FLUID THERAPY IN GASTRO-ENTERITIS WITH SEVERE DEHYDRATION

A BIOCHEMICALLY CONTROLLED TRIAL OF A REGIME IN 40 PATIENTS

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The infant and pre-school mortality rates remain high in countries where populations live under unhygienic conditions and where the general state of nutrition is poor.[1-6] In these communities gastro-enteritis is a major cause of death, particularly in children under the age of 5 years. The magnitude of the problem of dealing with the high incidence of gastro-enteritis in Cape Town has been stressed by Truswell,[5] Robertson *et al.*,[6] and Bowie.[7] A scheme for the management of gastro-enteritis in outpatient departments has been described previously by Hansen.[8] Bowie[7] reviewed the scheme in 1,135 cases and found it to be clinically satisfactory. This scheme was essentially the administration of parenteral fluids to combat dehydration. The fluids were given either intravenously or subcutaneously depending on the severity. The fluid used was a 1 : 1 mixture of 5% dextrose and full-strength Darrows solution, thus giving a half isotonic Darrows and 2½% dextrose solution.

The purpose of this paper is to show the biochemical effects of the 1 : 1 dextrose and Darrows mixture when used intravenously in the treatment of severe dehydration. The effects of 1/6 molar sodium-r-lactate in the patients with severe acidosis are also studied, and it is also intended to show that potassium given intravenously from the commencement of therapy need not have serious complications and is probably beneficial.

A. Case Material

During the period August 1960 to January 1961, 40 patients were chosen at random from outpatients awaiting treatment. They were neither consecutive nor selective. The only criteria for inclusion was that they had to be (1) under 2 years of age, (2) dehydrated as a result of gastro-enteritis, and (3) should have received no parenteral fluids during the previous 10 days.

Each patient was clinically assessed and recorded as to: (*i*) severity of his condition, (*ii*) evidence of acidosis, and (*iii*) the degree of dehydration as manifested by loss of tissue turgor, sunken eyes, fontanelle, etc. The latter was designated at 5% (moderate) or 10% (severe) dehydration. There were 30 patients in the 'severe dehydration' group.

B. Treatment

All patients received 'bicillin forte' and sulphadiazine or chloramphenicol suspension. Milk feeds were stopped and a 1 : 1 mixture of Darrows solution and 5% dextrose was given by mouth. The amount of oral fluid was determined purely by the demand of the infant and was not measured. If there was severe vomiting, oral fluids were withheld for from 3 to 6 hours.

Intravenous fluids were administered through a scalp vein. The fluid used was identical to that given orally, i.e. half isotonic Darrows and dextrose (Table I).

The amount of fluid given over the first 24-hour period was calculated on the basis of replacement and maintenance.

TABLE I. HALF ISOTONIC DARROWS AND DEXTROSE SOLUTION

Sodium	**61 mEq./l.**
Potassium	**17 mEq./l.**
Chloride	**51 mEq./l.**
Lactate	**27 mEq./l.**
Calories	**100 mEq./l.**

TABLE II. 1/6 MOLAR-r-LACTATE

Sodium	**167 mEq./l.**
Lactate	**167 mEq./l.**

Replacement fluid: (i) in 5% (moderate) dehydration: 25 ml./lb. (*ii*) in 10% (severe) dehydration: 50 ml./lb.
Maintenance fluid: 70 ml./lb. for all patients.

Thus, a patient who was moderately dehydrated would receive 95 ml./lb., and a severely dehydrated patient would receive 120 ml./lb. at a drip rate calculated to last for 24 hours. In practice, as all patients received oral fluids, it was found more convenient to estimate the intravenous fluid requirement at 100 ml./lb., for 24 hours. Careful check as to the clinical state of hydration was made at regular intervals to ensure that no patient was becoming over-hydrated. The infusion was discontinued at approximately 24 hours, or when the patient was judged to be rehydrated on clinical grounds. The schedule was continued if dehydration was judged to be still present.

In addition, if any patient was thought to be severely acidotic, he was given 1/6 molar sodium-r-lactate. The amount given was 10 ml./lb. and was part of the 24-hour schedule. This would be given initially and then followed by the half isotonic Darrows mixture.

