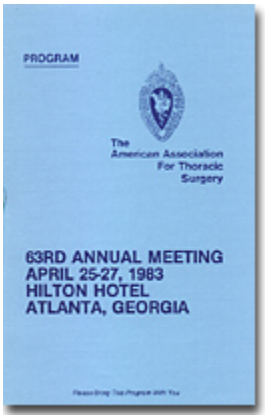
1983 ANNUAL MEETING PROGRAM



THE AMERICAN ASSOCIATION
FOR THORACIC SURGERY
1983 Annual Meeting

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American Association for Thoracic Surgery 63RD ANNUAL MEETING Scientific Program

MONDAY MORNING, April 25, 1983

8:30 a.m. Business Session (Limited to Members)
Grand Ballroom

8:45 a.m. Forum Session
Grand Ballroom

1. Is a Left Ventricular Vent Necessary for Coronary Artery Bypass Procedures Performed with Cardioplegic Arrest?

ROBERT H. BREYER, J. WAYNE MEREDITH*,*

STEPHEN A. MILLS, MICHAEL BARRINGER*,*

ZAK K. SHIHABI, ALBERTO TRILLO* and*

A. ROBERT CORDELL

Winston Salem, North Carolina

Before the widespread adoption of hypothermic cardioplegic arrest (HCA), the physiologic benefits of left ventricular venting (LVV) were convincingly shown. Now, more than 90% of cardiac surgeons utilize HCA during the performance of coronary artery bypass graft procedures (CABG) and many no longer use a vent. Equivalent myocardial cooling and ventricular performance, as assessed by radionuclide angiography, were recently reported in a series of vented (V) and non-vented (NV) patients undergoing CABG. However, animal studies by other investigators have demonstrated depressed left ventricular function (LVF) when left ventricular end diastolic pressure (LVEDP) was elevated to 20 mmHg during reperfusion, even though ventricular distension during the arrest period *per se* was not harmful. In clinical practice, LVEDP rarely exceeds 10 mmHg during reperfusion and radionuclide angiography may not be sufficiently sensitive to detect subtle deficiencies in myocardial protection. Consequently, the role of LVV in CABG remains unclear.

We evaluated the need for LVV using a canine model (35 dogs) that closely simulated the conditions during routine CABG. The dogs were placed on car-diopulmonary bypass, subjected to 60 min. of HCA followed by 30 min. of reperfusion. The left ventricle was vented in 18 of the 35 dogs, not vented in 17 dogs. Myocardial temperature (Temp.) and left atrial pressure (LAP) were recorded continuously. Before (pre) and 30 min. after (post) HCA: (1) LVF curves (6 V, 6 NV dogs) were generated at constant arterial pressure (MAP) and the pre and post values for left ventricular stroke work index (LVSWI) fitted to second order polynomials of LAP. (2) Maximum dP/dt was determined at constant LAP and MAP. (3) The left ventricle was biopsied for determinations of ATP (12V, 11 NV dogs) and semiquantitative grading of ultrastructure (6V, 6 NV).

LAP in NV dogs was 6.9 ± 3.9 mmHg during HCA and 5.0 ± 3.0 mmHg during reperfusion. Temp, during HCA was 10.0 ± 4.6 C in V dogs and 8.8 ± 2.2 C in NV dogs ($p = 0.5$). Comparison of fractional change in LVSWI (LAP = 8) demonstrated no differences in LVF curves between V and NV dogs ($p = 0.47$) and the V and NV dogs could not be separated by linear discriminant analysis ($p = .40$). Equivalent recovery of maximum dP/dt was seen in both groups, 86.2% in V

dogs and 97.4% in NV dogs ($p>0.1$). Post ATP was 96.7% of control (4.30/μmol/gm) in V dogs and 94.6% (4.37 μmol/gm) in NV dogs ($p = 0.7$). Mitochondrial ultrastructure was equally preserved in both groups. The absence of LVV did not lead to left ventricular distension or to more rapid rewarming. The equivalent myocardial protection (preservation of LVF, ATP and ultrastructure) seen in all dogs is contradictory to recent reports that suggest LVV is necessary during reperfusion after HCA.
*By invitation

2. Autogenous Pericardial Patch Enlargement of the Aortic Root or Annulus During Aortic Valve Replacement: Long-Term Follow-Up

