6	.183‡
At resolution of illness	3 ± 4	1.9 ± 4.3	.219‡
% weight gain‖ 2 wk after discharge	6.8 ± 10.3	6.2 ± 8.0	.807‡
Duration of diarrhea	54 ± 28	93 ± 56	<.001†
Males	54 ± 31	87 ± 59	.020†
Females	55 ± 24	105 ± 48	.006‡

* Values are means ± SD.
† Wilcoxon nonparametric test.
‡ *t* test.
§ Calculated by using the following formula: percent weight gain = (weight at specified interval minus admission weight) divided by admission weight and multiplied by 100.
‖ Calculated by using the following formula: percent weight gain = (weight 2 weeks after discharge minus weight at resolution of illness) divided by weight at resolution of illness multiplied by 100.

nous hydration. One patient in group A and one in group B required intravenous therapy because of continued high stool output (10.5 mL/kg/h and 12.6 mL/kg/h, respectively) by the third day of therapy. One group B patient, while receiving soy-formula, required intravenous therapy because of persistent vomiting 88 hours after hospitalization.

2. Seven patients (two patients in group A and five in group B) had continuous diarrhea for more than seven days. Diarrhea resolved in three patients (one patient in group A and two in group B) between the eighth and ninth day of therapy while the patients were receiving the soy-based, lactose-free formula. The other four patients who were considered to be treatment failures were switched to a hydrolysate formula (Pregestimil, Mead Johnson); in all patients, the diarrhea resolved within two to four days.

None of the patients with prolonged diarrhea had persistently low pH (<6) nor detectable levels of reducing substances in stools. However, all patients who successfully completed treatment and those who were considered to be treatment failures due to persistent vomiting or prolonged diarrhea had occasional stools with a pH below 6 and low concentrations of reducing substances (0.25% to 0.50%).

DISCUSSION

Controversies about the role of feeding during the acute phase of diarrheal illness have existed for the past six decades,[2] Park[10] in 1924 and Chung and Viscorova[11] in 1948 argued that feeding should be continued during the course of diarrheal illness, because there is a net increase in absorption of nutrients when feeding is introduced despite the fact that stool output may increase. In recent years, pediatricians have been concerned about the transient deficiencies of the disaccharidases, especially of lactase, that frequently accompany acute diarrhea.[12] This fact, coupled with the anecdotal observations that have suggested that stool output decreases when patients are starved during diarrheal illness, has discouraged pediatricians from using feeding in treatment of patients during the acute phase of the illness. One of the reasons for these conflicting opinions is the limited data available from controlled studies comparing different forms of feeding. The practice of withholding foods from infants with each diarrheal illness may not impact greatly on the nutritional status of well-nourished children from affluent countries. However, this practice could have a substantial negative impact on growth development in undernourished infants living in developing countries; such infants may experience several episodes of diarrhea in 1 year.[13,14]

Our study indicates that introduction of a soy-based, lactose-free formula during diarrheal illness is not only safe but offers a distinct advantage over the standard practice of starving patients. In group A patients, who were given soy formula after the initial four hours of rehydration, there was a reduc-

tion in stool output of more than 50%, and the duration of diarrhea was likewise reduced by more than 40%. We were not able to detect any difference in weight gain between the groups. This was probably because all of our patients were relatively well nourished, and the nutritional injury to the patients in the control group was minimal.

The improved outcome in the formula-fed group was probably the result of enhanced absorption of fluids, electrolytes, and nutrients from the gut. This, in turn, may have resulted from the hydrolytic products of protein (soy) and carbohydrates (corn syrup solids and sucrose) contained in the formula. These hydrolytic products, such as amino acids, dipeptides, and tripeptides may have potentiated the absorption of water and electrolytes from the gut.

Recent studies have shown that adding glycine to the standard WHO-ORS reduces stool output by 50% when this solution is administered to infants with diarrhea.[15] The same effect has also been noted when rice powder is used instead of glucose in the WHO-ORS.[15] Corn syrup solids contain polymers of glucose in the form of amylose and amylopectin, which are hydrolyzed by pancreatic and salivary amylase to smaller saccharides. These saccharides and sucrose are effectively hydrolyzed by brush border enzymes, sucrase, isomaltase, and glucoamylase. These hydrolyzed monosaccharides may also play a role in enhancing absorption of sodium, chloride, and water from the gut. This mechanism, however, less likely explains the improved outcome of formula-fed infants as maximal absorption of water and sodium occurs when ORS contains 2% to 3% glucose.[16] The WHO-ORS used in this study has a glucose concentration of 2%. Finally, the continued feeding of formula may also have prevented the reduction of mucosal disaccharidase levels which can occur with prolonged starvation.[17]

Other studies from different areas of the world have also found that it is safe to introduce soy-based, lactose-free formulas during diarrheal illness. Leake et al[18] compared the effect of introducing soy-based, lactose-free formula with the effect of introducing lactose-based formula after a period of fasting in patients with diarrhea. Of the group given soy-based, lactose-free formula, 91% successfully completed therapy while only 36% of the group given a lactose-containing formula successfully completed therapy. However, Leake et al did not standardize the initial period of fasting in their patients.