The electrolyte content of the intravenous solutions can be seen in Tables I and II.

C. Blood Samples

Venous blood was taken before the commencement of the intravenous infusion and after its completion. Sodium, potassium, chloride, urea, carbon dioxide combining power, and sugar, were determined by the routine hospital laboratory. All patients were weighed before and after infusion.

D. Biochemical Methods

Sodium and potassium were estimated using an Eel flame photometer. Chlorides were estimated by the titration method of Schales and Schales. Carbon dioxide was measured by van Slyke's method. Sugar was estimated by the method of Hagedorn and Jenson, while urea was estimated by a modified Robb method.

RESULTS

The full details of the results are set out in Table III and Figs. 1 - 6.

(*a*) *Amount of fluid given, weight gain and duration of infusion.* The average amount of fluid given intravenously was 953 ml. No check was kept on the oral intake. The average weight before therapy was 11 lb., while after therapy it was 12 lb. 4 oz. The average gain

in weight was thus 1 lb. 4 oz. (Table III). The duration of intravenous fluid therapy ranged from 11 to 48 hours. Excluding one patient who was on therapy for 44, and another for 48 hours, the range was from 18 to 30 hours. In one instance, the patient's condition was assessed as satisfactory after 11 hours.

(*b*) *Sodium (Fig. 1).* The mean serum sodium con-

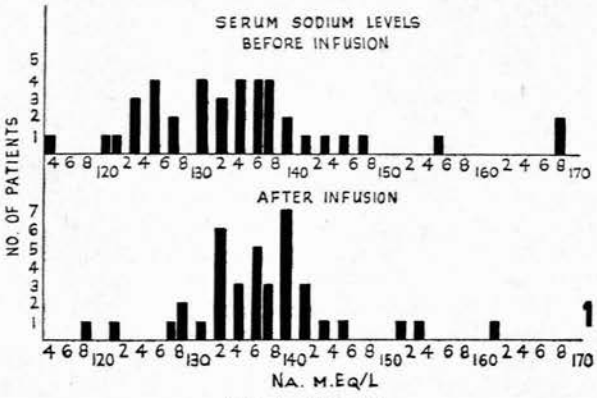

Fig. 1. See text.

centration before and after intravenous therapy was similar — 134·5 and 137·7 mEq./l. This was due to the wide range. There were 3 patients with levels of more than 150 mEq./l. After infusion they were all improved, although only one was restored to normal. One patient, previously normal, was rendered hypernatraemic. Twelve (30%) had levels of less than 130 mEq./l. All but one patient improved and in only 5 the sodium levels remained low after therapy.

(*c*) *Potassium (Fig. 2).* The mean levels of potassium

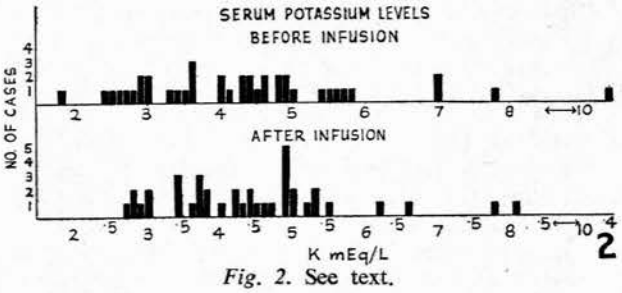

Fig. 2. See text.

were the same before and after therapy. The range was wide — 1·7 - 10·4 mEq./l. before, and 2·7 - 8·1 m.Eq./l. after therapy. There were 8 with less than 3 mEq./l. Of these, only 3 remained low after treatment. There were

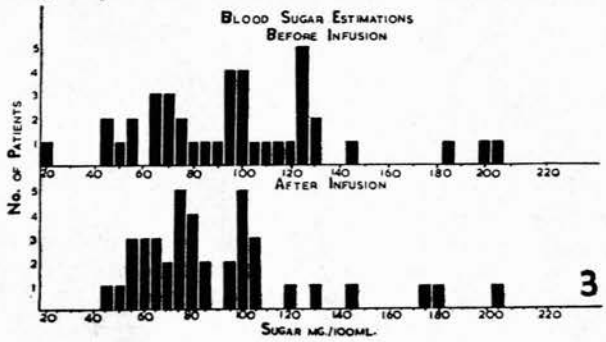

Fig. 3. See text.