JEFFREY M. PIEHLER, GORDON K. DANIELSON,*

JAMES R. PLUTH, THOMAS A. ORSZULAK,*

FRANCISCO J. PUGA, HARTZELL V. SCHAFF*,*

WILLIAM D. EDWARDS and CLARENCE SHUB**

Rochester, Minnesota

Patch enlargement of the aortic root or annulus is a widely accepted technique when restrictive anatomy is encountered during aortic valve replacement. Patches made of prosthetic material have been almost exclusively utilized, and patches of autogenous pericardium have not received wide acceptance. Although pericardium is advantageous because of its low cost, ready availability, non-porosity, and ease of handling, its long-term durability has not been fully established. From 1965-1981, 96 patients had autogenous pericardial patch enlargement of the aorta during aortic valvular replacement. There were 44 males and 52 females with a mean age of 46.5 years. In 81 patients, the patch was placed solely in a supravalvular location to facilitate aortic closure around the valvular prosthesis, while in 15 patients the patch was placed in a transannular fashion, and thus in both subvalvular and supravalvular locations, allowing for annular expansion and insertion of a larger prosthesis. The four operative deaths were from low cardiac output and were unrelated to the use of the patch. In a mean follow-up of 5.4 years (range 6 months to 15 years), no patient has had clinical evidence of sudden patch failure and none has had patch aneurysms detected by routine chest roentgenography. One patient required reoperation for peri valvular leak where the prosthesis had been sutured to the patch. Objective data concerning the late postoperative status of the patch were available in 48 patients. Twenty-four underwent a subsequent cardiac operation where the patch was visualized; of the remaining 24, 16 underwent two-dimensional echocardiography, 2 aortic root angiography, and 6 postmortem examination. In each patient, the patches were well incorporated into the adjacent tissues and patch aneurysms were universally absent. Postmortem histologic examination of the patches revealed a thick fibro-elastic neo-intimal layer to the pericardium with a smooth transition to adjacent aortic intima. This proven durability suggests that autogenous pericardium is an excellent patch material when required during aortic valve replacement.

*By Invitation

3. Experimental Mitral Regurgitation: Physiologic Effects of Correction on Left Ventricular Dynamics

JOHN A. SPRATT, CRAIG O. OLSEN*,*

GEORGE J. TYSON, DONALD D. GLOWER, JR.**

*and J. SCOTT RANKIN**

Durham, North Carolina

Sponsored by: DAVID C. SABISTON, JR.,

Durham, North Carolina

It has been suggested that mitral valve replacement for mitral regurgitation (MR) can precipitate acute myocardial failure by increasing left ventricular (LV) afterload. To investigate this hypothesis, 9 dogs were surgically instrumented with ultrasonic transducers to measure LV diameter (D), electromagnetic flow probes to measure ascending aortic blood flor (Q), and micromanometers to measure LV and pleural pressures (P). At the time of implantation, an 8 mm stainless steel shunt was inserted through the LV myocardium at the base of the anterior wall and sutured to the left atrial (LA) appendage, producing simulated MR of 30-50% of total LV output. A balloon occluder was placed around the LA appendage. One to 7 days after implantation, each dog was studied in the conscious state, and data were recorded during acute occlusion of the shunt. Heart rate was maintained constant by atrial pacing. Data were analyzed digitally, mean ejection wall tension (T) was calculated as ejection LVP x D, and Q was integrated to compute forward cardiac output (FCO). Systolic D shortening (Î”D) was calculated as the change in D from beginning to end ejection. After shunt occlusion, mean LV ejection pressure (EP) increased* in all studies. LVT increased* by an average of 10%, Î”D decreased* by 24%, and FCO increased* by 13%. Thus, the higher afterload after elimination of MR produced an acute fall in stroke shortening and total LV output. However, FCO increased in all studies, implying improved pump efficiency and overall cardiac function. These data suggest that FCO should increase with correction of MR and that the associated augmentation in afterload is probably not a major factor causing low cardiac output after correction.

Shunt Status	EP (mmHg)	LVT (cm mmHg)	EDO (mm)	Î”D (mm)	FCO (ml/min)
Open	92.4 ± 19.0	3070 ± 443	35.1 ± 3.5	18.4 ± 4.7	1756 ± 741
Occluded	101. 3 ± 19.7*	3385 ± 404*	34.9 ± 3.7	13.9 ± 6.2*	1953 ± 572*

* = significant difference using Student's t test for paired data ($p<0.05$).

