operation suggest that major surgical interference, with burns colonized by bacteria, early in the after-burn period may lead to the establishment of systemic infection.

Biochemical, Histological, and Serological Investigations in Asymptomatic Carriers of Australia Antigen

D. M. GOLDBERG (*The Royal Hospital, Sheffield*), R. I. RUSSELL AND J. G. ALLEN (*Royal Infirmary, Glasgow*), R. N. M. MACSWEEN (*Western Infirmary, Glasgow*), AND J. WALLACE (*Glasgow and West of Scotland Regional Transfusion Centre*) Thirty-nine blood donors found to be Australia antigen or antibody positive were studied for evidence of hepatic disease. Twenty donors gave a history of possible exposure to infection with viral hepatitis. Seven had severe and five had minor biochemical abnormalities; eight of these 12 subjects had Australia antigen and four antibody; 23 subjects had normal biochemistry. Of 11 donors who underwent hepatic biopsy, three were found to have evidence of chronic persistent hepatitis. Two of these three had antigen and one had antibody. Minor histological abnormalities were found in a further five donors. Five donors with normal biochemistry were biopsied, and one of these was found to have chronic persistent hepatitis.

The results suggest that subjects with persistently positive tests for Australia antigen are at risk of developing chronic hepatic disease, and that those with positive tests for antibody are at similar risk. Close follow up of blood donors and other subjects found to have positive tests is thus indicated. Although abnormalities in biochemistry may be present in such subjects, normal biochemical tests do not exclude abnormal hepatic histology. Conventional 'liver function tests' were of little value, and all other antibody tests yielded negative results. Serum enzymes were far the most sensitive indices of probable hepatic involvement, aspartate transaminase, isocitrate dehydrogenase, glutamate dehydrogenase, and adenosine deaminase being the most useful of the eight enzymes studied.

Recent Developments in Globin Structure

H. LEHMANN (*Addenbrooke's Hospital, Cambridge*) The duplicated β-chain is the δ-chain. They differ by 10 out of 146 residues and one forms part of Hb A ($\alpha_2\beta_2$), and the other of Hb A_2 ($\alpha_2\delta_2$) (Lehmann and Lang, 1973). On unequal crossing-over of two chromosomes with the genes (δ'-β' two types of fusion chromosomes result, one—($\delta\beta'$)—with the gene for the δ/β chain of Hb Lepore $\alpha_2(\delta/\beta)_2$ but no genes for a δ or a β chain, and the other $\delta'(\beta\delta')\beta'$ with a gene for the β/δ fusion chain of Hb anti-Lepore and additional genes for a δ and a β chain.

If the α chain is duplicated no chemical difference has so far been described. On unequal crossing-over, fusion chromosomes with a gene for one (α') and three α chains ($\alpha'\alpha'\alpha'$) should arise corresponding to those for the chromosomes for haemoglobin Lepore and anti-Lepore.

A heterozygote for the first would have three, and a homozygote two, instead of four α chain genes: and a heterozygote for the latter five, and a homozygote six instead of four. The first two conditions would be expected to cause an α-thalassaemia, and the latter a globin chain imbalance with a surplus of α chains. The resulting 'β-thalassaemia' would show a normal proportion of δ to β chains, ie, a normal haemoglobin A_2. One would not expect the cells to be hypochromic. The doubly abnormal heterozygote (α') ($\alpha'\alpha'\alpha'$) would be expected to be normal clinically but a carrier capable of handing a thalassaemic disorder to his offspring.

Reference

Lehmann, H., and Lang, A. (1973). Different aspects of alpha thalassemia. (Third Conference on Cooley's Anemia, 9-10 April 1973). *Proc. N.Y. Acad. Sci.*, in press.

The Early Diagnosis of Monocytic Leukaemia Based on a study of 91 Cases

J. G. HUMBLE (*Westminster Hospital, London*) In the past 36 years, 91 cases of monocytic and myelomonocytic leukaemia have been seen by the author at Westminster Hospital. There were 54 males and 37 females. The age distribution ranged from 6 weeks to 93 years. A slight preponderance of males was seen in the decade 59-69 whereas the female peak was seen in the decade 49-59. Using conventional staining methods and also by examining the living cells (Pulvertaft and Humble, 1960; Humble, 1967) the cases were divided into the two categories: true monocytic leukaemia (Schilling) 26 cases and myelomonocytic (Naemgeli) 65 cases. The series was studied to show the initial symptoms, signs, earliest blood count, and survival from the earliest symptom that could be confidently attributed to leukaemia to the death of the patient. It was found that in the true monocytic form the overall survival is much worse than in the myelomonocytic form. As to the presenting blood counts, 62% of the cases had a haemoglobin level of less than 9 g/dl and 45% had a white cell count of less than 10 000 per microlitre. Aetiological features in this series, and especially pre-leukaemic disturbances in the blood counts, will be discussed, as will the effect of treatment on survival.

References

Humble, J. G. (1967). Diagnosis and treatment of monocytic leukaemia. (Summary). *Proc. Roy. Soc. Med.*, **60**, 1310.