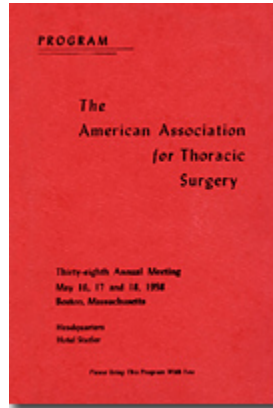


1958 ANNUAL MEETING PROGRAM



THE AMERICAN ASSOCIATION *for* THORACIC SURGERY 1957-58

OFFICERS

President..... BRIAN BLADES, Washington, D. C.
Vice-President..... MICHAEL E. DE BAKEY, Houston, Texas
Treasurer..... JULIAN JOHNSON, Philadelphia, Pa.
Secretary..... HIRAM T. LANGSTON, Chicago, Ill.
Editor..... EMILE HOLMAN, San Francisco, Calif.

COUNCIL

WILLIAM E. ADAMS (1958) H. BRODIE STEPHENS (1960)
WARRINER WOODRUFF (1959) FREDERICK G. KERGIN (1961)
CAMERON HAIGHT (1958)

MEMBERSHIP COMMITTEE

DUANE CARR, *Chairman*
EDWARD J. BEATTIE, JR. HERBERT C. MAIER
EDOUARD D. GAGNON LAURENCE M. SHEFTS
FRANK L. GERBODE JOHN W. STRADER

ASSOCIATION REPRESENTATIVES ON THE BOARD OF THORACIC SURGERY

JOHN C. JONES JOHN W. STRIEDER
EDWARD M. KENT WILLIAM M. TUTTLE

Friday Morning, May 16, 1958

8:30 A.M. Business Meeting - Imperial Ballroom

Scientific Session: REGULAR PROGRAM

Eulogy - Evarts A. Graham
by Tom Burford

1. Further Studies in the Surgical Management of Carcinoma of the Lung.

EDWARD D. CHURCHILL, RICHARD H. SWEET, J. GORDON SCANNELL

and EARLE WILKINS (*by invitation*), Boston, Mass.

Eight years ago the authors presented before this society the results of surgical treatment of carcinoma of the lung at the Massachusetts General Hospital in the years 1930 to 1950. The years since that report have been characterized by a greater awareness of the disease on the part of the medical profession. The authors, therefore, propose to review the surgical experience with primary cancer of the lung at this hospital in the years 1950-1956 with this in mind. During this period approximately 600 proven cases were seen, of which about 300 came to thoracotomy, comparable in number to the earlier series. In addition, it is possible to present a long-term follow-up of the survivors of the 1930-1950 group.

2. Should we Insist on "Radical Pneumonectomy" as a Routine Operation for Carcinoma of the Lung?

JULIAN JOHNSON, CHARLES K. KIRBY and WILLIAM S. BLAKEMORE
(*by invitation*), Philadelphia, Pa.

In an effort to improve the results of resection for carcinoma, of the lung, many surgeons have turned to the so-called "radical pneumonectomy," attempting to apply the principle of "en bloc" resection for this lesion. A widely circulated motion picture on the subject implies that the surgeon who does not practice "radical pneumonectomy" does an incomplete operation. In discussing this subject around the country, we have been impressed with a general reluctance of thoracic surgeons to admit that radical pneumonectomy is not their routine for carcinoma of the lung, and those who do admit it, tend to do so sheepishly.

We have maintained a conservative approach to the problem of cancer of the lung in a series of about 700 patients admitted to the Hospital of the University of Pennsylvania. Pulmonary resection was carried out in 38 per cent of these patients with a 7 per cent hospital mortality. The five year survival rate was 25 per cent for those undergoing resection, or 9 per cent for all patients seen more than five years ago.

We have been unable to find results significantly superior to these as far as five year survival is concerned in any unselected series, including those of the strongest proponents of the radical approach. We shall discuss this problem in some detail. We believe that the presence or absence of blood vessel invasion in the surgical specimen is more important in the prognosis than the extensiveness of the mediastinal resection. This leads us to believe that the advocacy of radical pneumonectomy as the routine operation for cancer of the lung by all thoracic surgeons throughout the country is unjustified at the present time.

3. Results in the Treatment of Bronchogenic Carcinoma-An Analysis of 1008 Cases.

SOL CENTER (*by invitation*), Miami, Fla., and
THOMAS H. BURFORD, St. Louis, Mo.

One thousand eight patients with bronchogenic carcinoma were seen and treated at the Barnes Hospital Chest Service between January 1, 1948 and January 1, 1956. The diagnosis of cancer of the lung was verified in one hundred percent of cases by microscopic section. The date of follow-up was July 1, 1957. All patients were followed for a minimum of one and one-half years to a maximum of nine and one-half years. Operability and resectability rates are presented. Factors influencing prognosis are analyzed and discussed. Survival figures are given.

4. Coronary Artery Disease After 25 Years.

CLAUDE S. BECK, Cleveland, Ohio

This work is based upon approximately 6,000 experimental operations on the coronary arteries over the past 25 years. The motivation of this work was to improve the crippled coronary circulation. To improve the abnormal circulation requires understanding of the normal circulation. This work had this common denominator and resulted in the establishment of a new physiology for the disease.

Some of the concepts are new but others are given a practical application and are different but not new. They are as follows: Reduction of coronary artery inflow - a, under normal conditions, b, under conditions in which a red cell can go anywhere within the heart's substance. The theory of oxygen differentials and the production of electric currents in the heart. Effect of measured quantities of blood on the electric stability of the heart. Amount of blood that can pass by openings

of 1.5 and 1.0 mm. in the coronary artery system. Total coronary inflow versus even distribution. Even distribution produced by inter-coronaries. Predominance of even distribution over total inflow.

Operative methods to produce intercoronaries. What operation can do and its limitations. Selection of patients. Clinical results. Mortality - in the last 178 consecutive patients operated upon September 1, 1955 to July 1, 1957 there were 2 operative deaths, 1.2%. The mortality was 3 for 25 minutes but the death factor was reversed and it fell back to 2 again.

5. The Clinical Significance of Cor Pulmonale in the Reduction of Cardiopulmonary Reserve Following Extensive Pulmonary Resection.

WILLIAM E. ADAMS, ROBERT W. HARRISON (*by invitation*), EDWIN T. LONG (*by invitation*) and BENJAMIN BURROWS (*by invitation*), Chicago, Ill.

Extensive pulmonary resection occasionally results in an immediate marked reduction of cardiorespiratory reserve leading to cor pulmonale, right heart failure and death. Many individuals are able to compensate to reduction of lung volume in the early postoperative period only later to develop symptoms and disability attributable to reduced cardiopulmonary reserve. Thirty patients who had survived extensive pulmonary resection (pneumonectomy or bilateral lobectomy) two to fourteen years were studied in an effort to define the resulting physiologic alterations. The functional capacities of these individuals ranged from near normal to complete disability. Observations included pulmonary function studies, electrocardiograms and cardiac catheterization with measurement of pulmonary artery and right ventricular pressures, cardiac output and peripheral arterial oxygen saturations at rest and during exercise. Ventilatory studies failed to reveal marked abnormalities other than reduction in vital capacity and maximum breathing capacity commensurate with the amount of lung tissue resected. Generally, there was little relation between changes in peripheral arterial oxygen saturation during exercise and functional capacity. Right ventricular or pulmonary artery pressures at rest were found to be increased 25 to 200 per cent above normal levels. During exercise the right heart pressures rose markedly attaining levels 50 to 140 per cent higher than the resting levels. There appeared to be a close correlation between the amount of elevation in right heart pressures during exercise and the individual's functional capacity; i.e., dyspnea and fatigability on exertion.

Conclusions drawn from such observations are that pulmonary hypertension, especially occurring during exertion, is a major factor in the production of the disability occurring at long periods of time after extensive pulmonary resection.

6. Effect of Severe Unilateral Hypoxia on the Partition of Pulmonary Blood Flow in Man.

AARON HIMMELSTEIN, P. HARRIS (*by invitation*), H. W. FRITTS, JR. (*by invitation*) and ANDRE COURNAND, New York, N. Y.

Using a method previously reported from this laboratory, the effect of severe unilateral hypoxia on the partition of the pulmonary blood flow has been studied in five subjects with minimal lung disease. The method combined bronchspirometry, cardiac catheterization, and arterial cannulation. During a control period, one lung breathed a 25% and the other a 21 % oxygen in nitrogen mixture. During hypoxia, a 5% mixture was substituted for the one containing 21%. The total pulmonary flow was measured by dividing the combined oxygen uptakes of both lungs by the arterio-venous oxygen difference. The flow through the lung breathing 25% oxygen was calculated by assuming full saturation of the blood in its pulmonary veins. The flow through the hypoxic lung was then obtained by difference. In three subjects the level of the arterial saturation during hypoxia first fell, then rose as hypoxia was continued. This rise was associated with a reduction in the fraction of blood flowing through the hypoxic lung. In the fourth patient the result was equivocal. In the fifth, the saturation fell to and remained at 80%, no change in flow was evident, but pulmonary arterial hypertension developed. The implications of these observations will be discussed.

Friday Afternoon, May 16, 1958

2:00 P.M. Scientific Session: REGULAR PROGRAM -Imperial Ballroom

Address by the President

Brian Blades, Washington, D. C.

7. Surgical Treatment of "Atypical" Patent Ductus Arteriosus.

WILLIAM P. YOUNG (*by invitation*), GEORGE G. ROWE (*by invitation*),

ANTHONY R. CURRERI and JOSEPH W. GALE, Madison, Wis.

Closure of a patent ductus has usually been considered to be contraindicated when pulmonary artery pressure has approached or equaled aortic pressure or when arterial oxygen saturations indicate reverse flow through the ductus. We have found that the majority of such "atypical" ducti can be closed safely.

A rather simple test has been carried out at the time of operation to determine the prognosis if closure were to be done in those patients with pulmonary hypertension. The response of the pressures in the pulmonary artery and the aorta to temporary compression of the ductus is the significant finding. There was 95 per cent survival of the 23 cases in which the pulmonary artery pressure fell and the aortic pressure rose. Only 25 per cent of four cases survived when these changes did not occur. It is felt that the ductus should not be closed if the pressure in the pulmonary artery rises with temporary compression of the ductus.

A total of 188 patients have had their ducti closed. Twenty-seven of these had significant pulmonary hypertension. A mortality rate of 1.2 per cent in those without pulmonary hypertension contrasts with a mortality rate of 15 per cent in those with pulmonary hypertension. The first 93 ducti were closed prior to our use of cardiac catheterization and therefore none of them were "atypical". Twenty-seven or 28 per cent of the subsequent 95 ducti had associated pulmonary hypertension - indicating the importance of the problem.

8. Intrathoracic Aneurysms of the Aorta - Analysis of 160 Cases Treated by Resection.

MICHAEL E. DEBAKEY, DENTON A. COOLEY, E. STANLEY CRAWFORD

(*by invitation*) and GEORGE C. MORRIS, JR. (*by invitation*),

Houston, Texas

During the seven year period since our first successful resection of an aneurysm of the aorta, we have employed this method of therapy in 160 cases of intrathoracic aneurysms. This report is based upon certain observations derived from an analysis of this experience.

The cases are divided into four categories according to the type and location of the aneurysm: aneurysms involving the arch, 48 cases; aneurysms involving the descending thoracic aorta, 63 cases; thoracoabdominal aneurysms, 19 cases; and dissecting aneurysms, 29 cases. Although the underlying principle of therapy is similar for all cases and consists essentially in resection of the aneurysm, the method of application is somewhat different for these various groups of cases. Emphasis is placed upon recent developments in the technical application of resection and graft replacement, particularly in relation to the use of controlled extracorporeal circulation and the permanent bypass principle.

The gross mortality in this series of cases was 29 per cent. Most important among the factors influencing mortality are age, heart disease, hypertension, and the type, extent and location of the aneurysm. Follow-ups on all patients surviving operation are available, and long-term survival rates

are presented. Physiologic considerations in terms of cardiac, neurologic, and renal function are also presented.

9. Experiences with the Davila-Glover Purse String in the Correction of Mitral Insufficiency: A Critical Appraisal.

EDWARD M. KENT, WILLIAM B. FORD, JOHN F. NEVILLE, JR. (*by invitation*)

and DON L. FISHER (*by invitation*), Pittsburgh, Pa.

The purse-string technique for the control of severe mitral regurgitation has been employed in 33 patients. The preoperative diagnosis had been confirmed in 30 of these individuals by means of data obtained at left heart catheterization, including T1824 blue dye injection into the left ventricle and immediate recovery of the dye from the left atrium. The information obtained by these methods will be discussed.

The early and late surgical mortality rate has been high (approximately 50%). The survivors will be discussed from the clinical standpoint with reference to evidences of continuing effective control of regurgitation. The postoperative evaluation of these survivors has also included late repetition of the left heart catheterization and intraventricular blue dye injection as consistently as possible. The degrees of adequacy of control of mitral insufficiency as demonstrated by these techniques will be presented. The results of the operation in our hands have been unfavorable and we have discontinued its use.

10. Technical Considerations in Decortication for the Pleural Complications of Pulmonary Tuberculosis.

PAUL C. SAMSON, DUANE L. MERRILL (*by invitation*), DAVID J. DUGAN,

Oakland, E. J. SHABART (*by invitation*), Livermore, LOUIS BARBER

(*by invitation*), Stockton, and JAMES YEE (*by invitation*),

Oakland, Calif.

Since 1946 pulmonary decortication has been performed under our direction on approximately 225 patients either as the sole operation, or combined with thoracoplasty and/or resection. The overall mortality in the patients whose main operation was decortication, was approximately three per cent. There was a good to excellent result with pulmonary re-expansion, hemithoracic restitution and primary healing in more than 85 per cent.