*By Invitation

4. A Comparison of Valved with Nonvalved Extra Cardiac Conduits: An Experimental Study

ANDREW C. FIORE, PAMELA S. PEIGH*,*

ROBERT J. ROBISON, MICHAEL D. GLANT*,*

*HAROLD KING and JOHN W. BROWN**

Indianapolis, Indiana

Extra cardiac conduits (ECC) are essential in operations for congenital discontinuity between the right ventricle (RV) and the pulmonary artery (PA). The disturbing degree of obstruction reported in clinical series of ECC containing porcine valves has been largely attributed to stenosis at the RV anastomosis, early valve deterioration and the development of a thick neointimal lining within the Dacron graft. The purpose of this study is to compare transconduit resistance and thickness of the neointimal lining in right ventricular ECC with and without a porcine valve.

Sixteen millimeter woven Dacron conduits were implanted in 15 adult mongrel dogs followed by proximal PA occlusion with Dacron tape. In 6 dogs, the ECC contained a porcine valve, while in the other 9, it did not. Cardiac output (C), transconduit gradient (G) and resistance (G/CO) were measured at operation, 6 and 12 months postoperatively. After one year, the ECC was removed, the Dacron graft opened longitudinally and the cross-sectional thickness of the neointimal lining (excluding suture lines) was measured microscopically. Data were collated by time and group. Groups were compared statistically using a T test and two way analysis of variance.

		Operation	6 Months	12 Months
Valved	G/CO	5.30 ± 1.25	6.26 ± 1.11	7.32 ± 1.42
(N = 6)	NT			1370 ± 313.1
Nonvalved	G/CO	8.84 ± 1.78	7.90 ± 1.27	9.56 ± 1.14
(N = 9)	NT			367.7 ± 28.07

CO in liters/minute; G in mmHg; NT is neointimal thickness in microns.

Each value expressed as a mean ± SEM.

Cardiac output and resistance were not significantly different between the two groups. The thickness of the neointimal peel was nearly four-fold greater in valved conduits ($P<.005$). Nonvalved conduits had uniform neointimae, while conduits containing valves had fenestrated intimal linings and varying degrees of valve cusp thrombosis and calcification. The neointimal thickening seen in valved conduits was greatest distal to the valve.

This study demonstrated: (1) the presence of a porcine valve in an extracardiac conduit is associated with a thicker and fenestrated neointima than when the valve is absent, and (2) the absence of a valve in the conduit did not adversely affect the cardiac output or resistance in our model at one year. These data suggest that right ventricular extra cardiac conduits with a different valve design or without valves warrant further investigation.

*By Invitation

5. Internal Mammary Artery Versus Saphenous Vein Graft: Comparative Performance in Patients with Combined Revascularization

RAM N. SINGH*, JULIO A. SOSA* and
GEORGE E. GREEN

Pittsburgh, Pennsylvania; Albany and New York, New York

Thirty-three patients with coronary artery disease undergoing combined myocardial revascularization with internal mammary artery (IMA) and saphenous vein grafts (SVGs) underwent angiographic studies up to ten years after surgery. Each patient had one IMA graft and one or more SVGs. The studies were analyzed to assess the state of preservation of the grafts as well as patency, and findings were correlated with symptoms. Eleven asymptomatic patients, studied one month to five years (mean 1.9 years) after surgery, had intact IMA grafts and SVGs in a good state of preservation. Of the six patients developing symptoms in the first year of surgery,

three had evidence of poor flow in the IMA graft due to large side branches and the other three had stenosis or occlusion of the SVGs. Sixteen patients became symptomatic after several years of symptom-free status and were studied three to ten years (mean 6 years) after surgery. In this group, one IMA graft was occluded and the remaining 15 were in excellent condition. Of the 23 SVGs in this group, 17 (74%) were either occluded or severely stenosed and only six (26%) were in good condition. SVG failure is the predominant cause of late development of symptoms in patients with combined revascularization. Our findings suggest that late SVG failure is a result of an intimal proliferative process from which the IMA grafts tend to remain free. The long term performance of the IMA grafts is far superior to the SVGs.
*By Invitation

