NEONATAL PLASMA IRON FOLLOWING TOTAL DOSE INFUSION OF IRON DEXTRAN TO MOTHERS

by

U. N. PATHAK*, D.G.O., M.M.S.A., M.R.C.O.G.,

During pregnancy, there is selective transplacental transport of iron from the mother to the foetus against a concentration gradient. Normally, this transfer is proportional to the size of the foetus and occurs at the expense of the maternal bone marrow, Bothwell *et al* (1958). But in maternal iron deficiency states it has been shown that although the process of iron transfer to the foetus is unchanged, both in humans and in experimentally induced iron deficiency in the rat, there is increased iron deficiency (Guest and Brown, 1957 and O'Dell *et al*, 1960). On the other hand there is evidence suggesting that there is no preferential gain of iron by the foetus at the expense of the mother with iron deficiency (Dawson and Desforges, 1958 and Sturgeon, 1950).

This problem is of considerable practical importance in a population having a high incidence of iron deficiency, as may occur in tropical countries such as Jamaica. In an attempt to correct maternal iron deficiency, intravenous infusion of iron-dextran has been found to be effective therapy, as reported previously (Pathak, 1966; Pathak *et al*, (1967). Further studies of this method have included observations on the effect of these maternal iron infusions on the foetal iron status. This paper presents the results obtained in the study with a discussion of their bearing on the transplacental passage of iron in iron deficient mothers.

Material and Methods

All the patients studied were Negroes attending the antenatal clinics at the University Hospital of the West Indies, Kingston, Jamaica. The patients were divided into two groups:

Group I—This included patients who maintained satisfactory haemoglobin levels during their pregnancy by use of routine oral iron supplements (510 mg. compound ferrous sulphate, B.P.S. in tablet form).

Group II—This consisted of patients matched for age and parity with those in the first group, who showed an iron deficient anaemia, in spite of oral iron supplements, at the 32nd week of their pregnancies. This diagnosis was made after an examination of a blood film, bone marrow aspiration biopsy, serum iron and total iron binding capacity estimations. These patients were treated with iron dextran complex, administered as total dose infusion. The details have already been described (Pathak, 1966; Pathak, 1967; Wood, *et al*

*Senior Lecturer, Dept. of Obst. & Gynec. University of the West Indies, Jamaica.

Received for publication on 1-7-1969.

TABLE I
Summary of clinical material

Groups	No.	Mother: Parity 0	Parity 1-4	Parity 5+	Hb. value in Gms. Mean	Hb. value in Gms. Initial	Hb. value in Gms. Pre-drip	Hb. value in Gms. Term	Hb. value in Gms. Post-natal	Newborn: No.	Sex M	Sex F	Weight in Lbs. Min.	Weight in Lbs. Max.	Weight in Lbs. Mean
I Non-treated	25	6	15	4	3.4	10.8	11.1	11.2	11.2	25	13	12	6—0	8—2	7.1
II Treated	25	6	15	4	3.7	10.6	9.3	11.2	11.7	25	14	11	6—0	8—3	7.2

1968). These infusions were given between 32-36 weeks of gestation, and patients did not receive any further oral iron for the rest of their pregnancy.

It is almost certain that the diet in almost all the patients in their group fell short of the basic requirement. Both groups had vitamin supplements prescribed as a routine. No premature babies were included in the series. There were two sets of twins in Group II.

Laboratory Procedure

Soon after delivery of the placenta, 5 ml of blood was collected from the umbilical vein, using a disposable syringe and appropriate containers. No attempt was made to strip the cord. Specimens with gross clots or haemolysis were discarded. The following determinations were made on the samples: haemoglobin (Hb); packed cell volume (PCV); mean corpuscular haemoglobin concentration (M.C.H.C.); serum iron (S.I.); total iron binding capacity (T.I.B.C.); percentage saturation (Sat. per cent). Serum iron estimations were done by the method of Beale *et al*, (1961, 1962). Haematological values were estimated by routine methods (Dacie and Lewis, 1963).

Results

Some of the pertinent clinical data for the two groups of patients studied is shown in Table I. The ages ranged from 18 to 41 years and they were matched for parity. It will also be seen that sex and birth weight range of the infants were very similar. Table II gives the mean values and ranges of the various estimations of iron status made in both groups. It is in-

TABLE II
Comparison of results

Estimates	Group I		Group II	
	Mean	Range	Mean	Range
Hb. Gm. per cent	15.6	13.6-17	15.4	13-18.2
Red cells /c.mm.	4.57	4-6.1	4.8	4.1-6.1
P.C.V.	50.5	45-53	49.5	45-53
M.C.H.C. per cent	30.9	29-32	31.5	29-32.8
Serum iron mg./100 ml.	187	150-210	193	154-2.8
T.I.B.C. mg./100 ml.	231	200-250	249	200-265
Saturation per cent	87.3	70-95	86.5	70-98

teresting to see that the mean values in the two groups are remarkably similar. The improved iron status of the mother in the treated group did not seem to make any significant difference to the parameters examined.