The circumstances in which decortication has been employed include: Incidental decortication; post-pneumothorax unexpandable lung with or without fluid; "false re-expansion"; pure or mixed tuberculous empyema. In the latter two categories it was soon realized that adequate antituberculosis chemotherapy was of the greatest value in preventing the advent of complications.

The use of decortication alone or in combination with thoracoplasty or resection depends upon the status of the underlying lung with particular reference to the presence or absence of active tuberculous disease as well as its capability for re-expansion. Careful estimation of probable expansibility is necessary since prompt complete re-expansion of the lung is the best insurance against space problems and empyema. This evaluation often required the careful assessment of the information obtained from all the available methods of examination.

Decortication in tuberculosis is frequently a much more complicated operation than when carried out for other indications. Particularly it is often necessary to remove the parietal peel preferably without entering the empyema sac, and thus without contaminating the operative field. This can be accomplished in nearly all instances. The technique is similar at its outset to

pleuropneumonec-tomy which can likewise be employed to remove both lung and empyema envelope en masse without spillage.

Attention is called to the value of decortication as an incidental procedure with resection often required to insure prompt and unhampered re-expansion of remaining lobes encased in a thin but inelastic peel.

11. Achalasia of the Esophagus - Further Thoughts on Surgical Management.

WILLIAM M. TUTTLE, Detroit, Mich., and ROBERT T. CROWLEY

(by invitation), Williamson, W. Va.

The varied methods of surgical approach to this disease which has not responded to dilatation leads one to believe that the approach has not been adequate. Many and sundry operative procedures have been used. Within the last few years we have employed a transthoracic anterior Heller type of operation, but instead of leaving the muscularis open we have sutured this layer in a horizontal plane. It has been our feeling that this has been a satisfactory operation and, as will be demonstrated by x-ray and clinical histories, has prevented regurgitation and thus esophagitis which has been the plague of other operative approaches. Thirty-five patients have been so approached. The results have been gratifying.

Saturday Morning, May 17, 1958

8:30 A.M. Scientific Session: THORACIC SURGERY FORUM - Imperial Ballroom

12. The Simplified Stationary Screen Pump Oxygenator.

JEROME HAROLD KAY *(by invitation)* and ROBERT M. ANDERSON

(by invitation), Los Angeles, Calif.

In 1956 at The Surgical Forum of The American College of Surgeons, one of us (JHK) described a simplified screen-type pump oxygenator with flow equal to normal cardiac output. This apparatus oxygenated blood by passing a film of blood on stationary screens in an atmosphere of oxygen. The blood was pumped by two roller pumps. The results with this apparatus have been very good. Recently we have greatly simplified and improved this apparatus so that the entire oxygenator, coronary sinus reservoir, venous reservoir, air trap and filter are combined into one chamber. This eliminates the connections on the arterial side and decreases the priming volume. One of the most important improvements consists of an automatic filmer. This completely eliminates saline to start a film. The apparatus is far simpler to use than a bubbler and is less traumatic to the blood elements. It can be run by one person. It is now being used clinically.

13. Comparison of Relative Merits of Occlusive and Non-Occlusive Pumps for Open Heart Surgery.

PAUL C. HODGES *(by invitation)*, C. WALTON LILLEHEI, RICHARD CARDOZO

(by invitation), ANDRE THEVENET *(by invitation)*, Minneapolis, Minn.

Both occlusive and non-occlusive types of pumps have been recommended for open heart surgery. In general these recommendations have been based upon personal preference or sentiment rather than on factual comparative data.

At the University of Minnesota Heart Hospitals more than 450 patients have had open heart surgery utilizing a pump perfusion system. More than 50 of these clinical perfusions have exceeded a duration of 50 minutes. This clinical experience has been obtained using either an occlusive or non-occlusive pump of several designs. The clinical experience has been supplemented by an extensive laboratory investigation encompassing controlled observations on variables.

Analysis of this experimental and clinical data makes possible some definite conclusions as to the relative merits of occlusive versus non-occlusive pumps. These conclusions are based upon analysis of (1) ability to maintain blood pressure in the perfused subject, (2) evidence of blood trauma (hemolysis, protein denaturation, bleeding, and platelet destruction), (3) ease of flow regulation, and (4) operative risk.

14. Cardiac By-Pass Without Artificial Oxygenator.

W. T. MUSTARD (*by invitation*), W. SAPIRSTEIN (*by invitation*)
and DENISE PAY (*by invitation*).

Sponsored by F. G. KERGIN, Toronto, Canada

Cardiac by-pass is accomplished without the use of an oxygenator by perfusing the aerated lung fields. Avoiding an artificial oxygenator reduces extra-corporeal handling of the blood and consequently the effects of blood damage. An extended period of cardiac by-pass is thus permitted enabling intracardiac surgical correction of some of the more complicated defects.

An operative technique is described together with experimental results. Using conventional cardiac pumps, vena caval return is perfused through the pulmonary circuit. Oxygenated blood returning to the left atrium is cannulated into a reservoir from where it is further pumped into the systemic system. With the heart in asystole, a distended rubber balloon has been developed to occlude the mitral valve orifice, ensuring dry right and left ventricles even in the presence of septal defects.

The results of animal experiments suggest the superiority of this by-pass procedure for the treatment of selected cardiac anomalies over presently used systems with extracorporeal oxygenation.

15. Monitor and Control of Blood Oxygen Tensions and pH During Total Body Perfusion.

LELAND C. CLARK, JR. (*by invitation*), SAMUEL KAPLAN (*by invitation*),

EDWARD C. MATTHEWS (*by invitation*), F. KATHRYN EDWARDS (*by invitation*)

and JAMES A. HELMSWORTH, Cincinnati, Ohio

There is a need for a relatively simple method to continuously monitor the oxygenation of blood and carbon dioxide exchange during total body perfusion for direct vision intracardiac surgery. Measurements of blood saturation are unsatisfactory because during perfusion the oxygen tension of blood is frequently considerably above normal arterial tensions.

For the last several years we have used polarographic methods to record arterial and venous blood oxygen tensions during cardiopulmonary bypass. This method which is applicable to any extracorporeal system has the following advantages: (1) Continuous records of absolute oxygen tension over long periods of time are obtained. (2) The oxygen electrode adds only a few square millimeters of polyethylene to the system. (3) Neither the cathode nor the anode is in contact with the blood, and no voltage is impressed in the blood. (4) The electrode gives a linear response in current flow to a linear increase in oxygen content. (5) The speed of response is full scale in approximately five seconds. (6) The electrode is insensitive to changes in pH.

Continuous records of the pH of arterial blood are routinely obtained during cardio-pulmonary bypass. This information has been very helpful in the monitoring of carbon dioxide exchange in bubble oxygenators. Respiratory acidosis or alkalosis can be prevented by varying the gas flow and size of the oxygen bubbles in the extracorporeal circuit.

The details of these two methods will be described and their value will be demonstrated in the monitoring of human perfusions.

16. Experimental and Clinical Studies of Controlled Hypothermia Rapidly Produced and Corrected by a Blood Heat Exchanger During Extracorporeal Circulation.

IVAN W. BROWN, JR. (*by invitation*), WIRT W. SMITH (*by invitation*),

W. GLENN YOUNG (*by invitation*) and W. C. SEALY,

Durham, N. C.

A simple blood heat exchanger capable of use with any type of heart-lung machine has been developed by our laboratory. Experimental studies with the exchanger of the cooling and rewarming velocities of various tissues such as the brain, heart, kidney, liver, intestinal tract, and muscle masses have been determined for various body flow rates, inflow blood temperatures and arterial inflow sites. The exchanger permits a lowering of vital organ temperatures as rapid as 1.0° C. per minute for the range from 37° C. to 30° C. with a slightly slower rewarming velocity. In most instances, the mid-esophagus temperature closely follows the intracardiac temperature curve. Changes of heart, kidney, intestinal, cerebral, and mixed body A-V oxygen differences during cooling and rewarming were determined for various total body inflow rates. No toxic or deleterious effects on the blood have been noted on rapidly removing or adding heat to the inflowing blood over a wide range of temperatures from 0.5° C. to 42° C. A specially designed bubble trap used with the exchanger for excluding oxygen bubbles which might be released from the oxygen saturated rewarming plasma will also be described. A summary of our clinical data from more than 12 open heart operations will be presented which indicates the many complementary advantages of combining hypothermia and extracorporeal circulation particularly when controlled hypothermia can be achieved rapidly at any time during operation and similarly corrected.

17. Cerebral Blood Flow and Brain Volume Changes in Extra-corporeal Circulation.

MAX M. HALLEY (*by invitation*), KEITH REEMTSMA (*by invitation*),

MANUEL BRESLER (*by invitation*) and OSCAR CREECH, JR.,

New Orleans, La.

Cerebral Damage may be evident in the early postoperative period following extracorporeal circulation. This phenomenon is sometimes suggestive of cerebral edema, and may be completely reversible.

Two groups of animal experiments are being conducted in order to clarify the pathogenesis of the syndrome: The first group involves direct measurement of cerebral blood flow during extracorporeal circulation by means of a magnetic rotameter. It includes determination of cerebral oxygen consumption at different rates of flow; determination of the arterial-venous difference of glucose, pyruvate, and lactate in the cerebral circulation; and measurement of arterial and venous pO₂ and pCO₂.

The second group involves the measurement of central venous pressures and cerebro-spinal fluid pressure during extracorporeal circulation, and subsequent determination of brain volume

changes by the method of White, immediately and at intervals after extracorporeal circulation. Correlation between brain volume changes and the variables of both groups is being attempted.

Results to date in the first group indicate that (a) Cerebral flow during extra-corporeal circulation is directly proportional to systemic blood pressure and is reduced to very low levels during hypotension (b) Perfusion rate is not a good index of cerebral flow (c) Cerebral oxygen consumption falls markedly at low levels of cerebral blood flow (d) Vasopressors, such as nor-adrenalin increase cerebral flow by elevating the systemic blood pressure (e) Glucose levels of venous and arterial cerebral blood show no definite variation during extracorporeal circulation (f) Increase in pCO₂ does not appear to increase cerebral flow.

Results in the second group to date indicate that (a) Brain volume may be significantly increased after extracorporeal circulation (b) Spinal fluid pressure may be elevated to various degrees during extracorporeal circulation, even in the presence of normal venous pressures, particularly at high rates of perfusion (c) Marked spinal fluid pressure increases occur in the presence of elevated venous pressures, particularly in the superior vena cava (d) No definite correlation has yet been established between venous or spinal fluid pressure changes and post-operative increase in brain volume.

18. Clotting Deviations in Man During Cardiac By-Pass: Fibrinolysis and Circulating Anticoagulant.

KURT VON KAULLA (*by invitation*) and HENRY SWAN, Denver, Colo.

Serial coagulation studies before, during, and after heart surgery using a fixed screen oxygenator with DeBakey pumps reveal that two major deviations from the normal coagulation process may occur. One is a very marked increase in the fibrinolytic activity of the plasma reaching a peak during or immediately following the by-pass procedure. This was a persistent finding in patients with a perfusion rate of 55-70 ml/kg/min. (9 out of 9 patients). Fibrinolysis was less pronounced with a rate of 100-110 ml/kg/min. (1 out of 1), and was absent with a rate of 190-200 ml/kg/min. (2 out of 2). The euglobulin technique, permitting early recognition of fibrinolytic activity in heparinized blood, was used. The plasminogen activator in the urine rose to high levels shortly before or during the fibrinolytic phase; dropped to zero in the following hours; and returned to normal after several days. It is suggested that a tissue activator is released during the operative trauma, particularly during the pump-dependent phase, which subsequently activates the blood plasminogen. The intensity of the reaction may be related to the CO_a tension.

The second phenomenon, independent of the perfusion speed, is the appearance of a powerful anticoagulant as measured by the thrombin time. It develops after protamin neutralization of the administered heparin and may persist from minutes to hours. Neither fibrinolysis nor the circulating anticoagulant *per se* necessarily create clinical hemorrhage, but hemorrhage can occur if both phenomena overlap. In 2 patients out of 20, clinical hemorrhage was evident.

19. Elective Cardiac Arrest: The Relationship of Elevated Intra-cardiac Pressures to Subsequent Myocardial Function and Pathologic Pulmonary Changes.

JOHN ROSS, JR. (*by invitation*), JOSEPH W. GILBERT, JR. (*by invitation*),

EDWARD H. SHARP (*by invitation*) and ANDREW G. MORROW,

Bethesda, Md.

Cardiac failure, ventricular fibrillation and pulmonary parenchymal changes sometimes follow elective cardiac arrest during cardiopulmonary bypass. A study was undertaken to define more precisely the central hemodynamic effects of perfusion with cardiac arrest and to determine the relationship of these changes to the above complications.

With a Melrose pump-oxygenator, 31 dogs were subjected to total cardio-pulmonary bypass. Pressures in the left atrium, right ventricle, and aorta were recorded throughout perfusion and recovery. Control and post-perfusion lung biopsies were obtained.

Seven of the 31 dogs underwent perfusion without cardiac arrest or cardiomy. Left and right heart pressures remained low during perfusion, cardiac failure following bypass was not encountered, and post-perfusion lung biopsies were normal. In the remaining 24 dogs, cardiac arrest was induced with potassium citrate. In seven of these animals, right atriotomy was maintained throughout the arrest and recovery periods. Intracardiac pressures did not arise, and cardiac failure occurred in only one dog. In four dogs in which cardiac arrest was induced without atriotomy, left and right heart pressures showed progressive elevations during arrest. Following arrest, ventricular fibrillation occurred in two dogs, and three animals developed cardiac failure. Lung biopsies in two of these showed perivascular edema and hemorrhage. In the remaining 13 dogs, right atriotomy was performed immediately before arrest was terminated. Although the elevated intracardiac pressures returned to normal with right atriotomy, complications were no less frequent. Clamping the pulmonary artery during arrest prevented right ventricular pressure elevation, and augmented left atrial pressure increase.

