Vickery (*5*) and that collected by Martell and Calvin (*4*) on ethylenediamine tetraacetic acid chelates. The ionic radii of the lanthanons have all been obtained from the work of Templeton and Dauben (*6*) and those of the other elements from Ketelaar (*7*). Davies considers that covalent bonding occurs in those cases in which the stability of the bonds is *greater* than that expected for an alkali-like ion of identical Z^2/r value. It is apparent from the figure that the lanthanon chelates are ionic in nature, the stabilities actually being less than expected. Their behavior contrasts markedly with that of the transition elements included in the figure for comparison. A possible explanation for the behavior of the lanthanon chelates lies in the small size of the ions and their large charge. The required number of chelate groups are prevented from approaching one another as closely as expected in the resultant complex because of their mutual repulsions. An extension of the figure would show that the Fe^{III} and Cr^{III} complexes are less stable than expected for ionic bonding. This does not prevent considering these as ionic complexes in spite of the optical stability of any resolved complexes. Such stability is primarily dependent upon the magnitude of the instability constant rather than the type of bonding.

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New Hemoglobin Possessing a Higher Electrophoretic Mobility than Normal Adult Hemoglobin

We have observed a new abnormal hemoglobin, moving with a higher electrophoretic mobility than the normal adult hemoglobin, in two members of a Chinese family. This is the sixth abnormal hemoglobin discovered since Pauling *et al.* (*1*) characterized the first abnormal hemoglobin in sickle cell anemia.

Five members of this family have been studied since we first saw one of them in our office on 23 Mar. 1954; they are represented by solid enclosures on Fig. 1. They were seen because of a severe hypochromic, microcytic anemia that was indistingishable on stained smears from hereditary leptocytosis, first noted in patient 9. A similar picture was detected in the blood of patient 10; and both patient 9 and patient 10 give a lifelong history of easy fatigability and

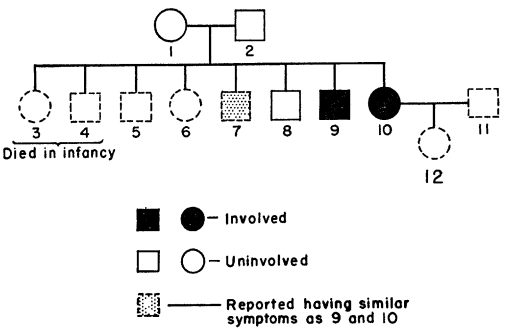

Fig. 1. Genetic table of family showing occurrence of new abnormal hemoglobin.

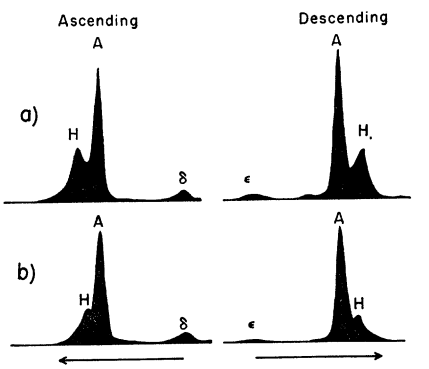

Fig. 2. Electrophoretic patterns of hemoglobin: (*a*) from subject 10; (*b*) from subject 10 mixed with normal.

both have splenomegaly. Blood from subjects 1, 2, and 8 showed no abnormality.

Electrophoretic analyses of hemoglobin from subjects 9 and 10 performed at that time in Veronal buffer *p*H 8.6, ionic strength 0.1, revealed two distinct hemoglobins, as is shown in Fig. 2*a*. The slower one has the same electrophoretic mobility as the normal adult hemoglobin, as can be seen in Fig. 2*b*, where hemoglobins from a normal individual and patient 10 were mixed. The faster hemoglobin, accounting for 35 percent of the total, is abnormal and is hitherto undescribed. Electrophoretic patterns of subjects 1, 2, and 8 showed only adult hemoglobin.

Since hemoglobin G is the most recently described (*2*), it is proposed that the abnormal hemoglobin described here be designated as hemoglobin H. The genetic pattern does not seem to follow that of other abnormal hemoglobins, since neither parent possesses the abnormal hemoglobin.

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