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The use of Ringer's lactate in the treatment of children with cholera and acute noncholera diarrhoea

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Two regimens of fluid and electrolyte therapy were studied in children with severe cholera and noncholera diarrhoea. In one, lactated Ringer's solution was the sole intravenous fluid, additional water, glucose, and potassium being given by mouth. In the other, three different intravenous solutions were employed to meet all fluid and electrolyte requirements. The response to therapy was satisfactory with each regimen. Because of prolonged stupor or vomiting about 15 % of children treated by the first regimen were unable to ingest a sufficient quantity of glucose solution by mouth, and intravenous supplementation with a hypotonic glucose–saline solution was necessary. It is concluded that lactated Ringer's solution is suitable as the sole intravenous solution for children with acute cholera and noncholera diarrhoea provided oral supplementation, as described, is possible. The study also provides useful observations on the means of evaluating fluid requirements in such children and specific guidelines for such therapy.

Several successful treatment regimens for paediatric cholera have been described (Lindenbaum et al., 1966; Wallace & Oleinick, 1966; Gutman et al., 1969). Each requires the administration of special intravenous fluids. In many areas in which cholera is prevalent such solutions are unavailable, and the case-fatality rate from paediatric cholera in such areas is usually high. This report describes the use of a single, commercially available, intravenous solution, lactated Ringer's solution, in the treatment of paediatric cholera. The clinical and biochemical features of cholera and acute noncholera diarrhoea in children so treated are compared with those in a second group of children treated by a regimen based on recently reported metabolic balance studies of paediatric cholera in which three different intravenous solutions were used (Mahalanabis et al., 1970). The observations in this report also permit a useful comparison of several means by which fluid requirements and the adequacy of fluid replacement may be judged in children with cholera.

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METHODS

Selection of patients

The children selected for study were of either sex, were less than 6 years of age, gave a history of watery diarrhoea (which was usually of less than 24 hours duration), and presented physical signs of moderate or severe extracellular fluid loss requiring immediate rehydration. None had received parenteral fluids or antibiotics prior to selection. After a brief history had been taken and a physical examination made, each child was weighed nude and then placed on a metabolic bed. Paediatric urine collectors were employed to facilitate separate collections of urine and stool. Femoral artery blood was collected in a heparin-rinsed syringe, a stool specimen was obtained by a sterile rectal catheter, and rehydration was begun. Femoral artery blood was again obtained after 6, 12, 24, and 48 hours. Stool and urine outputs were weighed at 6-hour intervals. Rectal swabs were obtained for culture each day until discharge. Body

weight was determined after 6, 12, 24, and 48 hours, the children being weighed nude on a simple pan balance of local manufacture.

Treatment regimens

At admission, patients were assigned randomly to one of two treatment regimens (Table 1). The first treatment group (Group MS) was placed on a rehydration and maintenance regimen in which all water and electrolyte requirements were given intravenously. Three different intravenous solutions were utilized. Initial rehydration was with lactated Ringer's solution (Na 130, Cl 109, K 4, Ca 2.7, and lactate 28 mEq/litre), of which 20–30 ml were given per kg of body weight during the first hour to expand plasma volume and correct the base deficit. The remainder of the initial deficit was replaced with a hypotonic electrolyte solution in 5% glucose (Na 106, Cl 74, and HCO_3^- 32 mEq/litre), 60–80 ml/kg being given during the next 7 hours. The same solution was used to replace, on a volume-for-volume basis, all stool losses occurring after admission until diarrhoea ceased. The replacement of urinary and insensible losses was initiated 8 hours after admission by means of a third solution, containing 31 mEq of sodium chloride per litre in 5% glucose. This was given at a rate of 75 ml per kg of body weight per day.

