RHESUS SENSITIZATION IN THE BANTU

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The extent to which the Bantu are affected by Rh sensitization is being considered and full agreement on this controversial subject has still to be reached. From observations made over a period of 14 years, during which time 36,000 Bantu patients were delivered in hospitals in Port Elizabeth, it became increasingly apparent that cases of *erythroblastosis foetalis* due to Rh sensitization in the Bantu were few and far between. In fact, during this period not a single case of *hydrops foetalis*, kernicterus or haemolytic disease was personally encountered among these infants, either directly or indirectly, in contrast with the common experience with White infants.

On the other hand, it has been stated by Zoutendyk[1] that there is a mistaken impression that Rh sensitization is excessively rare in the Bantu and that antibody production is at a generally lower level and the clinical condition less severe. He also found that the Rh-sensitization rate in the Bantu population as a whole was approximately one-third that of an unselected White population and that, although the condition can be as severe in the Bantu as it is in the White, a significantly larger proportion of sensitized Bantu seem to remain below the critical antibody level, which is a titre of 1/16 - 1/32.

It was decided to investigate the problem from the clinical aspect without becoming submerged in the complexities of Rh serology.

MATERIAL AND METHODS

The patients were unselected and consisted of 500 Rho(D)-negative mothers (5·6%) in a consecutive series of 8,946

Bantu patients who had been delivered at the Livingstone Hospital during 1960 - 62 and whose ABO and Rh groups had been recorded. With few exceptions these women were Xhosas.

All Rh-negative patients who attended the antenatal clinic were tested for antibodies at regular intervals during the last trimester. At birth the cord blood was examined as a routine for the blood group, Rh factor, direct Coombs test, maternal antibodies, bilirubin and haemoglobin levels. The Rh tests were carried out in tubes with saline agglutinating sera and, if a Rho(D)-negative specimen was found, a second test was performed with the incomplete sera. Sensitization to other Rh antigens is not included in this series. The techniques used for antibodies were the saline, indirect Coombs and ficin.*

Every effort was made to confine this investigation to parents of genuine Bantu descent and to exclude the genetic influence of mixed ancestry, but one of the chief obstacles encountered was the elusiveness of the father.

RESULTS

Rh Sensitization

Rh antibodies were present in 7 (1·4%) of the 500 Rh-negative mothers. In the total series of 8,946 unselected patients this is an incidence of 0·08%. Five of the 7 infants were Rho(D)-positive and had negative direct Coombs tests; the laboratory results on 2, including 1 stillbirth, were not recorded (Table I). It will be seen that the antibody titre of 6 of the 7 sensitized mothers remained below the level of 1/16 - 1/32 and that 6 of the 7 infants were clinically unaffected. The only stillbirth in this group was that of a macerated foetus occurring in a gravida 2 with an antibody titre of 1 in 4. This foetus was not hydropic and the cause of death remained in doubt. According to Boggs,[2] 'If the stillborn foetus of a sensitized woman is not hydropic, then some cause other than the haemolytic disease should be sought as the explanation of the foetal death'.

TABLE I. RH-SENSITIZED MOTHERS

Patient	*Gravida*	*Child*	*Titre of Rh antibodies*	*Previous obstetric history*
L.P.	4	Alive	$\frac{1}{16}$ inc.	C 2. D 1 at 8 months
I.Z.	2	,,	$\frac{1}{4}$,,	D 1 at 9 months
R.M.	4	,,	$\frac{1}{4}$,,	C 1. M 2
N.D.	6	,,	$\frac{1}{4}$,,	C 4. D 1 at 18 months
C.S.	2	,,	$\frac{1}{8}$,,	C 1
N.M.	2	Macerated	$\frac{1}{4}$,,	C 1
M.S.	6	Alive	$\frac{1}{128}$,, abnormal	C 4. M 1 at 7 months (5th pregnancy)

C = No. of living children; D = Died; M = Miscarriage; Inc. = Incomplete saline antibodies.

The only patient (M.S.), gravida 6, with a high antibody titre (1 in 128) gave birth to a normal, healthy infant at term. Her fifth pregnancy had ended in a 7 months' miscarriage at home and, previously, she had been delivered of 4 healthy children. Two years after her sixth confinement her antibody titre in the non-pregnant state was 1 in 32.

Between them these 7 mothers had had a total of 26 pregnancies—22 children were born alive and well and none required a blood transfusion; 3 subsequently died from respiratory or gastro-intestinal infections, but not before 8 months had elapsed. There were 3 miscarriages, of which the details are not available, and 1 stillbirth.

*The various tests were carried out at the Port Elizabeth branch of the South African Institute for Medical Research.

SURVEY OF THE 500 RHO(D)-NEGATIVE MOTHERS AS A GROUP

In view of the unusually small number of Rh-sensitized mothers, a survey of the whole group of 500 will show how slight the influence of the Rh-negative factor was on haemolytic disease and on perinatal mortality. The risk of developing antibodies is greatest in the second pregnancy when 4·5% of women carrying their second Rh-positive foetus develop antibodies; there is an additional 2·8% in the third pregnancy, and 1·3% in the fourth.[3] In the present series 393 women were multiparous (78·6%), including 47 ranging from gravida 7 to gravida 11, yet only 1 in 71 became sensitized.

