vascular disease. The healthiest doctors at the age of 65 have had a similar family history of cardiovascular disease, and much the same may be said of those who develop pulmonary tuberculosis; my inference is that the same experience would be found in the same number of doctors suffering from other diseases. Also, it can be generally stated that the medical men who develop pulmonary tuberculosis between the ages of 30 and 65 have no worse tuberculous family history than the healthiest doctors, nor indeed than those who develop coronary thrombosis. I believe the converse is true: that a family history of tuberculosis is negligible after the age of 30, and even before that age.

Onset of Coronary Thrombosis.—The records were then searched to find at what ages coronary thrombosis occurred, how many died of this illness, how many survived and were unfit to work again, and how long those worked who completed their convalescence.

Ages at Onset				Deaths	Unfit for Work	Returned to Work
35-45	45-55	55-65				
7	19	11		11	8 years, 1 5 " 3 4 " 1 1 " 3	18* Working : 4 years, 2 3 " 6 2 " 2 1 " 4 <1 " 2 Reached 65 2
Approx. No. of Living in Each Group in 100s :						
36	18	11				

* Average duration of unfitness for any work was 18 weeks.

From this it can be said that out of 37 who develop coronary thrombosis 11, or approximately 1/3, die, 8 become ambulatory but unfit for work, whereas 18, or half of them, are working after a year or for a few years. Liability to coronary thrombosis steadily rises with successive years, as one would expect, but only to the age of 45, when a plateau is reached which remains flat to the age of 65.

Onset of Tuberculosis.—At what ages do the 37 who suffered from tuberculosis develop their disease?

Ages at Onset				Deaths	Unfit for Work	Returned to Work
25-35	35-45	45-55	55-65			
16	12	8	1	6	10 (up to 9 years)	21* Working : 23 years, 1 12 " 1 7 " 1 5 " 3 4 " 5 3 " 2 2 " 2 <2 " 3
Approx. No. of Living in Each Group in 100s :						
14	36	19	11			

* Average duration of unfitness for any work was 65 weeks.

Summary

1. The causes of death among doctors before the age of 65 have been stated.
2. The causes and incidence of ill-health among doctors have been tabulated.
3. The title of "the doctor's disease," given to coronary disease, is challenged.
4. It was found that the parental history as regards cardiovascular degeneration was the same for 37 doctors who developed coronary thrombosis as for a control group of 37 doctors who were still working at the age of 65, both these groups having been passed fit at the age of 30.
5. It was also found that the parental history as regards pulmonary tuberculosis was the same for 37 doctors who developed pulmonary tuberculosis as for the control group of 37 doctors who were still working at the age of 65, both these groups having been passed fit at the age of 30.

During the Balkan campaign according to German experience (Weiler: *Dtsch. Militärarz.*, 1941, 11) the principal diseases occurring were dysentery and influenza. Acute influenza was often difficult to distinguish from typhoid. The percentage of cases of bronchopneumonia (41) was high. Half of the cases were treated with sulphonamides and the other half with "novalgin" and quinine. The results were the same in both groups. The author believes that the best results were obtained by intravenous injection of quinine-calcium.

THE CLINICAL SIGNIFICANCE OF THE Rh FACTOR

BY

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AND

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(Concluded from p. 538)

Haemolytic Transfusion Reactions Due to Rh Antibodies

As first pointed out by Wiener and Peters, the repeated transfusion of Rh-positive blood to an Rh-negative individual may be followed by a haemolytic reaction due to the production of Rh antibodies as a response to the first or subsequent transfusions. The following case is probably of the mildest type of this reaction.

A Mild Type of Reaction

Case 1.—A man aged 25 had had symptoms of ulcerative colitis for 8 years. In November, 1940, he had a relapse and received two transfusions totalling 2,500 c.cm. of citrated stored blood of group O. These transfusions were followed by clinical improvement, and the patient remained well for many months. He was readmitted to hospital in April, 1942, with a return of symptoms, however, and in view of the success of the previous treatment it was decided to give him a further transfusion. He was found to belong to group O Rh-negative. His serum was tested against a series of group O bloods and found to give no reactions at all at 37° C. or at any other temperature. It was therefore considered safe to proceed with the transfusion of blood from two of these donors. For purposes of record these bloods were also tested with anti-Rh sera and found to be Rh-positive.

