Renin and aldosterone response in human newborns to acute change in blood volume

Sir,

Referring to the paper by Dillon et al. (Archives, 1978, 53, 461), we should like to report our studies on 26 term neonates, who were exchange transfused and compared with 10 healthy controls. The aetiology of jaundice of the exchanged newborns was Rhesus incompatibility in 5 cases, ABO incompatibility in 11, G-6-PD deficiency in 5, and in the remaining 5 it could not be determined. The blood used for exchange transfusion was supplemented with citrate dextrose (ACD). Angiotensin I levels were measured, by the radioimmunossay technique of Haber et al. (1969), immediately before, in the middle, and at the end of the exchange transfusion. The middle and end transfusion specimens were taken after a 10-min interruption of the exchange transfusion. No loading of the baby's circulation was made by leaving a surplus. Serum Na, Hct, and Hb were also determined.

The angiotensin I levels of the exchange transfused infants at the beginning (1.36±0.18 SE ng/ml per hour), the middle (1.75±0.18), and the end of the exchange transfusion (1.47±0.18) did not differ significantly, although an increase was observed in the middle. Neither did the serum sodium levels differ.

The mean level±SE of angiotensin in the blood used for the exchange transfusion was 0.84±0.13. If the aetiology of jaundice is taken into consideration, the angiotensin I levels of the infants, whose jaundice was due to G-6-PD deficiency, were lower (0.87±0.23) at the beginning of the exchange transfusion than those of infants with jaundice due to Rhesus incompatibility (1.36±0.46), ABO incompatibility (1.52±0.27), or to unknown causes (1.47±0.30), or to the levels of the healthy controls (1.59±0.16). No correlation between the Hct and angiotensin levels was noted. Lower levels of angiotensin I were found before the exchange transfusion in infants on their first 2 days of life.

From the above data we can conclude that exchange transfusion, if done slowly and lasting 14 to 2 hours, does not influence the angiotensin levels of the newborn. Moreover this slow rate of exchange transfusion corresponds to the one (of 5 ml/kg per 3 min) recommended by Aranda and Sweet (1977) as producing only minimal, rapid, reversible changes in the blood pressure. The observed lower angiotensin I levels in the jaundiced infants with G-6-PD were probably due to the young age (24 days median) of the infants and not to increased haemolysis.

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References

Gluten challenge in treated coeliac disease

Sir,

In their interesting paper (Archives, 1978, 53, 449), Packer et al. state that (after a 3- to 4-month period of gluten ingestion) 'the finding of a normal postchallenge biopsy excludes coeliac disease', but they also counsel that such children should be followed for up to 2 years after challenge, with the implication that a normal biopsy at this time definitely excludes coeliac disease.

We have made a preliminary report of a similar study which is being published in greater detail (McNicholl et al., 1979). 40 children were challenged with gluten after a mean of 5.8 years on gluten-free diets and so far 37 have been confirmed to have coeliac disease by the occurrence of mucosal relapse. In some children, the mucosal relapse was much slower than in the patients of Packer et al. In one girl after 28 months on gluten the overall appearance of the mucosa was just acceptable as normal and the interepithelial lymphocytes and mucosal disaccharidases were normal; it was not until the 63rd month on gluten that the mucosa was sufficiently abnormal to indicate definite relapse. Throughout this period she showed no haematological abnormalities and remained on the 75th centiles for height and weight.

We strongly reinforce the recommendation of Packer et al. that coeliac disease should not be diagnosed without satisfactory biopsy and agree that it was necessary for them to attempt to prove or disprove the initial diagnosis of coeliac disease (probably made by others) by gluten challenge. However, we would regard the 6 children with normal biopsies after 2 years on gluten as unsatisfactory subjects for allowing conclusions to be drawn concerning gluten intolerance, in the absence of an initial diagnostic biopsy. We agree with them that there are no consistent biochemical, haematological, or anthropometric indices of mucosal relapse. Finally, our experience dictates (Egan-Mitchell et al., 1978) that children whose initial mucosa had been definitely abnormal and who had fulfilled other accepted criteria for coeliac disease, must be followed up for considerably longer than 2 years, to allow a primary diagnosis of coeliac disease to be discarded.

