One result of the situation in Liverpool (referred to in your editorial of Feb. 24) that seems to be overlooked is the fact that the time between a patient seeing her family doctor and seeing a consultant in outpatients is seldom more than ten days. Should she need admittance, that will be another ten to fifteen days, unless her case is urgent.

From what we hear of long waits in places where work is organised better, we wonder just what the public wants from the N.H.S. They may prefer a long wait for admission to an efficient unit, putting up with frustration and anxiety to attain what paternal managers assure them is better. It is just possible, however, that they prefer not to be so frustrated and anxious. What weight is to be given to the patient's voice in such matters? Will it be heard more clearly in the reorganised N.H.S.? I had hoped that you would have magnified it and stressed that " management " has no magic solutions to its problems.

Finally, although full-time myself, I hope that my clinical colleagues attract large numbers of private patients to themselves. Here, such people cannot be accused of queue jumping.

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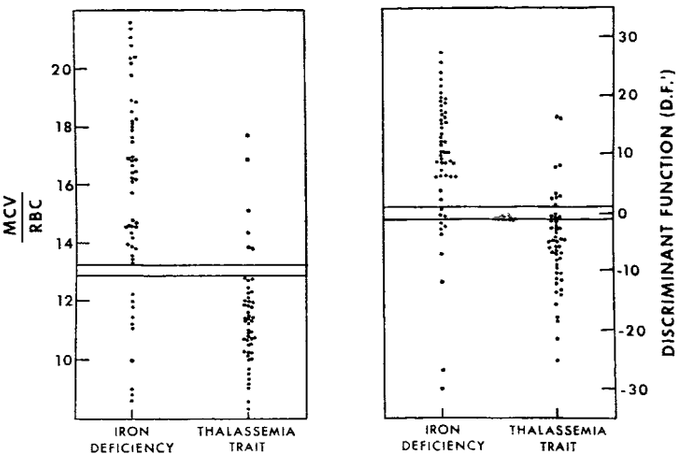
DIFFERENTIATION OF IRON DEFICIENCY FROM THALASSÆMIA TRAIT

SIR,—England and Fraser [1] reported that an index (D.F.′ = M.C.V. − R.B.C. − [Hb × 5] − 3·4) obtained from routine blood-counts could successfully distinguish thalassæmia trait from iron deficiency in patients with microcytosis. I have found that an even simpler index, M.C.V./R.B.C., is equally capable of distinguishing the two conditions.

The accompanying figure compares the efficacy of D.F.′ and M.C.V./R.B.C. in separating 53 iron-deficient patients seen at the San Francisco General Hospital from 50 patients with either α or β thalassæmia trait. Patients with iron deficiency, identified by review of the hospital clinical-chemistry records, had serum-iron levels below 50 mg. per 100 ml., transferrin less than 14% saturated with iron, M.C.V. of less than 80, and Hb greater than 9 g. per 100 ml. Patients with lower hæmoglobin levels were excluded to allow a more useful comparison with thalassæmia trait, where hæmoglobin levels are almost always above 9 g. per 100 ml. β-thalassæmia trait was confirmed by measurement of A_2 and F hæmoglobin, and α-thalassæmia trait by a family history of microcytosis associated with normal A_2 and F hæmoglobin levels and unresponsiveness to iron therapy. In some instances, the diagnosis was further confirmed by in-vitro globin-chain synthesis studies or by the presence of hæmoglobin H or Bart's hæmoglobin in relatives.

87 of 103 patients were correctly classified by M.C.V./R.B.C. and 86 of 103 by the D.F.′. Both indices appear to be equally accurate, but M.C.V./R.B.C. has the virtues of simplicity and ease of recall. Thalassæmia-trait patients were erroneously classified as iron-deficient when other causes of anæmia, such as hæmolysis, bleeding, or pregnancy were present, as previously found by England and Fraser. In 4 iron-deficient patients who were polycythæmic due to chronic obstructive pulmonary disease, M.C.V./R.B.C. and D.F.′ fell in the thalassæmia-trait range. Partially treated iron deficiency was also spuriously classified as thalassæmia trait. In general, coexisting diseases which influence the red count invalidate both M.C.V./R.B.C. and D.F.′ as diagnostic indices in iron deficiency and thalassæmia trait.

6 patients with evidence of both iron deficiency and thalassæmia trait were analysed separately. In 3 who were anæmic, M.C.V./R.B.C. and D.F.′ indicated iron deficiency, while in the other 3, who did not have anæmia, thalassæmia trait was indicated. The inconsistent performance of these indices in the combined presence of iron deficiency and thalassæmia minor may limit their usefulness in screening surveys carried out in parts of the world where both iron deficiency and thalassæmia minor are prevalent. Where other complicating diseases are not present, a decision as to which further laboratory diagnostic studies are required in patients with microcytosis should be greatly facilitated by determination of the M.C.V./R.B.C. or the D.F.′.



Comparison of M.C.V./R.B.C. (left) and D.F.′ (right) in patients with iron deficiency or thalassæmia trait.
Area of uncertainty indicated by horizontal lines.

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ACANTHOCYTES AND HYPOBETALIPOPROTEINÆMIA

SIR,—The interesting observation on simultaneous association of acanthocytosis and dietary hypobetalipoproteinæmia by Dr Gracey and Dr Hilton (March 24, p. 679) on Australian aboriginal infants has also been seen in familial low-density-lipoprotein abnormalities.[1] That study, involving 13 members of a kinship, showed typical acanthocytosis and anisocytosis in the propositus's fasting red cells when they were incubated in tissue-culture medium containing 10% autologous serum. In vitro, the abnormal erythrocytes recovered normal morphology almost instantaneously by addition of small amounts of hyperlipæmic sera or glycerol solutions. Furthermore, this patient's red cells remained normal under similar test conditions when her serum-cholesterol levels reached, by treatment, values of 100 mg. per ml. or higher.

It seems reasonable, therefore, to suggest that familial and possibly acquired acanthocytosis represent reversible phenomena resulting from temporary alterations in the biophysical characteristics of the red-cell membrane induced by reduction in circulating low-density lipoproteins.

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1. England, J. M., Fraser, P. *Lancet*, March 3, 1973, p. 449.

1. Mars, H., Lewis, L. A. Lazzarini Robertson, A., Jr., Butkus, A., Williams, G. H., Jr. *Am. J. Med.* 1969, **46**, 886.