ABSTRACTS OF CARDIOLOGY

Edited in collaboration with Abstracts of World Medicine

A Clinical Appraisal of Pentapyrrolidinium (M & B 2050) in Hypertensive Patients. E. D. FREIS, E. A. PARTENOPE, L. S. LILIENFIELD, and J. C. ROSE. *Circulation (N.Y.)*, **9**, 540–546, April, 1954.

The authors report the results of a comparative study of the effect of a new ganglionic blocking agent, pentamethylene 1:5-bis-(1-methylpyrrolidinium) bitartrate (pentapyrrolidinium, "M and B 2050") and hexamethonium chloride on the blood pressure of hypertensive patients. In 27 patients with severe fixed hypertension treated during 2 to 6 months solely with pentapyrrolidinium given orally in three doses a day, the mean total daily effective dose was 300 mg. (range 135 to 630 mg.). In these patients the average blood pressure fell from 230/135 mm. Hg (range 180/110 mm. to 260/160 mm. Hg) before treatment to 170/110 mm. (range 130/95 to 210/130 mm. Hg) after treatment.

Cross tolerance between the two drugs was very small, and tolerance developed to pentapyrrolidinium less often than to hexamethonium. The side-effects of the drug were similar to those of hexamethonium, impotence being particularly frequent and troublesome. One important advantage of pentapyrrolidinium was that its onset of action was far more predictable than that of hexamethonium and, in contrast to the latter, oral administration lowered the blood pressure significantly without producing prolonged collapse or paralytic ileus. It is emphasized, however, that critical adjustment of the dose is necessary, which is a definite drawback, since a slight excess may produce hypotensive reactions such as postural faintness, and slight underdosage may fail to lower the blood pressure significantly. The authors therefore recommend that its use be confined to cases of severe hypertension which have not been benefited by simpler measures.

A. Schott

Arterial Hypertension Treated with *Rauwolfia serpentina* and *Veratrum viride*. C. JOINER and R. KAUNTZE. *Lancet*, **1**, 1097–1099, May 29, 1954.

A clinical trial was conducted in which hypertensive patients taking veratrum viride in the form of "veriloid" were also given rauwolfia serpentina. This treatment was suggested by reports that rauwolfia possessed additive properties in lowering the blood pressure when given with other hypotensive drugs. A total of 24 patients were selected for the trial, but 8 had to be withdrawn before the end; thus the results in 16 cases only are present. All the patients received veriloid, 1 mg. per 10 lb. (4·5 kg.) body weight, throughout the 16 weeks of the trial. Rauwolfia was given in addition to the veriloid—to 10 patients in a nightly dose of 0·5 g. for the second 8 weeks of the study, and to 6 patients in a nightly dose of 0·5 g. for the first 8 weeks and of 1 g. for the remaining 8 weeks.

The addition of rauwolfia serpentina resulted in a bradycardia in 6 patients; in 6 others there was a fall in diastolic pressure, which was, however, clinically significant only in 3 (a fall of 14 to 45 mm. Hg). Increasing the dose of rauwolfia from 0·5 to 1 g. had no effect in the 6 cases in which this was done. No serious toxic effects were observed, but in 2 patients pruritus with urticaria developed.

The authors conclude that in the dosages used there is no clear evidence of an additive effect of the two drugs.

Arthur Willcox

The Uncommon Heart Diseases. NATHANIEL E. REICH, M.D. Oxford: Blackwells Scientific Publications, 1954. Pp. 516, Figs. 110. 75*s*.

This book is intended as a supplement to the standard textbooks on heart disease. A large amount of the material included would seem to have no place in a book with this title, as for example the effects of pregnancy, obesity, anæsthesia, or aortic insufficiency on the cardiovascular system, whereas many of the more uncommon types of heart disease are more adequately covered in several of the larger standard works available. The value of this book for reference purposes is limited by the fact that the bibliography is confined almost entirely to the American literature. *G. W. Hayward*

A Primer of Congestive Heart Failure. GEORGE E. BURCH. Oxford: Blackwells Scientific Publications, 1954. Pp. 126. 30*s*.

This monograph, one of the American Lecture Series, summarizes present ideas on the mechanism of congestive heart failure and discusses the difficulties in accepting many of the clinical concepts. The chapter on treatment is written from an essentially practical point of view and the chapters on digitalis and the mercurial diuretics describe clearly the mode of action and method of administration of these drugs. This book which was primarily intended for the young graduate in medicine and the general practitioner can be recommended to all physicians interested in the problem of congestive heart failure. *G. W. Hayward*

Painless Myocardial Infarction: a Review of the Literature and Analysis of 220 Cases. M. D. ROSEMAN. *Ann. intern. Med.*, **41**, 1–8, July, 1954.