During cardiac arrest bronchial arterial blood flows into the left heart, and also reaches the right heart through a collapsed pulmonary valve. Without cardiomy, the resulting change in diastolic myocardial fiber length imposed by dilatation may often result in cardiac failure, sometimes associated with lung damage. Both left and right heart distension can be prevented by right atriotomy alone, provided the pulmonary valve remains incompetent. The importance of these findings in the management of patients undergoing elective cardiac arrest will be discussed.

20. Lung Lymph in Experimental Pulmonary Edema.

A. ROBERT CORDELL (*by invitation*), Winston-Salem, N. C., RICHARD A.

BAHN (*by invitation*), Buffalo, N. Y., JAMES C. STEPHENS (*by invitation*),

Buffalo, N. Y. and H. H. BRADSHAW, Winston-Salem, N. C.

A technique has been developed whereby lymph can be collected from the right lymphatic duct in the neck of dogs. It will be shown by means of dye injection studies that in approximately fifty per cent of dogs this lymph comes directly from the lungs without mixing with chyle from the thoracic duct. Thirty-five animals have undergone successful right lymphatic duct cannulation.

Control studies of lung lymph have been carried out on sixteen dogs with the following results: Flow rate ranges from 0.1 to 1.3 cc. per hour. Positive pressure respiration doubles this output in the average dog. Total protein content is approximately one-half that of circulating plasma. Sodium level averages 145 milli-equivalents per liter and chloride content 140 milli-equivalents per liter. Potassium level varies from 2.8 to 4.2 milligrams per liter.

Experimental pulmonary edema has been produced by the intravenous injection of alpha naphthyl thiourea in the dosage of 50 milligrams per kilogram.

Studies of pulmonary lymph during development of this fatal condition have shown the following results: Pulmonary lymphatic flow rises sharply to levels of 12 to 15 cc. per hour. Protein content shows a rise from 3.0 to 4.0 grams per cent. Sodium, potassium, and chloride concentrations show relatively little change.

Pulmonary edema has also been produced by means of creating aortopulmonary artery fistulae. Studies of lung lymph in these animals have shown close correlation with those from dogs in which pulmonary edema was drug-induced.

This technique is presently being used in the laboratory in an attempt to increase our knowledge of pulmonary lymphatic physio-pathology.

21. The Effect of Position on Pulmonary Ventilation.

THOMAS F. NEALON, JR. (*by invitation*) and

JOHN H. GIBBON, JR., Philadelphia, Pa.

It has been stated that it is more difficult to provide adequate pulmonary ventilation for anesthetized patients in the lateral position than in the supine position. As the lateral position is frequently used in thoracic surgery, we undertook to determine whether a greater minute volume ventilation was required in this position than in the supine position.

For the purposes of this study, adequate pulmonary ventilation was taken to be the minute volume ventilation required to maintain the pCO₂ of arterial blood at its preoperative level. The pCO₂ of the expired gas was measured continuously by an infra-red absorption carbon dioxide analyzer. Periodic checks were made by gas analysis of arterial blood samples. Ventilation was provided by an intermittent positive and negative pressure ventilator attached to a closed-circle anesthetic machine with a cuffed endotracheal tube. The minute volume ventilation was measured by a gas flow meter in the expiratory line. Determinations were made on the same patients in the supine and the lateral positions before and after the intrathoracic portion of the operation.

No increase in ventilation was found to be required when the patient was turned to the lateral position from the supine position. An open thoracotomy did not alter these findings when the lung was not retracted or compressed.

22. Aortic Homograft Replacement of the Main Pulmonary Artery.

GEORGE ROBINSON (*by invitation*), PHILIP GLOTZER (*by invitation*),

MARVIN GILBERT (*by invitation*) and ELLIOTT S. HURWITT,

New York, N. Y.

Two techniques have been developed in dogs for replacement of the main pulmonary artery and its bifurcation by an aortic homograft. One method includes the use of cardiopulmonary bypass and elective cardioplegia. The second procedure may be accomplished without temporarily replacing the heart and lungs or interrupting the circulation, by employing a side-to-side anastomosis between the anterior wall of the main trunk of the pulmonary artery and the posterior wall of the mid-portion of the graft. Evaluation of the fate of the grafts in long-term surviving animals includes angiocardiology and histologic study. The potential clinical applications for pulmonary arterial replacement include atresia of the pulmonary artery (either as a solitary lesion or as a component of the Tetralogy of Fallot); chronic thrombotic occlusion of the pulmonary artery; coarctation of the pulmonary artery; and involvement of the pulmonary artery by aneurysms, tumors, or trauma.

23. Experimental Results with a Prosthetic Aortic Valve.

BENSON B. ROE, JOHN W. OWSLEY (*by invitation*) and PETER C.

BOUDOURES (*by invitation*), San Francisco, Calif.

A collapsible plastic intraluminal valve has been constructed for intravascular implantation and a suitable technique has been developed for placing this valve into the ascending aorta of experimental animals without total circulatory arrest or bypass. Three years' experience with various modifications of a tubular flexible tricuspid prosthetic valve has resulted in many improvements to reduce resistance and clotting. The physical properties of the silicone material are excellent in strength and resilience; stress studies reveal no significant fatigue, and no surface

clotting or loss of flexibility has occurred after several months of function. Problems of adjacent clotting and embolization will be discussed and preventive measures presented.

Satisfactory coronary artery flow is maintained with a completely competent valve distal to the ostia. At least three dogs are living with valves functioning for six months and one survived secondary avulsion of the anatomical cusps. A large number of shorter survivors have demonstrated tolerance to the position of the prosthesis, and pressure studies indicate minimal resistance across the valve and total competence to pressures well above 300 mm. Hg.

Technical and physiological material will be presented to demonstrate the valve action and competence under mechanical stress, while functioning in the ascending aorta, and after long-term implantation.

24. Postmortem Perfusion Studies in the Evaluation of Techniques of Aortic Valvulotomy.

W. GERALD AUSTEN (*by invitation*), ROBERT S. SHAW (*by invitation*),

W. M. THURLBECK (*by invitation*) and J. GORDON SCANNELL,

Boston, Mass.

During the past two years the hearts of 25 patients dying with severe aortic stenosis have been studied by perfusion of the aortic valve so as to determine quantitatively the degree of aortic stenosis and regurgitation. The valves were subsequently restudied after the performance of reconstructive procedures. The following observations were made. (1) Closed or "blind" commissurotomy or dilatation usually results in a forbidding degree of regurgitation and inconsistent relief of stenosis, often with release of gross calcareous emboli. (2) Debridement and commissurotomy under direct vision consistently allows adequate relief of stenosis without producing significant regurgitation and without the uncontrolled release of emboli. (3) Anatomic identification of the commissure is the key to successful commissurotomy. After debridement accurate definition of the valvular landmarks is possible. (4) Inspection of normally functioning valves and perfusion studies have shown that valve leaflets may be normally fused to a point 2 mm. from the aortic wall and that division of adherent leaflets beyond this point affords no decrease in valvular resistance to forward flow but may increase the degree of regurgitation.

The application of these principles in a limited number of operative cases is described.

25. Intercoronary Collaterals in Normal Hearts.

SVEN BELLMAN (*by invitation*) and HOWARD A. FRANK, Boston, Mass.

For an understanding of the vascular response to the narrowing or occlusion of coronary arteries and the variation in the adequacy of this response from individual to individual, an accurate knowledge of the numbers, dimensions, locations, pathways, and connections of intercoronary collateral channels in normal hearts seems essential. This information should also be helpful in the planning of surgical efforts to increase coronary collaterals.

Most current knowledge of intercoronary communications in normal hearts has been gained by indirect means, such as the measurement of retrograde pressures and flows, or the demonstration of the interarterial passage of test substances. The connecting vessels themselves cannot usually be demonstrated in normal hearts by standard dissection, histological or radiological technics. Microradiography, applied stereoscopically following the injection of suitable contrast media, has been used successfully to demonstrate the finest vascular elements of many tissues and is well suited to an investigation of the intercoronary vessels.

The present paper presents the results of a post-mortem stereomicroradiographic study of the intercoronary vessels in normal human hearts, and, for correlation of experimental with clinical observations, in the hearts of commonly used laboratory animals as well.

26. Changes in Pulmonary Artery Pressure During Cardiopulmonary By-Pass: An Experimental Study.

JAMES B. LITTLEFIELD (*by invitation*), J. FRANCIS DAMMANN, JR.,

PHYLLIS R. INGRAM (*by invitation*) and WILLIAM H. MULLER, JR.,

Charlottesville, Va.

Pulmonary complications associated with cardiopulmonary by-pass may be transient or result in fatal hemorrhage and edema. This experimental study was performed to evaluate pulmonary artery pressure changes during cardiopulmonary by-pass in dogs with a normal and increased collateral pulmonary circulation.

Acute experiments performed on seventeen dogs employed cardiopulmonary by-pass at intervals up to two hours, using a high flow, bubble, pump-oxygenator. The pulmonary artery and aorta were clamped simultaneously and cardiac standstill induced. Left auricular, pulmonary and femoral artery pressures were monitored and lung biopsies obtained. A right cardiotomy was employed. Left pulmonary artery ligation (termed "physiological pneumonec-tomy") performed in five dogs, several months before evaluation, simulated increased collateral pulmonary circulation seen in certain congenital cardiac patients.

RESULTS:	Pulmonary Artery Pressure mm. Hg.	
	Normal Dogs	"Physiological Pneumonectomy" Dogs
1. Before Standstill:	Below 24	Up to 48 (Intermittent)
2. During Standstill: (30 - 60 minutes)	Below 30	60-80 (Persistent with hemorrhage in 10-30 min.)
3. After Standstill:	Up to 65 (Intermittent)	30-50 (Intermittent)

SUMMARY:

- 1) Pulmonary hypertension was not a problem before standstill but persisted in the "physiological pneumonectomy" dogs during standstill, accompanied by severe hemorrhage.
- 2) Both groups of dogs showed intermittent pulmonary hypertension during cardiac recovery until the left ventricle functioned efficiently.
- 3) Accumulative periods of pulmonary hypertension may produce lung complications at any time during cardiopulmonary by-pass.
- 4) Pulmonary hypertension during cardiopulmonary by-pass may be reduced or prevented by: an unoccluded pulmonary artery, left auricular decompression, prompt return of left ventricular function, maintenance of normal systemic blood pressure and delayed cardiotomy closure.

Saturday Afternoon, May 17, 1958

2:00 P.M. Executive Session. (Limited to Active and Senior Members). Imperial Ballroom.

3:30 P.M. Scientific Session: REGULAR PROGRAM -Imperial Ballroom.

27. Intrathoracic Pheochromocytomas.

HERBERT C. MAIER and GEORGE H. HUMPHREYS, II, New York, N. Y.

Our experience with intrathoracic pheochromocytomas suggests that such tumors are not as rare as the literature would seem to indicate. Since there are few tumors in which the patient's outlook is more markedly influenced by correct diagnosis and appropriate drug therapy during operative removal the current experience with these neoplasms is summarized. Unless the possibility of a pheochromocytoma is recognized on the basis of clinical and operative findings, a serious hypertensive or hypotensive crisis may occur. Awareness of the characteristic location of pheochromocytomas, knowledge of the nature of the metabolic disturbances associated with these tumors, and information on the gross appearance of the lesion within the thorax should enable the surgeon to institute the correct therapy. These features will be discussed and illustrative cases of intrathoracic pheochromocytoma presented.

28. Management of Staphylococcal Tension Pneumatocoles by Intracavitary Suction Tube Drainage.

ELTON WATKINS, JR. and ALEXANDER C. HERING (*by invitation*),

Boston, Mass.

The rising incidence of Staphylococcal pneumonia indicates that tension pneumatocoles seen in the course of this illness may present a serious problem in the future. We have seen extreme ventilatory difficulty in infants as a result of these tension cysts. Monaldi drainage has proved lifesaving in preventing the ballooning of cysts appearing during the course of the acute pneumonic episode. Methods have been devised for introduction of these tubes accurately under local anesthesia in small infants. The method is preferable to lobectomy in the presence of bronchopleural fistula with Staphylococcal empyema or when the toxicity of the primary pneumonic process precludes immediate thoracotomy.

29. Topography of the Human Coronary Arteries Considered in Relation to Cardiac Surgery.

THOMAS N. JAMES (*by invitation*) and GEORGE E. BURCH (*by invitation*).

Sponsored by ALTON OCHSNER, New Orleans, La.

Disturbances in cardiac rhythm and conduction of impulses are recognized as important problems in cardiac surgery. Although every attempt is made to spare conduction pathways and the S-A and A-V nodes, little attention has been directed to the blood vessels supplying these areas. This has been largely due to meager knowledge regarding their circulation.

In a study of 58 human hearts by injection of vinylite into the coronary arteries, followed by hydrochloric acid corrosion, observations on the blood supply to the conduction system have been made. The study provided a spatially oriented preparation of the heart chambers and vessels.

To demonstrate how blood supply to conduction centers may be disturbed during cardiac surgery, some surgical procedures in current use, such as purse-string repair for mitral insufficiency, the circumclusion technic for closing interatrial septal defects, transatrial cannulation of the venae cavae for cardiac bypass, and right ventriculotomy, will be discussed.

Potential hazards associated with these procedures will be presented, as well as those of related procedures. Some hazards include occlusion of the artery to the sino-atrial node occlusion of the artery to the atrioventricular node, and occlusion of the posterior descending ventricular artery.

7:00 P.M. Banquet and Dancing, Hotel Statler -

Imperial Ballroom.

Attendance limited to Members of the Association and their ladies, Invited Speakers and their ladies.

Dinner dress preferred.

Sunday Morning, May 18, 1958

8:30 A.M. Scientific Session: REGULAR PROGRAM -Imperial Ballroom.

30. Open Operation for Mitral Insufficiency.

DONALD B. EFFLER, LAURENCE K. GROVES, WILLIAM V. MARTINEZ (*by invitation*)
and WILLEM J. KOLFF (*by invitation*), Cleveland, Ohio

Increasing experience with the pump oxygenator has permitted a relatively safe approach to the mitral valve. Whereas, there is little reason to utilize such a technique in the treatment of mitral stenosis, it does have real value in the treatment of mitral insufficiency.

