

described should apparently be identical to those found by Itoh in Japan: it may prove that supernumeraries of this type are not uncommon. On the other hand, it is possible that by coincidence the supernumeraries arose separately in the Japanese population and the Sudanese population from which our stock is descended. Alternatively, the chromosome complement which includes the extra chromosomes may, at one time, have been widespread, and the Japanese and Sudanese populations may be survivors of this type. In these and other localities where they might occur the survival of supernumerary chromosomes could be explained by an adaptive advantage conferred by supernumeraries in particular stocks in these limited environments.

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¹ Mather, K., *J. Genet.*, **34**, 203 (1940).

² Itoh, H., *Jap. J. Genet.*, **10**, 115 (1934).

³ Darlington, C. D., and Upcott, M. B., *J. Genet.*, **41**, 275 (1941).

⁴ White, M. J. D., "Animal Cytology and Evolution", 121 (1943).

False Positive Results in the Diagnosis of Rare Blood-Group Antigens

MR. I. DUNSFORD's communication in *Nature* of December 5, p. 1059, directs attention to the importance of the detection of the rare sub-group A_4 , when uncommon blood-group antigens are being considered, such as family or 'private' antigens, which many workers have described.

In the diagnosis of these rare blood-group antigens, false positive results due to polyagglutinability of the red cells should be considered. Polyagglutinability of red cells is a condition in which a person's erythrocytes, while not usually being agglutinated by their own serum, are agglutinated by other normal sera of homologous *ABO* group; this agglutination occurs on slides, in tubes, or in capillaries, at room temperature.

Reepmaker¹ has recently documented twelve cases which have been described and he adds another case. I myself have seen five cases, all of which were observed as a result of difficulties arising during *ABO* typing. When a rare blood group is thought to be detected, such as group A_4 , one should bear in mind that this might possibly be a case of polyagglutinability of the red cells, and they should be tested with a large number of normal sera from donors of compatible *ABO* group. This is necessary because the percentage of compatible sera with which the affected cells react varies, in my experience, from 9 to 60 per cent. False positive results due to this cause may also occur during the detection of other rare blood-group antigens.

Dunsford gives the frequency of A_4 blood as 1:30,000; it is probable that polyagglutinability of

the red cells is a rarer condition, but its true frequency is unknown.

It is assumed that the samples of blood which are examined for these rare blood groups are collected aseptically and have remained sterile from the time of collection until tested. The examination of infected cell samples can give rise to false positive results similar to those which occur with polyagglutinable red cells.

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¹ Reepmaker, J., *J. Clin. Path.*, **5**, 266 (1952).

'Hidden' Anti-Rhesus Saline Antibodies

A *Rhesus* antibody of any given specificity, for example, anti-*D*, occurs in two common forms or states: as a 'saline antibody' that will agglutinate red cells suspended in saline, and as an 'albumin antibody' that will agglutinate red cells only if they are suspended in a colloid medium or if they are first treated with a proteolytic enzyme. The blood of a sensitized woman may contain either or both, but only the albumin antibody is demonstrable by standard methods in the blood of her newborn baby. Recently, we found saline anti-*Rhesus* antibodies in the blood of three such babies, following replacement transfusion in two and simple transfusion in one. Albumin antibody but no saline antibody was demonstrable in the blood of each of the three prior to transfusion. None of the donors' bloods contained anti-*Rhesus* saline antibodies or irregular agglutinins of any other type.

As an example, newborn baby C., with an albumin anti-*D* antibody of titre 16 and no saline antibody in its cord blood, received a replacement transfusion from donor S. At the end of the transfusion the titre of the antibody in the baby's blood was 4 in albumin, 8 in saline. On further investigation, we found that the change in the antibody in the cord blood could be reproduced in the test-tube. When one part of serum of donor S. was incubated with one part of cord serum of baby C., for 30 min. at 37°C., the antibody titre was 16 in albumin, 32 in saline. A similar experiment was carried out with the serum of donor S. plus the serum of Mrs. C., with similar results. Development of a saline antibody in the test-tube by this means has been carried out with a number of other sera.

We are calling serum of a person such as donor S. an 'antibody-transforming' serum, and the saline antibody that developed a 'hidden saline' antibody. Investigations to date indicate the following.

(A) Concerning antibody-transforming sera: (1) Perhaps one in ten normal sera has antibody-transforming activity in it to some extent. (2) Antibody-transforming activity is somewhat weakened but not abolished by heating at 56°C., for up to two hours. (3) Antibody-transforming activity is not affected by repeated freezing and thawing. (4) When *Rhesus*-positive red cells are incubated with an antibody-transforming serum, they are not altered so as to be agglutinated by an albumin antibody in the capillary or on the slide. (5) When *Rhesus*-positive red cells are sensitized by an albumin antibody and then washed three times in saline, an antibody-transforming serum will not agglutinate them.

(B) Concerning the 'hidden saline' antibodies: (1) We have so far demonstrated hidden saline anti-