1,000 c.cm. of citrated blood from these two donors was given slowly over a period of 5 hours. Following the transfusion the patient had a slight headache and a rise of temperature to 100° F. but no other symptoms, and when seen on the next day expressed himself as feeling better for the transfusion. A venous sample was obtained 48 hours after the transfusion for comparison with the pre-transfusion sample. It was found that only insignificant changes in haematocrit and haemoglobin values had occurred; at the same time it was noted that the serum of the later sample was distinctly yellow, in contrast with that of the pre-transfusion sample, which was pale. A test was made to see whether the Rh-positive blood was surviving in the recipient's circulation by testing a suspension of blood taken from him with a potent anti-Rh serum. Many moderate-sized clumps were found in the suspension, thus indicating the survival of a substantial part of the transfused blood, since a control test with the recipient's blood before transfusion showed no trace of agglutination.

A further sample was obtained upon the fifth day, and it was noted that the serum was even more icteric; a quantitative van den Bergh test showed that it contained 1.5 mg. per 100 c.cm. Examination of the serum with a series of group O bloods revealed the presence of an Rh antibody. A survival test showed that only a small amount of Rh-positive blood was now present in the circulation. On the seventh day after transfusion there was no trace of the donor blood in the recipient's circulation, and the Rh antibody had risen further in titre.

Throughout the period of observation the patient continued to improve slowly, and he left hospital on the eighth day. Blood urea estimations were carried out upon the fifth and seventh days after transfusion; strictly normal values were found.

Reactions in Recently Delivered Women

The frequency with which intra-group haemolytic transfusion reactions occur in women whose infants are affected with erythroblastosis foetalis has been stressed by Levine, Katzin, and Burnham, and corroborated by Wiener. In the present series of investigations 4 such cases have been encountered. Two illustrative cases are here quoted to demonstrate the variation in severity of the reactions.

Case 2.—A woman aged 25: two previous pregnancies. The first was normal throughout, resulting in the birth of a normal baby. The second resulted in a baby which developed jaundice during the first week of life, but subsequently improved without relevant treatment. After delivery the mother was found

to have high blood pressure, oedema, and albuminuria. During the third pregnancy she remained well until about the eighth month, when swelling of the ankles, albuminuria, and hypertension developed. At this time the uterus became suddenly more bulky and hydramnios was diagnosed. On June 9, 1942, she was delivered of twins, the first of whom died after 2 to 3 hours and the second after 8 hours. Both infants had gross ascites and all the other features of hydrops foetalis. On the fifth day after delivery it was decided to give the mother a transfusion because of her anaemia. It was found that the patient's group was O Rh-negative, and that her serum contained an Rh antibody. (The twins were group A Rh-positive.)

Two group O and supposedly Rh-negative donors were selected by tests made with the patient's serum and with another human serum containing only moderately active Rh antibodies. A concentrated cell suspension was prepared from the blood of these two donors and transfusion was begun at a drip rate. By the end of an hour approximately 250 c.cm. had been given without any untoward reaction. Shortly afterwards, however, the patient started to complain of violent lumbar pain, difficulty in breathing, and generalized discomfort. The transfusion was stopped, approximately 300 c.cm. having been given, but she started to shiver and had a violent rigor followed by a rise in temperature. A venous sample was obtained 3 hours after the beginning of the transfusion, and it was found that the serum had become intensely icteric. Examination of the recipient's blood confirmed that her cells were group O Rh-negative, type MN, and that her serum contained an Rh antibody. The blood of both the donors was now retested with a sample of human serum containing very potent Rh antibodies, and it was discovered that one of the donors was Rh-positive (and the other Rh-negative). Both belonged to type N. By means of the differential agglutination technique it was possible to show that by the day after the transfusion the recipient's blood stream contained only half the expected number of type N cells (the calculation being made from the known amount and known red cell count of the blood transfused). Moreover, there was no trace of reaction with the potent Rh antiserum. It thus was clear that the blood of the Rh-positive donor had been eliminated, whereas that of the Rh-negative donor had survived. The fate of the latter cells was investigated by tests made during the following few weeks; it was found that they were only slowly eliminated.

Since the post-transfusion sample contained no free haemoglobin in the serum it was not surprising that the patient's urine was free from this pigment. The subsequent course of the case was entirely favourable.

