Maternal histidinaemia

Sir,

Your correspondent, M. D. Armstrong (*Archives*, 1975, **50**, 830), asks the right question, namely, 'Are children born of histidinaemic mothers at risk of harm during gestation?' but does not provide anything other than anecdotal evidence in his attempt to answer it. The children of our 37-year-old woman with histidinaemia were exactly like his, i.e. 'None had shown any signs of slow or abnormal development during infancy or childhood. . .'. We suspected nothing but did get full-scale IQ measurements done on both parents and the 4 older children.

As reported in our paper (Lyon, Gardner, and Veale, 1974), the children's mean IQ was about 20 points less than the midparental value, a finding which came as a complete surprise to us. Before Dr. Armstrong concludes that maternal histidinaemia is not harmful to the offspring, we feel he should make the appropriate investigations. Apparent normality of the children is not sufficient grounds for concluding that maternal histidinaemia is harmless.

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Administration of parenteral iron to newborn infants

Sir,

Scott *et al.* (1975) suggest that the possibility of reducing the anti-infective role of unsaturated transferrin may caution the use of iron by the parenteral route in newborn infants. Our clinical and laboratory experiences support their suggestion. A policy of giving infants considered to be at risk of iron deficiency anaemia 2 ml of iron dextran complex intramuscularly during the first week of life was adopted at the National Women's Hospital, Auckland, in 1970. At the same time a similar prophylactic treatment of low birth-weight infants was advanced from later than the first month of life to as early as the first week. There are about 5000 deliveries annually at the hospital and before 1970 there had been an average of 1-2 cases of *Esch. coli* meningitis annually. In 1971 and 1972 there was a total of 21 cases of *Esch. coli* meningitis, 18 of which arose within 2-5 days of the administration of iron dextran (Farmer, 1973). The incidence of *Esch. coli*

meningitis returned to the previous level when the treatment of term infants was made more selective and treatment of low birthweight infants was either discontinued or given only after 1 month of age. A similar experience with *Esch. coli* meningitis and septicaemia caused Barry and Reeve (1973) to abandon another programme of prophylactic administration of iron to newborn Polynesian infants.

We have studied blood taken from infants before and 24 hours after administration of iron dextran, and have confirmed a diminished bacteriostatic activity against *Esch. coli* accompanying very high iron levels in the post-treatment serum (Becroft, Dix, and Farmer, unpublished observations, 1976). No change was detected in the bactericidal and chemotactic properties of blood phagocytes nor in serum components concerned with opsonization and chemotaxis.

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Dr P. H. Scott and co-workers comment:

Thank you for allowing us to comment on the letter from Dr. Farmer and Dr. Becroft. The antibacterial effect of iron binding proteins, whether in blood or milk, has been mainly demonstrated *in vitro* or in small groups of animals; epidemiological evidence concerning the significance of this mechanism in preventing infection during early childhood has been scanty. Their records are therefore valuable.

It seems that parenteral iron should be used only when it is essential and always with caution, particularly in children with low plasma concentrations of transferrin, such as the newborn, the malnourished, and certain protein losing states.

We wonder about the effect of oral iron which we give to preterm babies. Few such babies, however, achieve a high saturation of transferrin with iron (Brozović *et al.*, 1974).

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