The Association of Clinical Pathologists: 85th General Meeting

The 85th general meeting of the Association of Clinical Pathologists was held at Imperial College, London on 24 and 25 September 1970. Most of the papers given as free communications are abstracted below. There were two symposia, the first on 'Prenatal diagnosis' (Chairman, C. O. Carter) and the second, 'Uses and control of the newer antimicrobial drugs' (Chairman, Mark Ridley). There was also a seminar on 'Growing points' with Air Vice Marshal W. P. Stamm in the chair, and a slide seminar on 'Lesions of the central nervous system' with Professor P. M. Daniel in the chair. The Dyke Foundation Lecture entitled 'Trends in pathology—a reciprocal essay' was given by A. G. Signy, and is published on page 744. Magnus Haines delivered the Presidential Address on the theme 'The emergence of pathology in gynaecology'.

SH (Australia) antigen in early life

G. C. TURNER, ANNE M. FIELD, RAGAA M. LASHEEN, R. MCL. TODD, AND G. B. BRUCE WHITE (*Liverpool and Colindale*)

After receiving an injection with an unsterile syringe a young man in Liverpool developed serum hepatitis with a positive test for SH (Australia) antigen. About two months later his wife developed antigen-positive hepatitis; the day after she became jaundiced she gave birth to a female child after 35 weeks' gestation.

Apart from immaturity and physiological jaundice the child was normal and no antigen was detected at birth or after 32 days. At 59 days, however, the test for antigen was strongly positive and also at 80 days, by which time a marked elevation of SGPT had occurred. The child remained quite well with no jaundice or other clinical evidence of disease but positive tests for antigen and elevated SGPT levels persisted for at least eight months.

Electron microscopy of the first positive sera from the child showed three types of particle: a few small round forms (about 22 nm), rather more long forms, and larger double-shelled particles 42-44 nm with evidence of internal structure. There was no sign of clumping at this stage but eight months later, when some weakening of the precipitation and complement-fixation reactions for antigen had occurred, there was clumping which involved all

three forms of particle. This suggested slow development of the response which in adults is associated with elimination of the antigen. It was too early to say whether the child would become a chronic carrier or whether the liver damage revealed only by elevated transaminase levels would develop into chronic disease.

The reappearance of malaria in the USA

G. J. CUNNINGHAM (*Richmond, Virginia*)

Following the period between 1956 and 1965 when the malaria incidence in the USA was uniformly low there was a marked increase in 1966 which has been maintained. In 1969, there were 3,806 cases. These cases were almost entirely in war veterans returning from South East Asia, mainly Vietnam. Although troops going into Vietnam were given prophylactic doses of chloroquine and primaquine a sharp outbreak of falciparum malaria had occurred in late 1965. This was attributed to the chloroquine resistance of the local strain of falciparum. On returning to the USA a number of veterans developed malaria, *P. vivax* being the cause in about 80%. It was suggested that many of these men on their return had failed to complete their course of prophylaxis as prescribed.

Of these cases occurring in the USA a few have been fatal. All were caused by *P. falciparum*, some being in soldiers returning from Vietnam, others in civilians who had recently been to West Africa. One disturbing feature is the number of cases, a few of which have been fatal, where malaria has developed following blood transfusion. In the future more stringent conditions will have to be enforced on prospective donors who have suffered from malaria. There have been a few 'introduced' cases, and although the local mosquitoes have been infected by parasites, the density appears insufficient to provide a reservoir for local infection on a large scale.

A new colorimetric method for blood alcohol determination

SIDNEY B. ROSALKI (*London*)

A new colorimetric procedure for blood alcohol determination is described. Blood proteins are precipitated and the protein-free supernatant incubated with nicotinamide-adenine dinucleotide (NAD), alcohol dehydrogenase (ADH) nitro blue tetrazolium (nitro BT), and phenazine methosulphate (PMS). Ethanol in the sample reduces the NAD, and the reduced NAD so formed reduces nitro BT to a coloured formazan, PMS serving as an intermediate electron carrier. The reaction is allowed to proceed to completion and colour formation quantitatively relates to

sample alcohol concentration. An ethanol standard of known concentration is included with each batch of determinations and sample alcohol concentration calculated from this. The method permits the examination of large numbers of samples with rapidity and precision and without the need for specialized apparatus. Comparison with an ultraviolet enzymatic procedure for blood alcohol determination gave excellent agreement.

Vascular lesions of malignant essential hypertension

N. G. SANERKIN (*Cardiff*)

The individual vascular lesions of malignant essential hypertension are essentially identical with those of renal cortical necrosis. They show all the features expected in ischaemia with failed reflow (implicating obliterative spasm of over three hours' duration) and of ischaemia with good reflow (implicating obliterative spasm of one to three hours' duration). With the lower grades of ischaemia, there is selective necrosis of medial muscle cells. 'Fibrinoid change' and proliferative endarteritis are secondary or reactive lesions, arising as a result of the primary ischaemic lesions. A full account is being given elsewhere (Sanerkin, 1970)

Reference:

Sanerkin, N. G. (1970). Vascular lesions of malignant essential hypertension. *J. Pathol.* in press.

An amyloid lung

A. P. PRIOR

After an influenza-like illness a middle-aged housewife developed cough with sputum and increasing breathlessness. Symptoms and signs worsened despite treatment and she died in eight months.

At necropsy changes were confined to the lung. There was gross alteration both in the macroscopic and microscopic features. Amyloid had affected the parenchyma and this in turn showed ossification and calcification.

