ABSTRACTS OF CARDIOLOGY

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The authors have carried out a study at the University of Minnesota Medical School, Minneapolis, of the mechanics of ventilation, with particular reference to the cause of dyspnea in patients with heart disease. Their method was based on the theory that the intrathoracic pressure at any time during breathing represents the sum of (1) the retractive forces of the lungs, (2) the pressure required to overcome tissue friction of the lung, and (3) the pressure gradient required to produce a flow of air through the bronchial tree. Intrathoracic pressure was measured by means of a balloon in the esophagus, the difference between this pressure and that in the mouth being recorded by a differential strain gauge. The breathing was interrupted suddenly at any point by either a hand-operated three-way respiratory valve or an electrically operated solenoid valve. A spirometer tracing of the changes in lung volume was made. To differentiate between the pressure required to overcome tissue friction of the lung and the pressure gradient required to maintain the flow of gas through the bronchi it was necessary to make the subjects breathe two gases with different physical properties but about equal kinematic viscosity; those chosen were air and a mixture of oxygen (20%) and argon (80%). The increase in lung volume was plotted against the static intrathoracic pressure in order to produce a pressure-volume curve for each subject.

In all, 10 normal men, 6 normal women, and 16 patients of both sexes with heart disease, mainly rheumatic, were examined. It was found that the pressure required to produce a change in lung volume of 100 ml. was significantly greater in patients with heart disease. This factor was found to show a high degree of correlation with the reciprocal of the vital capacity both of normal subjects and of patients, but no relation was found between the reduction in vital capacity and the increased resistance to air flow.

Comparison of the pressure-flow relationships in patients with heart disease when breathing the two gas mixtures showed that tissue friction was a negligible factor and that the degree of movement of the lungs was determined by the degree of resistance to gas flow. Since there may be no increase in resistance to air flow in patients with heart disease and exertional dyspnea the authors conclude that the latter is most closely related to a reduction in vital capacity and to the altered elastic properties of the lung; on the other hand it is thought likely that increased resistance to air flow may play an important role in the dyspnea of cardiac asthma.

H. E. Holling


This small handbook on cardiac resuscitation, based on the course given at Cleveland, Ohio, is designed to provide the anesthetist and surgeon with the knowledge that will enable them to treat promptly and efficiently a case of cardiac arrest occurring under anesthesia. After introductory chapters on the physiological aspect of cardiac arrest, predisposing factors, and preventive measures, the rest of the book is devoted to a detailed and practical description of a two-part plan for restoring the normal circulation, the first being the re-establishment of the "oxygen system" and cardiac massage, and the second the treatment of cardiac standstill in ventricular fibrillation. The author claims that if the plan is properly carried out any normal heart that has stopped can be made to beat again.

Graham Hayward


The scope of the book has been widened since the first edition appeared fourteen years ago, as in addition to an extensive revision of the original text there are descriptions of the vascular disorders of the skin, brain and spinal cord and viscera. The wisdom of including a chapter on the vascular diseases of the brain is doubtful as it is so short as to be of little value to either the physician interested in peripheral vascular disorders, as they are usually understood, or to the neurologist. The early part of the book consists of chapters on methods of examination and on the different vascular diseases. Nearly two-thirds of the book is devoted to treatment and the dust cover claims that every known method of treatment is included. There are full and practical descriptions of medical, surgical, and physical methods of treatment but the absence of any cross references in the text necessitates frequent use of the index if the treatment of any particular disorder is sought. The value of the book for reference purposes is limited by the inadequacy of the index which is incomplete and so arranged that it is difficult to find any particular piece of information.

Graham Hayward


This atlas is based on 131 angiograms obtained at necropsy by injection of arteries, or in some cases veins, with radio opaque materials. The reproductions are excellent and
in addition to a full description in the text there are summaries in English, French, and Spanish at the end of each chapter. A large number of the illustrations were obtained from patients with disease of the heart or lungs and the sections on acquired heart disease, due to hypertension, coronary disease, or mitral stenosis, or on the various types of congenital heart disease, will be of great interest to the cardiologist. Other chapters illustrate the changes in the blood vessels of the kidneys in various diseases and in the portal circulation in portal hypertension and cirrhosis of the liver. This book should be studied by all who are interested in the study of the circulation by means of the injection of radio-opaque substances.

