infants they describe are similar, but not identical, to ours, particularly with respect to presence of symptoms, age at diagnosis, and type of nutrient used. Their *x*-ray shows rachitic changes and it is unlikely that marked rib abnormality was present in the absence of respiratory difficulty. We too have found that the serum levels of 25-hydroxyvitamin D (25-OHD_3) were normal in all infants in whom it was measured; the value for Case 3 was 19·5 ng/ml. 3 of 7 other patients with bone disease, but without respiratory distress, also had normal concentrations (range 16·5–34·5 ng/ml).* These findings do not of themselves exclude the possibility of an abnormality in vitamin D metabolism, but do suggest that treatment with 25-OHD_3 is unlikely to be beneficial.

We are confronted by a difficulty however, since radiological changes in Cases 1, 2, and 4 were definitely those found in classical nutritional rickets. Another infant with similar bone disease but no respiratory distress had biochemical changes consistent with rickets, including reactive hyperparathyroidism, which returned to normal after therapy with 1α-hydroxyvitamin D (Glasgow and Reid, 1977). Curiously this child also had a normal level of 25-OHD_3.

Deficiency of trace elements may also be an important aetiological factor. Several authors have described infants with copper deficiency and noted a variety of features, namely costochondral beading, demineralisation, rib fractures, expanded metaphyses, muscular hypotonia, and apnoeic episodes (Al-Rashid and Spangler, 1971; Griscom *et al.*, 1971; Ashkenazi *et al.*, 1973). In addition, however, anaemia was a prominent finding in each of these reports; it was usually of some severity, resistant to iron treatment (sideroblastic anaemia), and associated with erythroid and myeloid hypoplasia. Hypoalbuminaemia also was sometimes present. Any anaemia in our infants was mild with features such as leucocytosis and reticulocytosis which are not consistent with copper deficiency. Plasma proteins were always normal.

The questions which none the less remain to be answered are first, whether the infants we describe with a potentially fatal respiratory disorder have a deficiency of, say, 1,25-dihydroxyvitamin D; and second, is this compounded in some of the lightest and sickest infants by deficiency of trace elements such as copper, manganese, or silicon? Further prospective study of affected babies, careful review of existing histological material, and measurements of trace elements in existing serum samples are required.

References

Alexander, S. W. (1974). Copper metabolism in children. *Archives of Disease in Childhood*, **49**, 589–590.

Al-Rashid, R. A., and Spangler, J. (1971). Neonatal copper deficiency. *New England Journal of Medicine*, **285**, 841–843.

Ashkenazi, A., Levin, S., Djaldetti, M., Fishel, E., and Benvenisti, D. (1973). The syndrome of neonatal copper deficiency. *Pediatrics*, **52**, 525–533.

Forfar, J. O., and Arneil, G. C. (1973). *Textbook of Paediatrics*, p. 1970. Churchill Livingstone, Edinburgh and London.

Glasgow, J. F. T., and Reid, M. (1977). 1α-Hydroxyvitamin D in nutritional rickets. *Lancet*, **2**, 302.

*Kindly carried out by Dr Angela Fairney, St Mary's Hospital, London.

Glasgow, J. F. T., and Thomas, P. S. (1977). Rachitic respiratory distress in small preterm infants. *Archives of Disease in Childhood*, **52**, 268–273.

Griscom, N. T., Craig, J. N., and Neuhauser, E. B. D. (1971). Systemic bone disease developing in small premature infants. *Pediatrics*, **48**, 883–895.

Hambidge K. M. (1976). The importance of trace elements in infant nutrition. *Current Medical Research and Opinion*, **4**, Suppl. 1, 44–59.

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Neonatal records and the computer

Sir,

Readers will no doubt have noticed an error in my paper (*Archives*, 1977, **52**, 452). Owing to a faulty provisional print out from which the data on jaundice were derived, the 1975 entries for 'absent' and 'mild' jaundice and their percentages and ranges were transposed in Tables 3 and 4. The accompanying text should read 'In 1975 as many as 26% (range 17–39%) of 21 030 babies had bilirubin levels between 86 and 204 μmol/l (5 and 12 mg/100 ml). . . .' This change, however, does not alter the significance ($P<0·001$) of the increase in jaundice between 1974 and 1975 as reported. I apologise for this oversight on my part, and trust it has not caused confusion among your readers.

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Neonatal plasma bilirubin chart

Sir,

There is a tendency for phototherapy to be used too often or for too long in the management of babies with neonatal jaundice. The accompanying chart has been constructed in order to give some guidance as to when phototherapy may be indicated. It has been found useful in practice. In term infants plasma bilirubin levels lying below the diagonal line on the chart are unlikely to cause anxiety and do not call for phototherapy. Bilirubin levels above the diagonal line are an indication for a careful decision to be made in individual cases as to what treatment, if any, should be started.

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