TABLE II. PERINATAL MORTALITY

Total No. of infants	504 (4 sets of twins)	
Living	492 (97·6%)	
Stillborn (fresh)	6	Perinatal mortality = 2·4%
Stillborn (macerated)	4	
Died	2	

Perinatal Mortality

The stillbirth rate of 1·98% and the gross perinatal mortality of 2·38% (Table II) compare favourably with figures available elsewhere for the population as a whole. This is particularly noteworthy, since these Bantu mothers come from a low socio-economic class. In 1954 the foetal mortality rate for the United States was 2·0% for the White population and 3·91% for the non-White population.[4] The perinatal death rate in Holland in 1960 was 2·7%.[5]

An analysis of the perinatal deaths in the present series is shown in Table III.

TABLE III. CAUSES OF PERINATAL DEATH

Fresh stillbirths (6)

1. Prematurity (one of twins)
2. Prematurity (chronic nephritis)
3. Prematurity (pre-eclampsia)
4. Dystocia (postmaturity)
5. Anencephaly
6. Cause unknown

Macerated stillbirths (4)

7 and 8. Prematurity
9. Cause unknown
10. Cause doubtful—Rh antibodies 1/4

Deaths (2)

11. Cerebral haemorrhage, (breech delivery)
12. ABO haemolytic disease

Apart from Case 10, no Rh antibodies were present during pregnancy or in the cord blood and no signs of Rh erythroblastosis were found in any of the foetuses or infants.

DISCUSSION

In Zoutendyk's series of 11,125 Rh-negative Bantu patients, 491 were Rh sensitized (4·4%) compared with 1·4% in the present series. In terms of unselected antenatal cases Zoutendyk found the sensitization rate in the Bantu to be 3 per 1,000 births and in the present series it was 0·8 per 1,000 births. These rates are well below those of the White population in South Africa and in other countries, where they vary from 6 to 9 per 1,000 births. With the one doubtful exception of the macerated stillbirth in this series, none of the other babies showed any signs of Rh-haemolytic disease.

At first sight it is difficult to account for the marked disparity in the incidence of sensitization between the 2 series, even when the considerable difference in numbers has been taken into account. When searching through the records certain sources of error were revealed, which in turn would be transmitted to the laboratory records, and might explain some of the discrepancies.

Sources of Error

1. Some patients with Bantu names, and classified as Bantu, were found to be of mixed descent. This includes those Coloured women who had Bantu husbands and who regarded themselves as Bantu.

2. The racial group of the husband was a considerable source of error. In urban areas Bantu women are often found married to Coloured husbands, of whom some, together with the newborn infant, were classified as Bantu in the records.

3. The most significant factor was the high proportion of single women (33% in this series), indicative of the number of fathers of unknown racial origin.

It may be appropriate to point out at this stage that to compare Rh sensitization in American Negroes with that in West African Negroes or in the Bantu of Southern Africa can be misleading. According to the latest estimate, only 10% of American Negroes are of pure Negro descent and that is the only section that should be compared with its African counterpart.

Low Incidence of Rh Sensitization

When, as far as possible, all the above sources of error have been excluded, it is nevertheless remarkable that in this series of 500 Rh-negative Bantu mothers, the incidence of sensitization should be so low. That out of 504 children born to these women, 492 were alive and well; that not a single infant required a blood transfusion for Rh haemolytic disease; that 6 out of the 7 sensitized mothers were below the critical level of the antibody titre and that only 1 infant (stillborn) could be described as an unconfirmed case of erythroblastosis, is further evidence of the low frequency of Rh sensitization in the Bantu.

It is not disputed that severe cases do occur and it is agreed that many a sensitized mother can have a succession of unaffected children, but the higher incidence of sensitization in Zoutendyk's series requires an explanation. It possibly lies in the manner in which the material was gathered. After the elimination of several sensitized and non-sensitized Rh-negative patients from the present series due to incorrect racial classification, it can definitely be stated that the material collected is a true reflection and a typical sample of the unselected local Bantu population. In the period under review (1960 - 62) there were no sensitized Rh-negative patients admitted from other towns or rural areas, consequently there was no loading of statistics. In Zoutendyk's series all sensitized Bantu women were admitted to the Bridgman Memorial Hospital at 38 weeks, not only from the antenatal clinic of the hospital itself but also from 'neighbouring municipal native clinics, townships and from distant rural areas'.[6] This does suggest an undue proportion of Rh-sensitized patients, which would naturally result in a higher incidence, unless all the antenatal patients from these various clinics, townships and rural areas during that period (1946 - 1960) were included in the over-all series.

Future investigations on this subject should give accurate and conclusive results, if they are carried out in an area with a homogeneous Bantu population.

SUMMARY

1. In an unselected consecutive series of 8,946 Bantu patients delivered in hospital, 500 (5·6%) were Rho(D)-negative, of whom 7 (0·08%) were sensitized.

2. Six of the 7 sensitized patients had an antibody titre below the level of 1/16 - 1/32.

3. Six of the 7 babies born to these sensitized mothers were totally unaffected.

4. The gross perinatal mortality for the group of 500 Rh-negative patients was 2·38%.

5. Not a single baby required a blood transfusion for Rh haemolytic disease.

6. The view that the Bantu have a high degree of immunity to Rh sensitization and that on the whole the children of Bantu parents are little affected is given additional support.

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