STUDIES IN THE ANÆMIAS OF INFANCY AND EARLY CHILDHOOD
(From the Children's Hospital and the Department of Diseases of Children of the University, Birmingham).

Part VI.—Nutritional anæmia in mother and child

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The physiological relationships which exist between the mother and her child during pregnancy and lactation are intricate and complex. The normal material requirements for the adequate growth of the foetus in utero are various, but under healthy conditions are remarkably constant and are met by placental transmissions. The fact that nature also demands that the mother shall transfer to the foetus, particularly during the latter months of the pregnancy, certain materials to provide stores available for use during the early months of extra-uterine life, is a further example of the natural dependence of the infant upon its mother. On occasion these processes act adversely on the mother, and well recognized clinical disorders may be either produced or rendered apparent. The adverse effect of pregnancy in the osteomalacic mother is a clear though rare example of this, and it is now realized that examples in relationship to the blood are more common.

The disorders of the blood produced by pregnancy vary considerably in type and degree, simple hypochromic anæmia being of relatively common occurrence, whereas the more serious megalocytic anæmia of pregnancy fortunately is rare. In the hypochromic anæmia of pregnancy it is probable that transference of the maternal reserve of haemopoietic substances to the foetus is an important ætiological factor, and this is the more evident in the pregnant mother who through previous or present nutritional defect probably has poor reserves of iron, etc. Rapidly repeated pregnancies or multiple pregnancies are also contributory causes. Strauss and Castle1 have shown that another important factor is the occurrence of gastric anacidity or hypoaacidity. These writers also think that the megalocytic anæmia is due to a temporary lack of the specific intrinsic factor in the gastric juice, and that lack of the extrinsic factor from the diet may produce the same effect. Finally, they produce evidence to show that both varieties of anæmia may
exist in the same woman. Prescription of iron-containing foods or of medicinal iron, including copper, in adequate amounts during pregnancy will prevent the development of hypochromic anaemia; and an adequate diet, including yeast or marmite, and a potent preparation of liver, is therapeutically valuable in the megalocytic anaemia of pregnancy. It is, therefore, an important part of ante-natal supervision to enquire into the state of the maternal blood and to aim at the provision of essential haemopoietic substances in adequate quantity in the dietary of the pregnant person. Several clinical workers have observed the ease with which maternal nutritional anaemia is produced in pregnancy; its prevention is usually also equally simple.

Anaemia in pregnancy may have far-reaching effects upon the fœtus in utero. Clinical observations which we have made show that nutritional anaemia may show itself in the new-born child, or become apparent during the milk-feeding period of early life. In our view there appears little doubt that a congenital anaemia may be a true nutritional anaemia, and that it may be due to deficiency in the maternal diet. The transference of haemopoietic substances from the mother to the fœtus is progressive throughout pregnancy, and is considerably increased in the later weeks. This transference of iron and its allied haemopoietic substances occurs in order to make provision for foetal haemopoiesis and to accumulate a store (principally in the liver) which will be available during the milk-feeding period of early life. It is recognized that such foetal storage principally occurs in the latter weeks of the normal full-term pregnancy. It is, therefore, clear that if the total available quantity of iron, etc., in the maternal tissues is insufficient, there will be a deficiency of these substances in the developing fœtus, and a condition of simple anaemia may be expected to be present in the infant at or soon after birth. According to the majority of authorities infants born of anæmic mothers have normal blood pictures at birth: thus Baar and Stransky\(^2\) state that anaemia of the mother does not produce anaemia in the new-born child, a statement with which Strauss and Castle agree.

We believe that this conception is erroneous, although an infant born of an anæmic mother is not necessarily anæmic. We base our belief partly on the evidence detailed in Part II of this series (p. 95) showing that nutritional anaemia can develop shortly after birth in rats whose mothers have been apparently cured of nutritional anaemia by yeast; and partly on the clinical evidence that we submit in the present paper.

