A Historic Patient: Angina Pectoris (?)
Shadow and Substance of 'Priority'
Two Centuries Ago (circa)

"Captain Charles, aged 50, had retired from the army in full vigour and health some years previously, but, leading a more sedentary life, he had put on weight. . . For some years he had complained of difficulty in breathing which gradually increased until he could not walk 100 yards at all quickly without provoking a sense of suffocation which was relieved by halting for a few moments. . . On 23 February 1768, after lunching with the élite of his friends he was late in setting out for another gathering held at a house about 700 yards away. Hurrying there, he was seized by an oppression and leant against the doorway, rejecting the help of a servant who came to his aid, after which he hurried up two flights of stairs and took his seat at the meeting much oppressed. He appeared to his friends to be dying, was carried out, and found to be dead.

. . . "The body was opened by a surgeon the following evening in the presence of Rougnon, Athalin, the Rector of the University, and other physicians and surgeons whose curiosity had been aroused. . . The heart was larger than normal by a third, due to dilatation of the thin-walled right ventricle. All the valves and the left ventricle were normal. . . There is no mention of the coronary arteries. . .

"The necropsy was far from tranquil and the audience engaged in a lively discussion. Some saw only fat on the heart, others noted the dilated vena cava, and some saw nothing unusual, saying 'M Charles est mort parce qu'il est mort'. In a lengthy dissertation, Rougnon concluded that ossification of the costal cartilages had interfered with inspiration and so prevented the free passage of blood through the lungs, causing stasis of the right heart which struggled against the obstacle in the lungs. . .

"[in a present century review] Professor Hans Kohn . . . concluded that Captain Charles did not suffer from angina but from emphysema. . . . He quoted Claude Bernard that priority in science did not consist of facts but of conclusions drawn from them. . .

"To sum up our conference, of the ten authorities I [Bedford] have cited, five are in favour and five are opposed to the claim that Rougnon was the first to describe angina pectoris. . .

"It is quite clear that Captain Charles had chronic lung disease with heart failure and he may well have had pulmonary hypertension. Readers will form their own opinion of the symptoms and whether they accept paradysspnoec oppression in the chest as anginal as many French authorities have done. All will probably degree that Rougnon did not describe Heberden's angina of effort, indeed his account added little to the old idea of asthma dolorificum."

Minuscule Review


Despite the common nature of congenital heart disease (1 in every 125 births), there have been few meticulous studies of the incidence of heart defects among the relatives of affected individuals, particularly for offspring. Data on offspring would tend to be less available because of the decreased life expectancy of patients, but there should be a sizable adult population with atrial septal defect, coarctation of the aorta, pulmonary stenosis, aortic stenosis, and patent ductus arteriosus. The present report, though dealing with a relatively limited sample, does provide the type of information that has long been needed by the genetic counselor and the practicing cardiologist. There were five affected children among the 190 children of 73 parents with atrial septal defect and six affected children among the 162 children of 57 parents with ventricular septal defect (in no case was more than one parent affected, but the authors do refer to a marriage of two of their patients with ventricular septal defect). The respective frequencies in offspring were 2.6 and 3.7%, the former being 37 times, and the latter, 21 times, the frequency of the individual defect in the general population. All affected children had the same defect as the parent, though several children in each group had additional cardiac defects. The authors draw attention to the close approximation of these risk figures to the expected values for multifactorial inheritance (square root of the incidence in the general population) and wisely point out that these risk figures pertain only to the first occurrence of congenital heart defect, the risk becoming significantly higher once an affected child has been born. Interestingly, patients with congenital heart defects appear to marry less frequently or later, and this observation suggests to the authors that this may result in lowered fertility. (It would likewise be of interest to determine differences in marrying age and relative fertility between those patients with atrial septal defect having surgery in childhood and those not having operation. To satisfy this interest would necessitate a very large sample and special treatment of data.)

Although not mentioned by the authors, it seems better in counseling patients with ventricular and atrial septal defects to compare these empiric risk figures for offspring to the overall incidence of congenital heart defects in newborns, rather than to the risk figures for the specific lesion. In other words, parents are probably more interested in knowing how their risk for having a child with congenital heart disease compares to that of a nonaffected parent, not just for a specific defect, but for any type of congenital heart defect. The former comparison involves a 21 to 37-fold increase in risk, while the latter is in the range of a threefold to fivefold increase. It is hoped that the authors and others will provide comparable data for other cardiac malformations, as well as more extensive material on the two defects dealt with in this study.

