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thalassaemia. The mother's blood contained 23% haemoglobin D, the remainder being mainly haemoglobin A; this is consistent with haemoglobin D trait (Chernoff, 1958). In the three siblings haemoglobin A was completely lacking; the haemoglobin present was almost entirely haemoglobin D. These findings indicate that all three siblings inherited a D beta gene from the mother and a beta thalassaemia gene from the father. The substitution of D beta chains for normal beta chains in the haemoglobin composition of the three siblings represents genetic interaction between the two inherited abnormalities which in this instance led to a complete suppression of haemoglobin A synthesis.

Since the laboratory findings in haemoglobin D–thalassaemia are little different from those of homozygous haemoglobin D disease, it is essential to carry out a family study if the true nature of the abnormality is to be determined. It is interesting to note that all three siblings were doubly heterozygous for haemoglobin D and thalassaemia and that none of other possible genetic alternatives occurred.

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