6. Pulmonary Artery Balloon Counterpulsation for Right Ventricular Failure

LELAND G. SIWEK, G. KIMBLE JETT*,*

ANTHONY L. PICONE, ROBERT E. APPLEBAUM**

*and MICHAEL JONES**

Newton and Boston, Massachusetts; Bethesda, Maryland

Sponsored by: W. GERALD AUSTEN, Boston, Massachusetts

Right ventricular (RV) failure frequently occurs in patients undergoing correction of congenital cardiac defects, as well as in other settings. RV hypertrophy (RVH) was created in sheep by pulmonary artery banding. RV failure was then produced by performing a right ventriculotomy. Unlike normal hearts, those with RVH and moderate RV outflow obstruction all developed intractable RV failure after ventriculotomy. All six unassisted controls died. Six experimental animals received mechanical assistance by pulmonary artery balloon Counterpulsation (PABCP). A Dacron graft anastomosed end-to-side to the proximal PA served as a reservoir for a 40cc Intra-Aortic Balloon Pump. PABCP effectively reversed RV failure, low cardiac output, and systemic hypotension. Measurements were made in experimental animals during consecutive alternating periods with or without PABCP. Each animal served as its own control. The data from experimental periods were compared by the paired t-test. PABCP increased cardiac output from 1.45 ± 0.16 to 2.03 ± 0.13 L/min ($p<0.0001$), and increased aortic systolic pressure from 78 ± 7 to 99 ± 6 mmHg ($p<0.001$). PABCP produced significant reduction in RV afterload, decreasing RV systolic pressure from 56 ± 5 to 41 ± 3 mmHg ($p<0.0001$) and the RV systolic pressure time index from 1140 ± 79 to 710 ± 65 mmHg·sec·min^{-1} ($p<0.0001$). While RV systolic pressure decreased, PA systolic pressure distal to the band increased from 31 ± 1.5 to 40 ± 1.1 mmHg ($p<0.0001$). RA pressure decreased from 13.9 ± 0.7 to 11.4 ± 0.6 mmHg ($p<0.0001$) with PABCP and RVEDP similarly fell from 14.9 ± 0.6 to 10.7 ± 0.5 mmHg ($p<0.0001$). PABCP produces dramatic hemodynamic improvement and should prove clinically useful in managing otherwise refractory RV failure.

10:15 a.m. Intermission - Visit Exhibits - Lower Level (Galleria) - Complimentary Coffee

*By Invitation

11:00 a.m. Scientific Session
Grand Ballroom

7. Late Functional and Hemodynamic Status of Surviving Patients Following Insertion of a Left Heart Assist Device

DANIEL M. ROSE, STEVEN M. COLVIN*,*

ALFRED TO CULLIFORD, O. W. ISOM,*

JOSEPH N. CUNNINGHAM, JR. and FRANK C. SPENCER

New York, New York

Since 1978 we have inserted a left atrial to ascending aorta left heart assist device in 35 patients with 17 acute and 13 long-term survivors. Thirty-three patients could not be successfully weaned from cardiopulmonary bypass and required insertion of the assist device. Two patients had refractory ventricular arrhythmias and profound cardiac failure in the early postoperative period and required insertion of the assist device. We employ a left heart assist device modified from the assist device initially described by Litwak in 1976. With this type of device, flow rates of 3.5 to 4.0 L/min can be attained. Once the patient's own cardiac function recovers satisfactorily, the patient can be weaned from the assist device. Surviving patients were maintained on the assist device for 16 - 92 hours. There were no significant complications related to the use of the assist device.

Seventeen patients were successfully weaned from the assist device. Four early deaths occurred 60-120 days following removal of the assist device (one from cardiac causes, and three from sepsis). Of the 13 long-term survivors (8 months - 42 months), 4 patients have mild to moderate recurrence of their angina (NYHA Classification II-III) and 9 patients are completely asymptomatic (NYHA Classification I) and 7 are working full time. Three of 13 patients have significantly decreased ejection fractions from their preoperative level, while 10 of 13 have either maintained or increased their ejection fraction from their preoperative level.

We conclude that use of a left heart assist device not only can improve acute survival in patients with profound cardiac failure but can also help preserve long-term ventricular function.

*By Invitation

8. The Implantation of the Total Artificial Heart for the Treatment of Endstage Cardiomyopathy

WILLIAM C. DeVRIES and L. D. JOYCE**

Salt Lake City, Utah

Sponsored by: DAVID C. SABISTON,

Durham, North Carolina

On December 2, 1982, a 61 year old man with endstage cardiomyopathy (Class IV-B) was taken to the operating room during bouts of symptomatic ventricular tachycardia, at which time his heart was removed and an orthotopic pneumatic driven total artificial heart was implanted. Preoperatively, the patient's myocardium had progressively deteriorated clinically and histologically (endomyocardial biopsy) despite therapeutic trials of digoxin, furosemide, hydralazine hydrochloride, amiodarone, azathioprine, and prednisone. After extensive medical, social, psychiatric and financial screening, the patient was approved for surgery. The postoperative period was complicated by intermittent renal failure and pulmonary insufficiency, seizures, valve breakage, compensated disseminated intravascular coagulation and anticoagulation difficulties. The events leading to the surgery, the surgical procedure and the complicated postoperative management will be discussed. The effect of the device upon the formed blood elements, as well as the physiological and pathological compensations in this patient will also be presented.