The metabolic processes of calcium and iron are very similar, but there does appear to be one striking difference between them. The mother cannot retain calcium at the expense of the fœtus, but has to surrender it even if in doing so she contracts osteomalacia: whereas the mother can, and sometimes does, retain iron to the detriment of the fœtus. In the paper by Strauss and Castle to which reference has already been made, there is a chart
giving a graphic representation of the haemoglobin percentages of the new-
born infants of fourteen anaemic mothers. We have calculated out these
haemoglobin values and find that they range from 21.48 grm. per 100 c.cm.
(15.4 per cent. Haldane scale) to 12.5 grm. per 100 c.cm. (91 per cent.). These
results were obtained during the first few days after birth since there is
normally a rapid decrease in haemoglobin and erythrocytes in the first weeks
of life. Mackay has recently pointed out that the normal haemoglobin of
the new-born child is about 140 per cent. on the Haldane scale (193.3
grm. per 100 c.c.). Using this standard there is evidence of at least one anaemic
baby in the series of Strauss and Castle, despite their statement to the
contrary. Moreover, there is definite evidence that in later infancy the
children of anaemic mothers may show anaemia: thus in examining a series of
pregnant women Mackay found nine whose haemoglobin was between 54
and 70 per cent., and the babies of these pregnancies showed a lower
haemoglobin level at every month for the first six months of life than the
babies of non-anaemic mothers. Again, Strauss found that infants who had
not received treatment, and whose mothers were not anaemic during
pregnancy, had an average haemoglobin of 67 per cent.; whereas those whose
mothers were definitely anaemic had an average haemoglobin of 46 per cent.

The condition of the blood in these cases of simple nutritional anaemia of
infancy is that of a hypochromic anaemia and has been fully described in a
preceding paper (Part III, p. 117). Intercurrent infection may cause some
change in the blood picture and immature forms of white cells may be seen
on such occasions. Enlargement of lymphatic glands is absent, but the
spleen may be palpable. The general nutrition of the baby may be otherwise
good.

It is, therefore, a matter of interest to study cases of anaemia in the
infant which are related to the maternal state, especially as treatment, both
prophylactic and curative, is as a rule highly successful. In the present
paper some of the examples which we have encountered are recorded and
discussed.

In our series of cases nutritional anaemia has occurred in mother and/or
infant (in some instances, in twins) in all possible combinations and
permutations which can be represented as follows, where N stands for
normal, and A for anaemic:—

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Examples of all these variations are recorded in the text.
Anæmia in twin births.

Consideration will first be given to a number of instances of anæmia in twins: in some the mother was anæmic, and in others the mother was not anæmic.

Twins anæmic: mother anæmic.—We give first two examples of the twins of anæmic mothers showing anæmia.

Family 1: (Chapman).—The mother had had three pregnancies in six years: (1) a girl; (2) twins, both male: one died, no known anæmia; (3) twins, male and female (the patients).

These infants came under observation in the first instance because the mother brought the male twin, Gordon, to hospital complaining that he was pale. The mother regarded herself as being in good health, although it was quite obvious that she had anæmia. The other twin, Greta, was considered to be healthy and was only brought to hospital on request.

Mrs. C., aged 30. Never any serious disease. Had always taken a normal mixed diet. During and after the last pregnancy, however, she had not felt in good health and had at times considerable shortness of breath. Treatment by means of 20 grn. doses of iron and ammonium citrate, thrice daily, and ordinary diet resulted in cure.

Gordon C. Brought to hospital at the age of 5 months. Normal birth. Full term. Birth weight, 4 lb. Slight icterus neonatorum for a week, never hemorrhage. Breast fed one month and then Grade A milk and rusks. When seen at hospital, good general nutrition; weight 14 lb.; very pale, no enlarged glands, no abnormal visceral signs, spleen not palpable, liver normal; no rickets.
ANÆMIA IN MOTHER AND CHILD

Treatment consisted in giving 10 grn. doses of yestamin and 5 grn. doses of iron and ammonium citrate thrice daily. The child during that time was given cow’s milk.

Greta C. (twin to Gordon). Birth weight 4 lb. Same history, previous to first hospital observation, and treatment given was similar except that in this case yestamin was not given.