R.C.A.
Minuscule Review

Davis E: Criteria of rheumatic fever.

Pressures to revise the Jones criteria continue to be exerted. The 1965 revision, by a committee of the American Heart Association, emphasized evidence of streptococcal infection, thereby putting to practical use accrued documentation from research laboratories of the requirement of a streptococcal insult for rheumatic fever to develop initially or to recur.

In this recent article Davis proposes that polyarthritis, subcutaneous nodules, and erythema marginatum be demoted as major manifestations of rheumatic fever on the basis of nonspecificity or infrequency of occurrence, or both. In actual fact, there is no specificity of any of the manifestations, including the two, carditis and chorea that Davis considers to be the most important diagnostic features. Erythema marginatum and subcutaneous nodules, though admittedly rare, are diagnostically helpful when present. His suggestion that the presence or absence of carditis be specified has merit since this has some bearing on the prognosis. However, since patients who have polyarthritis as the major manifestation of rheumatic fever may develop carditis with subsequent attacks, it does not seem reasonable to designate migratory polyarthritis as a separate disease entity.

The limitations of the Jones criteria are spelled out in the 1965 revision. There is little to be gained by reshuffling the major and minor manifestations, by adding family history as a criterion, or by other maneuvers until we have a better understanding of the disease. The failure of investigators to provide clinicians with a specific diagnostic test leaves us in the fumbling position of using imperfect guide lines such as the Jones criteria, with discretion.

LEWIS W. WANNAMAKER, M.D.


17. Lasser RP, Haft JI, Friedberg CK: Relationship of right bundle-branch block and marked left axis deviation (with left parietal or peri-infarction block) to complete heart block and syncope. Circulation 37: 429, 1968


Role of Research in Medical Education

. . . . research serves additional vital functions in the education of physicians. In a field in which content changes so fast that what one learns in school is at best a foundation for the medicine of a decade later, it is important to have men capable of distinguishing assertion from evidence and dogma from adequately documented fact. Nothing fosters the development of the necessary critical ability to distinguish, as does an adequate experience in trying to develop evidence oneself, that is, in research. Such experience for every student of medicine is probably impossible, desirable as it might be. But it is absolutely essential as a part of the background of the teacher of medicine if he is to separate the relevant and probable from the mass of inevitably conflicting information and to transmit to his students the open-minded skepticism that will determine the adequacy of their future growth.

Minuscule Review


In the course of taking ultrasound echograms of the maternal abdomen for routine obstetrical reasons, the authors have found it possible to identify cardiac features of the fetus. Thus, the ratio of the cross-sectional area of the fetal heart to that of the fetal thorax was found to be 0.21 (SD, 0.06) on the basis of over 400 examinations, with no significant change in ratio during the last 8 weeks of pregnancy. The ratio of greatest transverse diameter of the fetal heart to the corresponding chest diameter was found to be 0.52 (SD, 0.05) on the basis of 96 examinations, again with no evident change during the last 8 weeks of pregnancy. In 20 of these 96 cases the authors were able to identify the interventricular septum clearly enough to measure the greatest diameters of the ventricles, the size ratio being 1.23 in favor of the left ventricle. Although the number of cases (423), would make it statistically likely that several cases of congenital heart defect would be included, no follow-up information is given regarding the outcome of the pregnancies.

R.C.A.
Centennial of Bert's Comparative Physiology of Respiration
Resistance à l'Asphyxie (Problème de Harvey)

La conclusion générale par laquelle nous terminons notre dernière Leçon, nous allons avoir à la reproduire, avec plus de force encore, après avoir étudié une question dont se sont occupés des hommes éminents, et qui posée par Harvey, a mérité, par son importance, de recevoir le nom de "problème de Harvey"; je veux parler de la longue résistance à l'asphyxie que présentent certains mammifères nouveau-nés.

Le véritable problème de Harvey était celui-ci: Pourquoi un foetus laissé dans le liquide de l'amnios, ou plongé dans l'eau avant d'avoir respiré, peut-il continuer à vivre pendant un temps très-long, faculté qu'il perd après avoir fait une seule inspiration dans l'air? . . .