Case 3.—A woman aged 36; four previous pregnancies. The first was terminated by craniotomy of the foetus because of disproportion. The second child was born prematurely after induction of labour a week or so before term. The child was very jaundiced, and died on the second day. The third pregnancy terminated in the birth of a stillborn child at 8 months. The fourth pregnancy resulted in the birth of a macerated foetus at 7 months. The fifth pregnancy was terminated by Caesarean section on Aug. 26, 1942. A living female child was delivered, but slight jaundice was apparent. At the end of 24 hours it was more jaundiced, and on the 28th a blood count showed R.B.C. 1.89 millions per c.mm., Hb 52%, and normoblasts approximately 16,000 per cmm. The infant died on Aug. 29.

Following the Caesarean section the mother became rather collapsed, and 20 oz. of citrated blood of group O were given. An hour after the end of transfusion she had a rigor, and 6 hours later complained of severe abdominal pain and backache and the respirations rose to 56 a minute. On the day after transfusion she was slightly jaundiced, and 29 oz. of dark brown urine were obtained by catheter. During the next 4 days only 12 oz. of urine altogether were passed, and the patient died suddenly on Aug. 31. Microscopical examination of the kidneys showed the typical features of necrosis of the tubules and the presence of masses of pigment in the tubular spaces.

Investigations carried out on Aug. 30 showed that the patient belonged to group A and was Rh-negative; her serum contained an Rh antibody. Her husband's group was O Rh-positive. A test was made by the "inagglutinable count" technique to see whether any of the group O blood given 4 days previously was surviving. It was found that this blood had been completely eliminated from the recipient's circulation. It was thus clear that the blood had been incompatible, and on examination it was found, as expected, that the donor was group O Rh-positive.

Transfusion of the Affected Infant

Levine, Burnham, Katzin, and Vogel have already reported that after the transfusion of Rh-negative blood the infant

maintains higher values of R.B.C. count and Hb concentration than after the transfusion of Rh-positive blood. Nevertheless, from observations of changes in R.B.C. counts and Hb concentration it is difficult to reach decisive conclusions unless considerable and consistent differences are found. A more reliable method of demonstrating the different survival of Rh-negative and Rh-positive blood in the circulation of infants affected with erythroblastosis is to use the method of differential agglutination. By an elaboration of this method it is possible after transfusing a mixture of Rh-negative and Rh-positive bloods to estimate the survival of the two types separately. The result of these experiments will be reported later (Mollison), but the following preliminary conclusions are set out, since they appear to be important from the point of view of treatment.

The rate of destruction of Rh-positive blood in the circulation of affected infants varies enormously from one case to the next. In some cases only slight differences have been found between the survival of Rh-positive and Rh-negative blood. More often, however, very great differences have been observed. The fact that there is a group in which the period of survival of Rh-positive blood is so much shorter than that of Rh-negative blood makes it very desirable to use Rh-negative donors as a routine for transfusion in cases of erythroblastosis foetalis.

Discussion

Iso-immunization in Pregnancy

Dienst (1905) first noted that when the mother's serum contained an agglutinin that was incompatible with the infant's erythrocytes the potency of her iso-agglutinins increased considerably during the fourth to eighth days after delivery. Jonsson (1936) made a study of the sera of women 2 to 3 months after delivery and found evidence of specific stimulation in some cases of "hetero-specific pregnancy": that is to say, when the mother belonged to group O and the infant to group A, for example, the mother's anti-A agglutinins were found to be stimulated. Jonsson did not titrate the sera, but simply used the presence of iso-haemolysins as evidence of an immune response. For this reason probably, he found evidence of specific stimulation in only a certain proportion of cases. As mentioned above, Levine and Stetson first suggested that the presence of atypical agglutinins in the serum of a recently delivered woman might be the result of immunization to an antigen in the foetus; and Levine *et al.* soon afterwards described many cases in which the atypical agglutinin had been found to be anti-Rh, and thus developed their theory of the aetiology of erythroblastosis foetalis, as outlined above. Naturally some explanation is needed for the fact that the presence of immune anti-A agglutinins in the serum of a mother carrying a baby of group A usually leads to no obvious abnormality in the baby, whereas the presence of immune anti-Rh agglutinins is so often associated with erythroblastosis of the (Rh-positive) infant. Wiener has suggested that this is because the Rh factor is found only in the erythrocytes, whereas the A and B substances are present in all the body tissues and secretions (in the majority of individuals whose erythrocytes contain the A or B factor), and thus protect the erythrocytes from damage. Levine *et al.* have suggested that in those individuals of group A, B, or AB whose body fluids do not contain the group-specific substances (the non-secretors of Schiff) the presence of immune anti-A or anti-B agglutinins in the mother's serum may lead to erythroblastosis. It may be pointed out that in any case in which the immunizing factor, be it Rh or A or B, is present only in erythrocytes, it is necessary to postulate the actual entry of foetal erythrocytes into the maternal circulation for immunization to occur.