The condition of the blood in the members of this family at the time they came under observation and the improvement under treatment is shown in Graph I. It will be seen that the mother and Greta were given iron, and Gordon (the more anemic of the twins) was also ordered yestamin. In the course of twelve weeks all three became normal, and in excellent clinical condition. A recent observation found the children and mother in good health. The good nutritional state of the mother and twins apart from the anæmia was remarkable. The possible effect of recurrent twin pregnancies as a factor in the hypochromic anæmia of the mother is suggested. The efficacy of the iron preparation, which contains copper, indicates that the cause of the anæmia was a simple deficiency of these essential substances. Probably the storage of iron in the maternal tissues before and during pregnancy was low, and a further depletion consequent upon the foetal demands was sufficient to create the anæmia. The division between the twins, in utero, of what was probably a subnormal transplacental supply of iron is an adequate explanation of their hypochromic anæmia.

Family 2: (Allmark).—In this case also, the mother, although obviously anemic, had sought no medical attention for herself, but brought her remaining twin to hospital because of his pallor and weakness.
Mrs. A., aged 38 years. Had six children in twelve years: four single pregnancies, and the last a twin pregnancy. During this last pregnancy she had been far from well with swollen legs, and was said to have nephritis of pregnancy. On a low diet all through the later months. Seen at hospital 12 months after the pregnancy and noticed to be pale, poorly nourished and weak. Urine showed a faint haze of albumin, but no cells or casts. Blood urea 42 mgm. per cent. Wassermann reaction negative. The condition of her blood and the subsequent effects of iron and yeast therapy are shown in Graph II.

On December 17th, 1931, she was ordered 1 dr. of yestamin daily, but on January 1st, 20 grn. doses of iron and ammonium citrate, t.d.s., were substituted for this.

The twins. One died at the age of 10 months with diarrhoea. Both were pale, even as young babies. No icterus neonatorum. Full term pregnancy; normal labour. Harold A. (surviving twin). Birth weight 4 lb. Fed on Cow and Gate milk, and later bread, butter, potato and gravy. At the first observation at hospital the infant weighed 18 lb.; pale and weak, constipation, no infection. No signs of visceral abnormality. No glandular nor splenic enlargement; no icterus; no rickets. Graph II shows the result of treatment.

The clinical improvement (see Graph II) in mother and twin was marked. In this case there is a history of defective nutrition during pregnancy which was probably responsible for the development of maternal anaemia; further, true congenital anaemia appears to have been present in the twins. The mother and surviving child are now well.

Twins anaemic: mother normal. The following cases are examples of anaemia in the twins and normal health in the mother; nevertheless, the nature of the anaemia is essentially the same as in the previous instances.
ANÆMIA IN MOTHER AND CHILD

Family 3: (Salmon).—Mrs. S. Healthy. Aged 27 years. Two previous single pregnancies and normal babies. Has had four children (including the present twins) in 8 years. During the twin pregnancy she had a little oedema of the legs. No anaemia. Full varied diet and always felt well and strong; when first seen at hospital 7 months after the pregnancy she was in excellent health and showed normal blood. Twins, normal labour.

Olive S. Full term. Birth weight 4 lb. Was very pale at birth and the mother says she didn’t think it was strong enough to live. Never any haemorrhage. No icterus neonatorum. Fed on Grade A cows’ milk. Brought to hospital because of general weakness at the age of 6 months, and then weighed 11 lb. Observed to have considerable pallor without any visceral disease or glandular or splenic enlargement and no signs of any infective process. Graph III shows the blood condition, treatment and progress. Gained 10 lb. in weight during treatment.

Kathleen S. Birth weight 4½ lb. Same food as twin sister. This child was brought up for observation at our request at the age of 11 months and found to be extremely pale. Weight 19 lb. 1 oz. No glandular nor visceral enlargement. Had been pale since a ‘very small baby’ and become progressively whiter. There had been no icterus at any time. Occasional simple diarrhoea. No evidence of any infection. Weight increased 7 lb. during treatment by reduced iron.