Cette résistance à l'asphyxie est vraiment des plus remarquables, et l'on ne saurait trop fréquemment en citer des exemples. Des petits Chiens naissants ont pu, dans les expériences de Haller et de Buffon, rester pendant une demi-heure immergés dans l'eau tiède, et en être retirés vivants. C'est à peu près la même durée pour les Chats et les Rats naissants, sur lesquels j'ai fait de nombreuses expériences. . . .

Pour nous, c'est dans la résistance vitale inégale des divers tissus ou éléments anatomiques que nous trouvons l'explication de la différence qui existe entre les animaux adultes et les nouveau-nés. Un nerf, un muscle, séparés du corps, se conduisent différemment, quant à la persistance de leurs manifestations physiologiques suivant qu'ils appartiennent à l'un ou à l'autre de ces êtres. Ce fait explique suffisamment que cette persistance soit inégale encore lorsque l'organisme vivant tout entier est soumis à des causes de mort. . . .


We will consider a question posed by Harvey and called "The Harvey Problem": the prolonged resistance to asphyxia manifested by certain newborn mammals.

The real problem of Harvey was this: Why does a fetus, left in amniotic fluid or submerged in water before breathing, continue to live for a very long time, a faculty which it loses after having taken a single breath in air?

This resistance to asphyxia is a most remarkable phenomenon. Newborn puppies, in experiments by Haller and by Buffon, were submerged half-an-hour in lukewarm water and were alive when removed. Cats and rats, on which I have made numerous experiments, stay alive about as long.

It is in the unequal vitality of tissues and organ systems that we may find explanations of the physiologic differences between mature and newborn animals.

A nerve, a muscle, separated from the body, behaves differently with regard to persistence of function, according to its source from an adult or newborn. This is sufficient explanation for the unequal survival of the entire animal subjected to various methods of dying.

Translation by Henry Blackburn
Minuscule Review


The authors report an altered affinity of hemoglobin for oxygen in two subjects with hyperthyroidism, one with experimentally induced hyperthyroidism, one with Grave's disease. The oxygen dissociation curve of the hemoglobin was shifted to the right and the oxygen tension at which the hemoglobin was 50% saturated (P50) was higher than normal. When the volunteer was made hyperthyroid, the P50 was increased 4.5 mm Hg and the red blood cell 2,3 diphosphoglycerate (DPG) was increased 32%. On the basis of these observations and others, including similar elevations of P50 and of 2,3 DPG in infants and young children with heart failure, the authors conclude that 2,3 DPG is a primary oxygen releasing factor in human red blood cells.

The authors infer from their studies that the A-V oxygen difference would be larger than normal in hyperthyroidism, with the corollary that a large proportion of the increased oxygen consumption could be accounted for by the enhanced release of O2 from hemoglobin. (The theory is good but many hyperthyroid patients have a relatively narrow A-V oxygen difference which has been conjectured to have been related to a high relative skin flow as part of heat regulation adaptation.)

In summary, Dr. Miller and associates contribute significant data to the flood tide of information on the role of 2,3 diphosphoglycerate in oxygen hemoglobin binding, and labelling this substance “releasing factor” makes the directional change easier for me to remember. One's curiosity in regard to the dynamics (what turns the mechanism off-and-on and what are the rate constants?) is further whetted. (An editorial by Mulhausen outlining some basic aspects of factors modifying the hemoglobin affinity for oxygen is published in the August issue of CIRCULATION.)

H. B. B.
50 Years Ago

Nobel Prize to Dr. Krogh—Physiology of the Capillaries

"My own first contribution to the problem . . . was published in Danish in 1918 . . . and appeared in the British Journal of Physiology (1919). . . . I found it possible to observe at least the superficial capillaries of muscles both in the frog and in mammals through a binocular microscope. . . . Resting muscles observed in this way are usually quite pale, and the microscope reveals only a few capillaries at fairly regular intervals. These capillaries are so narrow that red corpuscles can pass through only at a slow rate and with a change of form from the ordinary flat discs to elongated sausages. When the muscle . . . is stimulated to contractions a large number of capillaries became visible and dilated. . . . Since capillaries, even in a group fed by the same arteriole, do not all behave in the same way, the changes obviously cannot be due to arterial pressure changes. . . ."