In a study in Nigeria, Noah[19] introduced either a lactose-containing formula or a soy-based, lactose-free formula to children after withholding food for 24 hours. No difference in duration of diarrhea was observed between the groups. In developing countries, soy-based formula may either not be available or may be too expensive. Under such circumstances, the use of dilute cow's milk and/or a variety of locally available foods should be evaluated. Both in the developed and developing countries, breast-feeding infants should be encouraged to continue drinking breast milk during diarrheal illness.

The present study confirms observations previously made by us[5] and by others[20] that infants with diarrhea frequently required intravenous therapy when their stool output exceeded 10 mL/kg of body weight per hour. The study patient in group B who had persistent vomiting along with the four patients who were switched to a non-soy-based formula on the eighth day of therapy all may have had an intolerance to soy protein. Alternatively, if treatment had been allowed to continue using the soy-based, lactose-free formula, all may have resolved their illnesses. We did not find either stool pH or the presence of reducing substances in the stool helpful in predicting the success or failure of therapy.

We conclude that: (1) soy-based, lactose-free formulas can be safely introduced during a diarrheal illness, and, (2) the introduction of a soy-based, lactose-free formula during diarrheal illness shortens the duration of diarrhea and decreases stool output considerably.

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REFERENCES

1. Puffer RR, Serrano CV: Patterns of mortality in childhood, in *Report of the Inter-American Investigation of Mortality in Childhood*, Pan American Health Organization Scientific Publication No. 262, 1973
2. Brown KH, MacLean WC Jr: Nutritional management of acute diarrhea: An appraisal of the alternatives. *Pediatrics*

1984;73:119
3. Torres-Pinedo R, Lavastikda M, Rivera CL, et al: Studies on infant diarrhea: I. A comparison of the effects of milk feeding and intravenous therapy upon the composition and volume of the stool and urine. *J Clin Invest* 1966;4:469
4. Robson AM: Parenteral fluid therapy, in Vaughan VC III, McKay RJ Jr, Behrman RE (eds): *Nelson Textbook of Pediatrics*, ed 11. Philadelphia, WB Saunders Co, 1979, pp 284–306
5. Santosham M, Daum R, Dillman L, et al: Oral rehydration therapy of infantile diarrhea: A controlled study of well-nourished children hospitalized in the United States and Panama. *N Engl J Med* 1982;306:1070
6. Moseley SL, Echeverria P, Seriwatana, et al: Identification of enterotoxigenic *Escherichia coli* by colony hybridization using three enterotoxin gene probes. *J Infect Dis* 1982; 145:863
7. Yolken RH, Stopa PJ, Harris CC: Enzyme immunoassay for the detection of rotavirus antigen antibody, in *Manual of Clinical Immunology*, ed 2. Washington, DC, American Society for Microbiology, 1980, Chap 94, p 692
8. Yolken R, Lawrence F, Leister F, et al: Gastroenteritis associated with enteric type adenovirus in hospitalized infants. *J Pediatr* 1982;101:21
9. Yolken RH, Bartlett JG, Laughon B, et al: Enzyme-linked immunoassay for detection of *Clostridium difficile* toxins A and B in fecal specimens. *J Infect Dis*, in press 1985
10. Park EA: New viewpoints in infant feeding. *Proc Conn State Med Soc* 1924;20:190
11. Chung AW, Viscorova B: The effect of early oral feeding versus early oral starvation on the course of infantile diarrhea. *J Pediatr* 1948;33:14
12. Brown KH, Black RE, Parry L: The effect of acute diarrhea on the incidence of lactose malabsorption among Bangladesh children. *Am J Clin Nutr* 1980;33:2226
13. Black RE, Brown KH, Becker S, et al: Longitudinal studies of infectious disease and physical growth of children in rural Bangladesh: I. Patterns of morbidity. *Am J Epidemiol* 1982;115:3052
14. Black RE, Brown KH, Becker S, et al: Longitudinal studies of infectious disease and physical growth of children in rural Bangladesh: II. Incidence of diarrhea and association with known pathogens. *Am J Epidemiol* 1982;115:315
15. Mahalanabis D, Patra FC: In search of a super oral rehydration solution: Can optimum use of organism solute-mediated sodium absorption lead to the development of an absorption promoting drug? *J Diar Dis Res* 1983;1:76
16. Sladen GE, Dawson AM: Inter-relationship between the absorption of glucose, sodium and water by the normal human jejunum. *Clin Sci* 1969;36:119
17. Knudsen KH, Bradley EM, Lecocq FR, et al: Effect of fasting and refeeding on the history and disaccharidase activity of the human intestine. *Gastroenterology* 1968;55:46
18. Leake RD, Schroeder KC, Benton DA, et al: Soy-based formula in the treatment of infantile diarrhea. *Am J Dis Child* 1974;127:374
19. Noah MA: Soy-based formula for infantile diarrhoea: A controlled trial. *Niger Med J* 1979;9:243
20. Sack DA, Islam S, Brown KH, et al: Oral therapy in children with cholera: A comparison of sucrose and glucose electrolyte solutions. *J Pediatr* 1980;96:20