In 10 out of 220 cases of myocardial infarction proved by necropsy or serial electrocardiography at the Boston City

Hospital, the patient complained of no pain or discomfort. Only in 5 of the 10 was it possible to obtain a reliable history. Of these, 2 patients had a long history of angina but had no pain at the time of the infarction, and it is considered that in these cases slow and progressive narrowing of the coronary vessels may have resulted in relative anæsthesia of the infarcted area from destruction of the vessels and nerves, so that the final occlusion caused no pain. The other patients may have been hyposensitive individuals in whom the marked dyspnœa which accompanied the attack overshadowed a moderate amount of pain. From this series and a survey of the literature it is concluded that painless myocardial infarction is rare. *C. W. C. Bain*

Endomyocardial Fibrosis in Africa: Its Diagnosis, Distribution and Nature. A. W. WILLIAMS, J. D. BALL, and J. N. P. DAVIES. *Trans. roy. Soc. trop. Med. Hyg.*, **48**, 290–311, July, 1954.

From their experience at Mulago Hospital (Makerere College Medical School), Kampala, Uganda, the authors consider endomyocardial fibrosis to be one of the commonest causes of death from heart failure among Africans in this area. Analysis of a seris of 231 necropsies on patients dying of heart failure showed that the four main causes were endomyocardial fibrosis, 33 cases (14%); renal hypertension, 37 (16%); syphilitic aortitis, 34 (15%); and rheumatic heart disease, 20 (9%). They do not consider it likely that the disease is peculiar to this small area of Africa, but attribute the comparatively high incidence here reported to the rise in the number of necropsies performed and the increased awareness of the condition at Kampala. Only fragmentary information is available on the incidence of endomyocardial fibrosis elsewhere; most of this is from Africa, but a few cases have been reported from the United States and from Europe.

Since in most cases only the end-results of endomyocardial fibrosis are seen, the nature of the onset and development of the condition are unknown. Post mortem, the left ventricle shows endo- and myo-cardial fibrosis and the posterior cusp of the mitral valve may be adherent to the ventricle wall. The right ventricular lesion usually produces obliteration of the cavity in the region of the apex, and ocasionally the the posterior cusp of the tricuspid valve becomes adherent to the ventricle. Clinically, these pathological changes result in left, right, or bilateral heart failure, with or without mitral incompetence or tricuspid incompetence. X-ray screening shows a large, globular heart shadow and greatly decreased pulsation, which is presumably due to poor myocardial contractility. Low-voltage electrocardiograms are common, but arrhythmia is rare.

The pathogenesis is largely speculative; two possible sequences are discussed, namely, endocardial damage leading to thrombosis followed by fibrosis, or subendocardial muscle injury resulting in endocardial thrombosis and again fibrosis. The roles of infection, allergy, malnutrition, and toxic agents as possible ætiological factors are considered. Of infection, the authors state that " the possibility of virus infection (by itself or with some other contributing factor) initiating a fibrosis–thrombosis or thrombosis–fibrosis cycle involving the inner layers of the heart wall cannot be dismissed," In conclusion a plea is made for further information about the geographical and racial distribution of this condition, and the difficulties involved in studying the lesion in its early stages are emphasized. *J. Warwick Buckler*

Endomyocardial Fibroelastosis. A Study of Thirty Cases. W. R. HALLIDAY. *Dis. Chest.*, **26**, 27–40, July, 1954.

" Endomyocardial fibroelastosis " is regarded by the author as the most descriptive of the many names which have been applied to a condition of which the essential pathology consists in " a diffuse or focal thickening of the endocardium and subendocardial myocardium, consisting predominently of fibrous and elastic tissue." He was able to collect 30 cases (18 male, 12 female) from the post-mortem records of the Children's Hospital, Denver, Colorado, over a period of 5 years. The age at death varied between a few hours and 20 years and it is suggested that occasional patients may survive even longer.

The left side of the heart was more frequently and more severely involved than the right. In 17 cases the disease was associated with a great variety of congenital cardiovascular defects (which were the cause of death in 16) including coarctation of the aorta in 7 cases, patent foramen ovale in 8, patent ductus arteriosus in 9, and infundibular stenosis in 2. Anomalies of the coronary arteries were present in 5 cases, but these could not have produced myocardial ischæmia. In 11 cases (including 2 of these 17) various non-cardiovascular abnormalities were present, which were fatal in 4 cases. The remaining 10 patients died of endomyocardial fibroelastosis. In several cases hypoplasia of one or more chambers of the heart was present, but it could not be determined whether this was a result of the disease or of the associated abnormalities. *F. Starer*

Pressures in the Left Auricle and Ventricle in Mitral Stenosis before and after Commissurotomy. B. I. LATSCHA, F. DE'ALLAINES, and J. LENÈGRE. *Arch. Mal. Cœur.*, **47**, 385–409, May, 1954.

The pressures within the left auricle, left ventricle, and aorta were measured with an electromanometer by direct needle puncture after the pericardium had been opened at operation in 70 patients with mitral stenosis undergoing mitral commissurotomy.

In pure mitral stenosis the left auricular pressure was roughly halved as a result of operation on the valve, the pressure being usually in the region of 30 mm. Hg before operation. The left ventricular systolic pressure was found to be low initially in pure mitral stenosis, the average being 78 mm. Hg, and rose after satisfactory valvotomy to an average of 115 mm. Hg. Except when the mitral stenosis was accompanied by aortic regurgitation, the left ventricular diastolic pressure was normal initially and was unchanged after operation. When mitral incompetence was predominant, the left ventricular pressure was often raised.