Dr. Krogh’s Stillman Memorial Lectures were published in book form two years later with the following titles

I. Introductory. The Distribution and Number of Capillaries in Selected Organs
II. The Independent Contractility of Capillaries
III. The Structure of the Capillary Wall
IV. The Innervation of Capillaries
V. The Reactions of Capillaries to Stimuli
VI. The Reactions of Capillaries to Stimuli (Continued)
VII. The Hormonal Control of the Capillary Circulation
VIII. The Mechanism of Some Capillary Reactions, Especially in the Skin of Man
IX. The Exchange of Substances through the Capillary Wall
X. The Exchange of Substances through the Capillary Wall (Continued)
XI. Some Applications of the Physiology of Capillaries to Complex Processes in Health and Disease

Minuscule Review


In the past years, several reports have indicated the possibility of a link between spontaneous closure of a ventricular septal defect (VSD) and the formation of an aneurysm of the membranous septum. This idea has now been further substantiated by a report by Dr. Misra and associates. The authors documented their observations in three patients, aged 5, 14, and 22 years old, respectively, each with a small VSD and an aneurysm of the membranous septum. In initial studies of each, a VSD without septal aneurysm was demonstrated. In follow-up studies, varying from 3 to 7 years, an aneurysm at the base of the ventricular septum with reduction in the caliber of the VSD was demonstrated.

The observations by Dr. Misra and associates are interesting since they document, for the first time, the combination of (1) spontaneous diminution in size of a VSD and (2) simultaneous appearance of an aneurysm of the membranous septum. The question may be raised, however, whether the aneurysms demonstrated are truly of the membranous septum. An alternative explanation is that the defect could become diminished in size by adhesion of the septal leaflet of the tricuspid valve to the edges of the defect, while the demonstrated “aneurysmal” outpouching is of the very tricuspid tissue which reduces the size of the defect. This alternate explanation is appealing, since it relates one recognized mode of closure of a VSD to the appearance of an aneurysmal pouch (as illustrated in figure 2; Simmons RL et al: Circulation 34: 38-45, 1966).

ANTON E. BECKER, M.D.
MIES J. BECKER, M.D.
Minuscule Review
(An Etiologically Perplexing, but Distinctive Cardiovascular Syndrome)


In a clinicopathologic conference of the Massachusetts General Hospital, there is presented the case of a 47-year-old man with cardiac disease and systemic and pulmonary embolism. The pathologic examination revealed a large heart without coronary or valvular disease. The endocardium of the dilated left ventricle showed fibroelastosis and the myocardium of both ventricles showed scarring, extensive in the left ventricle and minimal in the right.

The diagnosis offered as to the primary disease is endomyocardial fibroelastosis. The implication is that the endocardial change represents an entity. An opposite view may be taken, namely that the myocardial disease is primary and that the endocardial thickening is a response to tension upon the connective tissue of the endocardium incident to dilatation of the failing left ventricle.

There are other cases with similar clinical patterns and myocardial change but without endocardial fibroelastosis. Do such cases represent an entity different from that described in the case in point or is likely that for the same stimulus leading to proliferation of connective tissue (such as comes with stretching of the endocardium) there is an individual variation? Certainly, individuals differ with regard to production of connective tissue in the healing of wounds. Some are keloid formers, others not. Perhaps an analogous difference occurs among individuals with regard to the development of endocardial fibroelastosis when a chamber is dilated. If one were to accept this line of reasoning, cases of the type described should be considered examples of primary myocardial disease, with the endocardial fibroelastosis representing a response to the fundamental myocardial condition rather than a specific entity.

JESSE E. EDWARDS, M.D.
**Minuscule Review**


_Amer J Cardiol_ 23: 830, 1968.

Increasing interest is being directed toward a unique cause of pulmonary hypertension in children since the first description in 1965 of two patients with severe obstruction of the upper respiratory tract by enlarged tonsils and adenoids. Arterial oxygen unsaturation, alveolar hypoventilation with carbon dioxide retention, increased pulmonary arteriolar resistance, and right heart failure are the clinical sequelae. Reversal of the process follows removal of tonsils and adenoids. The authors report on four additional patients exhibiting this syndrome. All were Negroes aged 2½ to 8 years. Carbon dioxide elevation (mean Pco₂, 50 mm), hypoxemia (mean Po₂, 65 mm), and pulmonary hypertension (mean PA pressure, 26 mm) were demonstrated in three patients in whom such studies were done. These abnormalities were not severe and the postoperative values were only slightly different (Pco₂, 50 mm; Po₂, 77 mm; and PA mean pressure, 20 mm). Improvement followed removal of tonsils and adenoids. One child was a mongoloid, and three had mental retardation.

