whilst control patches (in one case of plain gauze and in another of gauze impregnated with yellow soft paraffin) were negative.

When such untoward reactions may occur, however rarely, it seems reasonable to doubt whether the continued incorporation of balsam of Peru in tulle gras dressing *B.P.C.* is justified; moreover, its beneficial effects are probably slight.

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Haemolytic Disease of Newborn: Delivery at Term of Living Affected Infant after Previous Stillbirths

A rhesus-negative woman whose husband is homozygous rhesus-positive, and who has had one or more infants still-born owing to haemolytic disease, is usually told that the chance of having a live infant from any future pregnancy is virtually nil. However, this view has been challenged by Watson, Crosse, and Hatchuel (1954), and by Watson (1956); they advocate premature induction of labour in such mothers, or delivery of the infant by caesarean section if foetal movements are diminishing. Williams (1956) described one case successfully managed by induction at the 34th week of pregnancy, followed by immediate exchange transfusion of the infant. The mother in question had previously had three stillbirths. Watson (1956) also mentions two other similar cases in which the infants were saved by inducing labour at the 36th week. Buffa, Francois, and Davidsohn (1956), in studying the outcome of pregnancy in 167 women whose sera contained immune Rh antibodies, reported that of 15 mothers who had had previous stillbirths five were delivered of affected but living infants, all of whom survived after treatment. They comment that these findings contradict the extreme pessimism often expressed to such mothers on the likelihood of their having a liveborn infant.

A further case is described which supports the new-found optimism expressed by these writers. It is of interest partly because the mother, after two previous stillbirths, was delivered at term of a liveborn affected infant, successfully treated, and partly because the mother's antibody titre in any pregnancy, whether it produced an affected live or stillborn infant, was relatively low throughout.

CASE REPORT

The mother's previous obstetric history is summarized as follows. 1941: caesarean section (India) for disproportion. Given 2 pints (1,140 ml.) of blood at operation; infant liveborn and normal. 1943: normal delivery; infant liveborn, no transfusions; severe jaundice for over a week; retarded development with incontinence and ataxia until death at 2½ years, presumed kernicterus. 1947: normal delivery; infant liveborn, jaundiced; simple transfusion first day; generalized spasticity and incoordination of movements, diagnosed as kernicterus by paediatrician; died at 4 years. 1949: normal delivery; exchange transfusion (Salisbury); infant alive and well. 1951: pregnancy terminated at 4 months, reason unknown. 1952: macerated stillbirth at 36 weeks. 1953: macerated stillbirth at 36 weeks. 1954: spontaneous abortion at 16 weeks.

She became pregnant again in November, 1955. Her attendances at the antenatal clinic were irregular, and,

although induction of labour at 36 weeks was considered, she did not present herself for admission until term: labour was induced surgically and a liveborn male infant weighing 6 lb. 15 oz. (3,150 g.) delivered.

Serology.—This woman was group O cde/cde; her husband was group B, cDE/cDE (presumptive). Antibody tests had been done at frequent intervals in her various pregnancies. The highest titre was 1/20 either in albumin or by indirect antiglobulin techniques. In her fourth, fifth, sixth, and seventh pregnancies no saline antibodies were demonstrated; but in 1956 an antibody titre of 1/20 in saline was detected in March, and remained at this level, as did the titre in albumin, until delivery. The specificity of the antibodies (both in 1953 and in 1956) was anti-D only. It is probable that this woman's sensitization dates from her blood transfusion (probably Rh-positive blood) in 1941.

The baby at birth (August, 1956) looked normal, but within an hour jaundice at the base of the cord was noticed. Cord blood findings were: haemoglobin, 13.9 g. per 100 ml.; serum bilirubin, 5.7 mg. per 100 ml.; direct Coombs test positive; group B, Rh (D) positive; blood film showed erythroblastemia. Exchange transfusion was carried out within three hours of birth, 306 ml. of blood being withdrawn, and 296 ml. of compatible (saline, albumin, and indirect antiglobulin techniques) blood injected. Samples of cord blood immediately before and after this exchange showed bilirubin levels of 8.7 and 7.8 mg. per 100 ml. respectively. Twelve hours later the infant was deeply jaundiced, and the bilirubin level was 24 mg. per 100 ml. (heel prick). A further exchange transfusion was therefore given, 400 ml. of blood being withdrawn and 380 ml. injected (O Rh negative); immediately after the exchange the bilirubin was 10.3 mg. per 100 ml., and haemoglobin 13.6 g. per 100 ml. The next day (40 hours) the haemoglobin was 15.8 g. per 100 ml. and the jaundice not increased; thereafter the jaundice faded rapidly during the next three days.

COMMENT

This case clearly demonstrates that it is possible for an Rh-negative woman, having had previous stillbirths due to Rh incompatibility, to have a liveborn affected infant at term. It is generally agreed (Mollison and Walker, 1952) that premature induction of labour is not indicated in most mothers with antibodies. However, induction of labour at the 36th or even 34th week in certain selected cases—that is, those who have had previous stillbirths due to haemolytic disease—is the treatment of choice, followed by immediate and large exchange transfusion of the infant. That the infant described here required two exchange transfusions within 12 hours is almost certainly due to the relatively small first exchange. Preliminary investigations here (J. R. H. P.) based on estimation of percentage of foetal haemoglobin before and after exchange transfusion, show that the efficiency of exchange transfusions is not as high as is generally supposed, even with exchanges of 50–60 ml. per lb. (110–132 ml. per kg.) of body weight. It seems desirable to aim at 80 ml. per lb. (176 ml. per kg.) for the more seriously affected infants.

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