Hyperglycaemia in infantile gastroenteritis

Sir,

We read with interest the paper by Rabinowitz *et al.*¹ Our own impressions were at variance with his figures. To verify our views we analysed 33 cases of acute gastroenteritis treated within our hospital in the last six months. All patients admitted with acute gastroenteritis have urea and electrolytes determinations before treatment is started. In our laboratory, blood glucose estimation is performed routinely on all blood samples sent for urea and electrolytes measurement. Blood glucose concentration is measured by glucose oxidase method.

The age range of our patients was between 1 and 22 months (mean 7.3 months). Twenty of these were below the 3rd centile of the Boston weight chart. One stool sample grew *shigella* but none of the others grew any pathogenic bacteria. The degree of dehydration was calculated from the weight of the child at admission, and weight when diarrhoea had stopped and the child was fully rehydrated. There was good correlation between this method and the clinical assessment of dehydration at admission.

Twelve children had severe dehydration with more than 10% loss of body weight and the rest had mild to moderate dehydration. Eleven children had high urea (more than 6.6 mmol/l), 14 had low potassium (less than 3.8 mmol/l), 10 had low sodium (less than 133 mmol/l), and 24 had low bicarbonate (less than 22 mmol/l) concentrations, but none had a blood glucose value of more than 10 mmol/l.

An occasional patient with high blood glucose concentration has been seen by all of us but we were sure that in our experience the incidence could not have been as high as 55%. All the clinical and biochemical parameters of our patients were quite similar to those reported by Rabinowitz *et al.* The differences between our and their experience and the similarities between their normal and hyperglycaemic children with acute gastroenteritis show that there must be some other factor(s) responsible for this phenomenon. More work needs to be done to find out whether it is the type of virus, the race of the patient, or over zealous use of oral rehydration powder that is responsible for this disparity.

Reference

- 1 Rabinowitz L, Joffe BI, Abkiewicz C, Shires R, Greef MC, Seftel HC. Hyperglycaemia in infantile gastroenteritis. *Arch Dis Child* 1984;59:771-5.

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Dr Rabinowitz and co-workers comment:

We suggest that the following points may explain Singh *et al.*'s failure to observe hyperglycaemia, compared with our high prevalence, in infantile gastroenteritis:

1. The nature of any underlying viral infection and ethnic group of the children, as acknowledged. Indeed, it is well established that certain viral infections may produce pancreatic beta cell destruction. None of our infants was given oral rehydration powder before admission.
2. A difference in seasonal incidence may also be relevant. Our patients presented in the late summer months but it is not clear when the Saudi Arabian children presented.
3. All the children in our study had severe disease, necessitating hospital admission. This evoked marked stimulation of stress hormones, one of the main pathogenetic factors in the evolution of hyperglycaemia.
4. Finally, the mode of collection of samples for blood glucose estimation appears to have differed. If their samples were sent to the laboratory in 'U and E' tubes without fluoride preservative, it is conceivable that substantial in vitro glycolysis occurred (especially in a hot climate), thereby spuriously lowering blood glucose readings.

Functional palatal incompetence and teratogenesis

Sir,

The interesting report by Dr Pearl and colleagues¹ of palatal incompetence in the fetal anticonvulsant syndrome prompts us to describe two children who have similar problems.

The children are sisters born to first cousin Caucasian parents. Throughout each pregnancy their mother smoked 80 cigarettes and drank 5 or 6 'shorts' of alcohol daily. At various times during the pregnancy of the elder girl she also took diazepam, nitrazepam, amitriptyline, and phenelzine.

Abnormal features present in both sisters include short stature and microcephaly, severe developmental delay, ataxia with an intention tremor, delayed bone age, and a similar facies with arched eyebrows, peri-orbital fullness, anteverted nares and thin upper lip. The elder girl has pulmonary hypertension with an atrial septal defect and patent ductus arteriosus. The younger girl has a normal heart and unilateral ptosis. Both girls have hypernasal dysarthria with nasal escape and very reduced palatal movement. Structurally their palates are intact with no submucous cleft.

The parental consanguinity makes it difficult to exclude an autosomal recessive disorder in these sisters, although their abnormalities do not readily conform to any inherited disorder known to us. They do, however, closely resemble the fetal alcohol syndrome in which speech problems including disorders of articulation and 'voice dysfunction' are common,² although we are unaware of any report clearly documenting palatal incompetence in this condition.

In the patients described by Dr Pearl and colleagues, teratogenesis was attributed to anticonvulsants. One of the mothers also smoked heavily and abused alcohol. Alcohol