The authors suggest that a central nervous system deficit resulting in inadequate respiratory response to hypoxia is an important causative factor in this syndrome. In addition, a pulmonary vascular bed that is hyperreactive to hypoxia may be important in the genesis of the pulmonary hypertension. The authors report that all of the patients had pulmonary edema. This diagnosis was based only on roentgenograms since pulmonary artery wedge pressures were normal in the two patients in which this measurement was made. There was no evidence of left ventricular dysfunction by clinical or electrocardiographic studies. Pulmonary edema in respiratory insufficiency has been described by Rao and associates (Amer J Med 45: 229, 1968), but this was observed in the presence of left ventricular failure which was absent in the patients described in this report. Clearly more data are needed before one can be certain that either pulmonary edema or left heart failure is present in this syndrome.

An important question regarding etiology is the role of upper airway obstruction alone versus recurrent bouts of pneumonitis in the genesis of the pulmonary vascular changes. The latter process can cause destruction of the pulmonary vascular bed and may result in ventilation-perfusion deficits by airway obstruction. The failure of complete recovery following relief of upper airway obstruction in many of these patients suggests that recurrent pulmonary infection may be an important additional etiologic mechanism.

_Herbert N. Hultgren, M.D._
American Board of Internal Medicine

The following candidates were certified in the Subspecialty of

CARDIOVASCULAR DISEASE

Atlanta, Georgia

July 10-11, 1970

CALIFORNIA
San Diego
Sobel, Burton Elias

Travis AFB
Flamm, Melvin Daniel, Jr.

CANAL ZONE
Balboa Heights
DeCastro, Carlos Manuel

COLORADO
Aurora
Marshall, Robert Martin

Littleton
Lesage, Charles H., Jr.

FLORIDA
Miami
Myerburg, Robert Jerome

Miami Shores
Wald, Stewart

GEORGIA
Atlanta
Nutter, Conal Owen

Augusta
Postell, William Newton, Jr.

ILLINOIS
Chicago
Fallico, Raul Esteban

Riverside
Pouget, Jean Maurice

KANSAS
Manhattan
Varriale, Philip

Prairie Village
Bell, Hubert Harvey

MARYLAND
Baltimore
Ross, Richard Starr

Bethesda
Glancy, David Lucas

MASSACHUSETTS
Salem
Kaulbach, Maximiliaan

Gustav

NEW JERSEY
Englewood
Grayzel, Joseph

Jersey City
Haider, Bunyad

NEW MEXICO
Albuquerque
Goss, Jerry Eldon

NEW YORK
Great Neck
Gabor, George Eugene

Rochester
Murphy, Gerald William

OHIO
Cincinnati
Conway, Gene Farris

Columbus
Weissler, Arnold

OREGON
Portland
Sutherland, Donald Wood

 PENNSYLVANIA
Wynnewood
Krause, Robert Leonard

SOUTH CAROLINA
Spartanburg
Sbar, Sheldon Stuart

TEXAS
Houston
Raine, Michael Bennett

VIRGINIA
Roanoke
Hollingsworth, John Hayden

WASHINGTON
Seattle
Murray, John A.

WISCONSIN
Madison
Corliss, Robert James

Milwaukee
Botticelli, James Thomas
News from the
American Heart Association

44 East 23rd Street, New York, New York 10010
Telephone: 477-9170

AHA Scientific Sessions
Program Features Listed

The 43rd annual Scientific Sessions of the American Heart Association, scheduled from Thursday morning, Nov. 12 through Sunday noon, Nov. 15 in Convention Hall, Atlantic City, will feature seven programs on Clinical Cardiology and concurrent sessions on various phases of cardiovascular research.

Abstracts of papers to be presented at the Scientific Sessions and at the Council on Arteriosclerosis meeting, as well as other selected abstracts, will be published as a supplement to the October issue of Circulation.