The authors have encountered two major types of mitral insufficiency: congenital and acquired.

The *congenital type* of mitral insufficiency may be associated with an atrial defect of the ostium primum type. Failure to recognize the mitral insufficiency at the time of surgical closure may result in congestive failure. The authors' experience and technique in the transatrial correction of combined mitral insufficiency and ostium primum defect is described.

The *acquired forms* of mitral insufficiency encountered at surgery are most frequently caused by rheumatic fever. The etiology, however, is of less importance than the topography of the diseased valve. Mitral insufficiency may be represented by ruptured chordae tendineae, dilatation of the annulus, destruction and distortion of either valve cusp, and by surgical accident in the treatment of pure mitral stenosis. Operations for correction of mitral insufficiency were first attempted through the left atrial appendix utilizing the pump oxygenator and elective cardiac arrest (Melrose technique). Recent experience with the pump oxygenator utilizing the right sided approach, with or without elective cardiac arrest, has shown distinct advantages. The operation is performed by a right thoracotomy alone utilizing retrograde cannulation of the left common femoral artery. The exposure is good and postoperative complications reduced. The authors' experience and techniques are detailed.

31. Direct Vision Correction of Mitral Regurgitation.

EARLE B. KAY and CID NOGUEIRA (*by invitation*), Cleveland, Ohio

Many ingenious techniques have been devised during the past few years for the surgical correction of mitral regurgitation, none of which have been wholly successful. The obvious disadvantage in the past has been the inability to achieve complete or permanent correction of the insufficiency by closed or blind techniques. The application of direct vision techniques, utilizing a

mechanical pump oxygenator, for the correction of mitral regurgitation was a natural development in this field, similar to the transition from closed to open techniques that has taken place in the surgical correction of other cardiac lesions.

In the developmental phase of any new surgical technique many aspects of the problem have to be tried and evaluated. Much of this can be accomplished in the experimental laboratory. However, the final evaluation is dependent upon the results of clinical application. The main problems, apart from those inherent in extracorporeal circulation and perfusion techniques associated with the open technique in the correction of mitral regurgitation, are the approach or exposure, the control of air embolism, the advisability of elective cardiac arrest, and the technical correction of the insufficiency.

Eleven patients with mitral valvular disease have now been operated upon. Eight of the patients had mitral regurgitation and three mitral stenosis. A left-sided approach was employed in four, and a right-sided approach in seven. Elective cardiac arrest has been employed in seven and in four the operative correction was performed with the heart beating. The advantages and disadvantages of the various exposures and the advisability of cardiac arrest will be discussed. At the present it is felt that the right-sided approach allowing the heart to beat will be associated with the greatest degree of success. In this group there was only one operative death, the first in the series.

32. Satisfactory Surgical Correction of Pectus Excavatum Deformity in Childhood - A Limited Opportunity.

KENNETH J. WELCH (*by invitation*), Boston, Mass.

From 1951 to 1958, 138 children with moderate to severe pectus excavatum deformity have been evaluated with regard to the desirability of surgical correction. Seventy-six patients have been considered suitable candidates and have been operated upon by the author. The first thirty-seven patients were operated upon between 1951 and 1955, using the Sweet multiple chondrotomy technique. The second group of thirty-nine patients was operated upon employing a modified Garnier-Lester-Ravitch technique during the period 1954 to 1957. The average age at the time of operation, in both groups, was 3.7 years.

A concept of etiology (chondro-dystrophy) and a plan of therapy is outlined. Detailed physiologic data on twelve patients, ranging in age from two to twelve years, is presented, including pre and postoperative pulmonary function and cardiac catheterization studies. Growth studies have been carried out in selected cases.

It is concluded that surgical correction of pectus excavatum deformity is desirable and effective in a limited age group - two to five years.

a. It is unnecessary and more hazardous (if adequately performed) in patients less than twenty-four months of age.

b. It is of little value after the age of five years, in terms of objective improvement.

c. The risk of operation is minimal in this age group and this risk is defined by: (1) The hazard of a one-and-a-half to two-hour endotracheal general anesthesia in a temporarily flail chest; (2) the accuracy of whole blood replacement in this same period of gravimetrically estimated amounts, in the range of 250 to 500 cc.; (3) the danger of surgically-introduced infection which can be minimized by the avoidance of external traction devices and inadvertent unilateral or bilateral thoracotomy.

33. Pectus Excavatum: An Appraisal of Surgical Treatment.

PAUL C. ADKINS (*by invitation*) and OWEN GWATHMEY, Washington, D. C.

A review of thirty-nine patients seen with a pectus excavatum deformity during a four year period is presented. In twelve patients the operation was deferred to a later age, or was not indicated because of minimal deformity. Twenty-seven patients were subjected to various operative procedures for correction of the defect. The technical aspects of the operative procedure are discussed briefly and the necessity of individualization of each case is emphasized. The indications for the use of the minimal procedure of excision of the xiphoid and division of the substernal tendon in infants under the age of one year are given. The necessity for postoperative physiotherapy for correction of residual postural defects is emphasized. A critical evaluation of the over-all results in the twenty-seven operated cases is presented. The best results were obtained by plastic reconstruction of the sternum and costal cartilages with the use of a homologous rib graft as a strut.

34. Hypoventilation, Hypoxia and Acidosis Occurring in the Acute Postoperative Period.

R. MAURICE HOOD (*by invitation*) and ARTHUR C. BEALL, JR

(*by invitation*), Oakland, Calif.

Sponsored by FRANK GERBODE, San Francisco, Calif.

Hypoventilation during open chest operations, with resultant carbon dioxide retention and respiratory acidosis, has received considerable attention; however, comparatively little interest has been manifested in this problem during the acute postoperative period. The present investigation was undertaken to determine the ventilatory efficiency during this period and its result on arterial oxygen saturation, carbon dioxide levels, and pH.

Forty patients were studied. Thirty were patients undergoing intrathoracic procedures; ten were having major abdominal operations. These were unselected but not consecutive cases. Samples of arterial blood were obtained prior to surgery, before removal of the intratracheal tube, and at fifteen-minute intervals for one hour thereafter. Each sample was analyzed for arterial oxygen (O₂) saturation and carbon dioxide (CO₂) content, and the pH of each sample determined. At the time of removal of each sample, a two-minute spirogram was made, and the tidal volume and minute ventilation were calculated.

The results were analyzed statistically and individually. Fifty per cent showed a significant reduction in arterial pH occurring after surgery. The same patients showed an increase in arterial CO₂ content. Twenty per cent showed a decrease in O₂ saturation below 85 per cent during the hour of observation. Spirograms vividly demonstrated the basis for hypoxia, CO₂ retention, and acidosis. Not infrequently the tidal volume dropped below 300 cc and approached the level of the anatomic dead space. Spirograms revealed, in addition, periods of apnea and irregularity of individual breath excursions.

The writers are of the opinion that postoperative hypoventilation with its resulting metabolic changes may be as significant as that occurring during surgery. Slides illustrating statistical results and several individual cases will be shown. Etiological factors and corrective measures will be discussed.

35. Clinical and Hemodynamic Evaluation of the Surgical Correction of Aortic Stenosis.

DWIGHT E. HARKEN, HARRISON BLACK, WARREN J. TAYLOR (*by invitation*),

WENDELL B. THROWER (*by invitation*), HARRY S. SOROFF (*by invitation*),

STEVEN LUNZER (*by invitation*) and ROY H. CLAUSS (*by invitation*),

Boston, Mass.

Experience with various operations to correct aortic stenosis, including trans-ventricular valvulotome fracture dilatation, open aortic operation and various closed transaortic operating tunnel techniques has varied from moderately encouraging to disastrous throughout the world. A critical analysis of an operation that we have used recently encourages us.

This report on 100 consecutive transaortic operations using an Ivalon tunnel points up pathologic hemodynamic and technical factors that influence the quality of repair. The quality of repair and mortality have steadily improved throughout the series. There have been but three deaths in the last 65 operations, and two of these three patients were operated in overt congestive failure (e.g., an end diastolic pressure of 45 mm. Hg. and bilateral hydrothorax). Many patients in congestive failure did not succumb. Even this type of terminal patient can attain encouraging clinical and hemodynamic improvement.

The limiting factors to totally satisfactory correction of aortic stenosis will be presented and prosthetic valves discussed.

36. The Surgical Treatment of Anomalous Pulmonary Veins.

HENRY T. BAHNSON and FRANK C. SPENCER (*by invitation*),

Baltimore, Md.

Eighteen patients with anomalies of the pulmonary venous return have been treated with open intracardiac surgery. Hypothermia was used in fifteen patients and extracorporeal circulation in three. There were fifteen survivors and three deaths.

Eleven patients had all or part of the right pulmonary veins entering the right atrium in conjunction with an atrial septal defect. The anomalous drainage was corrected during closure of the atrial defect by transposing the atrial septum so that the veins entered the left atrium.

Seven patients had a total anomalous drainage of the pulmonary veins. The veins entered the left innominate vein in two patients, the superior vena cava in three patients, and the coronary sinus in three patients. Anomalous drainage into the superior caval system was corrected by partitioning the superior cava or right atrium so the veins drained through an atrial septal defect into the left atrium. The anomalous drainage into the coronary sinus was treated by excising the partition between the coronary sinus and the left atrium.

The anatomical abnormalities and the methods of surgical correction will be discussed and illustrated.

Sunday Afternoon, May 18, 1958

2:00 P.M. Scientific Session: REGULAR PROGRAM -Imperial Ballroom.

37. Pulmonary Resection in the Treatment of Tuberculosis - Experience with 1700 Cases.

RAYMOND J. BARRETT (*by invitation*), RICHARD JANKOSKA (*by invitation*),

J. CLAUDE DAY, PAUL V. O'ROURKE and E. J. O'BRIEN, Detroit, Mich.

A review of all pulmonary resections performed for tuberculosis at Herman Kiefer Hospital is presented. Resection is increasingly the method of choice so that at present only about 10% of cases are treated by thoracoplasty - the latter being reserved for cases deemed not suitable for resection.

A previous report giving experiences prior to 1950 with a mortality rate of 12% is incorporated in the present resume which gives an overall mortality rate of approximately 3% and a current mortality rate of below 2%.

The bronchopleural fistula rate, while it has been halved from 12% to 6% still remains the number one complication. Delays in expansion are a frequent complication although not, in the majority of cases, requiring further surgery. Fistula and delayed expansion are much more prone to occur in tuberculous than in non-tuberculous resections.

Spread or reactivation of disease has become a relatively rare happening, occurring in less than one per cent of cases in the past five years.

The extent of resection has been steadily refined so that pneumonectomy is now done very rarely, whereas segmental resection is rapidly becoming the most common operation.

38. Pulmonary Infarction Complicating Segmental Resection.

JOHN M. SALYER and HAROLD N. HARRISON (*by invitation*), Denver, Colo.

During the past three years 13 of 398 patients having segmental resections in the treatment of pulmonary tuberculosis have developed a peculiar sort of postoperative difficulty. Such complications are attributable to post-resection *segmental* or *subsegmental infarction*. In our recent experience such untoward events alone have given a complication rate of 3.2 per cent. Further consideration of this problem seems indicated since the course of events which results in serious postoperative complication, to our knowledge, has been alluded to only twice in the voluminous literature dealing with complications following pulmonary resection.

Study of such infarcted contiguous segments removed at subsequent thoracotomy have demonstrated that pathologic changes resulting from venous obstruction and/or thrombosis usually begin at the intersegmental surface and proceed centrally. Other changes such as congestion, hemorrhage, infarction, liquefaction, occasional abscess formation and associated organization and fibrosis have been demonstrated. Air leaks along the segmental plane may create tension pleural spaces. If proper management is delayed, empyema, bronchopleural fistula, delayed hemorrhage and perhaps progression of tuberculous disease can be expected. None so diagnosed has progressed beyond the tension pleural space stage. 12 of the 13 patients had secondary thoracotomy from 9 to 19 days after primary resection. One was not properly managed until 144 days had elapsed. All recovered uneventfully after the condition was corrected. To date none have had recurrence of tuberculous disease.

Suggestions for prevention and recommendations as to surgical management will be discussed.

39. Creation of a Temporary Artificial Ductus in the Surgical Correction of Ventricular Septal Defects Associated with Severe Pulmonary Hypertension. A Two-Stage Operation.

HOWARD D. SIRAK (*by invitation*) and DON M. HOSIER (*by invitation*).

Sponsored by H. WILLIAM CLATWORTHY, JR., Columbus, Ohio

Surgical correction of interventricular septal defects employing open-heart technique is associated with a high operative mortality whenever severe pulmonary hypertension is present. In contrast, cases of "atypical" ductus with a similar degree of pulmonary hypertension have been successfully treated. Therefore, a two-stage operation was devised in order to permit a more gradual

reversal of the pathophysiology. This procedure has been employed only in individuals who had essentially equal pressures in both the pulmonic and systemic circulations.

At the time of the open-heart surgical correction, but prior to the injection of heparin, an artificial ductus is created between the ascending aorta and pulmonary artery. This is accomplished by anastomosing a homograft, 5-6 mm. in inside diameter, in end-to-side fashion to each of these structures at a point about 4 cm. from the heart. After a few minutes to permit clotting at the suture lines, the open-heart surgery is accomplished in the usual fashion. At the conclusion of the by-pass, the artificial ductus, which had been temporarily occluded during the perfusion, is opened. After an interval of 3 to 6 months the artificial ductus is divided through a relatively minor operation performed through the right chest.

The smaller diameter of the artificial ductus as compared with the original septal defect reduces but does not obliterate the left-to-right shunt, thus allowing a partial return to the normal state. It also serves as an escape valve for the right ventricle should the resistance in the lungs be excessive. From a physiologic point of view, staging the repair reduces the final operation to closure of an "atypical" ductus.