Discussion

Plasma turnover in the pregnant woman reflects the needs of the placenta and the bone marrow. Following the intramuscular administration of iron dextran, iron uptake in both these areas is active but the foetus shows higher iron storage (Pribilla, 1958). No increase in iron content is seen in the placenta or membranes (Pathak *et al.*, 1967). It is likely that the bulk of iron storage probably occurs in the foetal liver and bone marrow as it happens in adults (Wood *et al*, 1968). Direct confirmation of this is difficult unless supported by observations at autopsy. The present study shows that the cord blood measurements of the various indices associated with iron metabolism are similar in both 'normal' mothers and those treated for iron deficiency anaemia. Even if the treated mother remains iron deficient the foetus appears to have normal blood values. On the face of it, this suggests that there is in fact a preferential absorption of iron across the placenta to the foetus. However, there is an alternative explanation. Even if the blood values are normal the total circulating haemoglobin mass may still be less in the infants born of mothers with iron deficiency. If so, then the body stores are less and the serum iron concentration might be expected to fall in the weeks immediately following birth. There is some evidence to show that this does occur and by as much as 40 per cent in some cases (Sisson and Lund, 1958). Similarly, infants of successfully treated mothers may have better iron storage at birth, even if this is not immediately reflected in the parameters we have measured in the circulating blood. If this inference is correct it is of practical importance in premature or multiple pregnancy, where the foetus is notorious for its deficient iron storage. Such babies will not and in our experience do not show any fall in their haemoglobin levels in early infancy or early childhood.

Sturgeon (1959) found no appreciable difference in the 6-12 month and 18 month old infants by supplementing the maternal diet with iron in the antenatal period. He concluded that iron deficiency of early

childhood results from environmental factors after birth, common both to the mother and the baby, and not the maternal anaemia in the antenatal period. There is only one criticism of the observation that the recovery of iron stores in anaemic patients following oral iron is not as satisfactory as following total dose infusion. Therefore, in a community like Jamaica where the diet is poor in iron content, improving the maternal iron status in the antenatal period may protect the foetus from developing the anaemia of the late infancy and childhood. Only a long term follow-up of these babies will provide a definite answer.

Summary

Cord blood samples were collected soon after delivery of normal non-anaemic mothers and those who developed anaemia in the antenatal period but were successfully treated with Iron-Dextran Complex given as total dose infusion.

The two groups comprised of 25 patients each, matched for age and parity. Various haematological determinations made failed to reveal any significant difference in the two groups. A suggestion is made that in spite of this, the babies of treated mothers may have better iron stores and thus be less prone to developing anaemia of late infancy.

References

1. Beale, R. N., Bostrom, J. O. and Taylor, R. F.: J. Clin. Path., **14:** 488, 1961.
2. Beale, R. N., Bostrom, J. O. and Taylor, R. F.: J. Clin. Path. **15:** 156, 1962.
3. Bothwell, T. H., Pribilla, W. F., Mebust, W. and Finch, C. A.: Am. J. Physiol. **193:** 615, 1958.
4. Dacie, J. V. and Lewis, S. M.: Practical Haematology, ed. 3 London, 1963, Churchill.
5. Dawson, J. P. and Desforges, J. F.: A.M.A.J. Dis Child **96:** 169, 1958.
6. Guest, G. M. and Brown, E. W. A.M.A.J. Dis. Child, **93:** 486, 1957.
7. O'Dell, B. L., Hardwick, B. C. and Reynolds, G.: J. Nutrition, **73:** 151, 1961.
8. Parrish, F. M. and Brame, D. D.: Am. J. Obst. & Gynec. **68:** 589, 1954.
9. Pathak, U. N.: J. Obst. and Gynec. India, **16:** 1, 1966.
10. Pathak, U. N., Wood, J. K. and Sorhaindo, B. A.: Obst. and Gynec. **29:** 500, 1967.
11. Pribilla, W. F., Bothwell, T. H. and Finch, C. A.: Iron Transport to the Foetus in Man: Iron in Clinical Medicine, Univ. of California Press 1958, p. 58.
12. Sisson, T. R. C. and Lund, C. J.: Am. J. Clin. Nutrit. **6:** 376, 1958.
13. Sturgeon, P.: Brit. J. Haematol **5:** 31, 1959.
14. Wood J. K., Milner, P. F. A. and Pathak, U. N.: Brit. J. Haematol, **14:** 119, 1968.