Highlights of the Scientific Sessions follow:

Thursday, Nov. 12


On Thursday evening, conferences are scheduled for small group discussion of cardiovascular problems.

Friday, Nov. 13


Saturday, Nov. 14


“Meet the Expert” session will be held on Saturday evening in individual rooms and in the ballroom, with separate tables for discussions on therapeutics.

Sunday, Nov. 15

Clinical Sessions: Report on the “Urokinase Pulmonary Embolism Trial” by John Blackmon and Sol Sherry; Symposium on “Experi-

Scientific and industrial exhibits will be displayed throughout the meeting. On Sunday morning, recently produced cardiovascular films will be shown.

The $50 registration fee is waived for members of Heart Association or AHA Councils. Medical students, house officers, research fellows, graduate students and members of the armed forces will be admitted without charge with letters of certification.

Registration and hotel reservation forms may be obtained from local Heart Associations or the AHA National Office, 44 E. 23rd St., New York, N. Y. 10010.

Meetings to Precede Heart Sessions

Preceding the American Heart Association’s Annual Meeting and Scientific Sessions in Atlantic City, the Association’s Council on Arteriosclerosis will hold its annual meeting on Tuesday and Wednesday, Nov. 10-11 in Howard Johnson’s Motor Lodge. In addition, five all-day Postgraduate Seminars will be held on Wednesday, Nov. 11 in the Hotel Shelburne, under sponsorship of AHA’s Councils on Basic Science, Cardiovascular Surgery, Circulation, Rheumatic Fever and Congenital Heart Disease, and High Blood Pressure Research.

Also scheduled for Wednesday, Nov. 11, is the Laennec Society meeting in the Hotel Shelburne, co-sponsored by AHA’s Council on Clinical Cardiology.

November 1 is Deadline To Apply for AHA Grants

November 1, 1970 is the deadline for submitting applications for American Heart Association Grants-in-Aid, to be awarded for the fiscal year beginning July 1, 1971. The deadline for applications for Established Investigatorships, Foreign Visiting Scientists and British-American Research Fellowships was September 15.

Grants-in-Aid are made to support and expand the research activities broadly related to cardiovascular function and disease, or to related fundamental problems. Support is available for all basic disciplines, such as physiology, biochemistry and pathology, as well as for epidemiological and clinical investigations which bear on cardiovascular problems. Limited funds are also available for support of research in the basic science disciplines which are independent of any apparent direct application to the field of cardiovascular disease.

Forms to apply for Grants-in-Aid may be obtained from the Research Department, American Heart Association, 44 E. 23rd Street, New York, N. Y. 10010.

Monograph Series Adds 2 Volumes

Two new volumes, Numbers 29 and 30, have been added to the Heart Association's Monograph Series as follows:

Volume 29, entitled “Coronary Heart Disease in Seven Countries,” reports on an international cooperative study on the epidemiology of the ailment. The study examined more than 12,000 middle-aged men in Finland, Greece, Italy, Japan, The Netherlands, United States and Yugoslavia. Included in the findings were that blood pressure and serum cholesterol were statistically important. Less important statistically in the incidence of the disease were body weight, cigarette smoking and physical activity, the study notes. The volume was edited by Ancel Keys.


The new volumes may be obtained from local Heart Associations or AHA’s Distribution Department.
Table-Top Unit Tests
Hypertension Diagnosis

A table-top “Diagnostic Testing Unit on Hypertensive Disease” has been prepared by the Heart Association's Committee on Medical Education for use at medical meetings, as a teaching exercise for medical students and house officers, and as a basis for discussion at hospital staff meetings.

The unit's four panels illustrate pertinent laboratory data on four patients. After reading their case histories and studying the panel's X-rays, eye grounds and electrocardiograms, the physician fills out a multiple choice questionnaire concerning each case. An accompanying 14-page booklet contains instructions for testing, case histories and questions and answers.

The 10-pound unit may be obtained on a loan or purchase basis from local Heart Associations or the AHA Central Office.

Digitalis is Topic
Of Ohio Symposium

An International Symposium on the "Basic and Clinical Pharmacology of Digitalis" is scheduled for September 29-30 at the Ohio State University College of Medicine in Columbus, under the direction of Bernard H. Marks and Arnold M. Weissler.