Six patients have been treated in this manner. One died because one of the leaflets of the tricuspid valve was distorted during closure of the septal defect and another because the hypertensive changes in the lungs were so far advanced that very little residual shunt through the interventricular septal defect remained. This patient was essentially an Eisenmenger's syndrome and should not have been selected for the operation. A discussion of clinical and laboratory findings of each of the cases and the operative technique will be presented.

40. Corrective Surgery in the Presence of Far Advanced Pulmonary Hypertension.

C. WALTON LILLEHEI, DONALD J. FERGUSON (*by invitation*) and

RICHARD L. VARCO, Minneapolis, Minn.

Pulmonary arteriosclerosis resulting in elevated pulmonary pressure due to greatly increased vascular resistance is a common accompaniment of many congenital and some acquired cardiac lesions and now that open heart methods have been perfected has proved to be the principle limiting factor to successful surgical therapy in these far advanced cases.

Two approaches to this problem are possible. The first is to define certain contra-indications for surgery and then reject for operation those patients who might be anticipated as having a high risk with the usual treatment.

The alternative, which we have preferred, is to consider that virtually all of these patients are basically curable if enough knowledge concerning their pathologic physiology can be acquired. Research upon this problem has been both experimental and clinical. The study of the lung biopsy in correlation with these other clinical and physiologic findings including the response to corrective surgery has been the key to progress.

First, the method of death in these patients has been accurately identified by serial pressure measurements and other observations during this event. Confirmation by reproduction of this picture in dogs has been carried out. Two factors of importance have emerged: abnormal vascular resistance and abnormal lung compliance.

With this knowledge two basic methods found of great therapeutic value clinically have evolved. First, are the measures directed to the lungs. These vary from (depending upon the severity of the pulmonary vascular disease and the patient's age) the use of a closed system with aid to the

respiratory effort ranging from supply of 100% oxygen in a closed system by a demand respirator to complete mechanical respiration at positive pressure.

Second, in the severest cases where the above methods have been insufficient, the construction of temporary decompressing shunts at the time of corrective surgery is necessary. The indications for, technique of, and physiology of these lifesaving shunts is presented.

As a consequence of these measures it has been possible to both reduce operative mortality and to extend corrective surgery to patients previously considered inoperable. Moreover, experience has indicated that if even these patients with such advanced states of pulmonary arteriosclerosis can be successfully guided through this crucial period of readjustment immediately post-correction, their lung pathology will heal in most cases in the succeeding months. Thus, the premium upon learning how to manage these patients though undeniably great has permitted the adoption of a considerably more optimistic attitude concerning pulmonary hypertension.

41. Surgical Treatment of the Tetralogy of Fallot by Open Intra-cardiac Repair.

JOHN W. KIRKLIN, F. HENRY ELLIS, JR. and DWIGHT C. MCGOON

(by invitation), Rochester, Minn.

Experience with the intracardiac repair of the tetralogy of Fallot is now sufficiently extensive to warrant an initial critical review, both in an effort to determine the efficacy of the method as compared to other methods and to assess the new technical knowledge which has been gained thereby.

At the time of writing of this abstract, 60 patients who had this particular intracardiac malformation have undergone surgical repair of it. Data concerning these patients, as well as others operated upon prior to the final preparation of the manuscript, form the basis of the report.

Features of the whole-body perfusion peculiar to this particular type of intracardiac surgery are discussed. Surgical technics for the repair of this defect are described. Although a low operative mortality rate can be quoted for selected groups within the series, or for patients operated upon recently, the nonsurvival rate for the entire series is approximately 30 per cent. This mortality rate is analyzed relative to the anatomic and technical factors which appear to have produced it. Among these is the adequacy of repair, documented in most cases by postcardiotomy studies of hemodynamics. The excellence of the result attainable for surviving patients by means of intracardiac repair is demonstrated.

An attempt is made to compare the hazards and results of this surgical technic with those of the shunt procedures, this comparison suggesting to the authors certain tentative conclusions regarding the surgical methods of treating the tetralogy of Fallot.

42. Atrial Septal Defect, Secundum. Observations on One Hundred Patients Treated by Open Operation.

HENRY SWAN, D. HYWEL DAVIES *(by invitation)* and S. GILBERT BLOUNT, JR.

(by invitation), Denver, Colo.

This report concerns 100 consecutive patients with atrial septal defect of the secundum variety not associated with pulmonic stenosis, all of whom were treated by repair of the lesion under direct vision, using general body hypothermia.

An analysis of the diagnostic features and a discussion of the nature of the hemodynamic and structural alterations due to the abnormality will be presented. Since almost all of these patients underwent cardiac catheterization preoperatively, and a majority were similarly studied postoperatively, considerable data has accumulated for study.

A wide variety of defects, of course, were observed. The frequency of multiple defects, of associated aberrant pulmonary veins and of aberrant entrance of the inferior vena cava into the left auricle emphasize the importance of visual guidance in the repair of these lesions.

Ninety-three patients, including the last 56 consecutive cases, survived the procedure and are alive and well at the present time. Careful postoperative observations on the effect of closure of the defects on the disordered circulation has led to some opinions concerning the relationship between the stage of the disease and the probable result of surgery. On the basis of these observations, and the demonstrated safety (less than 2% risk) of the current technique, our indications for surgery have approached those which are now accepted for patent ductus arteriosus.

The American Association for Thoracic Surgery 1957-58

HONORARY MEMBERS

ALLISON, PHILIP..... Radcliffe Infirmary, Oxford, England
BROCK, SIR RUSSELL C..... Guy's Hospital, London, England
CRAFOORD, CLARENCE..... Sabbatsberg Sjukhus, Stockholm, Sweden
DAVIES, H. MORRISTON
Broadgreen Hospital, Edgelane Drive, Liverpool 14, England
DENK, WOLFGANG..... Surgical University Clinic, Vienna, Austria
JACKSON, CHEVALIER..... Old Sunrise Mills, Schwenkville, Pa.
KELLER, WILLIAM L., COL., MC, USA, (RET.)
2930 Foxhall Road, N.W., Washington, D. C.
LERCHE, WILLIAM..... Larkhills, Cable, Wis.
SEMB, CARL..... Ullevaal Hospital, Oslo, Norway
SHENSTONE, NORMAN S..... 904 Medical Arts Bldg., Toronto 5, Ont.
THOMAS, SIR CLEMENT PRICE..... 69 Harley St., London, W. 1, England

ACTIVE MEMBERS

ABBOTT, OSLER..... Emory University Hospital, Emory University, Ga.
ADA, ALEXANDER E. W..... 139 East 94th St., New York 28, N. Y.
ADAMS, HERBERT D..... Lahey Clinic, 605 Commonwealth Ave., Boston 15, Mass.
ADAMS, RALPH..... Huggins Hospital, Wolfeboro, N. H.
ADAMS, WILLIAM E..... University of Chicago, 950 East 59th St., Chicago 37, Ill.
ALLBRITTEN, FRANK F., JR.
University of Kansas Medical Center, Kansas City 12, Kan.
ALLEY, RALPH D..... Albany Hospital, Albany, N. Y.
ASHBURN, FRANK S..... 1835 Eye St., N.W., Washington 6, D. C.
AUERBACH, OSCAR..... Veterans Adm. Hospital, East Orange, N. J.
AUFSES, ARTHUR H..... 812 Park Ave., New York 21, N. Y.
BADGER, THEODORE L..... 264 Beacon St., Boston 16, Mass.
BAHNSON, HENRY T..... 201 Cedarcroft Road, Baltimore 12, Md.
BAILEY, CHARLES P..... 249 N. Broad St., Philadelphia 7, Pa.
BALLON, HARRY C..... 1414 Drummond St., Montreal 25, Que.
BARKLEY, HOWARD T..... 4109 Montrose Blvd., Houston 6, Texas
BARONOFSKY, IVAN D., CDR. (MC), USNR
3135 Johnson Ave., New York 63, N. Y.
BEATTIE, EDWARD J., JR..... 1753 W. Congress St., Chicago 12, Ill.
BEECHER, HENRY K..... Massachusetts General Hospital, Boston 14, Mass.
BENEDICT, EDWARD B..... Massachusetts General Hospital, Boston 14, Mass.
BENSON, CLIFFORD D..... 1515 David Whitney Bldg., Detroit 26, Mich.

BETTS, REEVE H..... Christian Medical College, Vellore, So. India
 BISGARD, J. DEWEY..... 422 Doctors Bldg., Omaha 31, Neb.
 BLACK, HARRISON..... 67 Bay State Road, Boston 15, Mass.
 BLADES, BRIAN..... 901 Twenty-third St., N.W., Washington 7, D. C.
 BORTONE, FRANK..... 2765 Hudson Blvd., Jersey City 6, N. J.
 BOSHER, LEWIS H..... 1200 E. Broad St., Richmond 19, Va.
 BOYD, DAVID P..... 605 Commonwealth Ave., Boston 15, Mass.
 BRADSHAW, HOWARD H.
 Bowman Gray School of Medicine, Winston-Salem, N. C.
 BRANTIGAN, OTTO C..... 104 W. Madison St., Baltimore 1, Md.
 BREWER, LYMAN A., III..... 2010 Wilshire Blvd., Los Angeles 57, Calif.
 BRINDLEY, GEORGE V., JR..... Scott and White Clinic, Temple, Texas
 BROWN, A. LINCOLN..... 490 Post Street, San Francisco 2, Calif.
 BROWN, ROBERT K..... 1624 Gilpin St., Denver 6, Colo.
 BROWNRIFF, GARRETT M..... 47 Queens Road, St. Johns, Newfoundland
 BUCKINGHAM, WILLIAM W..... 314 Professional Bldg., Kansas City 6, Mo.
 BUGDEN, WALTER F..... Medical Arts Bldg., Syracuse 10, N. Y.
 BURFORD, THOMAS H..... Barnes Hospital, St. Louis 10, Mo.
 BURNETT, W. EMORY..... Broad and Ontario Sts., Philadelphia 40, Pa.
 BYRON, FRANCIS X..... 120 S. Lasky Dr., Suite 203, Beverly Hills, Calif.
 CARLSON, ROBERT I..... Veterans Adm. Hospital, Nashville 5, Tenn.
 CARR, DUANE..... 899 Madison Ave., Memphis 3, Tenn.
 CARTER, MAX G..... 670 George St., New Haven, Conn.
 CHAMBERLAIN, JOHN MAXWELL..... 23 East 79th St., New York 21, N. Y.
 CHAMBERS, JOHN S., JR..... 233 "A" St., Room 1205, San Diego 1, Calif.
 CHESNEY, JOHN G..... 2300 Biscayne Blvd., Miami 37, Fla.
 CLAGETT, O. T..... Mayo Clinic, Rochester, Minn.
 CLOWES, GEOGRE H. A., JR..... 3395 Scranton Road, Cleveland 9, Ohio
 COHN, ROY B..... Stanford University Hospital, San Francisco 15, Calif.
 COLEMAN, FRANK P..... 810 W. Franklin St., Richmond 20, Va.
 CONDON, WILLIAM B..... 1104 Republic Bldg., Denver 2, Colo.
 CONKLIN, WILLIAM S..... 1016 Standard Ins. Bldg., Portland 5, Ore.
 COOLEY, DENTON A..... Baylor University College of Medicine, Houston 25, Tex.
 COOPER, DAVID A..... 1520 Spruce St., Philadelphia 2, Pa.
 COTTON, BERT H..... 1321 N. Vermont Ave., Los Angeles 27, Calif.
 COURNAND, ANDRE..... Bellevue Hospital, 27th St. and 1st Ave., New York 16, N. Y.
 CRANDELL, WALTER B..... Veterans Adm. Hospital, White River Junction, Vt.
 CREECH, OSCAR, JR..... Tulane University School of Medicine, New Orleans 12, La.
 CRIMM, PAUL D..... Boehne Hospital, Evansville 12, Ind.
 CURRERI, ANTHONY R..... 1300 University Ave., Madison 6, Wis.
 DAILEY, JAMES E..... 4109 Montrose Blvd., Houston 6, Texas
 DANIEL, ROLLIN A..... 410 Medical Arts Bldg., Nashville 12, Tenn.
 DANIELS, ALBERT C..... 490 Post St., San Francisco 2, Calif.
 DAUGHTRY, DeWitt C..... 4201 Lake Road, Bay Point, Miami 37, Fla.
 DAVIS, EDGAR W..... 1150 Connecticut Ave., Washington 6, D. C.
 DAY, J. CLAUDE..... 307 David Whitney Bldg., Detroit 26, Mich.
 DE BAKEY, MICHAEL E..... Baylor University, Dept. of Surgery, Houston, Texas
 DECAMP, PAUL T..... 3503 Prytania Street, New Orleans 15, La.
 DELARUE, NORMAN C..... 25 Donlea Drive, Toronto 17, Ont.
 DENNIS, CLARENCE..... 989 Edgewood Ave., Pelham Manor, N. Y.
 DESHAIES, GEORGES..... 37 Bellingham Road, Montreal, Que.
 DETERLING, RALPH A..... 180 Ft. Washington Ave., New York, N. Y.
 DODRILL, FOREST D..... 621 David Whitney Bldg., Detroit 26, Mich.
 DOMM, SHELDON E..... 1918 W. Clinch Ave., Knoxville 16, Tenn.
 DORNER, RALPH A..... 710 Equitable Bldg., Des Moines 9, Iowa
 DORSEY, JOHN M..... 636 Church St., Evanston, Ill.

