some reflections on the rhesus problem

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The problem presented by the Rhesus baby is not usually one of diagnosis — this should have been made by the obstetrician before the baby has been delivered. However, it is not uncommon for one to be faced with the problem of a baby who becomes jaundiced in the first 24-36 hrs. after delivery. In such cases, the baby is deemed to be suffering from haemolytic disease of the newborn until proved otherwise. Estimation of the Hb and serum bilirubin, the Coombs Test, blood group and Rhesus factor of the mother and the baby, examination of a blood film, together with direct cross-match of the baby's red cells will enable one to arrive at the correct diagnosis in the large majority of cases. Besides haemolysis due to Rh incompatibility one must bear in mind ABO incompatibility and other rarer group incompatibility, such as Kell, Duffy and a few others.

In the case of ABO incompatibility there are three combinations which might cause trouble to the foetus —

mother B — infant A
mother A — infant B
mother O — infant A, B, or AB

In actual fact, only the mother O — infant A or B situations, with rare exceptions, leads to haemolytic disease. The offending antigen is almost always A, and what is more, A of the specific subgroup A1. Even so, as in the case of Rh antigens, only a small proportion develop any significent jaundice presumably because the foetus may develop protective mechanisms, or the facility for the mother to develop antibodies is fortunately somehow impaired, or it could be that the liver of most infants is capable of excreting the small additional load of bilirubin so that the jaundice never becomes clinically apparent.

Occasionally, hereditary spherocytosis, hereditary elliptocytosis, hereditary non-spherocytic anaemias, (eg. pyruvate kinase deficiency), congenital toxoplasmosis, cytomegalic inclusion disease (C.I.D. virus) and G6 P.D. deficiency can present an identical picture.

One also sometimes sees a baby with anaemia **at birth** in which the question arises whether he is suffering from haemolytic disease of the newborn. Indeed, anaemia and oedema, singly or in combination, with little or no jaundice, may be the predominant signs of haemolytic disease of the newborn. The absence of haemolysis as a cause of the anaemia is not difficult to settle by a proper evaluation of the results of the investigations already outlined. In such cases, one must exclude extraneous bleeding from the cord or from vasa praevia, or concealed bleeding into the placenta itself or even bleeding into the mother (foetomaternal transfusion) — this latter can be excluded by the Kleihauer test (a test for the presence of foetal RBCS in the maternal circulation). I was recently faced with this problem in a baby who was born at St. Luke's Hospital and was under the care of Dr. E.A. Cachia. The mother was Rhesus negative and had no detectable Rh antibodies during pregnancy. I was called to see the baby who was obviously pale soon after delivery. The Hb was in the region of 10gm% and the blood film showed no evidence of haemolysis. The baby was Rh negative. The Coombs test was negative and the serum bilirubin was 1mg %. The Kleihauer test was strongly positive and the baby was treated by simple transfusion. In this case, the mother was Rh negative but this was a red herring and haemolysis was quickly and decisively excluded as a cause of the anaemia.

Sometimes one is presented with anaemia in the first week or two of life which is due to haemolytic disease, when jaundice, (though present initially) has not been severe enough to draw attention to the underlying disease. Indeed, the jaundice may have been so mild as to be labelled simple physiological jaundice. Again in such cases, a proper evaluation of the routine investigations outlined above should not present any difficulty in diagnosis. The treatment is by simple transfusion.

Bleeding from one twin into the other twin (twin to twin transfusion syndrome) can also present problems soon after delivery — with anaemia in the one and polycythaemia in the other twin. The anaemia can be quite severe and I have seen it cause intrauterine death. Otherwise it may need correction by simple transfusion, soon after delivery if necessary. The polycythaemia twin may become quickly severely jaundiced, with the danger of kernicterus, and the increased blood viscosity can embarrass the circulation and cause various other complications.