Graham Hayward


In his preface the author states that "books on electrocardiography seem to possess one or more of several disadvantages for the beginner"; this book is no exception. We are told that introductory chapters on electrophysiology are often too intricate, but they may be so brief that electrocardiography becomes an empirical interpretation of patterns without rationale. We are told that in some books "only certain aspects of the subject are dealt with, for example, the arrhythmias may be entirely omitted"; in this book there is very little mention of the electrocardiographic findings in angina pectoris. Two pages are devoted to coronary insufficiency.

The author has endeavoured to present an easy technique of interpreting electrocardiograms. This is partially achieved but interpretation is surely confused by indiscriminate use of V leads and CF leads to illustrate the precordial electrocardiogram. Your reviewer agrees that it is useful to take advantages of the "emphasizing tendencies of the CR and CF connections, rather than to rely slavishly on the V leads because they are theoretically superior," but the auricular arrhythmias are not illustrated by CR1, which is especially useful in emphasizing P waves and CF leads appear too frequently in the illustrations.

This book is profusely illustrated and the author has achieved his object of presenting the pictures adjacent to the relevant text. However, many of the electrocardiograms are poorly reproduced and many are indifferent examples from the technical point of view. It is difficult to understand why chapters concerned with myocardial pathology, such as ventricular hypertrophy and myocardial infarction are separated by sections on the arrhythmias. This book is not burdened by an extensive bibliography but there is an index to illustrative electrocardiograms and a general index. Wallace Brigden


In continuation of a previous investigation (Ann. intern. Med., 1952, 38, 254; Abstracts of World Medicine, 1953, 14, 216) the authors have carried out, at the Beth-El Hospital, Brooklyn, New York, a post-mortem study of the relationship between the serum beta-lipoprotein level, the somatotype of the patient, and the degree of coronary atherosclerosis in 157 apparently healthy persons who died suddenly from accident, suicide, homicide, or coronary occlusion; somatotypes were determined according to Sheldon's methods. In most of the cases blood for analysis was obtained within six hours after death.

Of the 157 subjects, 73 died suddenly from coronary accident, and in 58 of these there was an abnormal beta-lipoprotein pattern. The over-all coefficient of correlation between the serum beta-lipoprotein pattern and the degree of coronary atherosclerosis was 0.84. When the cases were classified by somatotype the correlation for the mixed group of mesomorphs and endomorphs was 0.91, whereas for the group consisting of ectomorphic males it was only 0.6. The authors conclude from this study that "any male or female, with the exception of the ectomorphic male, who has an abnormal serum beta-lipoprotein pattern must be seriously regarded as a potential victim of atherosclerosis. This is particularly true of the mesomorphic male."

Z. A. Leitner


The efficacy of supposed coronary vasodilator drugs was studied by observing whether they modified the electrocardiographic response to a standard Master two-step exercise test. The ability of these drugs to relieve pain on exertion was primarily considered. The patients selected included 60 with coronary disease in whom, on repeated testing under identical conditions, there was a relative constant positive electrocardiographic response to exercise which could be favourably modified by a sublingual dose of glyceryl trinitrate taken just before the test (glyceryl trinitrate being accepted as a potent coronary vasodilator). Of all the drugs tried, only two in addition to glyceryl trinitrate gave responses which justified their use in the management of angina pectoris. Thus papaverine, 1 to 2 gr. (0.06 to 0.13 g.) intravenously or 3 to 8 gr. (0.2 to 0.52 g.) by mouth, was effective in 14 out of 24 patients, but was ineffective in the usual therapeutic dosage. The only drug that was of any value for more prolonged prophylaxis was pentaerythritol tetrinitrate ("peritrate"), 10 to 20 mg. having a marked effect for 4 to 5 hours by this test.

J. N. Agate


"Heart Disease in Infancy" is based on the study of some hundred cases seen at Zurich. The first chapter is of practical use for therein we find many valuable facts about the normal findings of the heart at this age.
Enlargement of the heart as a result of various congenital defects comes next. In fact it is from the point of view of enlargement that all these developmental errors are considered. Whether this is really a good approach is perhaps doubtful, although naturally in some ways this secondary effect does gauge the severity of the defect. The third chapter is more interesting as it deals with the myocardial defects. Here “enlargement” is considered as it occurs apart from the malformation. Two more chapters deal with causes of enlargement as a consequence of primary extracardiac diseases, and with treatment. This is a good book with plenty of information. The matter is well arranged and illustrated by diagrams, many in colours. There are plenty of references, and numerous photomicrographs and pictures of specimens. One hundred and sixty-one case histories are included. An amazing amount of knowledge has accumulated about heart disease in infancy; my 1924 edition of Von Reuss has only ten pages.