The second treatment group (Group R) received a single intravenous solution, lactated Ringer's solution, to replace the initial isotonic fluid deficit, to correct the metabolic acidosis, and to replace any stool losses following admission. Rehydration was accomplished using 20–30 ml per kg of body weight

Table 1. Outline of treatment for the two groups

Treatment group	Solution	Volume (ml/kg)	Hour of treatment	Electrolytes replaced (mEq/kg)		
				Na^+	Cl^-	HCO_3^-
		INITIAL REHYDRATION				
MS	Ringer's lactate	20–30	0–1	2.6–3.9	2.2–3.3	0.6–0.8
	hypotonic electrolyte in 5 % glucose [a]	60–80	1–8	6.4–8.5	4.4–5.9	1.9–2.6
	totals	80–110		9.0–12.4	6.6–9.2	2.5–3.4
R	Ringer's lactate	20–30	0–1	2.6–3.9	2.2–3.3	0.6–0.8
	Ringer's lactate	50–70	1–8	6.5–9.1	5.5–7.6	1.4–2.0
	totals	70–100		9.1–13.0	7.7–10.9	2.0–2.8
		MAINTENANCE WATER REPLACEMENT				
MS	0.18 % NaCl in 5 % glucose (intravenously)	75	8–24, daily thereafter			
R	5 % glucose (orally)	90–120	8–24, daily thereafter			
		REPLACEMENT OF CONTINUING STOOL LOSSES [b]				
MS	hypotonic electrolyte in 5 % glucose [a]	equal to stool volume				
R	Ringer's lactate	75 ml per 100 ml of stool volume				

[a] Electrolyte content: Na^+ 106, Cl^- 74, HCO_3^- 32 mEq/litre.

[b] Each group received oral potassium hydrogen citrate, 4 mEq/kg/day, in 4 divided doses, until discharged.

during the first hour and 50–70 ml/kg during the next 7 hours. All stool losses following admission were replaced by giving 75 ml of lactated Ringer's solution for each 100 ml of stool, until diarrhoea ceased. Oral administration of a 5% solution of glucose was commenced in most cases within 5 hours after admission. This was given at a dosage of 90–120 ml per kg of body weight in an attempt to provide water for insensible and urinary losses and to replace approximately 25% of the volume of stool losses following admission.

Patients in both groups received oral potassium replacement at a dosage of 4 mEq kg of body weight per 24 hours in 4 divided doses in the form of potassium hydrogen citrate until discharge from the hospital. Oral administration of tetracycline was commenced in both groups about 6 hours after admission at a dose of 50 mg per kg per 24 hours in 4 divided doses. It was continued for 48 hours. Refeeding with a normal diet was begun in most cases after 24 hours.

Any patient in Group R who was unable to ingest liberal quantities of fluid by mouth safely because of prolonged vomiting or altered consciousness was given maintenance water requirements parenterally as described for Group MS, using hypotonic sodium chloride (31 mEq/litre) in 5% glucose. In children with persistent vomiting, oral replacement of water was attempted for up to 24 hours before a change was made to complete intravenous maintenance. Children with deep coma lasting longer than 8 hours were changed shortly thereafter to complete intravenous maintenance. Determinations of arterial-blood pH, plasma specific gravity, plasma concentrations of sodium, chloride, and potassium, and plasma osmolarity were performed as previously described (Mahalanabis et al., 1970). Whole blood standard bicarbonate concentration was determined by the Astrup equilibration technique, utilizing a Radiometer pH meter 27 and microtonometer. Stools and rectal swabs were cultured for enteric pathogens as previously described (Mahalanabis et al., 1970).

RESULTS

Fifty-three children were studied. Table 2 gives the numbers treated in each age group and by each treatment regimen. *Vibrio cholerae*, biotype El Tor, Ogawa serotype, was isolated from the stools of all children with cholera. With one exception, children with noncholera diarrhoea yielded no recognized enteropathogen on stool culture. A nonagglutinating vibrio was isolated from the stool of one patient in Group R.

Table 2. Number of patients treated according to age and treatment regimen

Age (years)	Cholera		Noncholera	
	MS	R [a]	MS	R [a]
0–2	5	6 (1)	4	1
2–4	8	5 (1)	3	2
4–6	8	6 (1)	3	2 (1)
total	21	17 (3)	10	5 (1)

[a] Numbers in parentheses indicate patients who were changed to complete intravenous maintenance for reasons given in the text.

