(52 cases); (d) benign prostatic hypertrophy—clinical diagnosis only (60 cases); (e) other cancers (57 cases); (f) other urological illness (22 cases); (g) miscellaneous disease (116 cases); (h) undiagnosed (15 cases). The mean activity (SD) of the miscellaneous group was 1.97 (1.07) IU/litre at 37°C, giving an upper normal limit (mean + 2 SD) of 4.1 IU/litre. The AcPase values in this group did not correlate with age ($r = 0.040$). Eight patients with untreated carcinoma of prostate (22%) had values ≤ 4.1 IU/litre. Six of these had no prostatic enlargement and no metastases; the remaining two had enlarged prostates but no evidence of metastases. All 28 patients in this series with soft tissue (7) or bony (21) metastases had AcPase > 4.1 IU/litre. Values > 4.1 IU/litre were found in seven cases (6%) of benign hypertrophy, three cases (5%) of non-prostatic cancer, two cases (2%) of miscellaneous illness, and one case (6%) of uncertain diagnosis. No values > 4.1 IU/litre were found in the patients with other urological illness.

Sixty per cent of patients (26 cases) with untreated prostatic carcinoma had elevated serum alkaline phosphatase activity (APase), ie, > 15 KAU/100 ml as determined by an AutoAnalyzer adaptation of the method of Kind and King (1954). Correlation between the two phosphatases was not significant ($t = 0.294$; $P > 0.05$). APase was elevated in the presence of normal AcPase in only three cases of prostatic cancer, but all three were without metastases. Two further cases with minimal elevation of AcPase had APase > 50 KAU/100 ml. While APase is less frequently elevated than AcPase in prostatic cancer patients, it provides valuable confirmation of the diagnosis when the latter is normal or equivocal.

References

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A Survey of Hepatitis in Laboratories

N. R. GRIST (*Department of Infectious Diseases, University of Glasgow*) A preliminary enquiry from ACP members suggested a rising incidence of hepatitis which was reported from 58 of 285 laboratories for 1964-69 from 32 of 289 for 1970. Of 127 cases, 87 involved technicians and 48 affected workers in biochemistry. A continuing more detailed enquiry was initiated with the following results:

Information for 1970 from 244 laboratories showed 13 hepatitis cases in 12 laboratories. Attack rates were highest in science microbiologists (1/126 = 0.79%) and biochemistry technicians (7/1438 = 0.49%), the overall rate being 13/11253 (0.116%).

Information for 1971 from 215 laboratories showed 18 hepatitis cases in 15 laboratories. Attack rates were highest in science biochemists (2/315 = 0.63%), medical haematologists (2/315 = 0.64%), and medical morbid anatomists (2/390 = 0.51%); no cases were reported among biochemistry technicians.

Hepatitis B ('Australia') antigen was found in 11 of 22 cases tested over the two years and was associated with less mild illness; there was no death.

Among laboratories with hepatitis cases, more than average tested specimens from haemodialysis, transplant, and haemophilia units, and from drug addiction centres (1971 but not 1970). Risk was unaffected whether high-risk specimens were tested by general staff, special unit staff, or not accepted, or whether they were tested on mechanized apparatus in general use or segregated or not tested on such apparatus. Only slightly increased risk was found in laboratories testing for Australia antigen.

Bacteraemia in the Burned Patient

H. J. K. M. MAMATTAH, C. H. BEARD, AND D. M. JONES (*Withington Hospital, Manchester*) Burns are regularly colonized by a variety of bacteria, often without clinical evidence of infection. In some patients, due to various factors, colonization progresses to invasion and septicaemia. A study has been made of the influence of operative procedures, ie, desloughing and excision of necrotic tissue on the infective process. A series of unselected operations were monitored by taking a series of blood cultures while the operations were in progress. Out of 52 blood cultures, 17 (33%) were positive and nearly half of these grew more than one organism. Bacteraemia was detected in 40% of the operations studied and often appeared to be of 10 to 20 minutes' duration. The bacterial species grown by blood culture were always present colonizing the burn although not all the colonizing species were recovered in the blood cultures. Attempts were made to relate the findings to the severity of the burn, to the magnitude of the operative procedure, and to the time that had elapsed between the burn and the operation. In spite of bacteraemia none of the patients studied were treated with antibiotics and in no case was systemic infection established. Immunoglobulin levels are depressed for a few days after the patient is burnt but in the patients studied immunoglobulin levels were found to be normal at the time of operation and bacteraemia. The average timing of the operations was 20 to 25 days after the burn. Comparison of these results with observations made when septicaemia became established following

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 A₂ ($\alpha_2\delta_2$) (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 A₂. 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.

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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

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