Terence East

Probleme und Beispiele biologischer Regelung. R. Wagner. 1954. Georg Thieme Verlag, Stuttgart. 219 pages, 38 illustrations. 29.40 DM.

This is a physiological study of the problems of biological control. The author first discusses the mechanism of the control of voluntary movement. The second main part of the book considers the mechanism of the control of the circulation. First comes the regulation of the level of the blood pressure, and the control of the heart. The author thinks that the methods of control of voluntary movement and those of the circulation have much in common. Such an integration is always welcome as a move towards simplicity; but here the expression hardly merits that quality. There are whole pages unbroken by any paragraphs, which make reading heavy. Anyone who is prepared to tackle this philosophical physiology will no doubt find something interesting.

Terence East


Working at the University of Texas, Galveston, Texas, the author has studied the diuretic effect of acetazolamide (“diamox”) when given orally to patients with and without heart failure in doses ranging from 500 mg. to 6 g. in 24 hours. With large doses the urinary volume was more than doubled in 24 hours, accompanied by a marked rise in sodium and potassium excretion. The diuresis lessened on the following day, but electrolyte excretion continued in large amounts. In some cases diuresis and weight loss continued for periods up to 3 weeks without further administration of diuretics. Toxic effects were mild paraesthesia, drowsiness, nausea, vomiting (in 3 out of 15 cases), and the occurrence of a reversible psychosis in one patient with nephrosclerosis and uremia. In 12 patients with heart failure receiving either a single dose or repeated dosage (500 mg. of diamox at 8-hourly intervals for 4 doses) diuresis was only slightly more effective in the second and third 8-hour periods, but with repeated dosage sodium excretion was more effective during the 24 hours and the following day than with the single dose.

Acetazolamide diuresis is attributed to the inhibition of carbonic anhydrase in the renal tubules, producing an alkaline urine and excessive distal tubular excretion of potassium, with a decreased tubular reabsorption of sodium, bicarbonate, and consequently of water.

I. Ansell


The exact mechanism of auricular fibrillation in man has never been satisfactorily demonstrated. As a contribution to this subject the authors took the opportunity during the surgical treatment of patients with mitral stenosis and auricular fibrillation at the Cedars of Lebanon Hospital, Los Angeles, to obtain direct electrocardiograms from the fibrillating auricles and also to make high-speed cinematographic records. By neither method was any evidence adduced for the “circus movement” postulated by Lewis. The electrocardiograms showed large waves occurring irregularly at a rate of 250 to 400 per minute, with in addition highly irregular small waves along the base line. It has been shown previously that auricular flutter is due to the presence in the auricle of a rapidly discharging ectopic focus, and that auricular tachycardia, auricular extrasystoles, and auricular flutter are essentially of the same nature, in that the contraction pursues an orderly course from the site of origin to the extremities of the auricles. It is argued that if the rate of contraction of the ectopic focus exceeds a critical threshold—which varies in different patients—there is a complete breakdown of orderly mechanical and electrical activity, and auricular fibrillation results. This has been experimentally demonstrated on the heart of the dog.

The application of these findings to treatment is considered, and in a discussion of the action of drugs it is suggested that if the action of quinidine is gradually to slow the rate of discharge from an ectopic focus all the effects of this drug in varying doses can readily be explained. It is considered probable that procaine amide acts similarly.

C. Bruce Perry


The clinical features and necropsy findings in 15 cases of myocarditis, seen for the most part at Malmö General Hospital, Sweden, between 1944 and 1952, were analysed. The cases were divided into two groups according to the necropsy findings: (1) rheumatic myocarditis, in which Aschoff granulomata were found (7 cases); and (2) non-rheumatic myocarditis, in which non-specific inflammatory changes were noted (8 cases). Cardiac enlargement was present in all except two.

The most frequent electrocardiographic changes
were abnormalities in the S-T segment and T waves. In several of the cases of non-rheumatic myocarditis the condition was not diagnosed during life. The authors point out that in these cases sudden death may follow an apparently mild infection of the respiratory tract, such infection being apparently the commonest associated cause of death.