Comparison of treatment groups at admission

Table 3 summarizes the historical, physical, and biochemical data obtained at the time of admission. There were no significant differences between treatment groups with regard to any of the compared characteristics. The predominant clinical picture was one of severe isotonic extracellular fluid deficit with associated metabolic acidosis.

Comparison of response to therapy

Children with cholera. Children in both treatment groups responded rapidly to the initiation of intravenous water and electrolyte replacement. Those patients with a feeble or impalpable radial pulse regained a normal pulse volume within 30–60 minutes. Systolic blood pressure was also normal, or near normal, in both groups after 1 hour. Rectal temperature uniformly returned to normal within 12 hours after admission. Skin turgor was normal in about 70% of the patients in both groups within 6 hours and in all but 2 patients in Group MS within 12 hours. Skin turgor in these 2 was normal after 24 hours. Most children in both groups first spontaneously voided urine between 12 and 24 hours after the beginning of treatment.

Children in both treatment groups who had a depressed sensorium at admission regained normal consciousness rather slowly. Nine of 10 children in Group R, admitted with a depressed sensorium, were responsive within 6 hours but remained lethargic until at least 12 hours after admission. One child remained in deep coma for 56 hours before recovering satisfactorily. In Group MS, 8 of 10 children

Table 3. Comparison of treatment groups at admission *

Characteristic	Cholera		Noncholera	
	MS	R	MS	R
number	21	17	10	5
age (years)	3.8 (0.5–6)	2.9 (0.4–5.5)	2.9 (0.2–6)	3.5 (2–4.2)
weight (kg)	8.8 (4.2–12.1)	8.5 (3.3–13.9)	9.3 (3.7–13.5)	7.7 (5.6–12.1)
duration of illness before entry (hours)	11 (4–36)	9 (3–30)	11 (5–25)	19 (6–36)
absent radial pulse (%)	33	53	40	20
stupor or coma (%)	43	53	50	80
moderate or marked loss of skin turgor (%)	86	76	80	100
sunken eyes (%)	95	100	100	100
rectal temperature (°C)	38.7 (37.0–39.4)	38.4 (36.9–40.0)	39.1 (37.6–40.6)	38.3 (36.7–39.0)
systolic blood pressure (mm Hg)	32 (0–76)	35 (0–90)	48 (0–100)	54 (0–84)
plasma specific gravity	1.033 (1.028–1.038)	1.035 (1.030–1.038)	1.032 (1.027–1.037)	1.031 (1.027–1.035)
arterial-blood pH	7.18 (6.97–7.37)	7.18 (7.03–7.29)	7.21 (6.95–7.37)	7.27 (7.11–7.46)
plasma urea nitrogen (mg/100 ml)	27 (12–41)	21 (12–27)	28 (14–51)	53 (34–72)
plasma osmolarity (mOsm/litre)	293 (278–306)	289 (271–292)	293 (277–307)	289 (282–295)

* With the exception of the number of patients, the figures given are means and, where applicable, the ranges in parentheses.

admitted with depressed sensoria were responsive within 6 hours. Of the remainder, one was fully responsive within 12 hours and the other did not become so until 24 hours after admission.

Only one difference in the clinical course of illness in the two treatment groups was noted. Most of the children in Group R became very thirsty during rehydration with lactated Ringer's solution and drank glucose solution avidly. By contrast, the children in Group MS were placid and displayed no signs of thirst.

Arterial blood and plasma determinations at admission and 6, 12, and 24 hours after admission are summarized in Table 4. At admission the children in both treatment groups showed evidence of isotonic dehydration, as manifested by an elevated plasma specific gravity, a normal plasma sodium concentration, and a normal plasma osmolarity (Table 3). One child in each treatment group had hypoglycaemia at admission (the plasma glucose levels were 18 and 23 mg/100 ml, respectively).

The course of recovery was similar in both groups, although the return of plasma bicarbonate and pH values to normal was somewhat faster in Group MS. Plasma sodium values were normal throughout the course of illness in both groups. Plasma potassium values were normal at admission but were slightly low in both groups for at least 24 hours thereafter. Mean haematocrit values in Groups MS and R were 47% and 46%, respectively, at admission, and fell to 33% and 32% within 24 hours. Although plasma specific gravity did not fall appreciably after 6 hours, mean haematocrit values continued to fall significantly for at least 12 hours in Group MS ($P<0.01$ compared with 6-hour value) and 24 hours in Group R ($P < 0.05$ compared with 12-hour value).