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References
Hazards of hypertonic magnesium enema therapy

Sir,

Serious electrolyte disturbances can follow the use of enemas in children who have chronic bowel problems associated with disturbed absorption (Moseley and Segar, 1968). The hazards of hypertonic phosphate enemas were shown by Loughnan and Mullins (1977) and we have recently seen a case.

A 4-year-old boy had been attending medical outpatient departments with chronic functional constipation. His father, weary of the child's chronic burden, had bought from a chemist, without prescription, a Fletcher's magnesium sulphate retention enema. The father then administered approximately 100 ml of the total 130 ml magnesium sulphate to the child per rectum. In the father's presence 20 minutes later, the child suddenly collapsed and apparently lost consciousness. He was subsequently rushed to hospital by ambulance.

On admission the child was found to be deeply unconscious with fixed dilated pupils. He was centrally cyanosed with shallow, irregular respirations. Heart rate was 120/minute. He was markedly hypotonic and deep tendon reflexes could not be elicited. The child was immediately intubated and intermittent positive pressure ventilation was successfully carried out. Fortunately the full preceding history was elicited early and 10 ml 10% calcium gluconate was administered intravenously. Within a few minutes consciousness was regained, the pupils began to react to light, the deep tendon reflexes returned to normal, and spontaneous respirations were rapidly re-established. Initial investigations were Ca 2-31 mmol/l; 9-24 mg/100 ml (normal range 2-1-2-6 mmol/l; 8-4-10-4 mg/100 ml), Mg 5-87 mmol/l; 14-3 mg/100 ml (normal range 0-7-1-0 mmol/l; 1-7-2-4 mg/100 ml). Serial serum magnesium levels remained grossly raised and, subsequently, more treatment in the form of further aliquots of intravenous calcium gluconate, followed by peritoneal dialysis, and then by forced diuresis, was carried out for the next 18 hours. Results 20 hours after enema were Ca 2-27 mmol/l (9-1 mg/100 ml), Mg 0-91 mmol/l (2-2 mg/100 ml). The child continued to make a full recovery without any abnormal sequelae.

We feel this case shows the danger resulting from the availability of such enema preparations, permitting well intentioned but ill advised use by parents and others. It would appear that the recent legislation covering Part III of the Medicines Act does not restrict the sale of disposable enemas.

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References


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References


SIR,

Packer et al. (Archives, 1978, 53, 449) reported their experience of 32 children previously diagnosed as having coeliac disease who were re-investigated by gluten challenge in a well-documented study. While I agree with their advocacy of a small intestinal biopsy some 3 months after return to a gluten-containing diet, I cannot agree that a normal small intestinal biopsy, after such a 3-month interval, suggests that coeliac disease is 'for practical purposes' excluded. I have published details of my own experience concerning 2 children who respectively had a normal small intestinal mucosa after the following intervals back on a gluten-containing diet: 6 months (Walker-Smith, 1972), and 14 months (Walker-Smith et al., 1978). Yet they subsequently developed an abnormal small intestinal mucosa 1 year 10 months and 2 years respectively on a gluten-containing diet. So in fact these children did have coeliac disease and a previously normal biopsy after gluten challenge did not exclude this diagnosis. Such circumstances are certainly not common, but I believe that it is vital to emphasise the importance of a final biopsy at least 2 years after returning to a normal gluten-containing diet (as in fact, Packer et al. did) before it can be stated that coeliac disease is excluded (Walker-Smith and Kilby, 1977). What is more, Egan-Mitchell et al. (1978) and others have described an occasional child who may take even longer than the 2-year interval before developing a histological relapse. Therefore, it is my view that it is of great importance that there should be long-term, perhaps indefinite follow-up into adult life, for those children previously diagnosed as having coeliac disease in whom the mucosa remains normal despite return to a normal gluten-containing diet for 2 or more years. It is possible that some may eventually relapse.

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References