![Graph IV.—Twins of Family 4.](image)

As indicated in Graph III each child showed rapid improvement, and it is interesting to observe that although each twin at the commencement of treatment had a good count of red corpuscles the haemoglobin level was low. The rapidity of the increase in haemoglobin production in Kathleen S. on reduced iron was remarkable, there being a rise from 30 to nearly 80 per cent. in about six weeks, with corresponding improvement in her general condition. It is clear that the cause of anaemia in these twins was an intra-uterine deficiency in iron and possibly other haemopoietic substances. In the previous single pregnancies none of the children had shown any infantile anaemia. The twins are now in excellent health.
Family 4: (Chamberlain).—In this family the parents both came to hospital and were obviously in perfect health, whereas each twin was remarkably pale.

Mrs. C., aged 28 years. Always in first class health; excellent colour. Blood normal. Living on full varied diet. This twin pregnancy was her first. The twins were born at 8 months; labour normal.


Both babies pale at birth but no icterus observed. At 3 months their pallor was recognized by the parents and became more marked, although no medical attention had been sought. Both boys were brought to the hospital for the first time at the age of 12 months, obviously suffering from anaemia with weakness, shortness of breath and anemic anoxemia. Their general nutrition, however, was not bad since John had reached 16 lb. and David 14 lb. 14 oz. The boys were so seriously ill that immediate admission to the hospital was advised. There were no haemorrhages nor purpura, and the retinae were clear. Each had been breast fed for one month, and then changed to a dried milk with occasionally rusks, custard and beef tea. Soon after admission each child was given a blood transfusion.

The condition of the blood is shown in Graph IV and a record of the progress in hospital and medicinal therapy is indicated. This was one of the earliest cases of the series in which the direct use of iron and copper salts was made. A weak solution of ferric chloride in milk was given, together with 0.75 mgrm. of copper sulphate. The use of these substances tended to cause diarrhoeal disturbance, but the red blood cells and haemoglobin increased. Liver extract was utilized at one point and appeared to have acted as an added stimulus to erythrogenesis. The exhibition of iron and ammonium citrate, however, caused a rapidly progressive
increase of haemoglobin and finally, after a total period of about 16 weeks, the blood had assumed a nearly normal level. Each boy has been examined again recently, and their health is perfect. John weighs 35 lb. and David 34 lb. (aged 2½ years).

The aetiological importance of prematurity and twinning in the rapid development of hypochromic anaemia is exemplified by these infants. The lack of full-term development and even more important the failure to receive the specially abundant supply of iron, etc., which occurs in the ninth month of gestation, appear to have caused the rapid development of anaemia. Physical growth after birth was not materially delayed; in fact the progressive increase in weight doubtless accentuated the effects of the anaemia. It is not known if these were identical twins, or what the placental arrangement was.

![Graph VI.—Anaemic twin in Family 6.](image)

**One twin anaemic; other twin normal; mother normal.**—The following two cases are instances of anaemia occurring in only one of a pair of twins.

**Family 5:** (Bate).—Mrs. B., aged 25 years. First pregnancy single child 18 months before the birth of twins. Always in excellent health and never anaemic. Quite normal during twin pregnancy and lived on a full diet. Twins: normal labour; full term; both boys.

Malcolm: Birth weight 6 lb. 8 oz. Excellent colour at birth and has remained perfectly healthy. Blood normal on examination.

Peter: Birth weight 5 lb. 2 oz. Pale at birth. No icterus. Brought to hospital at the age of 4 months because of marked pallor and weakness. No enlarged spleen nor lymph glands. Each twin was fed in exactly similar manner on cows' milk.
The progress under treatment of the anæmic twin is shown in Graph V. The remarkably low haemoglobin value and red blood cell count on initial observation is in extreme contrast with the normality of the other twin's blood. The same milk diet was continued in hospital and ferrum redactum, grn. 3, daily, was added. The response was rapid and progressive and in about 12 weeks the blood was normal. The boy now weighs 22 lb., but has not quite reached the weight of his normal twin brother Malcolm.

There is practically no doubt that this was a case of nutritional anæmia in the new born. An unequal distribution of haemopoietic substances to the developing twins in utero is evident. The anæmic twin was also 1½ lb. smaller at birth. In this case there was gross defect in the number of red blood cells combined with an extremely low haemoglobin.

Family 6: (Hewitt).—Mrs. H., aged 38 years. Two pregnancies in 16 years. Always good health, never anæmic; normal pregnancy; blood normal.