8 patients with concentrations greater than 5·5 mEq./l., all of which were reduced by the therapy.

(*d*) *Urea.* There were 22 patients (55%) with blood urea of more than 40 mg.% before treatment. After rehydration there were only 6.

(*e*) *Sugar.* The mean blood-sugar level (Fig. 3) was 98·7 mg. per 100 ml. before, and 91·6 mg. per 100 ml. after therapy. Before the infusion there were 3 patients with levels below 50 mg. per 100 ml., while after therapy there was only 1.

(*f*) *Carbon Dioxide (Fig. 4).* The range was from 2·5

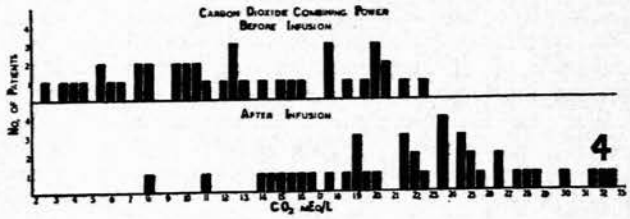

Fig. 4. See text.

to 22·6 mEq./l. before rehydration, with a mean of 11·9 mEq./l. After hydration the range was from 8·4 to 32·9 mEq./l., with a mean of 22 mEq./l. Thirty four patients (85%) were below 20 mEq./l. and thus acidotic, while 14 (35%) were less than 10 mEq./l. After treatment 14 patients had a CO_2 level of less than 20 mEq./l., and only 1, though improved, was still below 10 mEq./l.

There were 9 patients clinically assessed as severely acidotic and given 1/6 molar sodium-r-lactate. In all the CO_2 was raised significantly, and in only 2 instances was it still below 20 mEq./l. (Fig. 5).

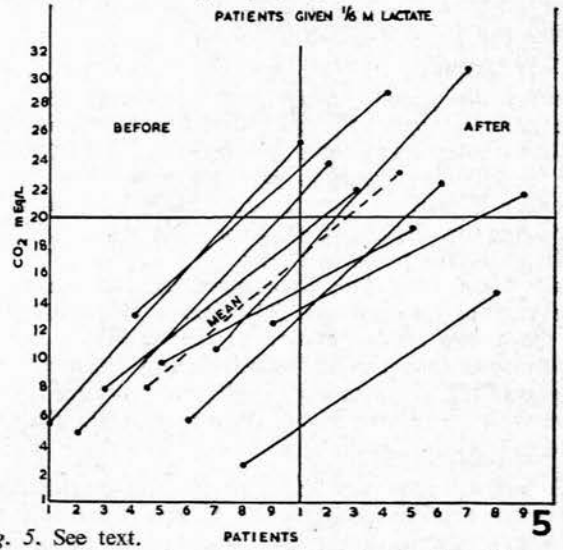

Fig. 5. See text.

In 8 other patients who had CO_2 levels of less than 10 mEq./l. and who did not receive 1/6 molar sodium-r-lactate, although their CO_2 levels were raised, in only one instance did it reach 20 mEq./l. (Fig. 6).

DISCUSSION

It is now more than 100 years since an electrolyte solution was first used intravenously for the correction of dehydration.[9] It was not until this century, however, that intravenous electrolyte therapy was put on a firm scientific basis.[10-15] With the advent of new knowledge, therapeutic

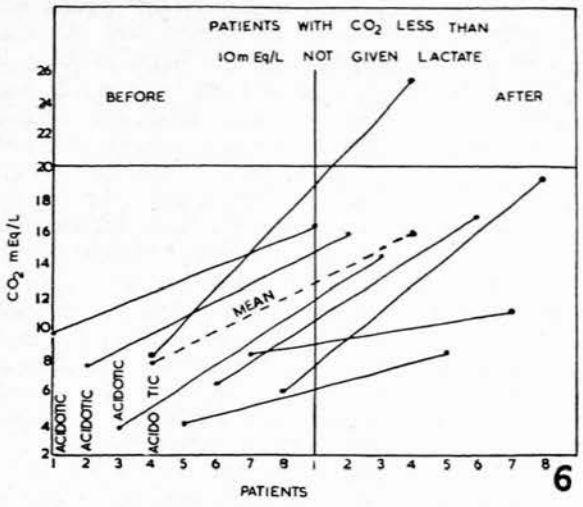

Fig. 6. See text.

