We are sorry to say that the elegant results of this clinical trial, although theoretically interesting, are of no practical use. Ironically, one of the important ‘advantages’ claimed by the authors, namely stability, is indeed the main disadvantage of sodium acetate as a component of an oral rehydration solution. Sodium citrate, on the other hand, being theoretically three times a better buffer than acetate or bicarbonate, and not being as hygroscopic as acetate, should be investigated in a similar clinical trial.

Reference

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Nature of complement deficiency in sickle cell disease

Sir,
The work of Larcher and colleagues\(^1\) was of great interest, as it confirmed and expanded our observations,\(^2\) cited by Dr Larcher \textit{et al.}, on the nature of complement deficiency in sickle cell disease. We feel, however, that in discussing their findings, Dr Larcher \textit{et al.} could have taken account of our further studies of this problem,\(^3\) which they seem to have overlooked. These studies, as well as our more recent work,\(^4\) indicate that about half of 'steady state' (intercritical) subjects with homozygous sickle cell disease have hypercatabolism of the alternative complement pathway, as judged by the \textit{in vivo} factor B metabolic turnover rate,\(^5\) and C3d levels in plasma.\(^6\) Mean factor D serum level is depressed in these patients,\(^7\) but this finding was statistically significant only when we

of the valproate dose. At this point she was discharged from hospital. Her other treatment at this time was diazoxide, 50 mg in the morning and 100 mg at night, and trimethoprime 60 mg at night.

She was seen again 2 months later and was noted to have a severe neutropenia of only \(0.2 \times 10^9/\text{l}\). This persisted for 3 weeks despite reducing her dose of sodium valproate and the drug was therefore stopped. Within 3 days her neutrophil count had returned to normal and has remained normal for 5 months. No alteration was made to her other drug therapy. There was never any evidence of viral infection as a possible cause of the neutropenia.

Neutropenia was first described during sodium valproate treatment by Jaeken \textit{et al.}\(^3\) who reported a single case in a 3 month old boy. More recently Coulter \textit{et al.}\(^8\) have reported a fall in the neutrophil count in 27 of 100 patients treated with valproic acid, but their minimum count of \(2.0 \times 10^9/\text{l}\) is of little clinical significance. Although the thrombocytopenia found with valproate therapy may be due to immune platelet destruction immunofluorescence studies have failed to show antibodies against granulocytes.\(^8\)

We would suggest that neutrophil counts as well as the customary platelet count should be performed on all patients treated with sodium valproate.

References
4 David N K Symon and George Russell
Department of Child Health,
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Sodium valproate and neutropenia.

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