Denis: Birth weight 6 lb. 6 oz. Always in excellent health; normal birth; full term; blood normal.

Doris: Birth weight 5 lb. 1 oz., breast fed. At three months pallor evident. When seen at hospital, aged 10 months, very pale, weak, and irritable. No enlarged glands; spleen just palpable. No infection. Never any icterus. Blood count showed an adequate number of red blood cells but the haemoglobin was only 34 per cent. Treatment effected a rapid improvement and after 14 weeks the haemoglobin had risen to 82 per cent. Graph VI illustrates the clinical course. At the age of 20 months she weighed 28 lb. and was in perfect health.

This is a typical case of severe hypochromic infantile anæmia in one twin, the other being from birth onwards free from any blood abnormality. The anaemia was due to defective haemoglobin formation consequent upon low iron storage at birth. Again the latter phenomenon was evident in the smaller twin. Since the pregnancy was carried to the full term no cause can be adduced other than an inadequate transfer, or the possibility of markedly unequal distribution, of iron to the foetuses from the healthy mother.

Anæmia in single births.

Infant normal; mother anæmic.—The picture presented when a severely anæmic mother is seen carrying her healthy good-coloured baby is an arresting one and immediately arouses the suspicion that nature has attempted to leave the infant in good condition at birth irrespective of maternal health. Such a process holds with remarkable constancy in healthy babies born of tuberculous mothers, true congenital tuberculosis being an extremely rare occurrence. In the following cases a healthy baby was born of a severely anæmic mother.

Family 7: (Davis).—Mrs. D., aged 37 years. Said to be always pale and when first seen at hospital she was pale and weak but of moderate general nutrition. She had developed more marked pallor during the pregnancy which otherwise had been normal. No renal disorder nor any evidence of systemic disease. No splenic nor glandular enlargement and no signs of infection. No haæmorrhages. Examination of the blood revealed less than 3,000,000 red blood cells per c.mm. and haemoglobin 35 per cent., giving a colour index of 0.6. Microscopically the red cells showed no marked aberration, were normal in size and no immature forms were present. Leucocytes normal. Advice was given about her diet and iron and ammonium citrate, 1 drm. daily, prescribed, with the result that there was a moderate rise in red
ANÆMIA IN MOTHER AND CHILD

cells and haemoglobin. Three weeks later yestamin was also added and a marked acceleration of the haemopoietic activity occurred so that at the end of a further period of four weeks an improvement was clinically obvious. One month later again the cure was definite and the mother said she had never had such a good colour nor felt so well.

Eric D., born a month prematurely, 5½ lb. at birth. He was a normal colour and has remained so, in sharp contrast with the mother's pallor. First seen at hospital at 4 months when he had reached 9 lb. in weight and appeared physically well. Feeds had consisted entirely of Grade A milk. The blood count showed 4,900,000 red cells per c.mm. and 85 per cent. haemoglobin. Leucocytes and platelets normal.

The result of the blood examination of mother and baby are shown in Graph VII. The fall in the baby's haemoglobin after the 30th day was due to coryza and bronchitis.

This case shows the interesting phenomenon of normal haemopoiesis in a baby born of a mother suffering from hypochromic anæmia preceding and during pregnancy. The adequate transfer here of haemopoietic substances in utero, is even more remarkable when it is understood that the baby was four weeks premature. This is, perhaps, the most striking case which we have seen as a clinical example of adequate transplacental nutrition and foetal storage of essential blood-forming substances at the expense of the mother: the latter being sufficient to increase the maternal anæmia during the pregnancy. The simple nutritional cause of the hypochromic anæmia is indicated by the rapid cure which followed an adequate supply of iron preparation. Yeast acted as an extra stimulant and illustrates the advantage which is sometimes seen when the two substances are prescribed together.
Infant anaemic: mother anaemic.—The following is a very good example of anaemia in mother and child.