schemes have become relatively complicated in an attempt to correct the laboratory estimated deficit.[16-18]

Powers[19] reported on one of the first satisfactory schemes of treatment for dehydration following gastro-enteritis, and with the introduction of fluid therapy there was a tremendous drop in infant mortality.[20] All subsequent schemes of rehydration have been but a modification of Power's original method. Though all the schemes advocated have been shown to be satisfactory, many require a certain amount of adjustment depending on laboratory results. This is not a practical procedure when large numbers of patients have to be dealt with, as in many clinics in under-developed countries. In such clinics it is essential to have one or two solutions that may be safely given to a patient whose electrolyte deficiency has to be estimated solely on clinical grounds.

Potassium depletion and the part it played was not appreciated until the work of Darrow[21] became known. The clinical effects of potassium depletion are now well recognized.[22-25] These effects were once described as toxaemia, and even recently referred to as such in European literature.[26]

The method of rehydration advocated by the majority of workers[10,11,17,24,25] is to give initially a simple isotonic saline solution. Thereafter a multi-electrolyte solution

TABLE III. FLUID THERAPY IN PATIENTS WITH SEVERE DEHYDRATION FROM GASTRO-ENTERITIS

Patient	Weight		Sodium		Potassium		Urea		Carbon dioxide		Sugar		Comment
	Before lb. oz.	After lb. oz.	Before	After	Before	After	Before	After	Before	After	Before	After	
1	8.2	9.4	141	136	4·6	5·2	87·5	18·7	11	23·6	45	65	
2	19.2	21.15	125	132·4	2·4	3·4	14·4	5·9	12·1	17·5	118	108	
3	10.9	11.8	132·4	137·9	2·7	3·4	92	27·8	10·8	21·7	122	54	
4	9.15	11.3	139·6	139·6	4·9	4·4	122	12·9	9·7	16·3	104	83	
5	18.11	20.1	136·2	139·6	4·0	3·84	57·7	16·5	7·6	15·8	75	100	
6	13.11	15.5	139·6	141·8	4·4	—	63·7	10·4	5·6	25·3	71	100	1/6 M Lactate
7	16.2½	16.9	137·9	132·4	5·4	6·2	22·6	12·5	17·5	20	85	82	
8	7.15	8.6	134·2	136	7·8	5	216·5	47	3·7	14·4	102	82	
9	11.14½	12.10	127	132·4	3·0	3·6	32·8	9·5	14·5	26·6	123	76	
10	7.3	8.13	123·3	143·5	3·3	4·9	12·4	14·9	8·3	25·4	76	101	
11	13	14.7	145·5	147·8	7	4·5	286	12·0	4·9	23·7	204	106	1/6 M Lactate
12	15.14	17.12½	168	161	5·7	3·8	262	10·2	22·6	31·6	185	124	
13	6.6	6.11¼	125	118	2·9	3	75·4	5·1	4	8·4	23	175	
14	6.2	7.8	127	136	4·3	5	38·8	7·3	18·7	25·8	56	67	
15	5.13	6.10	136·2	139·6	4·8	4·7	49	10·8	12·6	19	67	98	
16	14.7	15.15	123·3	128·8	2·5	2·8	90·5	45	7·9	21·9	206	73	1/6 M Lactate
17	4.15	5.9	134	132	6	5·5	195	40	6·5	16·9	69	62	
18	6.2½	7.7	120	121·6	2·6	3·7	90	45·6	8·3	110	120	147	
19	10.13	12.8	132·4	128·8	1·7	2·8	5	6·3	10	15·2	101	181	
20	10.5	11.9	137·9	134·2	4·4	6·7	9·9	6·3	15·3	—	54	57	
21	11.7¾	12.8	130·7	134·2	4·5	4·9	9·5	6·2	21·6	24·6	12·7	80	
22	11.7	13.2	134·2	139·6	4·8	7·8	124·4	12·1	13·1	28·8	148	71	1/6 M Lactate
23	13.15	15.8	143·5	153·9	4·9	5·3	23·2	19·0	6·0	19·3	93	75	
24	5.13	6.10	168	151·5	10·4	8·1	56·3	180	9·8	19·2	97	45	1/6 M Lactate
25	12.2	14.0¾	155·9	145·5	5·5	4·9	22·1	22·3	5·8	22·4	101	96	1/6 M Lactate
26	12.6½	14.4	137	132·4	3·6	4	89·4	6·2	16	28·2	126	55	
27	9.14	10.14	134·2	139·6	5	4·9	56	6·8	10·6	19·9	73	64	
28	15.13	17.15	147·8	147·8	5·6	5·3	54·2	10·2	17·5	23·5	95	56	
29	8.9¼	9.3	130·7	134·2	4·6	4·6	18·5	8·1	20·6	22·7	57	61	
30	10.1	11.3	132·4	139·6	4·1	5·1	20·0	5	20·1	27·8	70	67	
31	13.10	14.13	130·7	141·8	3·5	4·2	8·6	6·9	20	24·6	47	105	
32	12.14	13.2	123·3	136·2	2·9	3·7	9·7	4	20	32·9	113	207	
33	11.2	12.8	137·9	141·8	3	3·4	13·7	6	12·7	26·8	121	131	
34	10.1	11.15	130·7	127·0	3·6	3·7	17·2	6	17·5	24·8	98	78	
35	13.9	14.13	125	130·7	4	2·7	20·6	7·5	10·7	30·4	105	89	1/6 M Lactate
36	15.1½	16.2	136·2	137·9	2·8	2·9	53	11·7	20·8	18·8	121	104	
37	7.12	8.11	114·5	132·4	7	4·9	240	111·5	2·5	14·7	67	76	1/6 M Lactate
38	12.8	13	125	137·9	5·8	4·4	39	10	12·5	21·6	—	79	1/6 M Lactate
39	7.12	8.14	136·2	139·6	4·4	4·2	135	26·2	15·9	22·1	99	85	
40	9.6	9.13	121·6	136·2	3·4	4·3	59	6·5	19·8	32·1	84	100	
Averages	11	12·4	134·5	137·7	4·5	4·4	76·9	21·5	11·9	22	98·7	91·6	

