lungs in sudden infant death syndrome. *Arch Dis Child* 1985;**60**:471–3.
[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.
[3] Southall DP, Talbert DG, Johnson P, *et al.* Prolonged expiratory apnoea: a disorder resulting in episodes of severe arterial hypoxaemia in infants and young children. *Lancet* 1985;**ii**:571–7.
[4] Fagan DG. Post mortem studies of the semistatic volume—pressure characteristics of infants lungs. *Thorax* 1976;**31**:534–43.
[5] Fagan DG. Developmental and pathological studies of the structure and function of the lung in children. (Thesis). Cambridge University, 1973:26–37.
[6] Gruenwald P. The effect of age on surface properties of excised lungs. *Proc Soc Exp Biol Med* 1966;**122**:388–92.

Treatment for renovascular hypertension

Sir,

We read with interest the report of Awazu *et al*[1] that expands the experience with percutaneous transluminal balloon 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

[1] Awazu M, Shimizu M, Hojo H, Osano M, Kohda E. Percutaneous transluminal angioplasty for renovascular hypertension. *Arch Dis Child* 1985;**60**:627–30.
[2] Watson AR, Balfe JW, Hardy BE. Renovascular hypertension in childhood: a changing perspective in management. *J Pediatr* 1985;**106**:366–72.

A R Watson
City Hospital, Nottingham NG5 1PB
and J W Balfe
Hospital for Sick Children, Toronto

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 al*[1] of copper deficiency in four very low birthweight infants.[1] 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,

Table *Serial measurements of copper concentrations (μmol/l) in two studies of preterm infants*

Post-conceptional age (weeks)	*Plasma copper (Glasgow)*	*Serum copper (Belfast)*	
	Geometric mean (n)	*Geometric mean (n)*	*95% range*
30	5·5 (7)		
31	6·1 (16)		
32	5·6 (19)	5·9 (15)	2·8–12·4
33	6·1 (14)		
34	5·5 (17)		
35	6·5 (16)		
36	6·1 (12)		
37	7·3 (12)	7·8 (28)	3·8–16·0
38	7·3 (9)		
39	—		
40	—		
41	9·8 (3)		
42	—	11·1 (29)	6·7–18·4
43	10·2 (7)		
44	—		
45	11·5 (6)		
46	12·8 (2)		
47	13·9 (5)	12·5 (24)	8·1–19·3
48	—		
49	—		
50–54	—	13·5 (21)	9·9–18·4
55–59	—	16·0 (20)	9·1–28·2
60–69	—	16·7 (15)	10·1–27·7
70–79	—	17·5 (25)	10·5–29·2
80–89	—	18·5 (40)	11·2–30·6

Conversion: SI to traditional units—Copper: 1 μmol/l≈6·4 μg/100 ml.

although copper requirements for preterm infants have not been accurately ascertained, we suggest that deficiency is unlikely to occur in infants who are given at least 0·3 μmol of copper/kg/day.

One final point is that Sutton *et al* provided 254 mg of copper/kg/day for three of their infants diagnosed as having copper deficiency. We hope that this is just an unfortunate although hazardous typing error. We believe that they meant 254 μg/kg/day were provided.

References

[1] Sutton AM, Harvie A, Cockburn F, Farquharson J, Logan RW. Copper deficiency in the preterm infant of very low birthweight. Four cases and a reference range for plasma copper. *Arch Dis Child* 1985;**60**:644–51.

[2] McMaster D, Lappin TRJ, Halliday HL, Patterson CC. Serum copper and zinc levels in the preterm infant. A longitudinal study of the first year of life. *Biol Neonate* 1983;**44**:108–13.

[3] Halliday HL, McClure G, Reid M. Fluids and nutrition. In: *Handbook of neonatal intensive care*. 2nd ed. London: Bailliere Tindall, 1985:138–9.

[4] Balistreri WF. Neonatal cholestasis. *J Pediatr* 1985;**106**:171–84.

H L Halliday, T R J Lappin, D McMaster, and C C Patterson
Queen's University of Belfast, Institute of Clinical Science, Belfast BT12 6BJ

Dr Sutton and co-workers comment:

We thank Dr Halliday and his colleagues for bringing their work to our attention.

The appearance in our own article of 254 mg instead of 254 μg in respect of copper dosage (correctly shown as 4·0 μmol) was, indeed, unfortunate, and this error has already been drawn to the editor's attention by ourselves.

Dr Halliday's paper presents data with reference to postnatal age, whereas we consider that the maturity of the infant is important in determining body copper state and that gestational age is, therefore, of more value. The median gestational age of their group at 34 weeks is considerably more mature than our own group.

We would agree that though the WHO/FAO recommended absolute minimum daily copper intake is 0·79 μmol (50 μg)/kg, the evidence for this is limited and may not, in fact, apply strictly to the preterm infant. There are many pre- and postnatal factors that undoubtedly influence the copper requirements of the very low birthweight infant, not least among which is the bioavailability of the copper. While measurement of plasma copper concentration affords some indication of copper state, it would obviously be better if hepatic content and faecal and urinary copper excretions could readily be estimated at the same time. With regard to the biliary excretion of copper, we were under the impression that appreciable cholestasis is now an uncommon occurrence in the very low birthweight infant since the practice of early enteral feeding has evolved.

A recent nutritional study (unpublished) undertaken by certain of our group comparing results obtained using breast milk with copper content 5·8 μmol/l (37 μg/100 ml) with low copper, 0·47 μmol/l (3 μg/100 ml), and high copper, 6·1 μmol/l (39 μg/100 ml), containing formulae milks failed to show any significant differences between the plasma copper concentrations in the infants of either normal birthweight or low birthweight eight to 10 weeks after delivery, irrespective of the feeding regimen used. Various anthropometric measurements also failed to reveal any differences over the period of the study. We believe that while this information may support the contention of Halliday *et al* that 0·3 μmol (19 μg) copper/kg/day is, indeed, sufficient for preterm infants, we shall continue for the present to follow the schedule employing 1 μmol (60 μg)/kg/day as this larger intake has not, to the best of our knowledge, ever been shown to be deleterious. Unfortunately, the authors give no clinical details of the seven infants who managed well with only 0·3 μmol (19 μg) copper/kg/day.

We were gratified to find the results of the measurements of the Belfast workers' plasma copper concentrations were in close agreement with our own findings when the former were related to gestational age and expressed in a form similar to our own. As with many other biological varieties whose distributions are log normal rather than normal, we believe expression of results should, in such cases, be as the geometric mean together with appropriate confidence limits (for example 2·5% and 95·5%) as presentation of values arithmetically as means and standard deviations would be both inappropriate and misleading.