Changes in mean body weight after admission in both groups are summarized in Table 5. The greatest

Table 4. Arterial-blood values: cholera patients

Determination	Group	Time after admission (hours) [a]			
		0	6	12	24
pH	MS	7.18 ± 0.02	7.33 ± 0.02	7.40 ± 0.01	7.45 ± 0.01
	R	7.18 ± 0.02	7.34 ± 0.02	7.34 ± 0.02	7.39 ± 0.02
bicarbonate (mEq/litre)	MS	12 ± 0.5	17 ± 0.7	20 ± 0.8	23 ± 0.6
	R	11 ± 0.5	14 ± 0.6	16 ± 0.9	19 ± 1.0
sodium (mEq/litre)	MS	138 ± 3	138 ± 3	137 ± 3	135 ± 2
	R	136 ± 2	137 ± 2	135 ± 4	137 ± 3
potassium (mEq/litre)	MS	4.0 ± 0.4	3.3 ± 0.3	3.3 ± 0.2	3.6 ± 0.4
	R	4.2 ± 0.3	3.9 ± 0.2	3.3 ± 0.2	3.9 ± 0.3
chloride (mEq/litre)	MS	110 ± 3	110 ± 5	106 ± 2	103 ± 5
	R	114 ± 4	114 ± 4	117 ± 6	114 ± 4
glucose (mg/100 ml) [b]	MS	165 ± 13	203 ± 24	148 ± 17	84
	R	173 ± 20	93 ± 7	86 ± 10	105 ± 26
plasma specific gravity	MS	1.033 ± 0.001	1.026 ± 0.001	1.025 ± 0.001	1.025 ± 0.000
	R	1.035 ± 0.001	1.027 ± 0.001	1.026 ± 0.001	1.026 ± 0.000
haematocrit (percent)	MS	47 ± 1	39 ± 1	33 ± 2	33 ± 1
	R	46 ± 2	38 ± 1	37 ± 2	32 ± 1

[a] The figures given are means ± SE. In some instances the number of observations was too small to permit the calculation of the SE.

[b] In group MS 2 patients had very high blood glucose values at 6 hours (404 and 447 mg/100ml). These values were excluded in calculating the mean.

increase in body weight occurred during the first 6 hours. However, in both groups, body weight continued to increase until 12 hours after admission, after which it remained relatively stable.

Table 5. Percent increase in body weight during therapy

Group	Time after admission (hours) [a]			
	6	12	24	48
cholera:				
MS	5.6 ± 3.4*	8.0 ± 2.8	9.1 ± 2.6	8.4 ± 2.8
R	6.3 ± 3.4	7.3 ± 3.1	7.5 ± 3.0	7.3 ± 3.2
noncholera:				
MS	4.7 ± 2.3	5.9 ± 2.2	7.0 ± 2.2	7.9 ± 2.0
R	6.1 ± 1.9	6.9 ± 2.8	6.4	8.9 ± 0.9

[a] The figures given are means ± SD. Omission of the SD indicates that the number of observations was too small to permit its determination.

The mean 12-hourly stool output during the 48 hours following admission is shown in Table 6. During each 12-hour period the patients in Group R showed a slightly, though not significantly, greater stool output. There was a wide variation in total stool volume and duration of diarrhoea from child to child. However, in each group, about 75% of the total stool volume was passed during the first 24 hours and diarrhoea ended in all children within 48 hours. In Groups MS and R the maximum individual stool outputs during the first 24 hours were 23% and 28%, respectively, of the body weight at admission.