The authors made the surprising observation, by simultaneous recording of the pressures within the left ventricle and aorta, that the systolic pressure in severe

mitral stenosis may be higher in the aorta than in the left ventricle, this difference being reduced by valvotomy.

G. S. Crockett

The Electrocardiographic Effects of Intravenous Administration of Neostigmine and Atropine during Cyclopropane Anesthesia. E. JACOBSON and M. H. ADELMAN. *Anesthesiology*, **15**, 407–415, July, 1954.

Several reports have appeared of the occurrence of sudden death following the intravenous administration of neostigmine and atropine in anæsthetized and curarized patients. The present authors, in an attempt to explain the cause of these sudden deaths, have closely followed the sequence of events in 20 patients who were premedicated with morphine and atropine and anæsthetized with cyclopropane, electrocardiographic records being taken continuously before and during anæsthesia. Atropine was given to each patient at the height of neostigmine activity. The changes following the intravenous injection of neostigmine were: little change in blood pressure, the development of sinus bradycardia, first degree auriculo–ventricular block, increased amplitude of the QRS complex, and increased prominence of the T wave. There was no evidence to support the theory of Bain and Broadbent (*Brit. med. J.*, 1949, **1**, 1137) that atropine potentiated the cholinergic effects of neostigmine by central vagal stimulation. On the contrary, in every case here studied, intravenous atropine produced opposite effects; these were heralded by reactivation of sinus activity and followed by increased auriculo–ventricular conduction.

The authors speculate on the cause of death due to neostigmine. This substance alone, in a normal patient under cyclopropane anæsthesia, can produce profound disruption of auriculo–ventricular conduction, marked depression of the sinus node, and sinus arrest. These changes may easily terminate in cardiac arrest, particularly in patients suffering from jaundice, perforated peptic ulcer, and sinus bradycardia. In 2 of the 5 reported cases the patients were jaundiced.

In the authors' cases the response to intravenous administration of atropine was striking, showing runs of ventricular tachycardia and in one case a short burst of ventricular flutter. Several possible theories of these results are discussed. The tracings do not support the contention that atropine, by causing central vagal stimulation, potentiates the cholinergic action of neostigmine on the heart. The authors suggest that deaths following injection of neostigmine are attributable to either (1) the cardio-inhibitory action of neostigmine, or (2) the adrenergic effect of neostigmine and atropine, resulting in ventricular fibrillation.

W. Stanley Sykes

A Physiologic and Clinical Study of Cardio-Vascular Diseases. ALDO A. LUISADA, M.D. Pp. xlv+680, with 312 illustrations. Second edition. Price 114*s*. London: Baillière, Tindall & Cox.

This book has been extensively rewritten to cover the advances during the six years since the previous edition and there are new chapters on cardiovascular syphilis, prognosis of cardiovascular disease, and the social and legal aspects of heart disease. There are three appendices on prescriptions mostly of proprietary drugs, diets, and a summary of treatment in cardiovascular emergencies. Throughout the book there is more emphasis on technical and graphic methods of investigation such as phonocardiography than on physical examination, the importance of which the author considers has decreased since the newer methods of investigation have become available. The arrangement of the book is based on an anatomical rather than an ætiological classification of diseases and this has led to a great deal of repetition particularly as far as treatment is concerned. It is disappointing in a book with this title to find no adequate account of the hæmodynamic changes in heart failure or of present views on the pathogenesis of failure, one short chapter of 14 pages only being devoted to the whole of heart failure, including the manifestations, complications, diagnosis, prognosis, and treatment. The book is freely illustrated although some of the sketches are not very happily drawn, particularly the patient with a typical attack of angina pectoris who is shown clutching his left mammary region and left axilla. The bibliography is unusually complete and covers very adequately both the European and American papers.

G. W. Hayward

Comparison of the Oxygen Tension in Blood from the Left Atrium and a Systemic Artery. V. O. BJÖRK, G. MALMSTRÖM, and L. G. UGGLA. *Amer. Heart J.*, **48**, 8–12, July, 1954.

The authors have compared the oxygen tension in systemic blood with that in blood from the left atrium. In 11 cases of mitral stenosis blood drawn from the left atrium (through a long paravertebral needle) was found to have an oxygen tension which was on the average 7 mm. Hg higher than that in a systemic artery. The difference, it is suggested, might result from admixture of Thebesian-vein blood in the left ventricle. In the same patients pulmonary capillary blood, drawn through a wedged pulmonary arterial cather, had an oxygen tension averaging 21 mm. Hg higher than that in the left atrium. Here the factors involved might be: (1) double passage of blood through the alveoli leading to better oxygenation, or (2) bronchial venous admixture in the pulmonary veins lowering the oxygen content of blood reaching the left atrium.

J. McMichael

The Second European Congress of Cardiology will be held in Stockholm, September 10–14, 1956. *The general secretary will be*

Dr. Karl Erik Grewin,
Sodersjukhuset
Stockholm S, Sweden

but further details are not yet available.