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ORAL SUCROSE THERAPY FOR DIARRHŒA

SIR,—The successful maintenance of hydration in fifteen of eighteen patients with cholera and related diarrhœas using sucrose-electrolyte solution [1] represents an important advance in the prevention of diarrhœal death, especially in rural areas of the developing world. In Indonesia, not only is the price of glucose ten or more times that of sucrose, but, more importantly, it is available only in a few large chemical supply houses located in large urban centres. Sucrose, on the other hand, is found in every home.

We have compared the efficacy of glucose and sucrose electrolyte solution in a double-blind study of rehydration and maintenance of children who had been admitted to hospital with mild to moderate dehydration due to diarrhœa. Because initial results (I) indicated that the sugar concentration of 3% resulted in incomplete sugar absorption, composition was changed for trial II (see accompanying table).

BALANCE DATA DURING REHYDRATION AND MAINTENANCE WITH ORAL SUGAR-ELECTROLYTE SOLUTIONS

Oral therapy group*	n	Mean body-weight (kg)†	Mean stool output (l)†	Net fluid balance (l)†	No. with reducing substances > 250 mg/dl	Required I.V. fluids
Glucose I	10	7·32 (0·44)	0·65 (0·16)	1·55 (0·16)	4	1
Sucrose I	8	7·52 (0·39)	1·11 (0·50)	1·22 (0·16)	4	2
Glucose II	7	7·50 (0·57)	0·42 (0·10)	1·52 (0·26)	1	1
Sucrose II	8	8·28 (0·53)	0·66 (0·16)	1·76 (0·19)	5	1

* Solution composition (g/l):

	Sodium chloride	*Sodium bicarbonate*	*Potassium chloride*	*Glucose or sucrose*
I	2·0	2·0	1·0	30
II	3·5	2·5	1·5	20

† S.E.M. in parentheses.

Patients who failed to produce urine, who continued in negative fluid balance because of large stool losses, or who continued to have objective signs of dehydration (rapid weak pulse, poor skin turgor, or urine specific gravity above 1·028) for over six hours were given intravenous fluids and were considered failures of oral treatment. The results of rehydration of 33 children (see table) clearly indicate that there is little difference between sucrose and glucose containing electrolyte solutions in the treatment of dehydration due to childhood diarrhœa. None of these patients had cholera but all were deemed sufficiently dehydrated to require admission to hospital. The greater stool volume and larger number of patients demonstrating incomplete absorption of sugar in the sucrose group support the theoretical preference for glucose. However, the early urination and continuation of positive fluid balance in the face of continued substantial diarrhœal losses in both groups show that sucrose is almost as effective as glucose in enhancing gut fluid and electrolyte absorption and can act as a substitute where glucose is not readily available.

The failure of three of Nalin's eighteen patients to maintain positive fluid balance on the sucrose-electrolyte solution [1] is remarkably similar to the five of nineteen patients who required additional intravenous fluid in the initial report of oral glucose-electrolyte maintenance therapy for cholera by Nalin and colleagues in 1968.[2] Perhaps adjustment of the sucrose concentration downward as in our solution II will improve the results further by reduction in osmotic load.

On both theoretical and empirical grounds we agree with Nalin that the monosaccharide glucose is preferable to sucrose in oral therapy mixtures. We do not wish this preference to obscure the important finding that sucrose in low concentrations is effective in enhancing salt and fluid absorption in diarrhœal disease. The practical applications of this finding may be extensive in countering dehydration throughout much of the developing world.

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BLOOD-ALCOHOL AND SERUM OSMOLALITY

SIR,—In their instructive article (June 28, p. 1042) Dr Champion and others studied 565 acute trauma patients and confirmed the value of the readily available measurement of serum osmolality as an indicator of the blood-alcohol concentration. An even closer correlation should exist between the blood-alcohol concentration and the discrepancy between measured and calculated* serum osmolalities. Since the authors also measured the serum concentrations of sodium, urea, and glucose, they could easily determine this correlation. We would anticipate that they would thereby find a significant reduction in the incidence of misleading results, which they reported to be 21%. We look forward to the authors presenting this potentially superior correlation in the near future.

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* Strictly speaking, any formula using serum concentrations of sodium, urea, and glucose gives a result in terms of osmolarity (mosm/l of solution) rather than osmolality (mosm/kg of solvent).

AINHUM

SIR,—Your editorial (July 5, p. 19) and the letter from Dr Browne (July 19, p. 131) are of great interest. Ainhum continues to occur in the western hemisphere, and I have observed several cases in the Hospital das Clinicas of the Federal University of Bahia, Brazil, in the past ten years. Unbeknown to most of the medical world, the first complete clinical and pathological description of ainhum was provided by Dr Jose Francisco da Silva Lima in 1867.[1,2] He notes that "ainhum" was a Nagô African word meaning "to saw" and was used by the Black slaves in Bahia to describe this affliction of the fifth toe. In Bahia, as Silva Lima reported, this entity is still seen more often in males and more than one member of a family may be affected. The histological lesion of ainhum was first described by Otto Wucherer (of *Wuchereria bancrofti* fame) and included in this original publication.

As Dr Browne suggested, ainhum may be a *forme fruste* of a treponematosis. This particular idea was mentioned by Clarke in 1860 [3] on the basis of his observations in the Gold Coast, although he attached the term "dry gangrene" to the constriction of the proximal phalanx of the fifth toe. This ætiology, however, has not been closely examined.

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