Wiener (1942) has suggested that other atypical agglutinins besides anti-Rh may occasionally be the cause of erythroblastosis. The presence of atypical agglutinins, different from anti-Rh, in the serum of a recently delivered woman has been reported by Levine and Polayes (1941), among others.

In the series of cases described here in which the infant had definite erythroblastosis, an immune agglutinin incompatible with the infant's erythrocytes was found in the mother's serum in every case. In 44 out of 48 cases this agglutinin was anti-Rh, although in some of them immune anti-A or anti-B agglutinins were also found. In the remaining 4 only immune

anti-A or anti-B agglutinins were observed. Moreover, in these 4 the presence of other atypical agglutinins was unlikely, because the mother's serum did not agglutinate any of a series of 10 to 20 group O bloods when tested at different temperatures. This observation supports the idea that anti-A or anti-B agglutinins may occasionally cause the destruction of foetal erythrocytes of group A or B and thus be the chief aetiological factor in some cases of erythroblastosis foetalis.

The failure to demonstrate Rh agglutinins in the infant's serum in the majority of cases is not surprising, since these antibodies are not usually present in high titre in the mother's serum and since they will be expected to be absorbed on to the infant's (Rh-positive) cells. Nevertheless, as we have said, they could be readily demonstrated in one case in which the sera of identical twins who lived for only a few hours after birth were examined post mortem.

It will be noted that in the present series Rh antibodies have been found far more often than in the series described by Levine and his co-workers. The technique used by the latter authors consisted in incubation of the mixture of cells and serum (30 to 60 minutes at 37° C.), followed by centrifugation at low speed (500 r.p.m.) for one minute and by gentle shaking to resuspend the sediment. The tubes were then examined macroscopically for evidence of agglutination, and apparent negatives were examined microscopically. We have found that this technique is not so sensitive as that of simply allowing the mixtures to stand in the incubator for 2 hours, followed by examination of the pattern of the sediment and by a withdrawal of a portion of the sediment for microscopical examination, the latter operation being carried out with delicacy. Even the gentle shaking of the tubes advised by Levine *et al.* may, in our experience, completely abolish a weak reaction. It is of course necessary to carry out control tests with Rh-negative cells if weak reactions are to be accepted as evidence of the presence of an Rh antibody.

Selection of Blood Donors for those who have become Immunized to the Rh Factor

In the 3 cases described in which haemolytic reactions followed the transfusion of Rh-positive blood to an Rh-negative individual who had previously been immunized to the Rh factor, it will be noted that the essential feature is a rapid elimination of the donor blood from the recipient's circulation. This finding is in striking contrast to the survival time of compatible blood, which has been shown to be consistently 80 to 120 days (Wiener, 1934; Mollison and Young, 1942). From the 3 cases quoted above the impression may be gained that serious sequelae are the exception and not the rule following the transfusion of Rh-positive blood to a person whose serum contains an Rh antibody. However, it should be mentioned that in the 2 cases of which details have not been quoted the transfusion was followed by oliguria with a steadily rising blood urea. In these cases the patient, after being critically ill, at the end of 7 to 10 days after transfusion gradually started passing normal quantities of urine again, and recovered. Even in Case 1, in which there were apparently no untoward consequences at all, it is clear that the transfusion was of no benefit to the patient.

In Case 2 it was demonstrated that Rh-negative blood survived normally in the recipient's circulation after transfusion, and Wiener (1941) has published many such cases in which he has been able to demonstrate that Rh-negative blood survives normally.

For the transfusion of any recently delivered woman, and particularly in any case in which there is a suspicion that the individual has become immunized to the Rh factor, it is clearly very desirable to have available a panel of Rh-negative donors. At present large stocks of potent Rh test sera are not available, but if the recipient's serum is found to be incompatible with the donor's cells it should be possible to test the serum against a number of other individuals and to find a compatible donor.

