necessarily incompatible with those here reported. At a given time the ratio of synthesis of hæmoglobin *A* to hæmoglobin *F* must be greater than the ratio of these two hæmoglobins in the blood due to the life-span of the red cells, to the increasing percentage of hæmoglobin *A* and to the expanding blood volume of the fœtus. Should fœtal red cells containing hæmoglobin *A* have a relatively shorter life-span than those containing hæmoglobin *F*, the synthetic ratio, *A*/*F*, might be even higher. However, our observations do not bear upon the question of whether separate red cells contain exclusively one or the other types of hæmoglobin[2,4].

We have assumed the presence of only two hæmoglobins, *A* and *F*. It is now generally recognized that these two fractions are heterogeneous[8]. Experiments to characterize these hæmoglobin subfractions and to evaluate their importance in hæmoglobin synthesis are in progress.

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Plasma Antihæmophilic-Factor Concentrations in the Australian Aborigine and in Conditions associated with Hyperglobulinæmia

While studying the plasma antihæmophilic-factor concentrations of a large number of healthy individuals, it was noted that Australian aborigines in general have higher plasma concentrations of this factor than the white Australian population. The factor was estimated by a technique[1] based on the thromboplastin generation test and results were expressed as a percentage of the concentration in a standard normal plasma. The lower histogram in Fig. 1 shows the plasma concentrations in 86 healthy white adults adjusted to the nearest 10 per cent. The values are skewed with a fairly sharply defined lower limit and range from 60 to 200 per cent of standard normal. This group comprised equal numbers of males and females, and there was no significant sex difference in the values. These normal results are very similar to those found in a previous group of 80 normal white adults reported on from this laboratory[2].

Concurrent with the examination of this normal group, blood samples were examined from 28 Australian aborigines who were hospital in-patients.

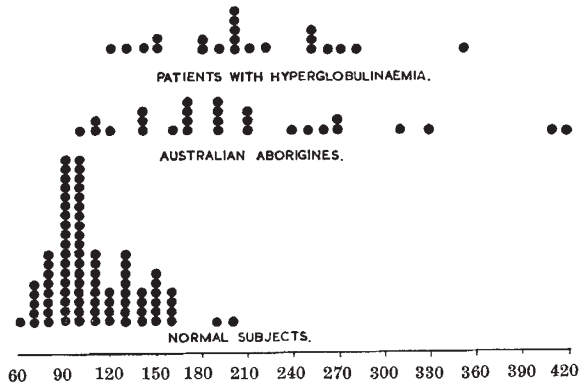

Fig. 1. Plasma antihæmophilic-factor concentrations in normal subjects, Australian aborigines and patients with hyperglobulinæmia

The plasma antihæmophilic-factor concentrations are shown in the middle histogram in Fig. 1. Values ranged from 100 to 420 per cent of standard normal and twelve samples showed values greater than the upper limit of the normal white range. The natives were of mixed purity from half caste to full blood. They were in hospital mostly for surgical or traumatic reasons. The higher values could be the result of different racial origin. However, the Australian aborigine has a higher concentration of plasma γ-globulin than the white Australian[3]. It was therefore decided to investigate white Australians who had hyperglobulinæmia as the result of disease.

The upper histogram in Fig. 1 shows the plasma antihæmophilic-factor concentrations in 22 white Australians who had hyperglobulinæmia as a result of varying diseases including lymphosarcoma, autoimmune disease, hepatic cirrhosis, nephrosis and multiple myelomatosis. Values ranged from 120 to 350 per cent of standard normal and approximated more to the aboriginal group than the white normal group. Nine samples showed values greater than the upper limit of the white normal range.

The plasma antihæmophilic-factor value is thought to be genetically determined and constant for the individual. The findings in the patients with hyperglobulinæmia indicate that the value can alter with disease. That plasma antihæmophilic-factor concentration may be related to the γ-globulin concentration is unexpected since the antihæmophilic-factor migrates electrophoretically with fibrinogen.

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Absorption–Elution Grouping of Dried Blood Smears

Thick blood smears (5–10 μl./cm.²) can be grouped in the ABO blood group system by an absorption-elution technique which has been evolved in this Laboratory over the past two years.

Dried blood smears, on standard microscope slides, prepared in the usual way, are fixed by plunging in *p*H 7·4 McIlvane buffer at 100° C. for 30 sec. The