received by the hospital, or had not been taken within the recommended time of 6 to 14 days. Of 1000 sequential samples received from the Glasgow area during 4 weeks in 1977, in 4.2% of cases the child was 6 days old when the specimen was assayed, and must therefore have been less than 6 days old when the sample was taken. 93.8% of samples were taken at an 'acceptable' age of 7 to 21 days old; but in the remaining 2% of cases, the child was over 3 weeks old before the test was done; in 0.8% of cases, representing 100 samples per year in Glasgow, or 700 in Scotland, the child was more than 2 months—in some cases, as much as 7 months old.

Obviously in these cases the value of the test is much reduced, with the possibility that permanent brain damage would already have occurred before it was performed. This has two consequences—firstly, doctors who are working with sick young children should be aware that they may not have had inborn metabolic errors excluded by the National Screening Programme; secondly, if neonatal screening is to be extended to other conditions of personal or economic importance, such as hypothyroidism or lead poisoning, a truly fail-safe and universal screening programme would be needed, if it is to justify its cost.

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Early onset of homozygous β° thalassaemia associated with neonatal jaundice

Sir,

I read with interest the paper by Furbetta *et al.* on this subject (*Archives*, 1978, **53**, 250). Although they stated that 'all other common causes of neonatal jaundice had been excluded', the ABO set-up (blood group of baby A and the mother O) which is the most common cause of jaundice in the newborn, was present. In ABO incompatibility, the direct Coombs's test is usually negative. Therefore this possibility should be ruled out before ascribing the neonatal jaundice to thalassaemia.

I should also like to add that among our 90 patients with thalassaemia major (until 1970) pallor and distended abdomen were noticed at birth in two, at 17 days of age in one, at 20 days in one, at 30 days in two, and at about 40 days in two; the observation of splenomegaly as early as 17 days of age in one, might indicate that Cooley's anaemia in this area becomes symptomatic very early as stressed by the authors.

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Professor Cao comments:

The letter from Prof. S. Ozsoylu raises the possibility that the neonatal jaundice could have been due to ABO incompatibility.

However there was evidence of a moderate haemolytic process with Hb level of 13 g/dl in the 5th day, and the jaundice was first noted on the 3rd day, while in ABO incompatibility there is usually little or no evidence of a haemolytic process, and hyperbilirubinaemia appears usually within 24 hours of birth. Moreover, microspherocytosis, a prominent feature of ABO haemolytic disease, was absent.

The direct Coombs's test on the infant's erythrocytes was negative, while in our laboratory, as in others, a weakly positive reaction in ABO haemolytic disease is common. Finally when there is ABO incompatibility it is our custom to test the eluate from the erythrocytes of the affected newborn with A (or B) adult cell. This reaction was negative, and not strongly positive as found usually in ABO haemolytic disease. ABO haemolytic disease cannot be ruled out with certainty in this case but the evidence makes this diagnosis highly improbable.

Prof. Ozsoylu comments that in the population under his care Cooley's anaemia often presented early. In the last year we diagnosed 80 new cases of Cooley's anaemia due to homozygous β^0-thalassaemia. An early presentation with criteria such as symptomatic anaemia (Hb $<$ 7–8 g/dl) at 75-90 days was observed in 30% of cases, but the severe anaemia (Hb $<$ 6 g/dl) at 60 days as seen in our patient was rather uncommon. Splenomegaly in cases with earlier presentation is an unusual feature in this area. The age and clinical findings at presentation in Cooley's anaemia varies in different ethnic groups. This may be due to the genetic heterogeneity of the disease and different environmental conditions.

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Successful restoration of immunity in the DiGeorge syndrome with fetal thymic epithelial transplant

Sir,

I read with interest the paper by Thong *et al.* on this subject (*Archives*, 1978, **53**, 580). The authors stated that correction of the hypocalcaemia was achieved by the use of

AT10 (vitamin D2); in which AT10 was considered as equivalent to vitamin D2. AT10 (dihydrotachysterol) is quite different from vitamin D2 which is ergocalciferol. Although both compounds, after their hydroxylation in the liver and kidney would be effective on serum calcium levels, they differ in that AT10 may be rachitogenic in long usage but vitamin D is the compound recommended for the prevention and treatment of rickets.

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Drs Thong and Robertson comment:

We thank Professor Ozsoylu for his correction of our error. AT10 (dihydrotachysterol) is a reduction product of vitamin D and differs from vitamin D in the geometrical configuration of the A ring. This product has been used in the treatment of hypoparathyroidism for many years, and is preferred to calciferol by some because of its more rapid onset of biological activity and its more rapid excretion (Root and Harrison, 1976). We have seen no evidence of a rachitogenic effects in patients with hypoparathyroidism treated with either dihydrotachysterol or calciferol, nor have we seen such a report in the literature.

Perhaps Professor Ozsoylu's concern is based on the *in vitro* work of Trummel *et al.* (1971) and Reynolds *et al.* (1973) who showed a direct effect of both dihydrotachysterol and its 25-hydroxylated metabolite, 25-hydroxy-dihydrotachysterol on bone resorption in culture, which was greater than the effect of 25 hydroxycholecalciferol.

Both calciferol and dihydrotachysterol have been used effectively in the long-term management of hypoparathyroidism. Since, in the absence of parathyroid hormone, the production of the most active form of vitamin D, 1, 25-hyhidroxycholecalciferol is impaired, a more rational form of therapy would be this product or its synthetic analogue, 1α-hydroxycholecalciferol which have been shown to be effective. These products are not yet generally available (Russell *et al.*, 1974).

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