It is probable that a modified compatibility test, as described above, will serve to detect the majority of cases in which Rh agglutinins are present. Clearly it will be of further help to know whether the prospective recipient is Rh-negative or not. It is strongly recommended that this modified compatibility

test be used as a routine before the transfusion of recently delivered women is undertaken. Where there are no facilities for carrying out this test two compromise procedures are available:

(a) In any case in which there is reason to suspect that the infant has erythroblastosis, or if there is a suspicion that any previous infant has had erythroblastosis, transfusion of the mother should be avoided if possible. Obstetric shock, post-partum haemorrhage, etc., should be treated by serum or plasma transfusion. Cases in which erythroblastosis is suspected should include all cases in which the infant is stillborn without obvious cause, and all cases in which the infant is born with oedema, jaundice, or obvious anaemia. (b) Rh-negative donors should be used as a routine either for the group mentioned in procedure *a* above or, better still, for the transfusion of all recently delivered women. This latter procedure has already been adopted by Levine *et al.* (1941) and Wiener (1941), but it must be pointed out that its immediate application on a large scale would present very great difficulties.

In discussing the choice of a donor for the affected infant the question of the suitability of the mother or father naturally arises, since they may be the only donors readily available. In this, as in all transfusions, the most important factor is the compatibility of the donor's erythrocytes with the recipient's serum. Compatibility in this case, however, has to be defined as "enjoying normal survival in the recipient's circulation," because it is not usually possible to demonstrate Rh agglutinins in the infant's serum, even in cases in which rapid elimination of Rh-positive blood is observed. From the point of view of the donor's erythrocytes the mother is suitable and the father unsuitable.

There is another factor to take into account, however—the compatibility of the donor's serum with the recipient's erythrocytes. This factor is ignored by some clinicians who regularly use, for instance, individuals of group O for transfusion to individuals of any group. Nevertheless, as demonstrated recently by Aubert, Boorman, Dodd, and Loutit (1942), the transfusion of serum containing potent incompatible agglutinins can result in the destruction of some of the recipient's erythrocytes. In infants who have developed a haemolytic anaemia as the result of the passage of Rh agglutinins through the placenta it is obviously undesirable to transfuse further amounts, and for this reason the transfusion of blood from the mother is to be deprecated.

Clearly, the ideal donor is an Rh-negative individual whose serum does not contain an Rh antibody and whose blood is also of the same group as the infant. However, when the infant is of group A and the mother is group O Rh-positive, and the destruction of the infant's erythrocytes is due to the mother's anti-A agglutinin, as we have supposed above, the most suitable blood for transfusion will be group O the serum of which contains low-titre agglutinins, since, by analogy, group A blood will probably be destroyed more quickly than group O blood in the infant's circulation.

To sum up, the most suitable donors for the majority of erythroblastic infants requiring transfusion are group O Rh-negative persons whose serum contains only low-titre agglutinins. Since they will also be the most suitable donors for transfusion for the majority of recently delivered women, it seems very desirable for every donor organization to collect a group of such individuals.

Incidence of Affected Infants in Different Families

When a case of erythroblastosis has occurred in a family some help may be given in the prognosis regarding future children by testing the blood of father, mother, and any available siblings. If, for instance, the mother is Rh-negative and the father Rh-positive and any of the children are Rh-negative, it is clear that the father's genotype is Rhrh, and therefore there is an even chance that future children will be Rh-negative and presumably not affected with erythroblastosis. If, on the other hand, all the children are Rh-positive, it will be more likely that future children will be affected.

Summary and Conclusions

In every case of erythroblastosis examined the serum of the mother has been found to contain an immune agglutinin which is incompatible with the infant's erythrocytes. In 44

out of 48 cases the agglutinin was anti-Rh. In the remaining 4 cases it was either anti-A or anti-B. Because of this latter finding it is considered that the anti-A and the anti-B agglutinins are capable of causing destruction of the infant's erythrocytes in a certain number of cases and thus, in some instances, of being the main causative factor of the erythroblastosis. The finding of weak Rh antibodies in the sera of the mothers of some babies with "physiological jaundice" supports the idea that there is no clear-cut distinction, from the clinical point of view, between mild and severe jaundice of the newborn.