DOUGLASS, RICHMOND..... Veterans Adm. Hospital, Castle Point, N. Y.
DRAKE, EMERSON H..... 18 Bramhall St., Portland 3, Maine
DRASH, EVERETT C..... University of Virginia Hospital, Charlottesville, Va.
DUGAN, DAVID J..... 459-30th St., Oakland 9, Calif.
EFFLER, DONALD B..... Euclid and East 93rd Sts., Cleveland 6, Ohio
EHRENHAFT, JOHANN L..... University of Iowa, Iowa City, Iowa
ELLIS, F. HENRY, JR..... Mayo Clinic, Rochester, Minn.
ELLISON, ROBERT G..... Medical College of Georgia, Augusta, Ga.
EVANS, BYRON H..... 2940 No. Fresno St., Fresno 3, Calif.
FALOR, WILLIAM H..... 623 Second National Bldg., Akron 8, Ohio
FELL, EGBERT H..... 122 So. Michigan Ave., Chicago 3, Ill.
FISCHER, WALTER W..... 170 East 78th St., New York 21, N. Y.
FORD, WILLIAM B..... 3500 Fifth Ave., Pittsburgh 13, Pa.
FORSEE, JAMES H., COL., (MC), USA..... 5207 Falmouth Road, Washington 16, D. C.
FRANK, HOWARD A..... 330 Brookline Ave., Boston 15, Mass.
FREEDLANDER, SAMUEL O..... 2460 Fairmount Blvd., Cleveland Heights 6, Ohio
GAENSLER, EDWARD A..... 229 Dudley Rd., Newton Centre 59, Mass.
GAGNON, EDOUARD D..... 902 Est, Rue Sherbrooke, Montreal, Que.
GALE, JOSEPH W..... Wisconsin General Hospital, Madison 6, Wis.
GARLOCK, JOHN H..... 3 E. 73rd St., New York 21, N. Y.
GEARY, PAUL..... 909 Park Ave., Plainfield, N. J.
GEBAUER, PAUL..... Leahi Hospital, 649 Pokole St., Honolulu, T.H.
GERBODE, FRANK L..... Stanford University Hospital, San Francisco 15, Calif.
GIBBON, JOHN H., JR..... 1025 Walnut St., Philadelphia 7, Pa.
GLOVER, ROBERT P..... 269 South 19th St., Philadelphia 3, Pa.
GORDON, JOSEPH..... 106 Girard Blvd., S.E., Albuquerque, N. M.
GRACE, ARCHIBALD J..... 530 Wellington St., London, Ont.
GRIMES, ORVILLE F..... University of California Hosp., San Francisco 22, Calif.
GROSS, ROBERT E..... 300 Longwood Ave., Boston, Mass.
GROW, JOHN B..... 3705 E. Colfax, Denver 6, Colo.
HAIGHT, CAMERON..... University Hospital, Ann Arbor, Mich.
HANLON, ROLLINS..... 1401 S. Grand Blvd., St. Louis 4, Mo.
HARKEN, DWIGHT E..... 67 Bay State Road, Boston 15, Mass.
HARPER, FREDERICK R..... 1104 Republic Bldg., Denver 2, Colo.
HARRISON, ALBERT W..... Medical Branch, Univ. of Texas, Galveston, Texas
HARRISON, ELLIOTT..... 1862 West Broadway, Vancouver 9, B. C.
HARRISON, HARLON W., CAPT. (MC), USNR
U. S. Naval Hospital, San Diego, Calif.
HARTER, JOHN S..... 212 Brown Bldg., Louisville 2, Ky.
HEAD, JEROME R..... 55 E. Washington St., Chicago 2, Ill.
HELMSWORTH, JAMES A..... Cincinnati General Hospital, Cincinnati 29, Ohio
HIGGINSON, JOHN F..... 2455 N. W. Marshall St., Portland 10, Ore.
HIMMELSTEIN, AARON..... 70 East 96th St., New York 28, N. Y.
HOCHBERG, LEW A..... 135 Eastern Parkway, Brooklyn 17, N. Y.
HOLINGER, PAUL H..... 700 N. Michigan Ave., Chicago 11, Ill.
HOLMAN, CRANSTON W..... 862 Fifth Ave., New York 21, N. Y.
HOPKINS, WILLIAM A..... 1293 Peachtree St., N.E., Atlanta, Ga.
HUDSON, THEODORE R..... 55 E. Washington St., Chicago, Ill.
HUGHES, FELIX A., JR..... Kennedy Hospital, Memphis 17, Tenn.
HUMPHREYS, GEORGE H..... 180 Ft. Washington Ave., New York 32, N. Y.
HURLEY, GERARD A. P..... 1538 Sherbrooke St., W., Montreal, Que.
HURWITT, ELLIOTT S..... Montefiore Hospital, New York 67, N. Y.
HURWITZ, ALFRED..... Maimonides Hosp., 4802 Tenth Ave., Brooklyn 19, N. Y.
JACKSON, CHEVALIER L..... 3401 N. Broad St., Philadelphia 40, Pa.
JANES, ERNEST C..... 250 Main St., East, Hamilton, Ont.
JARVIS, FRED J..... 1115 Columbia St., Seattle 4, Wash.

JOHNSON, ELGIE K..... 230 Hilton St., Hempstead, N. Y.
 JOHNSON, HOLLIS E..... 2122 West End Ave., Nashville 5, Tenn.
 JOHNSON, JULIAN..... 3400 Spruce St., Philadelphia 4, Pa.
 JOHNSTON, JAMES H., JR..... 710 N. State St., Jackson 2, Miss.
 JONES, JOHN C..... 1136 West 6th St., Los Angeles 17, Calif.
 JOYNT, G. H. C..... 399 Bathurst St., Toronto, Ont.
 KAY, EARLE B..... 10465 Carnegie Ave., Cleveland 6, Ohio
 KEELEY, JOHN L..... 30 North Michigan Ave., Chicago 2, Ill.
 KENT, EDWARD M..... 3500 Fifth Ave., Pittsburgh 13, Pa.
 KERGIN, FREDERICK G..... Toronto General Hospital, Toronto 2, Ont.
 KESSLER, CHARLES R..... 1321 -21st Way, South, Birmingham 5, Ala.
 KING, RICHARD..... 814 Doctors Bldg., Atlanta 8, Ga.
 KINSELLA, THOMAS J..... 1251 Medical Arts Bldg., Minneapolis 2, Minn.
 KIPP, HAROLD A..... Mercy Hospital, Pittsburgh 15, Pa.
 KIRBY, CHARLES K..... 3400 Spruce Street, Philadelphia 4, Pa.
 KIRKLIN, JOHN W..... Mayo Clinic, Rochester, Minn.
 KLASSEN, KARL P..... Ohio State University, Columbus 15, Ohio
 KLEPSEY, ROY G..... 1835 Eye St., N.W., Washington 6, D. C.
 KLOPSTOCK, ROBERT..... Veterans Adm. Hospital, Brooklyn 9, N. Y.
 LAIRD, ROBERT..... 399 Bathurst St., Toronto, Ont.
 LAM, CONRAD R..... Henry Ford Hospital, Detroit 2, Mich.
 LAMBERT, ADRIAN..... 768 Park Ave., New York, N. Y.
 LAMPSON, R. STARR..... 85 Jefferson St., Hartford 14, Conn.
 LANGSTON, HIRAM T..... 1919 West Taylor St., Chicago 12, Ill.
 LAUREY, JAMES R..... 5710 16th St., N.W., Washington 9, D. C.
 LEEDS, SANFORD E..... 2211 Post St., San Francisco 15, Calif.
 LEES, WILLIAM M..... 7000 N. Kenton Ave., Lincolnwood 30, Ill.
 LESTER, CHARLES W..... 70 East 80th St., New York 21, N. Y.
 LEVEN, N. LOGAN..... 1464 Lowry Medical Arts Bldg., St. Paul 2, Minn.
 LEWIS, F. JOHN..... Northwestern Univ. Med. School, Chicago 11, Ill.
 LILLEHEI, CLARENCE W..... University Hospitals, Minneapolis 14, Minn.
 LINDSKOG, GUSTAF E..... 50 Marvel Road, New Haven, Conn.
 LONGMIRE, WILLIAM P., JR..... UCLA School of Medicine, Los Angeles 24, Calif.
 LYNCH, JOSEPH P..... 1180 Beacon St., Brookline 46, Mass.
 MACKLER, SAUL A..... 104 S. Michigan Ave., Chicago 3, Ill.
 MACMANUS, JOSEPH..... 491 Delaware, Buffalo, N. Y.
 MAIER, HERBERT C..... 3 East 71st St., New York 21, N. Y.
 MAJOR, ROBERT C..... University Hospital, Augusta, Ga.
 MANNIX, EDGAR P., JR..... 12 Forest Turn, Manhasset, Long Island, N. Y.
 MAURER, ELMER P. R..... 827 Union Central Bldg., Cincinnati 2, Ohio
 MAUTZ, F. R..... 10515 Carnegie Ave., Cleveland 6, Ohio
 MAYER, JOHN H., JR..... 503 Plaza Parkway Bldg., Kansas City 12, Mo.
 MCDONALD, JOHN R..... Mayo Clinic, Rochester, Minn.
 MELTZER, HERBERT..... 505 Medical Arts Bldg., Edmonton, Alberta, Canada
 MERENDINO, K. ALVIN..... University of Washington, Seattle 5, Wash.
 MERKEL, CARL G..... 8 Church St., Saranac Lake, N. Y.
 MILLS, WALDO O..... 1445 Medical and Dental Bldg., Seattle 1, Wash.
 MINOR, GEORGE R..... Univ. of Virginia Hospital, Charlottesville, Va.
 MISCALL, LAURENCE..... 11 East 68th St., New York, N. Y.
 MUDD, JAMES L..... 634 N. Grand Blvd., St. Louis 3, Mo.
 MULLER, WM. H., JR..... Univ. of Virginia Hospital, Charlottesville, Va.
 MULVIHILL, DANIEL A..... 15 East 77th St., New York 21, N. Y.
 OATWAY, WILLIAM H., JR..... La Vina Sanatorium, Altadena, Calif.
 OCHSNER, ALTON..... Ochsner Clinic, 3503 Prytania St., New Orleans 15, La.
 OLSEN, ARTHUR M..... 102 2nd Ave., S.W., Rochester, Minn.
 O'NEILL, THOMAS J..... 269 South 19th St., Philadelphia 3, Pa.

O'Rourke, PAUL V..... 307 David Whitney Bldg., Detroit 26, Mich.
 OVERHOLT, RICHARD H..... 135 Francis St., Boston 15, Mass.
 PAINE, JOHN R..... The Buffalo General Hosp., 100 High St., Buffalo 14, N. Y.
 PAPPER, EMMANUEL M..... 622 West 168th St., New York 32, N. Y.
 PARKER, EDWARD F..... 86 Hasell St., Charleston 8, S. C.
 PAULSON, DONALD L..... 3810 Swiss Ave., Dallas, Texas
 PETERS, RICHARD M..... University of North Carolina, Chapel Hill, N. C.
 POOL, JOHN L..... 755 Park Ave., New York 21, N. Y.
 POPPE, J. KARL..... 1130 S. W. Morrison St., Portland 5, Ore.
 POTTS, WILLIS J..... 707 Fullerton Ave., Chicago 14, Ill.
 PROCTOR, OSCAR S..... Box 1053, Topeka, Kan.
 RAINE, FORRESTER..... 2230 E. Bradford Ave., Milwaukee 11, Wis.
 RAMSEY, BEATTY H..... 2210 Santa Monica Blvd., Santa Monica, Calif.
 RASMUSSEN, RICHARD A..... Blodgett Medical Bldg., Grand Rapids 6, Mich.
 RAVITCH, MARK M..... Johns Hopkins Hospital, Baltimore 5, Md.
 READ, CHARLES T..... 550 West Thomas Road, Phoenix, Ariz.
 RICHARDS, VICTOR..... Stanford-Lane Hospital, San Francisco 15, Calif.
 RIGGINS, H. McLEOD..... 140 East 54th St., New York 22, N. Y.
 RIPSTEIN, CHARLES B..... 54 Rose Ave., Great Neck, L. L., N. Y.
 ROBERTSON, ROSS..... 925 West Georgia St., Vancouver 1, B. C.
 ROGERS, W. L..... 490 Post St., San Francisco 2, Calif.
 ROSEMOND, GEORGE P..... 3401 Broad St., N., Philadelphia 40, Pa.
 RUMEL, WILLIAM R..... 807 Medical Arts Bldg., Salt Lake City, Utah
 SAMSON, PAUL C..... 3959 Happy Valley Road, Lafayette, Calif.
 SANGER, PAUL W..... Doctors Bldg., Kings Drive, Charlotte, N. C.
 SAROT, IRVING A..... III East 69th St., New York 21, N. Y.
 SCANNELL, J. GORDON..... Massachusetts General Hospital, Boston 14, Mass.
 SCHAFFNER, VERNON D..... 12 Cornwallis St., Kentville, Nova Scotia
 SCHMIDT, HERBERT WM..... Mayo Clinic, Rochester, Minn.
 SCOTT, HENRY W., JR..... Vanderbilt University Hospital, Nashville 4, Tenn.
 SEALY, WILL C..... Duke University Hospital, Durham, N. C.
 SELEY, GABRIEL P..... 540 Park Ave., New York 21, N. Y.
 SHAW, ROBERT R..... 3810 Swiss Ave., Dallas, Texas
 SHEFTS, LAURENCE M..... 503 Moore Bldg., San Antonio 5, Texas
 SHUMACKER, HARRIS B., JR..... Indiana Univ. Medical Center, Indianapolis 7, Ind.
 SKINNER, EDWARD F..... 899 Madison Ave., Memphis 3, Tenn.
 SKINNER, GEORGE F..... 36 Coburg St., St. Johns, N. B.
 SLOAN, HERBERT..... University Hospital, Ann Arbor, Mich.
 SOMMER, GEORGE N. J., JR..... 120 W. State St., Trenton 8, N. J.
 SOUTTER, LAMAR..... 80 East Concord St., Boston 18, Mass.
 STEELE, J. D..... Veterans Adm. Hospital, San Fernando, Calif.
 STEPHENS, H. BRODIE..... 384 Post St., San Francisco 8, Calif.
 STOREY, CLIFFORD F..... 1309 Medical-Dental Bldg., San Diego 1, Calif.
 STRANAHAN, ALLEN..... Albany Hospital, Albany, N. Y.
 STRIEDER, JOHN W..... 1180 Beacon St., Brookline 46, Mass.
 STRODE, JOSEPH E..... 1021 Kapliolani, Honolulu 14, T.H.
 SWAN, HENRY, II..... 4200 East 9th Ave., Denver 20, Colo.
 SWEET, RICHARD H..... 87 Chestnut Street, Boston 8, Mass.
 THOMPSON, SAMUEL A..... 850 Park Ave., New York 21, N. Y.
 TOUROFF, ARTHUR S. W..... 994 Fifth Ave., New York 28, N. Y.
 TUTTLE, WILLIAM M..... 307 David Whitney Bldg., Detroit 26, Mich.
 VAN HAZEL, WILLARD..... 224 S. Michigan Blvd., Chicago 4, Ill.
 VARCO, RICHARD L..... University Hospital, Minneapolis 14, Minn.
 VINEBERG, ARTHUR M..... 1390 Sherbrooke St., W., Montreal 25, Que.
 VORWALD, ARTHUR J..... College of Medicine, Wayne State Univ., Detroit 7, Mich.
 WADDELL, WILLIAM R..... 69 Woodland Road, Chestnut Hill, Mass.