Haemoglobin levels in nursery school children

D. M. D. EVANS (*Cardiff*)

A study of 1,074 apparently healthy children aged between 2 and 5 years revealed a wide range of haemoglobin levels, with a characteristic distribution curve pattern between 8.4g/100 ml (56% and 15.2g/100 ml (104%). In round figures the mean haemoglobin level was 12g/100 ml (81%). The effect of seasonal variation on haemoglobin level appeared negligible. If 10.5g/100 ml (71%) is taken as the figure below which anaemia is deemed to

exist, then 0.3% of infants at private nurseries were anaemic, compared with 3.7% in Local Authority nurseries, and 15% of children taken into care.

The relationship of haemoglobin level and hypochromia to iron response was studied.

Reaction to long-term oral anticoagulant therapy. A comparison of prothrombin and partial thromboplastin tests

R. D. EASTHAM (Bristol)

Over a period of three years 2,178 blood samples were tested from 88 patients, who between them underwent a total of 170 years of long-term oral anticoagulant treatment. The prothrombin ratio and the corresponding activated partial thromboplastin clotting time (PTT) were estimated on each blood sample, the daily dosage of oral anticoagulant being regulated to maintain the PTT between 50 and 70 seconds regardless of the prothrombin ratio. From the results obtained during each year of

treatment from each patient the PTT corresponding to a standard prothrombin ratio of 2.0 was calculated. This PTT result corresponding to a reference prothrombin ratio was significantly shorter in patients treated following attacks of venous thrombosis than in patients treated following myocardial infarction or in patients with mitral valve disease treated following embolic attacks. This reference PTT was even shorter in patients in whom a frank attack of acute thrombophlebitis developed during anticoagulant treatment, whatever the original condition necessitating anticoagulant treatment.

These results, taken with the known marked prolongation of the PTT by heparin, suggest that either a 'thromboplastic-like' substance or an anti-heparin substance is released into the circulation during an acute episode of thrombophlebitis, which results in reduction in the PTT unless larger doses of oral anticoagulant are given. It is also suggested that these findings might indicate longer periods of treatment with oral anticoagulants following venous thrombosis.

Cholestatic jaundice following treatment of chronic granulocytic leukaemia with busulphan

J. C. E. UNDERWOOD, R. T. SHAHANI, AND E. K. BLACKBURN (Sheffield)

A 25-year-old male with chronic granulocytic leukaemia developed cholestatic jaundice after over six years' treatment with busulphan, the total dose of this drug being 4.075 g of which 0.7635 g was given in the 12 months preceding the onset of jaundice as compared with an annual average intake of 0.6623 g of the drug during the previous five years. This virtually coincided with the onset of acute relapse. Histological findings at necropsy, while confirming acute relapse of leukaemia with only minimal hepatic involvement were, however, also those of cholestatic jaundice consistent with drug hypersensitivity. We have not found in the literature a similar instance of cholestatic jaundice probably due to busulphan.

Book reviews

Homeostatic Regulators Edited by G. E. W. Wolstenholme and J. Knight (Pp. viii + 329; illustrated.) 75s. London: J. and A. Churchill. 1969.

This book constitutes the edited proceedings of a symposium organized jointly by the Ciba Foundation and the Wellcome Trust early in 1969.

The subjects discussed ranged over a wider field than might have been expected from the title of the symposium. Presentations on cell population kinetics (L. F. Lamerton) and chaperones (O. H. Iversen) were to be expected. Less expected were discussions on regulatory systems in cell culture (M. G. P. Stoker), histones (E. W. Johns), nerve growth factors (C. A. Vernon and colleagues), cell surface structure and contact control (J. A. Forrester, L. Wolpert, and D. Gingell), and regulatory mechanisms in antibody synthesis (G. Möller). Surprisingly, hor-

mones are only discussed in passing. Both the definitions of homeostasis given in Stedman's Medical Dictionary (1 The state of equilibrium in the living body with respect to various functions and to the chemical composition of the fluids and tissues, eg, temperature, heart rate, blood pressure, water content, blood sugar, etc. 2 The process through which such body equilibrium is maintained) obviously refer to whole living organisms. The application of the term to regulatory mechanisms at the level of individual organs or tissues and to feedback control mechanisms at the subcellular level, as studied in cultured cells or by purely biochemical methods, seems to deprive it of much of its conceptual value.

Notwithstanding this criticism the book is valuable, because it highlights the poverty of our knowledge of homeostasis (by Stedman's definition) and because it provides an excuse for considering the role of factors such as interferon (N. B. Finter and D. C. Burke) and lysosomes (P. J. Jacques) as regulators. A remarkable feature is the professional quality of the editing which makes the discussions after each paper well worth reading.

FRANCIS J. C. ROE

Die progressiv-dystrophischen Myopathien By H. Heyck and G. Laudahn. (Pp. xv + 436; illustrated. DM118.00.) Berlin, Heidelberg, and New York: Springer-Verlag. 1969.

In the past decade there has been a great increase of interest in the pathophysiology of skeletal muscle, largely as a result of the stimulus provided by various organizations devoted to research into muscular dystrophy. One outcome of this has been the publication of several first-rate monographs upon the myopathies, among which must now be included the comprehensive analysis written by Heyck and Laudahn.

The first chapter, an historical and clinical review of muscular dystrophy, stems from Heyck's long interest in this subject as chief of the Department of Neurology in the Rudolf-Virchow Hospital in Berlin. He has also written the histopathological section, with the cooperation of Lüders (pathologist at the Wenckebach Hospital). The ultrastructural changes noted in muscular dystrophy are well described by Freund-Mölbert. Other chapters are devoted to the 'distal', 'ocular', and 'menopausal' myopathies,