Children with acute noncholera diarrhoea. The clinical response to intravenous rehydration of children with acute noncholera diarrhoea was entirely similar to that of children with cholera. Serial arterial blood and plasma values are summarized in Table 7. At admission arterial-blood pH values were slightly higher, and plasma specific gravity values slightly lower, than in children with cholera. Changes

Table 6. Stool output after admission

Group	Time after admission (hours) [a]			
	0–12	12–24	24–36	36–48
cholera:				
MS	293 ± 48	263 ± 47	72 ± 20	64 ± 21
	(16)	(14)	(5)	(4)
R	362 ± 47	358 ± 78	158 ± 42	105 ± 19
	(15)	(15)	(9)	(7)
noncholera:				
MS	286 ± 90	137 ± 43	[b]	[b]
	(8)	(5)		
R	215 ± 96	166 ± 74	[b]	[b]
	(3)	(3)		

[a] The figures given are means ± SE in grams for the entire group and, in parentheses, the number of patients passing liquid stool during the period.

[b] Noncholera patients voided very little semisolid stool or no stool after 24 hours.

in mean body weight after admission are summarized in Table 6. The mean total stool output during the 48 hours following admission of all children with acute noncholera diarrhoea was only 48% of that of all children with cholera ($P<0.01$). Eight of 15 children in this group had no diarrhoea after admission, and only 3 had diarrhoea lasting more than 24 hours after admission.

Circumstances requiring alteration of treatment regimen. The treatment regimen of 4 of the 22 children in Group R was altered. In 3 of these (2 cholera, 1 noncholera) prolonged stupor or vomiting, or both, prevented the required intake of glucose solution owing to the risk of fluid aspiration. These patients received their maintenance water requirements intravenously in the form of 5% glucose in 0.18% saline.

The fourth case was a 6-year-old girl admitted with a 10-hour history of vomiting and diarrhoea. Stool culture yielded *V. cholerae*. At admission she was

Table 7. Arterial-blood values: noncholera patients

Determination	Group	Time after admission (hours) [a]			
		0	6	12	24
pH	MS	7.21 ± 0.04	7.36 ± 0.03	7.42 ± 0.03	7.43 ± 0.02
	R	7.27 ± 0.07	7.35 ± 0.01	7.33	7.40 ± 0.05
bicarbonate (mEq/litre)	MS	12 ± 1	17 ± 1	20 ± 2	20 ± 1
	R	13 ± 2	15 ± 2	16	19 ± 2
sodium (mEq/litre)	MS	138 ± 5	137 ± 3	136 ± 3	136 ± 3
	R	130	136	140	141
potassium (mEq/litre)	MS	4.3 ± 0.4	3.5 ± 0.3	3.6 ± 0.5	3.8 ± 0.5
	R	3.4	3.1	4.3	3.3
chloride (mEq/litre)	MS	107 ± 5	111 ± 5	115	106 ± 8
	R	108	105	102	112
glucose (mg/100 ml) [b]	MS	139 ± 16	171 ± 11	194 ± 3	77
	R	120	181	53	46
plasma specific gravity	MS	1.032 ± 0.001	1.026 ± 0.001	1.025 ± 0.001	1.025 ± 0.001
	R	1.031 ± 0.002	1.025 ± 0.001	1.025	1.025 ± 0.001
haematocrit (percent)	MS	43 ± 2	35 ± 2	28 ± 1	30 ± 2
	R	44	31	32	32

[a] The figures given are means ± SE. In some instances the number of observations was too small to permit calculation of the SE.

[b] In group MS one patient each at 6 and 12 hours, and in Group R one patient at 24 hours, had very high blood glucose values (416, 690, and 598 mg/100 ml, respectively). These values were excluded in calculating the means.

stuporous and showed physical signs of severe extracellular fluid deficit, including nondetectable blood pressure. Three hours after intravenous rehydration was initiated she had a grand mal seizure, following which she remained comatose for 56 hours. During this period she was maintained solely by intravenous replacement. She subsequently recovered completely. A persistent cardiac arrhythmia was present during the first few days of convalescence but this had disappeared when she was re-examined after 3 weeks. Serum electrolytes were normal throughout her illness. Blood glucose at admission was 19 mg/100 ml, but was 56 mg/100 ml after 6 hours and 82 mg/100 ml after 12 hours. It was normal thereafter. Hypovolaemia was corrected within the first hour and plasma specific gravity, which was 1.038 at admission, was normal within 12 hours. Her arterial-blood pH and bicarbonate concentration, which were 7.03 and 8 mEq/litre, respectively, at the time of admission, were normal within 18 hours. Cerebrospinal fluid obtained by lumbar puncture on two occasions was normal. The initial transient hypoglycaemia was the only detected abnormality that might explain her seizure and prolonged coma.

