Local action. ADP was infused into the left brachial arteries of three subjects at rates of 200, 300, 450 or 600 μg per min for periods of 2 min, at the rate of 2 ml. per min. In all cases immediate and marked vasodilatation occurred in the left hand, but not in the right. Hand blood flow returned to the control level between 30 and 60 sec after the infusion was stopped (Fig. 2). The amount of radioactivity in samples of left forearm venous blood did not alter significantly in any subject, either during or after any infusion.

The lowest systemic doses of ADP with an effect on circulating platelet levels were equivalent to concentrations of 4×10^{-6} M ADP in the subjects' total blood volume. Allowing a maximum 120 ml. per min for total forearm and hand blood flow during ADP infusion, the amounts given intra-arterially would have produced minimum concentrations of ADP ranging from 4×10^{-6} to 1×10^{-5} M; yet these produced no change in platelet levels. It must be concluded that the reduction in the number of circulating platelets which follows the systemic administration of ADP is due to their transient concentration within either the splanchnic or the pulmonary circulation. These observations suggest that the spleen may be a site of such concentration, but the evidence obtained so far for this suggestion is inconclusive.

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M. G. Davey
Harry Lander

Department of Medicine,
University of Adelaide,
South Australia.

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Absence of Di^{a+} in Malayan Aborigines

Since the Diego (Di^a) blood group was first reported[1] in 1954, this factor has been demonstrated only in Asiatic or Mongoloid peoples[2]. The survey recorded here for the Diego blood group in Malayan Senoi and Jakun people was an attempt to establish some positive genetic evidence for their affinity to Mongoloid groups in South-east Asia.

The anti-Diego serum and the Diego positive (Di^{a+}) red cells used in this work were kindly supplied by Dr. Miguel Layrisse, of Caracas, Venezuela. The activity of both lots of anti-serum sent from Caracas was satisfactory by the indirect Coombs test on Di^{a+} red cells.

The Senoi and Jakun form two of the three main groups of Aborigines found in Malaya. The other group of Malayan Aborigines, the Negritos, have not been included in this survey. The affinities of the Senoi and Jakun with other races have long been disputed. Two divergent hypotheses have been advanced as to their origins. One theory is that they are related to Mongoloid peoples of South-east Asia[3], and the other is that they are related to the Veddas of Ceylon and to the Australian Aborigines[4]. Polunin and Sneath[5] in their blood group investigations of the Senoi and Jakun concluded that they are not predominantly Caucasoid or Australoid, but are probably similar to primitive Mongoloid groups in South-east Asia.

A total of 270 unrelated Aborigines (Senoi and Jakun) of both sexes and all ages was studied for the Diego factor. About half the population examined was taken from the Aborigines Hospital near Kuala Lumpur, and the rest from Aborigine soldiers stationed in Kuala Lumpur. No cases of Di^{a+} were found in the Aborigines surveyed. Of 27 unrelated, healthy Malayan Chinese examined, two were Di^{a+}, which is about the frequency expected.

The absence of Di^{a+} in the Senoi and Jakun examined does not support the hypothesis that they are related to Mongoloid peoples of South-east Asia.

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J. Chin

Institute for Medical Research,
Kuala Lumpur, Federation of Malaya.

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Occurrence of Hæmoglobin H and Hæmoglobin Bart's in Alpha-thalassæmia: a Family with Two Possible Homozygous Cases and with G-6-PD-deficiency

It has now generally been accepted that hæmoglobin H, which is a tetramer of normal β-chains, is found only in α-thalassæmia, where the deficiency of α-chains has led to a surplus of β-chains. The problem, however, why in only a small portion of cases of α-thalassæmia can Hb H be detected, has not been solved until now[1]. Fessas[2] reports that at birth all patients with α-thalassæmia have 5 per cent or more Hb Bart's, which is a γ-chain tetramer, but that these patients afterwards develop Hb H only rarely. In family studies of patients with Hb H disease often one parent shows a thalassæmic blood picture, but no Hb H.

The following observations might lead to the assumption that possibly the problem is often a technical one.

In a family of mixed Chinese, Siamese, Indonesian and European descent two children, a boy of 10 years old and his brother of 3 years old, were found to have Hb H disease (Table 1, Nos. 6 and 8). Both showed moderate hypochromic anæmia with low mean corpuscular hæmoglobin, marked aniso- and poikilo-cytosis of the red cells, and many target cells. The serum iron was normal. After incubation with brilliant cresyl blue, inclusion bodies in many erythrocytes were seen. They had not been splenectomized. Table 2 shows the results of hæmoglobin analysis. The alkali-resistant Hb fraction was normal as determined with the technique of Betke *et al.*[3]. Hb A_2, determined quantitatively on a DEAE-'Sephadex' column, was low (1·2 per cent and 1·5 per cent respectively). With horizontal starch-gel electrophoresis, in a phosphate buffer *p*H 7·7, 0·0054 mole/l. according to Gammack *et al.*[4], two small anodal fractions were found in both patients. Our idea that these fractions might represent Hb H and Hb Bart's respectively was confirmed by Dr. Lehmann.

The hæmatological results in the family of these patients are also summarized in Tables 1 and 2. Three children (Nos. 4, 5 and 7) had normal red cell morphology and a normal hæmoglobin pattern. The father (No. 1), mother (No. 2) and one daughter (No. 3) showed slightly increased aniso- and poikilo-cytosis of the red cells. In both parents the osmotic fragility of the erythrocytes was