At the present time, if serological tests are used as an aid in the diagnosis of doubtful cases of erythroblastosis, compatibility of the mother's serum with the infant's erythrocytes may be regarded as a strong point against the diagnosis, provided that the mother's serum is examined by a sensitive technique between 7 and 21 days after delivery. On the other hand, if the mother's erythrocytes are shown to be Rh-negative and her serum is found to contain anti-Rh agglutinins, the diagnosis is strongly supported.

A change in the method of selection of blood donors in certain cases is urgently needed. This applies particularly in the case of transfusion of recently delivered women and, above all, whenever there is any suspicion that the present infant is affected with erythroblastosis or when there is any previous history of the birth of jaundiced babies or of stillbirths without obvious cause.

At the same time, a modification of the present method of testing for compatibility between the bloods of donor and recipient must be made. Methods for carrying out these tests are described, and compromise procedures are discussed for cases in which there are no facilities for making these tests.

It is recommended that every blood bank takes steps to establish a panel of group O Rh-negative donors whose blood will be available, first, for the transfusion of the cases referred to above, and, secondly, for use as a routine in the transfusion of infants affected with erythroblastosis (when transfusion is required).

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The Queen has expressed her great interest in a plan launched by the Central Council for the Care of Cripples to open a new residential home and school for severely crippled boys, and has sent a donation of £1,000 which had been placed at her disposal by members of the "Bundles for Britain" branches in America to be used for the children of this country. The council has been deeply impressed with the great need for more homes in the reception areas for severely crippled children, who cannot easily move about and whose parents are often unable, under war conditions, to care for them adequately. The Shaftesbury Society, one of the council's affiliated members, which has had wide experience in running homes, has agreed to open this new home provided the necessary capital of £10,000 can be raised. About half the sum has now been promised.

OPTIMAL PROPORTIONS OF ANTIGEN AND ANTIBODY IN TESTS FOR Rh ANTIBODIES

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That the extent of the reaction resulting from mixing homologous antigen and antibody in a test-tube depends very largely on the proportions of the two reagents in the mixture has long been known. The proportions optimal for reaction can be determined (Dean and Webb, 1926; Duncan, 1932), but, while a particular antigen and its antibody will in suitable proportions produce a very well marked reaction, it may be possible, by varying the relative amounts of the same reagents, to get a mixture in which no reaction can be seen. Goldsworthy (1928) found that a specific antibody in the serum of a patient suffering from hydatid cyst of the lung could easily be missed unless the serum and the antigenic fluid, obtained from echinococcal cysts in the livers of cattle, were mixed in proportions suitable for producing flocculation. Workers with precipitins and bacterial agglutinins are familiar with zone phenomena due to the varying proportions of antigen and antibody in the different mixtures of titrations. Some human sera containing iso-agglutinins of the A-B-O system of blood groups show zoning: in titration with homologous red cells the best reaction does not take place in the mixture in which the iso-agglutinin is strongest, but in one in which it is more dilute, so that in a titration there is a pro-zone of one or more tubes in the series before those in which reactions are greatest. Although on very rare occasions a serum may be encountered which will show no agglutination with appropriate cells unless it is suitably diluted, zone phenomena give little trouble in testing for the A-B-O groups.

We have recently examined a sample of human serum containing antibodies for the Rh blood-group factor of Landsteiner and Wiener (1941). Used undiluted, this serum agglutinated the red cells of strongly positive reactors to anti-Rh, but failed to agglutinate cells from more weakly positive reactors. When, however, the serum was suitably diluted, definite and clear-cut agglutination was obtained with these weaker cells. Landsteiner and Wiener found marked differences in intensity in the reactions given by the red cells of different Rh-positive persons. Wiener (1942) mentions that with some human anti-Rh sera, used undiluted, reactions were inhibited until the sera had been heated at 56° C. for 10 to 20 minutes; the behaviour of the serum we describe was unaffected by heating. We obtained the serum in the middle of July, 1942, from a mother (B.)—of group O—who on Nov. 6, 1941, gave birth to a female child (her second), which died of erythroblastic anaemia on Nov. 16, 1941. B. is Rh-negative; a daughter born 2½ years ago is group O Rh-positive.

The Rh Factor

Red cells containing Rh react with immune sera prepared by injecting rabbits or guinea-pigs with the blood of rhesus monkeys. The factor is inherited as a simple Mendelian dominant and is present in about 85% of American whites. Boorman, Dodd, and Mollison (1942) have found a similar distribution in England. The examination of a small series of

* Working on behalf of the Medical Research Council.