REPORT

NOMENCLATURE OF ABNORMAL HEMOGLOBINS

In the course of the VIIIth International Congress of Hematology, September 1960, in Tokyo, Japan, 44 members met to discuss the nomenclature of the human hemoglobins. This field is expanding so rapidly that it is not possible even at an International Congress to obtain a full representation of all disciplines and groups of workers. The absence of several distinguished investigators in this field was acutely felt. Nevertheless the VIIIth International Congress of Hematology was considered the best possible occasion for such a gathering. The following recommendations are being sent to appropriate journals so that any of the distinguished investigators not present will be able to criticize them in public.

1. The recommendations on nomenclature made at the Symposium on Abnormal Hemoglobins in Istanbul, 1958, and published in 1959, are endorsed and the letters A–N (with the exception of B), and S are recognized as naming hemoglobins as there defined.

2. The description of the variants of hemoglobin S1 as M, M, Ms (from Boston, Milwaukee and Saskatoon respectively) is accepted, and it is suggested that new hemoglobins M are described with fully subscripted names until they have been shown to differ from these three when they should be given subscript initials (Mwat, possibly later M1).

3. The letters O, P and Q are being allotted to the hemoglobins described under these letters.

4. Until the next meeting of the International Congress the letters R–Z (excepting S) should not be allotted to new abnormal hemoglobins but these should be given names of localities. It should be left to the individual workers to choose the most meaningful name from the origin of the propositus, or the laboratory, hospital, town or district where the hemoglobin was found. A new name should not be allotted in this way unless it has been ascertained that the hemoglobin to be named is different from all those adequately described in the literature.

5. Of the two designations of the hemoglobin A2 variant: A2 and B2, the first is found more acceptable. If a third variant should be found it should be named A2 and not C2.

6. The names of the three known peptide chains of human hemoglobin are α, β, and γ, and it is suggested that the normal chains should be designated in that way (i.e. not α+ or α+, β+ and γ+). The genetic superscript for the normal gene product + (α+, β+, γ+) will not be used as it implies to the chemist a positive charge.

7. The expressions β1 for H and γ4 for Bart’s may be used when their identity is fully accepted. However, until the next meeting of the International Congress the traditional names should be mentioned at least once in each publication.
8. The present custom to describe an abnormal chain by adding the name of the appropriate abnormal hemoglobin in superscript should be maintained \((S = \alpha\beta_2; \text{Hopkins-2} = \alpha_2\text{Hopkins-2} \beta_2)\).

9. A polypeptide chain should not be designated with a new small Greek letter (such as \(\delta, \epsilon, \) etc.) until chemical evidence for complete separate identity from the \(\alpha, \beta\) and \(\gamma\) chains (such as exists between these chains themselves) has been established, genetic differences notwithstanding.

10. It is expected that the analysis of the aminoacid sequences in the globin molecule will eventually be followed by a precise chemical nomenclature. Meanwhile if a hemoglobin has been identified by the usual methods of electrophoresis, chromatography, spectroscopy, alkali denaturation, cold denaturation, and solubility tests, it should be described by the accepted capital letter as hitherto: \((S, C, D, G, E, \text{etc.})\) as recommended by the Working Party meeting at the VIth International Congress, Boston, 1956.

If comparison has been made on the same lines and with the same completeness with a hemoglobin carrying the name of a locality that name should be applied. If the abnormal chain is identified this should be indicated by a subscript; (for example: \(D_\alpha, D_\beta\)) and until the full identity by examining the aminoacid sequences has been established this subscript should be followed by a declaration of origin \((D_\beta \text{ Los Angeles}, G_\alpha \text{ Ibadan})\). This implies that \(D_\gamma\) will have to be renamed \(D_\beta \text{ Punjab}\). If two or more of such hemoglobins are then found identical by analysis of the aminoacid sequences, only the name of the hemoglobin which was first discovered and fully defined by the conventional methods should be retained. For example if \(G_\alpha \text{ Ibadan}\) and \(G_\alpha \text{ Azusa Kili}\) are found to be identical regardless of which the aminoacid sequence has been fully examined first \(G_\alpha \text{ Ibadan}\) should be the remaining name. However, until the next meeting of the International Congress the alternative names should be mentioned at least once in each publication.

11. No generally acceptable name was agreed upon for the familial condition in which hemoglobin F persists into adult age without morphological changes of the red cells and without anemia. The term non-microcythemic thalassemia was considered not specific enough as it might equally apply to the familial condition where hemoglobin A\(_2\) is raised without associated morphological changes. If the expression “high F gene” is used this should be done with reservation and only provisionally until further knowledge allows better terminology.

12. If several hemoglobins are present the phenotype should be designated by listing the hemoglobins in order of decreasing concentration regardless of genetical considerations (sickle-cell trait \(= AS\), sickle-cell anemia \(= SF\), sickle-cell thalassemia \(= SAF\), or \(SFA\), etc.)

REFERENCES

3. Pisciotta, A. V., Ebbe, S. N. and Hinz, J. E.: Clinical and laboratory features
OBITUARY


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PAUL CHEVALLIER
(1884-1960)

Early in his medical career, Paul Chevallier was introduced to the study of hematology by the well known histologist J. Jolly and the great clinician P. E. Weil. His first experiments, a study of iron assimilation in the spleen, were important for the development of Aschoff's work on the reticuloendothelial system. While physician to the Paris hospitals, Chevallier divided his activity between hematology and dermato-syphilology. The latter led to three observations which he applied to hematology: the acute peripheral leucolysin following arsenobenzol injection, the description of chronic inflammatory purpura, so much different than the thrombocytopenic purpura known at that time; and finally, while studying Quincke's edema and certain forms of leukoderma he proposed the idea of "metanemia", a non-anemic disease sometimes responding to iron therapy. This has been subsequently proven by serum iron determination. In recent years appointed Honorary Professor at the Faculty of Medicine of Paris, he wanted to do more than simply teach and to prepare his textbook Les Maladies du Sang. Despite poor health he traveled extensively, particularly to the Orient. While in Iran, he died in the midst of preparations for a series of lectures.

Keen researcher, great teacher and physician, Paul Chevallier brought the prestige of French medicine to new heights. He was the distinguished heir to the pioneers Hayem, Vaquez and Weil.

His numerous pupils in France and elsewhere will forever remember in their hearts this great teacher and friend.—G. Bilski-Pasquier, Paris, France.