containing potassium is used as maintenance fluid. Hartmann's regime[13] introduced an alkalizing solution (1/6 molar sodium-r-lactate) as the initial rehydrating fluid to correct acidosis. None advocate using potassium from the start. The danger of giving potassium in the initial rehydrating fluids has been repeatedly stressed,[10,13,24] and is based on the fact that in severe hydration there is shock, renal shutdown, and failure of the kidneys to deal with a potassium load.[10,27] Under these circumstances the serum potassium may initially be elevated, and it is feared that if potassium is given too early it may lead to hyperkalaemia and cardiac arrest. On the other hand, prolonged potassium depletion can have serious effects[22,27] and even lead to irreversible renal damage.[28]

In the present series of patients investigated, all but 9 (those who received 1/6 molar sodium-r-lactate) were given a solution containing 17 mEq./l. potassium from the initiation of treatment. Three of the 8 patients with elevated serum potassium concentrations (more than 5·5 mEq./l.) also received this solution. No toxic effects were noted. In all, the serum potassium fell to more normal limits. Severe hypokalaemia (less than 3 m.Eq./l.) was partially, if not totally, corrected.

It will be noted (Table III) that more than half the patients (5/8) with high serum potassium concentrations were also clinically severely acidotic and required 1/6 molar sodium-r-lactate initially, whereas only one patient with a low potassium was given 1/6 molar sodium-r-lactate. The inverse relationship between hyperkalaemia and acidosis is well recognized.[27,29,30] When using the method advocated by Hartmann,[31] Smith *et al.*[30] found that the solution (lactated KCl Ringers solution, which supplies 4 mEq./l. potassium), did not raise the serum potassium sufficiently. In the same paper it was shown that the Butler Talbot method may supply too much potassium. In considering the part played by potassium in dehydration, it must be remembered that the serum levels do not necessarily reflect the total body potassium, and potassium depletion in acute diarrhoea states may not be clinically evident.