Again, the problem of Rhesus disease in the newborn does not really lie with treatment. All the efforts of treatment should be directed at correcting the anaemia and preventing kernicterus. The indirect-reacting serum bilirubin should not be allowed to rise above 20 mg%, especially if respiratory distress, cyanosis and prematurity are also present. There is still no way of doing this except by the tedious procedure of exchange transfusion. Recently, both phenobarbitone and exposure to light of so many candle-power (phototherapy) have been found statistically to lower significantly the serum bilirubin in premature and full term infants. Indeed, I have not seen any significant jaundice in our premature babies at St. Luke's

Hospital, and, I believe that our well-lighted nursuries are important in this respect. However, these methods (phenobarbitone and phototherapy) have no place at all in the management of haemolytic disease of the newborn. The anchor sheet of treatment is the exchange transfusion, which should be repeated as often as necessary to keep the serum bilirubin below 20 mg%. The provision of disposable exchange transfusion sets has done a great deal to make it less tedious and a lot less messy. With these sets there is no need to wash those glass syringes which are continually stuck! The disposable syringe provided with the set is silicone-coated. I prefer to use fresh heparinized blood, which obviates several of the complications of citrated blood.

In my opinion, in the majority of cases, the real problem in Rhesus factor disease is one of communication and coordination. This is understandable considering the number of people who are involved — the obstetric team, the blood transfusion department, the haematology and biochemistry department and, of course, the paediatric team. The antenatal ward, the maternity ward and the paediatric ward are also involved at one time or another in the care of the Rhesus baby. Cooperation should not be difficult if there is adequate and timely communication and coordination. I have not mentioned the general practitioner — I do not think that the mother who has previously delivered a baby affected with haemolytic disease, however mild, or who has antibodies during pregnancy, should be delivered at home. The delivery should be made in a hospital equipped with the staff and facilities for performing an exchange transfusion, as an emergency if necessary.

I will illustrate what I mean by referring to three cases I have seen at St. Luke's Hospital in the last few months.

Case 1

This baby was delivered at home. He was the third baby. The first baby was normal. The second baby was slightly jaundiced in the first day or two of life. The third baby was referred to this hospital (to the O.P.D.) in the fourth day of life for jaundice. In fact, the serum bilirubin was 26 mg%, Hb 66% and Coombs test strongly positive. The baby was Rh positive and the mother Rhesus negative. The mother had never had the Rh factor determined and this was the first time that her blood grouping was performed, after the delivery of her third baby.

Case 2

This also was the third baby, and was delivered at another hospital. The mother was Rh negative and had a rising titre of antibodies during her third pregnancy. He was born in the middle of the night three weeks prematurely, and soon after delivery was transferred to St. Luke's Hospital. On examination he was moribund, with marked pallor, oedema, some jaundice and in heart failure. Efforts at resuscitation proved fruitless and he died 4 hrs later. The serum bilirubin was 7.3 mg%, Hb not determined (clotted blood), Baby's group O Rhesus positive.

Such cases of hydrops are of course notoriously difficult to treat. On two or three occasions I have given such babies a mini-exchange transfusion with packed cells, exchanging 100 or 200 cc blood, together with digitalization and diuretics, and then performed a full exchange transfusion for the hyperbilirubin-aemia later on, at 24-48 hours of age. Initially, it is not so much the jaundice as the anaemia which is causing trouble.

Case 3

This was the eighth baby. There were at least 3 previously affected babies necessitating exchange transfusion, all of whom, in fact, died. This baby was also affected, though not severly so, but it took about 12 hours before the exchange transfusion could be started.

In each of these cases there has been a lapse in communication. The difficulties are obvious but I do not think that they are insurmountable. The blood transfusion department and the haematology and biochemistry departments, and paediatrician should be alerted as soon as the mother is admitted to hospital, or at least as soon as labour has started. The inexorable march of labour cannot be halted, but we can do a good deal to be prepared to treat the product of a Rhesus gestation.