It is believed that the incidence of acute myocarditis is decreasing, perhaps as the result of the use of antibiotics in respiratory infection; if, however, such an infection does not respond to antibiotics the possibility of myocarditis as a complication should be considered, and serial radiographs and electrocardiograms should be examined. Rheumatic myocarditis is generally more readily diagnosed than the non-rheumatic form because the patient’s history is suggestive. The authors advise retaining the term myocarditis for a wide variety of infections and hyperergic, allergic, toxic, and metabolic conditions in order to emphasize the “structural lesions of myocardial cells” (apart from ischaemic heart disease) “resulting in impairment of cardiac function.”

R. S. Stevens


The operation of atrio-septo-pexy as originally devised consisted in closing an atrial septal defect by suturing an invaginated portion of the right atrial wall to the margins of the defect. Increasing experience has shown, however, that if the defect is large there may not be a complete rim of septal tissue, and complete closure by this method may not be possible. Recently the authors have come to the conclusion that closure is not necessary so long as the systemic and pulmonary venous systems can be separated. In some cases this problem has been solved by suturing the atrial wall to the free edge of the posterior septal remnant, thus converting the right atrial cavity into a U-shaped chamber lying behind the defect. (This modification has been named the “Pompeian version of atrio-septo-pexy,” by analogy to the ancient Pompeian method of manufacturing water-pipes.) The modified operation is very satisfactory in patients with a persistent remnant of lower septal tissue, but is not so suitable when the septum primum is totally absent, whether or not a septum secundum is present. Attempts to apply the technique in such cases may lead to damage to the bundle of His as it runs along the free upper margin of the muscular part of the interventricular septum. In these cases it is safe to complete the intracardial channel except for a short interval between the upper lip of the ostium of the coronary sinus and the most anterior (membranous) portion of the interventricular septum; the residual gap may then be closed with a small bag of pericardial fat placed on the left atrial side of the defect after carrying out Kiriluk’s technique.

Anomalous pulmonary venous return may be dealt with at the same operation. If only one vein empties abnormally it may be re-implanted into the side of the normally emptying vein. However, if both right pulmonary veins drain into the right atrium they may be excluded by suturing the lateral atrial wall to the anterior margin of the septal defect, thus converting the right atrium into two compartments, of which the small posterior one drains the right pulmonary veins directly into the left atrium.

The authors believe that the closed technique is better than an open method at the present time, and that hypothermia gives no added advantage, especially in adults. A total of 46 patients have so far been operated on. Among 30 patients with septum secundum atrial defects there were 3 operative deaths, but all the survivors showed marked clinical improvement, with complete abolition of the shunt in the majority. Among the 16 patients with ostium primum septal defects there were 11 operative deaths.

In conclusion the authors state their belief that correction of an interatrial communication is not indicated in patients with a right-to-left shunt.

F. J. Sambrook Gowar


Marfan’s syndrome is characterized by a wide variety of abnormalities which may be present in different combinations. The commonest abnormality is congenital dislocation of the lens, others being unusual length of limb with poor muscular tone, arachnodactyly, talipes, pes planus or syndactyly, hernia, high-arched palate, and kyphosis causing either pigeon-breast or pectus excavatum.

In this paper from the Johns Hopkins University and Hospital the author discusses the cardiovascular abnormalities. These generally affect the ascending aorta, causing fusiform aneurysm, dissecting aneurysm, and dilatation of the aortic ring with aortic incompetence. Any of these may directly or indirectly cause death, possibly suddenly, in children or young adults. Less commonly there may be minor degrees of coarctation of the aorta, patent ductus, atrial septal defect, dilatation of the pulmonary artery, and mitral-valve deformities, which last may be complicated by bacterial endocarditis. It seems likely that the diagnosis of Marfan’s syndrome may be overlooked in cardiac patients in whom the significance of abnormalities elsewhere in the body is not appreciated.

The author suggests that the underlying abnormality in all lesions may be an abiotrophy of connective tissue, causing disruption of elastic fibres which may not become evident until late childhood or adult life. In a study of the familial aspects of the condition he traced 50 families in which there was one certain instance of Marfan’s syndrome; in 70 per cent of the families there were other certain or possible cases. The total number of definitely affected subjects was approximately 105, and in 46 of these the cardiovascular system was involved. 