Co-sponsoring the symposium are AHA's Council on Clinical Cardiology, Ohio State University Medical College, AHA-Ohio Affiliate and Central Ohio Heart Association. Further information may be obtained from Dr. Weissler, Ohio State University Hospital, 410 W. 10th Ave., Columbus, Ohio 43210.

Meetings Calendar

1970

September 29-30: Basic and Clinical Pharmacologic Concepts of Digitalis Action, Columbus, Ohio. Dr. Arnold M. Weissler, Ohio State University Hospital, 410 West 10th Ave., Columbus 43210.

October 2-3: Cardiovascular Symposium (3rd Annual) St. Louis. Sponsors: Great Plains Heart Ass'n. in cooperation with St. Louis Heart Ass'n. and AHA Council on Clinical Cardiology. St. Louis Heart Ass'n., 4643 Lindell Blvd., St. Louis 63108.

October 5-7: 3 Days of Cardiology (3 Key Cardiovascular Conditions), Course Directors: Oglesby Paul, Hans H. Hecht, Richard A. Carleton, Chicago. Co-sponsor, Northwestern Univ. Medical Center, Passavant Memorial Hospital, Chicago Heart Ass'n. Medical Education Dept., AHA, 44 E. 23rd St., New York 10010.


October 12-17: Canadian Heart Foundation (Annual Meeting), Ottawa. Dr. John B. Armstrong, 270 Laurier Ave. W., Ottawa, 4 Canada.


November 12-17: American Heart Association, Scientific Sessions, Nov. 12-15; Annual Meeting, Nov. 16-17, Atlantic City, AHA, 44 E. 23rd St., New York 10010.


December 2-4: 3 Days of Cardiology (Electrocardiography), Course Director: Charles E. Kossmann, Memphis. Co-sponsors, Univ. of Tennessee College of Medicine, Tennessee Heart Ass'n. Medical Education Dept., AHA, 44 E. 23rd., New York 10010.

December 10-12: Cardiovascular Seminar (Coronary Artery Disease), Miami. AHA Council on Clinical Cardiology, Univ. of Miami School of Medicine, Fla. State Board of Health, Amer. Academy of General Practice, Heart Ass'n. of Greater Miami, 5080 Biscayne B'vld, Miami 33137.
1971


April 29-May 1: 3 Days of Cardiology, Course Directors, William C. Roberts, Richard S. Ross, Bethesda and Baltimore. Medical Education Department, AHA, 44 E. 23rd St., New York 10010.


May 10-13: 4 Days of Cardiology (Advanced Electrocardiography), Course Directors: J. Willis Hurst, Robert C. Schlant, Atlanta. Emory Univ. School of Medicine, American College of Cardiology, Georgia Heart Ass'n., Medical Education Dep't., AHA, 44 E. 23rd St., New York 10010.

June 14-16: 3 Days of Cardiology (Fundamental Problems of Cardiology, Hypertension and Nephrology), Course Director: Walter M. Kirkendall, Iowa City. University of Iowa College of Medicine, Iowa Heart Ass'n., Great Plains Regional Heart Ass'n. Medical Education Dep't. AHA, 44 E. 23rd St., New York 10010.

September 8-11: International Symposium on Drugs Affecting Lipid Metabolism, Philadelphia. Dr. W. L. Holmes, Lankenau Hospital, Philadelphia 19151.

Abroad

1970

September 6-12: World Congress of Cardiology (VIth), London. The Conference Center, 43 Charles St., Mayfair S.W.1, London, England.


September 15-18: Congress on Internal Diseases, Bratislava. Dr. M. Ondrejicka, Mickiewiczovz 13, Bratislava, Czechoslovakia.

October 25-30: International Congress of Internal Medicine, New Delhi. Dr. K. K. Datey, KEM Hospital, Bombay 12, India.

1971


May 27-29: International Cardiac Surgical Conference, Melbourne, Australia. Dr. Graeme Sloman, Royal Melbourne Hospital, Grattan St., Parkville, Victoria, Australia.


September 9-11: International Cardiovascular Society (10th Int'l Congress), Moscow. Dr. Allan D. Callow, 171 Harrison Ave., Boston 02111.

November 29-December 5: Asian and Oceanian Congress of Neurology, Bombay. Dr. A. N. Patel, Deer Lodge Hospital, Winnipeg, Manitoba, Canada.

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