Family 8: (Prately).—Mrs. P., aged 36 years. Gave a history of pallor since aged 12 years, worse during past few years. Had always taken an ordinary diet, but rarely any meat foods. She had borne four children in 6 years, the present patient being the fourth. The first baby died of anaemia, the second and third were healthy. She had no haemorrhage. Menstruation normal. No dyspepsia. Had always been an average weight. She came under hospital observation when she brought the baby Dorothy, aged 1 year 11 months, for treatment, and appeared extremely pale and of poor physique. She had not sought any advice for herself and was surprised when it was suggested that she should also become a patient. A blood examination revealed red blood cells 3,500,000 per c.mm. and only 18 per cent. haemoglobin; colour index 0.25. The red cells were normal in size and no immature forms were present. There were no glandular enlargements, splenic nor hepatic abnormality, and no icterus. No signs of any infective process. When first seen was ordered 1 drm. of yeast daily and 20 grn. of iron and ammonium citrate, twice daily.

Dorothy P., normal birth, full term. Birth weight 3 lb. Only weighed 5½ lb. at eight weeks. Fed on Ostermilk and cow’s milk, and later bread, butter, potato and occasional gravy. She had been pale since birth. On first examination at the age of 23 months she was extremely pale and asthenic; no glandular nor splenic enlargement, and no evidence of avitaminosis; no purpura, nor haemorrhage. Blood Wassermann reaction of mother and child negative. The blood, as in the case of the mother, showed only 18 per cent. haemoglobin and red blood cells 3,080,000; colour index 0.25. Microscopically the erythrocytes were free from any abnormal features.
ANÆMIA IN MOTHER AND CHILD

regarding size, shape or nucleation, and only 0·8 per cent. reticulocytes were present. When first seen she was ordered 15 grn. of yeast daily, and 5 grn. of iron and ammonium citrate, thrice daily.

It is therefore evident that both mother and child were suffering from a severe grade of simple hypochromic anæmia, clearly of nutritional origin in the mother. In the child the anæmia was related to congenital malnutrition, and intensified by inadequate feeding during the latter year of her existence. Medicinal treatment was immediately given, the child being admitted for this purpose and the mother attending regularly in the out-patient department. Graph VIII illustrates the progress in each instance.

The therapeutic response is specially noteworthy. During the first four weeks the baby increased her red cells from 3,080,000 to 5,010,000, and haemoglobin 18 to 70 per cent. Mrs. P. showed similar but less rapid haemopoiesis and a reticulocytosis to 5 per cent. After 12 weeks treatment with an iron preparation and dried yeast the patient appeared clinically cured, and soon afterwards the respective blood counts were quite normal. There was coincidentally an increase in the baby's weight of 5 lb. in about 12 weeks and a corresponding improvement in general health. Both are now (one year after first attending hospital) in perfect health.

The intense degree which simple hypochromic anæmia of nutritional origin may reach is demonstrated in this family. The prolonged anæmia in the mother was considerably intensified during the pregnancy owing, doubtless, to the further withdrawal of maternal haemopoietic substances by the foetus. Few infants have entered life in a more pathetic state of health, weighing only 3 lb. (at full term), and being very ill supplied with the requisite substances for its blood formation, an effect continued by the poor dietary until attendance at hospital. The ultimate result, however, shows how well even the advanced case will respond to iron medication.

Conclusion.

The biological problems discussed in this paper once more emphasize the value of adequate infantile storage of iron, etc., at birth and the importance of a mixed dietary at least after the first nine months of life. The experiments of C. M. Davies6, which showed the natural inclination for babies, at about the age of nine months, to help themselves with a varied diet when such was provided, are important indications of the necessity for such a diet.

There seems no doubt that the foregoing cases are in themselves sufficient demonstration of the efficacy of the therapeutic agents employed. A great deal of experimental work has been carried out recently in the attempt to show the possible mechanisms of production and cure of the types of hypochromic anæmia seen in infants. Reference has been made in preceding papers in this series to the results of this work, but it may be recalled that we have shown that the addition of yeast to the milk diet of rats suffering from nutritional anæmia will cure the anæmia and allow reproduction to occur. The members of the resulting litter, however, showed nutritional anæmia. The addition of yeast and pure iron in adequate quantities to a milk diet not only ensures the cure of nutritional anæmia in anæmic rats, but also that the members of any litter they produce then have a haemoglobin above normal. The clinical cases recorded in this paper furnish direct human corroboration of these and other experimental studies,
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