2 Talbert DG, Southall DP. A bimodal form of alveolar behaviour induced by a defect in lung surfactant—a possible mechanism for sudden infant death syndrome. Lancet 1985;i:1330.

Treatment for renovascular hypertension

Sir,

We read with interest the report of Awazu et al1 that expands the experience with percutaneous transluminal angioplasty (TLA) in children. We recently reported 17 children with renovascular hypertension,2 seven of whom were treated with this technique since 1979. Cure (defined as normotensive for age with no drugs) was achieved in two patients with main renal artery lesions for which TLA is probably best suited. Two patients developed renal artery thrombosis after the procedure, one of whom was successfully treated by autotransplantation of the kidney to the ipsilateral iliac fossa.

Enthusiasm for angioplasty must therefore be tempered by proper selection of patients and restriction of the technique to centres where experienced radiologists and surgeons are available. Two out of the five patients in Awazu’s series still required treatment with antihypertensive agents. As cure is preferable to chronic drug treatment, surgical techniques such as vascular repair or autotransplantation should be considered. Transluminal balloon angioplasty is a welcome addition to the available treatment modalities for these rare patients, but its exact role requires further careful assessment and documentation.

References


Correspondence

Dr Awazu and co-workers comment:

We agree with Drs Watson and Balfe’s comment on restriction of angioplasty to certain centres and the proper selection of patients. With experienced staff and a special observance for the possible side effects, we have not experienced any complications up to the present, though the number of the patients is small. Also we think that the proper selection of the patients is an essential factor for treatment with angioplasty. In our study three out of five patients were cured (as defined by Drs Watson and Balfe). In their study two out of five patients (two out of seven patients who developed thrombosis are impossible to assess) were technically not feasible, so it is difficult to say that they were properly selected. Therefore two out of three remaining patients were cured. Those ratios are encouraging.

As for the two patients in our study who required antihypertensive drugs, it might be necessary to consider surgery in the future. One patient had bilateral disease, however, in whom it would be difficult to expect cure with surgical treatment, as the two patients with bilateral disease who underwent autotransplantation clearly show. Considering the younger age of the patients and the possible extension or multiple occurrence of the lesion in the future, our present treatment is justified. Also the repeatability and the relative non-invasiveness of angioplasty should be re-evaluated.

Copper and the preterm infant

Sir,

We have read with interest the recent report by Sutton et al1 of copper deficiency in four very low birthweight infants. These authors found that diagnosis was made more difficult because of the lack of a suitable reference range for copper for these infants. The studies quoted by Sutton et al provide limited data on serum trace metal concentrations in preterm infants up to 1981. In 1983 we reported serial serum copper and zinc concentrations in a group of 48 preterm infants during the first year of life.2 A comparison of the results for plasma copper determinations obtained in Glasgow and serum copper concentrations in Belfast adjusted to show the geometric mean is shown in the Table. It is reassuring to find such good agreement between these two studies.

Sutton et al suggest that very low birthweight infants should be given at least 1 μmol (6-4 μg/100 ml) of copper/kg/day during parenteral and enteral feeding. Our own findings suggest that this statement may be unwarranted. Our policy is to provide 0-3 μmol (1-9 μg/100 ml) of copper/kg daily for very low birthweight infants who are receiving parenteral nutrition.3 The last seven infants of <30 weeks’ gestation (mean gestation 27 weeks, mean birthweight 930 g) had serum copper concentrations checked at a mean postnatal age of 70 days (mean and median post-conceptional age 37 weeks). None had a serum copper concentration outside the 95% ranges for the Belfast and Glasgow infants. For the group as a whole mean serum copper concentration was 11-5 μmol/l (73 μg/100 ml) (range 6-6-14-8). Furthermore, as copper is excreted by the biliary system high serum concentrations may occur in preterm babies with cholestasis.4 Thus,

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