WALKER, JAMES H..... 1123 Virginia St., E., Charleston 1, W. Va.
 WARE, PAUL F..... 16 Norwich St., Worcester, Mass.
 WATERMAN, DAVID H..... 1918 West Clinch Ave., Knoxville 16, Tenn.
 WATSON, WILLIAM L..... 1088 Park Ave., New York, N. Y.
 WEBB, WATTS R..... University Hospital, Jackson, Miss.
 WEISEL, WILSON..... 324 E. Wisconsin Ave., Milwaukee 2, Wis.
 WHITE, MARION L., JR..... Huntington Bank Bldg., Huntington, W. Va.
 WILLAUER, GEORGE..... 1930 Chestnut St., Philadelphia 3, Pa.
 WILLIAMS, MARK H..... 63 Front St., Binghamton, N. Y.
 WILSON, JULIUS L..... Henry Phipps Institute, Philadelphia, Pa.
 WILSON, NORMAN J..... 135 Francis St., Boston 15, Mass.
 WIPER, THOMAS B..... 536 Mason St., San Francisco 2, Calif.
 WOODRUFF, WARRINER..... 8 Church St., Saranac Lake, N. Y.
 WOODS, FRANCIS M..... 135 Francis St., Boston 15, Mass.
 WRIGHT, GEORGE W..... 11311 Shaker Blvd., Cleveland 4, Ohio
 WYLIE, ROBERT H..... 903 Park Ave., New York, N. Y.

ASSOCIATE MEMBERS

ACKMAN, F. DOUGLAS..... 1374 Sherbrooke St., W., Montreal, Que.
 ADELMAN, ARTHUR..... 701 East 63rd St., Kansas City, Mo.
 ADLER, RICHARD H..... 100 High St., Buffalo 3, N. Y.
 AITCHISON, DAVID B..... Mountain Sanatorium, Hamilton, Ont.
 ANDREWS, NEIL C..... Ohio Tuberculosis Hospital, Columbus 10, Ohio
 BAFFES, THOMAS G..... Children's Memorial Hospital, Chicago 14, Ill.
 BATTERSBY, JAMES S..... 1040 W. Michigan St., Indianapolis 7, Ind.
 BELL, JOHN W..... Veterans Adm. Hosp., 4435 Beacon Ave., Seattle 8, Wash.
 BENOIT, HECTOR W., JR..... 503 Plaza Parkway Bldg., Kansas City 12, Mo.
 BERG, RALPH, JR..... 231 Medical Center Bldg., Spokane 4, Wash.
 BERGMANN, MARTIN..... 4500 Olive St., St. Louis 8, Mo.
 BERNATZ, PHILIP E..... Mayo Clinic, Rochester, Minn.
 BLOOMBERG, ALLAN E..... 1095 Park Ave., New York 28, N. Y.
 BOUSQUET, ERNEST O..... 5689 Boulevard Rosemont, Montreal, Que.
 BRUNEAU, JACQUES..... 847 Cherrier, Montreal 24, Que.
 BRYANT, JOSEPH R..... 321 West Broadway, Louisville 2, Ky.
 BURBANK, BENJAMIN..... 244 Henry St., Brooklyn 2, N. Y.
 CAHAN, WILLIAM G..... 444 E. 68th St., New York 21, N. Y.
 CHANDLER, JOHN H..... 616 W. Forest Ave., The Jackson Clinic, Jackson, Tenn.
 CHODOFF, RICHARD J..... 255 South 17th St., Philadelphia, Pa.
 CHUNN, CHARLES F..... 442 W. Lafayette St., Tampa 6, Fla.
 CINCOTTI, JOHN J..... Veterans Adm. Hospital, Albany, N. Y.
 CLATWORTHY, H. WILLIAM, JR.
 The Children's Hospital, 561 South 17th, Columbus 5, Ohio
 CONNAR, RICHARD G..... 706 Franklin St., Tampa 2, Fla.
 COOKE, FRANCIS N..... 25 S. E. Second Ave., Miami 32, Fla.
 COX, WILLIAM V..... 133 Court St., Auburn, Maine
 CRACOVANER, ARTHUR J..... 103 East 78th St., New York 21, N. Y.
 CRASTNOPOL, PHILIP..... 1221 East 21st St., Brooklyn 10, N. Y.
 CRECCA, ANTHONY D..... 376 Roseville Ave., Newark 7, N. J.
 CROSS, FREDERICK S..... 10465 Carnegie Ave., Cleveland 6, Ohio
 CUTLER, PRESTON R..... 807 Medical Arts Bldg., Salt Lake City 1, Utah
 DAFOE, COLIN S..... 508 Medical Arts Bldg., Edmonton, Alberta
 DAMMANN, JOHN F..... 632 Preston Place, Charlottesville, Va.
 DASCH, FREDERICK W..... 416 West Market St., Pottsville, Pa.
 DEATON, W. RALPH, JR..... 1027 Professional Village, Greensboro, N. C.
 DEBORD, ROBERTA..... 1240 Jefferson Bldg., Peoria, Ill.
 DECKER, ALFRED M., JR..... 8 Church St., Saranac Lake, N. Y.

DEMATTEIS, ALBERT..... 1216-13th Ave., Altoona, Pa.
 DIVELEY, WALTER L..... 410 Medical Arts Bldg., Nashville 12, Tenn.
 DODDS, G. ALFRED..... 807 Broadway, Fargo, N. D.
 EGGLEE, EDWARD P..... 105 East 53rd St., New York 21, N. Y.
 EMERSON, GEORGE L..... 26 Strathallan Park, Rochester 7, N. Y.
 FINNERTY, JAMES..... Brookhaven Medical Arts Bldg., Patchogue, N. Y.
 FISHBACK, FREDERICK C..... 1835 Eye St., N.W., Washington 6, D. C.
 FOX, ROBERT T..... 2136 Robincrest Lane, Glenview, Ill.
 FRENCH, SANFORD W., III..... Letterman Army Hospital, San Francisco, Calif.
 FRIEDLANDER, RALPH..... The Bronx Hospital, Bronx 56, N. Y.
 FRIESEN, STANLEY R..... 39th and Rainbow, Kansas City 3, Kan.
 FULLER, JOSIAH..... 205 W. 2nd St., Duluth 2, Minn.
 GERBASI, FRANCIS S..... 151 Sampson, Albertson, Long Island City, N. Y.
 GLENN, FRANK..... 525 East 68th St., New York 21, N. Y.
 GLENN, WILLIAM W. L..... 333 Cedar St., New Haven 4, Conn.
 GOLDMAN, ALFRED..... 416 N. Bedford Drive, Beverly Hills, Calif.
 GRACE, EDWIN J..... 121 Fort Green Place, Brooklyn, N. Y.
 GRAVEL, JOFFRE-ANDRE..... 11 Place George Vth, Quebec City, Que.
 GREER, ALLEN E..... 1200 North Walker, Oklahoma City, Okla.
 GROVES, LAURENCE K..... Cleveland Clinic, Cleveland 6, Ohio
 GWATHMEY, OWEN..... 901 23rd St., N.W., Washington 7, D. C.
 HAMPTON, FOSTER, JR..... Suite 101, Interstate Bldg., Chattanooga, Tenn.
 HANNER, JOSEPH M..... U. S. Naval Hospital, San Diego 34, Calif.
 HARDY, JAMES D..... University of Mississippi Medical Center, Jackson, Miss.
 HAUSMANN, PAUL F..... 2212 West State St., Milwaukee 3, Wis.
 HERBEN, GEORGE F..... House of Rest at Sprain Ridge, Yonkers, N. Y.
 HEROY, WILLIAM W..... East Gate Road, Lloyd Harbor, Huntington, N. Y.
 HERRERA-LLERANDI, RODOLFO E.
 Centre Medico, Guatemala City, Guatemala, C. A.
 HERTZLER, JACK H..... 4377 West Maple Road, Birmingham, Mich.
 HUN, HENRY..... 149 Washington Ave., Albany 10, N. Y.
 INGRAM, IVAN N..... 655 Sutler St., San Francisco 2, Calif.
 IOVINE, VINCENT M..... 1150 Connecticut Ave., N. W., Washington 6, D. C.
 JAHNKE, EDWARD J., JR..... 222 Brettonwood Drive, San Antonio, Texas
 JAMPLIS, ROBERT W..... 300 Homer Ave., Palo Alto, Calif.
 JENSEN, NATHAN K..... 1629 Medical Arts Bldg., Minneapolis 2, Minn.
 JENSIK, ROBERT J..... 224 S. Michigan Ave., Chicago 4, Ill.
 JOHNS, THOMAS N. P..... 6305 Towana Road, Richmond 13, Va.
 JOHNSON, CLIVE R..... 1216 Pennsylvania Ave., Fort Worth 4, Texas
 JOHNSTON, FRANK R..... Bowman Gray School of Med., Winston-Salem, N. C.
 JUDD, ARCHIBALD R..... 304 N. Fourth St., Hamburg, Pa.
 JULIAN, ORMAND C..... 25 E. Washington St., Chicago 2, Ill.
 KAUNITZ, VICTOR H..... 4083 Delaware Ave., Tonawanda, N. Y.
 KELLEY, WINFIELD O..... Uncas on Thames, Norwich, Conn.
 KEMLER, R. LEONARD..... 576 Farmington Ave., Hartford 5, Conn.
 KENNEY, LEO J..... 610 Medical Arts Bldg., Grand Rapids 2, Mich.
 KIRSCHNER, PAUL A..... 2 East 92nd St., New York 28, N. Y.
 KITTLE, C. FREDERICK..... University of Kansas Medical Center, Kansas City 3, Kan.
 KRAEFT, NELSON H..... 1501 Magnolia Drive, Tallahassee, Fla.
 KUNDERMAN, PHILIP J..... 185 Livingston Ave., New Brunswick, N. J.
 KUNSTLER, WALTER E..... 1538 Sherbrooke St., W., Montreal 25, Que.
 LEETCH, HENRY W..... 108 Main St., Saranac Lake, N. Y.
 LEIBOVITZ, MARTIN..... 812 Medical Arts Bldg., Tulsa 3, Okla.
 LEWIS, J. EUGENE, JR..... 1325 South Grand Blvd., St. Louis 4, Mo.
 LEWIS, RUBIN M..... 2380 Ellsworth, Berkeley 4, Calif.
 LONGACRE, JACOB J..... 1503 Carew Tower, Cincinnati 2, Ohio