J. A. Cosh

During the operation of mitral valvotomy on 7 patients at Mount Sinai Hospital, New York, the pressure gradient across the stenosed mitral valve was determined by simultaneous measurement of the left atrial and left ventricular pressures, No. 20 needles being inserted into the left atrium, the left ventricle, and the aorta and being connected to three matched strain-gauge manometers by sterilized vinyl tubing.

The difference in filling pressure across the healthy mitral valve is normally too small to be measurable, but when the valve is stenosed the pressure gradient may rise to 20 mm. Hg. A more accurate assessment of the changes in filling pressure under different conditions would be aided by the simultaneous measurement of cardiac output, but so far the authors have not found it possible to do this under the conditions obtaining in the operating theatre. They point out that the performance of valvotomy is accompanied by a reduction in the pressure gradient which is correlated with the adequacy of the surgical procedure. In all cases the reduction in the pressure gradient is mainly due to a reduction in left atrial pressure, but in a few cases an elevation of left ventricular diastolic pressure also occurs. The authors suggest that measurement of the pressure gradient during the course of a valvotomy provides an estimate of the likely extent of the residual stenosis and is thus of help in deciding whether the attempt to increase the valve opening should be continued.

H. E. Holling


In reporting their experiences of the treatment of constrictive pericarditis the authors stress the need for more radical surgery in this condition, regarding delay in improvement and need for a second operation as due to inadequate pericardectomy. They consider that when calcification is present, even if symptoms are mild, decortication should be performed, as cardiac disability is progressive. In tuberculous pericarditis decompression of the heart by aspiration or operation is indicated as soon as symptoms of compression appear. The operation they use is as follows. The pericardium is excised to beyond the left, right, and the inferior borders of the heart, and often over the vena cava at the base. A mid-line incision is made, with vertical division of the sternum, which is repaired with at least three steel stitches over which the pectoral fascia is reaproximated. The decortication is begun over the apex and the left side liberated first, excising through a plane just outside the muscle, sometimes in two layers. If the heart is irritable and becomes irregular, there should be a few moments’ delay. Hemorrhage from the heart can be controlled by stitching down a small flap of the mobilized pericardium. The mediastinum and the right pleural cavity are drained. Full chemotherapy is given before and after operation.

The authors have performed this operation on 26 patients with constrictive pericarditis, the oldest aged 55. There were no operative deaths, but 4 patients died subsequently, 2 from myocardial failure and 2 from unrelated causes. The other 22 patients—8 with tuberculous and 14 with non-tuberculous pericarditis—are all well although 3 in each group have some limitation of activity.

_M. Meredith Brown_


The authors describe the results of treatment in 38 cases of staphylococcal endocarditis at Johns Hopkins Hospital, Baltimore. These fell into three groups: (I) 22 cases treated before antibiotics became available; (II) 3 cases treated between 1944 and 1948, when effective doses of penicillin were used and most strains of staphylococci were sensitive to the drug; and (III) 13 cases treated in the period 1949–53. Of the total number, the pre-existing heart disease was congenital in 10, rheumatic in 9, and hypertensive-arteriosclerotic in 6; in 13 there was no pre-existing cardiac condition. All but one of the 22 patients in Group I died, treatment in the successful case being with sulphathiazole, antiserum, and blood transfusion; in Group II there were two deaths and one recovery; while in Group III 6 patients survived and 7 died.

The authors discuss problems in diagnosis and treatment, mostly on the basis of the 13 cases in Group III.

The bactericidal and bacteriostatic effect of the patient’s serum in different dilutions against the infecting staphylococcus is considered to be a useful guide to therapy. For testing the sensitivity of the staphylococcus to various antibiotics as well as to penicillin the tube dilution test was found to be more satisfactory than the disk method.

The authors conclude that in cases of bacterial endocarditis, even when there are indications that the organism is resistant to the drug, penicillin should always be given in doses ranging from 8 to 24 mega units a day. Erythromycin in large doses—up to 3-6 g. a day for five weeks—may also be given, with one of the broad-spectrum antibiotics in addition. As regards length of treatment, the authors state that they "would hesitate...to advocate stopping (treatment) any sooner" than before the 6th or 7th week.

G. S. Crockett


In this short monograph the author discusses those physiological factors that are thought to influence the formation of oedema fluid in the lung. The pathological physiology of human acute pulmonary oedema is described very briefly and a rather inadequate account of present views is given on the pathogenesis of pulmonary oedema due to cardiovascular disease. The chapters on clinical manifestations and treatment add little to the information already available in the standard textbooks.

Graham Hayward