One child died suddenly 48 hours after apparent complete recovery from cholera. Post-mortem culture of heart blood yielded a heavy growth of *Staphylococcus aureus*, which was the presumed cause of death.

DISCUSSION

Effective treatment of paediatric cholera requires the replacement of both the initial deficit and the continuing losses of water and electrolytes. Three different fluid losses occur: (1) the deficit of isotonic fluid at admission (Mahalanabis et al., 1970); (2) the subsequent loss of liquid stool, which is hypotonic in electrolyte content (Wallace & Oleinick, 1966; Griffith et al., 1967; Mahalanabis et al., 1970); and (3) insensible and urinary water losses (Mahalanabis et al., 1970). The treatment regimen employed with children in Group MS was designed to replace water and electrolyte losses as accurately as possible, utilizing a different intravenous solution for each type of loss described above. This treatment regimen was based on previous recovery balance studies of children with cholera (Mahalanabis et al., 1970) and on previously established principles of treatment of paediatric diarrhoea (Cooke, 1969). The course of illness in these children represents the response to a near-optimum treatment regimen and serves as a guide for judging the effectiveness or shortcomings of other treatment regimens.

The clinical course of illness in the children in the two treatment groups did not differ appreciably. In each, shock was corrected rapidly, serious acidosis was relieved promptly, and serum electrolyte concentrations were maintained within acceptable limits. With the exception of the one child with prolonged coma, the recovery of all children from cholera and acute noncholera diarrhoea treated by either regimen was uneventful.

The treatment regimen of children in Group R included additional replacement of water, potassium, and glucose by mouth. These were thought to be essential for the following reasons. The sodium content of lactated Ringer's solution exceeds that of paediatric cholera stool by about 25% (Griffith et al., 1967; Mahalanabis et al., 1970). When given on a volume-for-volume basis to replace stool losses this solution would provide excess sodium replacement and little " free water " to replace urinary and insensible losses (Mahalanabis et al., 1970). To avoid excess sodium replacement, children in Group R received only 75 ml of lactated Ringer's solution for each 100 ml of stool passed after admission. Water lost in the remaining 25% of stool volume, and all insensible and urinary losses, were replaced with orally administred glucose solution. A few children in whom prolonged altered consciousness or recurrent vomiting prevented oral intake received this solution intravenously. Although it was not attempted in this study, it is likely that the glucose solution could have been given to these children by slow nasogastric infusion, thus avoiding the need for a second intravenous solution (Nalin & Cash, 1971). Glucose was included in the oral solution to correct or prevent hypoglycaemia, which occurs in a small proportion of children with acute diarrhoea (Hirschhorn et al., 1966). It is also likely that oral glucose would facilitate intestinal sodium absorption, thus further improving water and electrolyte balance (Pierce et al., 1969; Nalin & Cash, 1971). Oral potassium replacement was given to prevent the consequences of serious hypokalaemia, which are not likely to be completely prevented by the low potassium content of lactated Ringer's solution. We conclude that a treatment regimen employing intravenous lactated Ringer's solution is entirely satisfactory for the treatment of cholera and acute noncholera diarrhoea in children, provided that additional water, glucose, and potassium are given by mouth.

A major problem in the management of cholera in children is the estimation of the fluid volumes required for initial rehydration and subsequent maintenance during continuing stool loss. This is especially difficult for physicians who have little prior experience in the treatment of cholera. In this study several techniques commonly used to estimate fluid requirements were compared. The results provided some useful guidelines for the estimation of fluid requirements in children.