LUCIDO, JOSEPH L..... 634 North Grand, St. Louis 3, Mo.
 LYON, CLAYTON..... 384 Post St., San Francisco 8, Calif.
 MACDONALD, NEIL..... Medical Arts Bldg., Windsor, Ont.
 MACPHERSON, LACHLAN..... St. John Tuberculosis Hospital, East St. John, N. B.
 MADER, VICTOR O..... 149 S. Park St., Halifax, Nova Scotia
 MADOFF, IRVING M..... 1180 Beacon St., Brookline 46, Mass.
 MANGIARDI, JOSEPH L..... 520 Franklin Ave., Garden City, N. Y.
 MASON, JAMES M., III..... 1023 South 20th St., Birmingham 5, Ala.
 McBuRNEY, ROBERT P..... 899 Madison Ave., Memphis 3, Tenn.
 MELICK, DERMONT W..... 1005 Professional Bldg., Phoenix, Ariz.
 MENDELSON, HARVEY J..... 2065 Adelbert Road, Cleveland 6, Ohio
 MENDELSSOHN, EDWIN..... 1301 Tabor Road, Philadelphia, Pa.
 MEYER, BERTRAND W..... 1136 West Sixth St., Los Angeles 17, Calif.
 MICHELSON, ELLIOTT..... 1801 Eutaw Place, Baltimore 17, Md.
 MILLER, CARROLL C..... 304 Humphrey St., Swampscott, Mass.
 MORROW, ANDREW G..... National Heart Institute, Bethesda 14, Md.
 MOULDER, PETER V..... 950 E. 59th St., Chicago 37, Ill.
 MOUSEL, LLOYD H.
 Dept. of Anesthesiology, The Swedish Hospital, Seattle 4, Wash.
 NEMIR, PAUL, JR..... University of Pennsylvania, Philadelphia 4, Pa.
 NEPTUNE, WILFORD B..... 135 Francis St., Boston 15, Mass.
 NEWMAN, MELVIN M..... State Univ. of N. Y. 450 Clarkson Ave., Brooklyn 3, N. Y.
 NEWMAN, ROBERT W..... Medical Arts Bldg., Knoxville, Tenn.
 O'NEILL, JAMES F..... 140 Roslyn Ave., Glenside, Pa.
 PAUL, JOHN S..... Baker VA Center, Martinsburg, W. Va.
 PHILLIPS, FRANCIS J..... 742 "K" St., Anchorage, Alaska
 PINKHAM, ROLAND D..... Suite 1445, Medico-Dental Bldg., Seattle 1, Wash.
 POLK, JOHN W..... 604 Medical Arts Bldg., Springfield 4, Mo.
 POLLOCK, WILLIAM C., COL., (MC), USA..... 1336 Cherry St., Denver 8, Colo.
 POTTER, BENJAMIN P..... 821 Bergen Ave., Jersey City 6, N. J.
 PRATT, LAWRENCE A..... 15621 Windmill Pt., Grosse Pointe Pk. 30, Mich.
 QUINLAN, JOHN J..... Nova Scotia Sanatorium, Kentville, Nova Scotia
 RANDELL, HERBERT T., JR..... Louisville General Hospital, Louisville 2, Ky.
 ROBBINS, S. GWIN..... 899 Madison Ave., Memphis, Tenn.
 ROBINSON, JOSEPH L..... 1136 West Sixth Street, Los Angeles 17, Calif.
 ROE, BENSON B..... 384 Post St., San Francisco 8, Calif.
 ROSS, RALEIGH R..... 2 Medical Arts Square, Austin 5, Texas
 RYAN, BERNARD J..... 375 East Main St., Bay Shore, N. Y.
 RYAN, THOMAS C..... 90 Shenango St., Greenville, Pa.
 SALYER, JOHN M..... Fitzsimons Hospital, Denver, Colo.
 SANES, GILMORE M..... 3500 Fifth Ave., Pittsburgh 13, Pa.
 SCHAFFER, PAUL W.
 c/o Message Center, Walter Reed Army Hospital, Washington, D. C.
 SEILER, HAWLEY H..... 517 Bayshore Blvd., Tampa 6, Fla.
 SEYBOLD, WILLIAM D..... Hermann Professional Bldg., Houston 25, Texas
 SHIPMAN, SIDNEY..... 490 Post St., San Francisco 27, Calif.
 SIMPSON, H. MURRAY..... 292 Queen's Ave., London, Ont.
 SKINNER, A. M..... Homer Folks Tuberculosis Hospital, Oneonta, N. Y.
 SNYDER, HOWARD E..... Wy/2 E. Ninth Ave., Winfield, Kan.
 SNYDER, JOHN M..... 1236 Moffitt Ave., Bethlehem, Pa.
 STARKEY, GEORGE W. B..... 319 Longwood Ave., Boston 15, Mass.
 STAYMAN, JOSEPH W..... 8815 Germantown Ave., Philadelphia 18, Pa.
 STENSTROM, JOHN D..... 2390 Bowker Ave., Victoria, B. C.
 SULLIVAN, HERBERT J..... Medical Arts Bldg., Hamilton, Ont.
 SWENSON, ORVAR..... 300 Longwood Ave., Boston 11, Mass.
 TABER, RODMAN E..... Henry Ford Hospital, Detroit 2, Mich.

TAYLOR, FREDERICK H..... 1012 Kings Drive, Charlotte, N. C.
 TEMPLETON, JOHN Y., III..... 1025 Walnut St., Philadelphia 7, Pa.
 TEST, FREDERICK C., II..... 20252 Meyers Road, Detroit 35, Mich.
 THOMAS, GORDON W..... Int. Grenfell Association, St. Anthony, Newfoundland
 TILLOU, DONALD J..... 311 W. Church St., Elmira, N. Y.
 TRICERRI, FERNANDO E..... 993 Park Ave., New York 28, N. Y.
 VALLE, A. R..... U. S. P. H. S. Hospital, Detroit, Mich.
 VAN FLEIT, WILLIAM E..... Emory University Hospital, Emory University, Ga.
 VEAL, J. ROSS..... 1028 Connecticut Ave., N.W., Washington 6, D. C.
 WALKER, GEORGE R..... 5 Beach St., Sudbury, Ont.
 WALKUP, HARRY E..... Veterans Adm. Hospital, Oteen, N. C.
 WATKINS, DAVID H..... Denver General Hospital, Denver 4, Colo.
 WATKINS, ELTON, JR..... 300 Longwood Ave., Boston 15, Mass.
 WHITESIDE, WILLIAM C..... 415 Medical Arts Bldg., Victoria, B. C.
 WILSON, JOHN L..... Dept. of Surgery, American University of Beirut, Beirut, Lebanon
 WITMER, ROBERT H..... 126 East Chestnut St., Lancaster, Pa.
 WOLFF, WILLIAM I..... 30 Central Park South, New York 19, N. Y.

SENIOR MEMBERS

ALLEN, DUFF S..... 18 S. Kingshighway, St. Louis 8, Mo.
 AMBERSON, J. B..... Bellevue Hospital, New York, N. Y.
 BALLON, DAVID..... 1538 Sherbrooke St., N., Montreal 25, Que.
 BARNWELL, JOHN B..... Room 866, Veterans Adm., Washington 25, D. C.
 BAZIN, A. T..... 4064 Dorchester St., Westmount, Montreal, Que.
 BECK, CLAUDE S..... 2065 Adelbert Road, Cleveland 6, Ohio
 BERRY, FRANK B..... 4301 Massachusetts Ave., Washington 16, D. C.
 BETTMAN, RALPH B..... 104 S. Michigan Ave., Chicago, Ill.
 BIRD, CLARENCE E..... 64 Alfred Stone Rd., Providence 6, R. I.
 BLALOCK, ALFRED..... Johns Hopkins Hospital, Baltimore, Md.
 BLOCK, ROBERT G..... Montefiore Hospital, New York 67, N. Y.
 BUTLER, ETHAN FLAGG..... 956 West Water Street, Elmira, N. Y.
 BYERS, H. RODDICK..... Ganonoque, Ont.
 CARLSON, HERBERT A..... 4241 East 14th St., Long Beach, Calif.
 CARTER, B. NOLAND..... Cincinnati General Hospital, Cincinnati, Ohio
 CHURCHILL, EDWARD D..... Massachusetts General Hospital, Boston 14, Mass.
 CLERF, LOUIS H..... 5575 Eighth Ave., North, St. Petersburg 2, Fla.
 COLE, DEAN B..... Professional Bldg., Richmond, Va.
 CURTIS, GEORGE M..... Ohio State Univ. College of Medicine, Columbus, Ohio
 DAVIDSON, LOUIS R..... 30 East 60th Street, New York 22, N. Y.
 DECKER, HARRY R..... 730 The Park Bldg., 355-5th Ave., Pittsburgh 22, Pa.
 DIEFFENBACH, RICHARD H..... 570 Mt. Prospect Ave., Newark 4, N. J.
 DOLLEY, FRANK S..... 2010 Wilshire Blvd., Los Angeles 57, Calif.
 DOVELL, CHAUNCEY, COL., MC, (RET.)..... 62 South Boxwood St., Hampton, Va.
 ELKIN, DANIEL C..... Elkin Place, Lancaster, Ky.
 ELOESSER, LEO..... 490 Post St., San Francisco 2, Calif.
 FAULKNER, WILLIAM B., JR..... 1802 Fillmore St., San Francisco, Calif.
 FERGUSON, R. G..... Balfour Apts., Regina, Sask.
 FLICK, JOHN B..... 225 South Fifteenth St., Philadelphia 2, Pa.
 HARRINGTON, STUART W..... Mayo Clinic, Rochester, Minn.
 HART, DERYL..... Duke University, Durham, N. C.
 HAYES, JOHN N..... 24 Church St., Saranac Lake, N. Y.
 HEINBECKER, PETER..... Washington University Medical School, St. Louis 10, Mo.
 HOLMAN, EMILE..... 722 Funston Ave., San Francisco, Calif.
 HUDSON, WILLIAM A..... 602 David Whitney Bldg., Detroit, Mich.
 JANES, ROBERT M..... Medical Arts Bldg., Toronto 5, Ont.
 JOHNS, FRANK S..... Johnston-Willis Hospital, Richmond 21, Va.

KERNAN, JOHN D..... 103 East 78th St., New York, N. Y.
 KING, DONALD S..... Hitchcock Clinic, Hanover, N. H.
 KNOEPP, LOUIS F..... Veterans Adm. Hospital, Alexandria, La.
 LEAHY, LEON J..... 105 Medical Arts Bldg., Buffalo 2, N. Y.
 LEWALD, LEON T..... 1200 Fifth Avenue, New York, N. Y.
 LOCKWOOD, A. L..... 300 Bloor St., E., Toronto, Ont.
 MEADE, RICHARD H..... Blodgett Medical Bldg., Grand Rapids 6, Mich.
 MEYER, HERBERT WILLY..... Box 507, Rancho Santa Fe, Calif.
 MILLER, ROBERT T., JR..... Mountain Lake, Lake Wales, Fla.
 MOERSCH, HERMAN..... 725 Tenth Ave., Rochester, Minn.
 MOORE, JULIAN A..... 404 Flatiron Bldg., Asheville, N. C.
 MOORE, RICHMOND L..... 180 Ft. Washington Ave., New York 32, N. Y.
 MURPHY, JAMES D..... U. S. Veterans Adm. Hospital, Baltimore 18, Md.
 MYERS, J. ARTHUR..... 730 La Salle Bldg., Minneapolis, Minn.
 NEUHOF, HAROLD..... Box 198, Huntington Road, Stratford, Conn.
 NIXON, JAMES W..... 1121 Nix Professional Bldg., San Antonio 5, Texas
 O'BRIEN, EDWARD J..... 307 David Whitney Bldg., Detroit 26, Mich.
 ORNSTEIN, GEORGE..... 965 Fifth Ave., New York, N. Y.
 PACKARD, EDWARD N..... 142 Park Ave., Saranac Lake, N. Y.
 PICKHARDT, OTTO C..... 66 East 79th St., New York, N. Y.
 RIENHOFF, WILLIAM F., JR..... 1201 N. Calvert St., Baltimore 2, Md.
 RIGLER, LEO G..... Cedars of Lebanon Hospital, Los Angeles 29, Calif.
 ROSS, DUDLEY E..... St. Adolphe de Howard, P.Q., Canada
 SMITH, DAVID T..... Duke University, Durham, N. C.
 THORBURN, GRANT..... 1602 West Genessee St., Flint, Mich.
 TUCKER, GABRIEL..... 250 South 19th St., Philadelphia, Pa.
 TYSON, M. DAWSON..... Hitchcock Clinic, Hanover, N. H.
 VAN ALLEN, CHESTER M..... State Hospital, Bikaner, Rajputana, India
 WANGENSTEEN, OWEN H..... University Hospitals, Minneapolis 14, Minn.
 WEINBERG, JOSEPH A..... Veterans Adm. Hospital, Long Beach 4, Calif.
 WELLES, EDWARD S..... 20 Church Street, Saranac Lake, N. Y.

MEMBERS DECEASED

RUDOLPH MATAS FRANK I. TERRILL

THE AMERICAN ASSOCIATION FOR THORACIC SURGERY

Charter Members

June 7, 1917

E. Wyllis Andrews	Arthur A. Law
John Auer	William Lerche
Edward R. Baldwin	Howard Lilienthal
Walter M. Boothby	William H. Luckett
William Branower	Morris Manges
Harlow Brooks	Walton Martin
Lawrason Brown	Rudolph Matas

Kenneth Bulkley	E. S. McSweeney
Alexis Carrel	Samuel J. Melter
Norman B. Carson	Willy Meyer (Founder)
J. Frank Corbett	James Alexander Miller
Armistead C. Crump	Robert T. Miller
Charles N. Dowd	Fred J. Murphy
Kennon Dunham	Leo S. Peterson
Edmond Melchior Eberts	Eugene H. Pool
Max Einhorn	Walther I. Rathbun
Herman Fischer	Martin Rehling
Albert H. Garvin	B. Merrill Ricketts
Nathan W. Green	Samuel Robinson
John R. Hartwell	Charles I. Scudder
George J. Heuer	William H. Stewart
Chevalier Jackson	Franz Torek
H. H. Janeway	Martin W. Ware
James H. Kenyon	Abraham O. Wilensky
Adrian V. S. Lambert	Sidney Yankauer

Meetings of the American Association for Thoracic Surgery

1918-Chicago President, Samuel J. Meltzer
 1919-Atlantic City President, Willy Meyer
 1920-New Orleans President, Willy Meyer
 1921-Boston President, Rudolph Matas
 1922-Washington President, Samuel Robinson
 1923-Chicago President, Howard Lilienthal
 1924-Rochester, Minn. President, Carl A. Hedblom
 1925-Washington President, Nathan W. Green
 1926-Montreal President, Edward W. Archibald
 1927-New York President, Franz Torek
 1928-Washington President, Evarts A. Graham
 1929-St. Louis President, John L. Yates
 1930-Philadelphia President, Wyman Whittemore
 1931-San Francisco President, Ethan Flagg Butler
 1932-Ann Arbor President, Frederick T. Lord
 1933-Washington President, George P. Muller
 1934-Boston President, George J. Heuer

1935-New York President, John Alexander
1936-Rochester, Minn. President, Carl Eggers
1937-Saranac Lake President, Leo Eloesser
1938-Atlanta President, Stuart W. Harrington
1939-Los Angeles President, Harold Brunn
1940-Cleveland President, Adrian V. S. Lambert
1941-Toronto President, Fraser B. Gurd
1944-Chicago President, Frank S. Dolley
1946-Detroit President, Claude S. Beck
1947-St. Louis President, I. A. Bigger
1948-Quebec President, Alton Ochsner
1949-New Orleans President, Edward D. Churchill
1950-Denver President, Edward J. O'Brien
1951-Atlantic City President, Alfred Blalock
1952-Dallas President, Frank B. Berry
1953-San Francisco President, Robert M. Janes
1954-Montreal President, Emile Holman
1955-Atlantic City President, Edward S. Welles
1956-Miami Beach President, Richard H. Meade
1957-Chicago President, Cameron Haight