ERRATA

In the article "Adolescents' Self-Assessment of Compliance Behavior" by Litt (*Pediatrics* 1985;75:693–696), there is an error in Table 4. The first two side entries are reversed: "Compliant" should read "Noncompliant" and vice versa.

In the article, "Single-Dose Versus Conventional Therapy of Urinary Tract Infections in Female Adolescents" by Fine and Jacobson (*Pediatrics* 1985;75:916–920), the last sentence of the first paragraph should read: Although adolescents have been included in the study samples of many therapeutic trials, their age-specific response to therapy has never been isolated, and no current guidelines exist regarding single-dose treatment in the adolescent population.

results indicate that more use of the scheme will be made for infant seats than child seats. Once capital costs have been funded the scheme is financially self-supporting.

Our intention is to present these findings to the government with a recommendation that the various other schemes in existence around New Zealand be expanded to cover the total newborn population.

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REFERENCES

1. Pless IB: Accident prevention and health education: Back to the drawing board. *Pediatrics* 1978;62:431–435
2. Geddis DC: Children in cars: Results of an observational study in New Zealand. *NZ Med J* 1979;90:468–471
3. Geddis DC: How children travel in cars in New Zealand. *NZ Med J* 1982;95:740–742
4. Geddis DC, Pettengell R: Parent education: The effect on the way children are transported in cars. *NZ Med J* 1982;95:314–316
5. Geddis DC, Spears GF: Why parents do not provide automobile restraints for their children. *Aust Paediatr J* 1980;16:114–116
6. Elley, WB, Irving JC: Revised socioeconomic index for New Zealand. *NZ J Educ Stud* 1976;11:25–29
7. Reisinger KS, Williams AF: Evaluation of programs designed to increase the protection of infants in cars. *Pediatrics* 1978;62:280–287
8. Cullen C, Cunningham JL: *Compliance with the Child Passenger Protection Law: Effect of a Loaner Program for Low Income Mothers*, US Department of Transportation Technical Report DOT HS805 801. Nashville, TN, National Technical Information Service, 1980
9. Hletko PJ, Hletko JD, Nyberg J, et al: The Effect of an In-Hospital Maternity Education Program on Observed Correct Crash Restraint Device Use. Proceedings of American Association for Automotive Medicine Meeting, Ottawa, 1983, pp 168–170
10. Colletti RB: Hospital-based rental programs to increase car seat usage. *Pediatrics* 1983;71:771–773
11. Hletko PS, Hletko JD, Nyberg Jm, et al: The Effect of a Toddler Child Restraint Device Rental Program on Observed Correct Use. Proceedings of American Association for Automotive Medicine, San Antonio, TX, 1983, pp 86–88
12. Geddis DC, Appleton IC: Use of restraint systems by preschool children in cars. *Arch Dis Child* 1982;57:549–550
13. Klein D: The role of the medical profession, in *Prospects for the Reduction of Road Losses due to Vehicle Crash in New Zealand*, Dunedin, NZ, Medical Research Council of New Zealand, Special Report Series No. 1, 1973, pp 41–49

ERRATA

In the article, "Role of Soy-Based, Lactose-free Formula During Treatment of Acute Diarrhea," by Santosham et al. (*Pediatrics* 1985;76:292–298), Wyeth Laboratories was acknowledged as having supported the study; they did not.

In the second paragraph of the right-hand column on p 309 of Dr St Geme's commentary (*Pediatrics* 1985;76:308–310), the fourth sentence should read: The American Board of Pediatrics defines a pediatric *specialist* (not *sub*-specialist, as originally printed) as one capable of "management . . . any medical and health maintenance problems . . . all common problems of disease . . . for all medically life-threatening situations."

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