

for above all Midwifery is never dull nor monotonous especially in India, where the abnormal labour is often more common than the normal, at any rate in our hospitals.

I am sending Miss Abram a case history of an abnormal labour which we had recently in our hospital and will you please send her the history of any case you have had which you think is even more interesting than the one printed in the last month's Journal.

It is my hope that when I am travelling in various areas I might come and visit you personally, whether you work on the district alone or in hospital, for that reason I shall be very glad to hear from you or from the Branch Secretaries.

Please write to me ;

Miss D.M. Coggins,
Baptist Mission Hospital,
Berhampur. Ganjam.

Ganjam is very important as there is another Berhampur Hospital in Bengal & even if they are members of the M.U. they won't want all my correspondence.

D. M. Coggins

Some Blood Changes in Pregnancy

Before discussing the anaemias of pregnancy it would be well to consider the Physiological changes to be expected in the blood of every pregnant woman. These changes are as follows :

1. There is an increase in the total volume of blood, particularly during the last three months of pregnancy.

The major part of this increase is in the plasma, and this is probably of great importance in the prevention of shock after labour. The increase in red blood cells is only about half of that of the plasma, i.e. approximately 12% red cells increase as against 25% plasma increase. This produces an apparent anaemia, but the haemoglobin percentage does not fall below 70% in a normal pregnancy. The correct term for this condition is hydraemia.

The leucocyte count also rises, sometimes up to 12,000 per c.m. This is thought to be one of the ways by which the body prepares itself beforehand to resist infection. It should be remembered that immediately after parturition there may be a temporary rise of leucocytes well above this 12,000, which is due to a combination of injured tissues, loss of blood and energy, and is not an indication of sepsis.

2. Along with this physiological increase of blood, there is a hyperplasia of bone marrow, again especially in the last three months. Numerous red cells are formed in this marrow and are stored ready to be poured into the blood stream should there be any haemorrhage.

It can therefore be seen that these physiological changes in the blood are entirely protective.

PATHOLOGICAL CONDITION

1. The Anaemias of pregnancy

1. *Microcytic anaemia* is due to iron deficiency. In this condition the red blood cells are smaller than usual and the colour index is below 1.

An increased amount of iron is always necessary during pregnancy. The foetus is a merciless parasite and takes what it needs from the mother's blood. A certain amount of iron is also lost during parturition and lactation. The result of this loss will be aggravated should the mother be taking or absorbing insufficient iron. The contributory causes therefore may be dietetic, especially in a woman who is capricious in her appetite. Or it may be due to hypochlorhydria or achlorhydria which is sometimes found in pregnancy and which tends to a decreased power of absorption.

2 *Macrocytic anaemia* is similar to true pernicious anaemia. Some authorities consider there is a temporary derangement of gastric secretion, or it may be wholly the result of failure to absorb the anti-anaemic principle. If this is untreated it predisposes to premature labour. In this condition the red blood cells are larger than normal and the colour index is above 1.

3 *Hypoplastic (Aplastic) Anaemia*. This is a rare condition. The bone marrow is only producing about half of its normal amount of red cells. When this is suspected, a small portion of bone marrow is taken for examination. The cause is not known, though in some cases toxins are thought to be responsible.

II Haemolytic diseases of the Newborn i.e. Hydrops foetalis

Icterus gravis

Congenital haemolytic anaemia

The Rhesus Factor.

In 1940, during a series of investigations in America, red cells from a Rhesus monkey were put into a guinea pig. When serum from this guinea pig was added to human blood, 85% of the cases showed agglutination. These were the Rhesus positive people, and the sign given to their blood was RhRh. The ones that did not agglutinate were called Rhesus negative and their sign was rhrh.

It will be remembered that during maturation of the germ cells, the chromosomes divide into two, so that the embryo receives an equal number of chromosomes from each parent. It is for this reason that a double sign is given to the Rhesus factor. A purely positive blood, RhRh is one which has inherited Rh positive factors from both parents. These people are called Homozygous. A child with a RhRh positive father and a negative mother will be only half positive i.e. haeterozygous and their sign will be Rhrh. The result of mating positive with negative people can be seen in the tables below.

Father. RhRh. Homozygous	Mother. rhrh.
Children.	
Rhrh. Haeterozygous.	Rhrh

Father. Rhrh. Haeterozygous.	Mother. rhrh.
Children.	
Rhrh.	rhrh

It can be seen from this that whereas in the first table, the child will always be haeterozygous, in the second ; it will depend on which factor is inherited from the father as to whether it will be haeterozygous or Rh negative.

In 1940 it was discovered that in 101 cases out of 111 haemolytic diseases, the mother was a Rh negative while the child was positive. The theory is that the child's positive Rh factor passes through the placenta to the mother's blood and there produces strong anti bodies. It is these anti-bodies which diffuse back into the foetal bloodstream causing havoc there and in the liver. The foetal red blood cells agglutinate and are destroyed. The antibodies are stored up in the foetus and so the condition worsens.

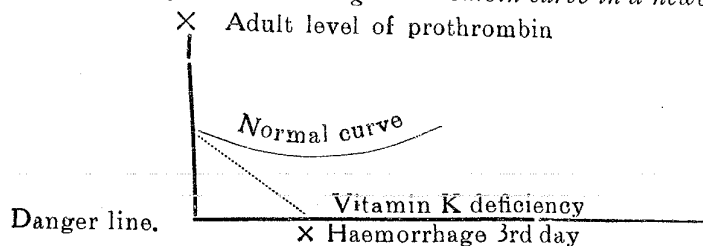
Recent discoveries point to there being numbers of different groups of Rh positive factors, each with their own antibodies so that the subject is becoming more complicated. But it must be remembered that every incompatible mating does not produce trouble. It only occurs in a very few cases and rarely with the first child.

III Vitamin K deficiency.

This is concerned with the formation of prothrombin which is necessary for the coagulation of blood. Apart from the intake of this vitamin in food, it is thought to be produced by bacterial action in the intestine. It is then converted into prothrombin in the liver.

In the foetus there is no bacterial action in the intestine and therefore there is a low prothrombin. What there is falls rapidly after birth, and then increases when bacterial action begins. Where there is grave K deficiency, the level of prothrombin will drop to danger point and haemorrhage will occur.

Diagram illustrating Prothrombin curve in a newborn infant.



The Treatment.

- I. The treatment of the anaemic conditions are obviously dietetic with additional liver therapy in the macrocytic variety, and iron therapy in the microcytic. In severe cases blood transfusions are given.
- II. The haemolytic diseases of the newborn are now given transfusions of Rh negative blood. Were Rh positive blood given, it would be quickly destroyed by the stored antibodies. In a successful case the antibodies will eventually disappear and the child's blood condition become normal.
- III. If there is a history of moloena neonatorum with previous children, Vitamin K, may be administered to the mother 4-12 hours before the birth of the child. After birth it may be given direct to the child by mouth or by injection. Intramuscular injections of blood are also sometimes given.

L G Carleton,