The solution advocated caused gain in weight and clinical improvement of all the patients. The mean gain in weight was 9% of the initial weight.

With regard to sodium, there were 3 patients, (7·5%) who were hyperosmolaric. This is a very low incidence as compared with a previous local experience of 30%.[32] American workers[33] usually quote 20%. Two of these patients who had high serum sodium levels received 1/6 molar sodium-r-lactate because of acidosis. In both instances the serum sodium was lowered to within normal limits, in spite of having been given a fluid containing 167 mEq./l. of sodium. The restoration of renal flow by rehydration probably prevents any dangerous rise in serum sodium in these patients.

The incidence of hypo-osmolarity was 30%, similar to previous findings.[33] This is higher than the usually quoted figure of 10%. The half Darrows and dextrose solution restored the vast majority of hyponatraemic patients to normal. Twenty-three patients had azotaemia with a blood urea of more than 40 mg. per 100 ml. Rehydration restored all the patients to a clinically improved status within 48 hours, and the majority within 30 hours. There were a number of patients with very low figures after treatment.

Acidosis as a manifestation of gastro-enteritis and dehydration has been recognized for a long time. It seems as if fewer than half of the patients with acidosis can be clinically recognized as such, and then only when the acidosis is very gross. There were 4 patients with CO_2 levels of less than 10 mEq./l. who were not diagnosed as acidotic and consequently given no molar lactate. Thus, even gross acidosis may not be clinically evident.

From the results it would seem that, to restore the acid/base balance to normal rapidly, 1/6 molar sodium-r-lactate is advisable. In spite of receiving essentially similar quantities of fluid, those who did not receive 1/6 molar sodium-r-lactate did not correct their acidosis as rapidly. This finding has also been noted by Smith *et al.*[30] who used 1/6 molar sodium-r-lactate followed by Hartmann's solution. This combination however, is far too low in potassium, supplying only 4 mEq./l. They recommend 1/6 molar sodium-r-lactate followed by Darrows solution as being probably the ideal replacement therapy.

The wide range of blood-sugar levels was not significantly altered by rehydration. This failure to raise the blood-sugar level is not surprising, since the solution used only supplied 100 cal./l. Three patients had, in fact, blood-sugar levels of less than 50 mg. per 100 ml. and thus were definitely hypoglycaemic. It may be advisable to give these infants more glucose by mouth, i.e. a 5% solution rather than a 2½% solution, especially on account of the fact that the caloric demands must be high during the illness. No children had any ill-effects from the low blood-sugar levels, but the number of patients was small.

SUMMARY AND CONCLUSIONS

The clinical and chemical effects of an intravenous fluid scheme on 40 dehydrated patients were studied. The solution used was made up of equal parts of Darrows solution and 5% dextrose. This was administered intravenously and orally. In severely dehydrated children 100 ml./lb. was administered. 1/6 molar sodium-r-lactate was used as the initial hydrating fluid in severely dehydrated and acidotic children.

In considering all the parameters, this mixture of half isotonic Darrows and 2½ dextrose, as advocated by Hansen[8] and Bowie[7] is a safe and satisfactory all-purpose rehydration fluid for handling large numbers of grossly dehydrated children. Careful biochemical control of patients treated with this solution has shown that it is effective in restoring normal serum chemistry in most instances. In a 24-hour period it did not fully correct acidosis as measured by CO_2-combining power. The addition of 10 ml./lb. of 1/6 molar sodium-r-lactate improved its efficiency in this respect.

The principle of giving intravenous potassium from the initiation of therapy does not appear to be harmful. Hypokalaemia, a common finding, even in severe dehydration, was corrected; in fact, in some cases more potassium could have been given. Where hyperkalaemia was present, i.e., in concentrations above 5·5 mEq./l., it was in every case lowered by this therapy.

The incidence of hypoglycaemia was low (7·5%). It is apparent that, where this complication is suspected, extra glucose should be included in the drip schedule.

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