The specific gravity of plasma (Gp) has been used as a guide to the volume of rehydration fluid required in adults and children (Watten et al., 1959; Gutman et al., 1970). The mean value for Gp in adults with severe dehydration is about 1.044 (Pierce et al., 1968). Phillips has suggested that the rehydration requirements of adults are approximately 4 ml per kg of body weight for each 0.001 elevation of Gp above 1.025 (Phillips et al., 1964). In the present study the Gp of children with severe dehydration averaged 1.034. Similar values for children have been reported previously (Griffith et al., 1967; Gutman et al., 1970; Mahalanabis et al., 1970). Assuming that severe dehydration in these children amounted to an average 9% loss of body weight, their rehydration requirements would be 10 ml/kg for each 0.001 elevation of Gp. This assumption is supported by the average stable weight gain demonstrated in the present study and by demonstrated water deficits in children with disease of comparable severity previously reported by Mahalanabis et al. (1970). These previously reported balance studies of somewhat younger children indicated an average rehydration requirement of 12 ml/kg for each 0.001 elevation of Gp. These observations indicate that Gp may be a useful guide to rehydration requirements in children, provided that calculated requirements take into consideration the considerably greater fluid deficit present for each 0.001 rise in Gp. The reasons for this difference between adults and children are not immediately apparent.

Fluid requirements to replace continuing losses, especially during the first 24 hours, must also be determined with reasonable accuracy. Although the water requirement for insensible and urinary losses is predictable, continuing stool losses vary greatly from patient to patient. In the present study, stool output during the first 24 hours ranged from zero to 28% of body weight. The direct measurement of stool volume, using a cholera bed, is a useful guide to fluid requirements. However, it is often impossible to collect stool accurately, separate from urine, in the setting in which cholera must be treated. Under such conditions the fluid requirements for replacing continuing stool losses can best be evaluated by frequent observations of the rate of stool production and by periodic determination of body weight. Repeated body weight measurements and bedside clinical examination, in our opinion, constitute the best means of accurately assessing fluid requirements. They are accurate, are easily performed, and circumvent the difficulties of stool measurement and blood sampling. Rehydrated body weight can be determined by adding the estimated initial fluid deficit to admission weight or by reweighing after initial rehydration is completed and careful re-examination indicates hydration to be adequate. Rehydrated body weight can then serve as an objective guide to further fluid requirements, the objective being maintenance of a nearly stable weight after rehydration. The maximum weight gain by children with severe volume depletion at admission should not exceed 10–11% and is usually somewhat less. In this study the weight gain after rehydration became stabilized within 24 hours and averaged 7–9% of the weight at admission.

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RÉSUMÉ

EMPLOI DE LA SOLUTION AU LACTATE DE RINGER POUR LE TRAITEMENT DES ENFANTS ATTEINTS DE CHOLÉRA OU DE DIARRHÉE AIGUË NON CHOLÉRIQUE

Une étude contrôlée a été faite chez 38 enfants atteints de choléra et 15 enfants souffrant de diarrhée aiguë non cholérique afin de vérifier si l'administration intraveineuse de solution au lactate de Ringer représente un procédé efficace de rétablissement de l'équilibre hydro-électrolytique. Certains des enfants (groupe MS) ont reçu successivement, par voie intraveineuse, trois solutions différentes, de composition optimale, destinées à combler le déficit initial en eau et en électrolytes et à compenser les pertes ultérieures par les selles et l'urine. Chez les autres (groupe R) seule la solution de Ringer a été injectée par voie intraveineuse, une solution glucosée étant donnée par voie orale. Les enfants des deux groupes ont reçu en outre un apport supplémentaire de potassium par voie orale.

La réponse au traitement a été satisfaisante dans les deux cas. On a noté simplement, chez les jeunes malades du groupe R, une plus grande fréquence de la soif et un retour plus lent à la normale des valeurs du bicarbonate plasmatique et du pH. Chez quatre enfants (15%) de ce groupe, incapables en raison de vomissements et d'un état de stupeur prolongés d'ingérer les quantités requises de solution glucosée, le traitement a dû être modifié et il a fallu recourir à des injections intraveineuse de soluté salin glucosé.

Les auteurs concluent que le traitement des enfants atteints de choléra ou de diarrhée aiguë non cholérique par administration intraveineuse de solution au lactate de Ringer donne entière satisfaction, à condition de donner simultanément, par voie orale, un complément d'eau, de glucose et de potassium. Seuls quelques enfants ne peuvent, en raison de leur état, en bénéficier.

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