11:30 a.m. Presidential Address - Grand Ballroom

INTELLECTUAL CREATIVITY IN THORACIC SURGEONS
FRANK C. SPENCER

12:15 p.m. Adjourn for Lunch - Visit Exhibits

*By Invitation

MONDAY AFTERNOON, April 25, 1983

2:00 p.m. Scientific Session
Grand Ballroom

9. Prevention of Local Recurrence and Survival in Surgically Treated Patients with Small Cell Carcinoma (SCC)

ROBERT J. GINSBERG, FRANCIS A. SHEPHERD,*

WILLIAM K. EVANS, RONALD FELD*, JOEL COOPER,*

RIIVO ILVES, THOMAS R. TODD*,*

*F. GRIFFITH PEARSON and PAUL F. WATERS**

Toronto, Ontario

There is a resurgent interest in adjuvant surgery as part of multi-model therapy for SCC. In patients with limited disease (N_2M_0 or better) treated by chemotherapy and mediastinal irradiation, a high rate of local recurrence (50%) has been found. Surgery may be able to reduce the incidence of local recurrence if it is added to treatment planning for limited SCC. Hopefully, this will ultimately improve the curability of this disease.

To assess the role of surgery in treating and preventing local recurrence in limited SCC, we analyzed retrospectively patients undergoing surgical resection for SCC between 1976 and mid 1982. Thirty-seven selected patients of over 1000 treated for SCC in our institutions have had surgical treatment over this 5½ year period. Most patients underwent resection for presumed non-small cell histology. Six later patients had preoperative planned combined modality therapy. All patients had presumed N, or less disease prior to surgery. Twenty-seven patients have been treated and followed for a minimum of one year. Most received adjuvant postoperative chemotherapy and/or radiotherapy. There were 12 stage I patients, 9 stage II and 6 stage III.

In 15 of 27 patients, relapse has occurred. The commonest site of first relapse was brain (7 of 15). Five of these patients had received prophylactic cranial irradiation. In only 2 patients, did relapse occur locally in the hemithorax and/or mediastinum - 1 in N_0 disease, 1 in N_1 disease. No local recurrence occurred in 6 patients with N_2 disease found at thoracotomy. Only 2 relapses have occurred beyond one year - both in brain. In those patients surviving greater than 2 years, no relapses have occurred.

In stage I surgically treated SCC, the estimated actuarial 5 year survival rate is 50%, in stages II and III, 23%. The median survival time for the whole group is 84 weeks. These survival times compare quite favorably to those for resected non-small cell carcinoma patients.

It appears that surgical excision may help in preventing local recurrence in SCC in $N_1$and N_2 disease. In our small series, surgical treatment of limited SCC has yielded estimated actuarial five year survival rates similar to those seen in the surgical treatment of non-SCC patients.

The eventual role of surgery in preventing local recurrence and improving the overall treatment of limited SCC awaits prospective randomized trials.

*By Invitation

10. Occult Lung Cancer

DENIS A. CORTESE, PETER C. PAIROLERO*,*

ERIK J. BERGSTRAHL, LWEIS WOOLNER*,*

MARY ANN UHLENHOPP, JEFFREY M. PIEHLER*,*

DAVID R. SANDERSON, PHILIP BERNATZ,*

DAVID E. WILLIAMS, WILLIAM F. TAYLOR*,*

*W. S. PAYNE and ROBERT S. FONTANA**

Rochester, Minnesota

During the past 10 years, 92 patients (all male) were found to have roentgenographically occult respiratory cancer. Upper airway cancer was found in 16 patients (17%)and lung cancer in 76. Sputum cytology in patients with lung cancer demonstrated marked cellular atypia in 16 patients and squamous cell carcinoma in 60. These latter 60 patients (positive sputum cytology, negative chest x-ray) form the basis of this report.

Ages ranged from 45 to 76 years (mean 62 years). The occult cancer was localized by bronchoscopy (range 1 to 5 times, mean 1.6 times) in 57 patients. One patient refused localization and is alive without treatment 6 years later. The remaining 2 patients were localized by chest roentgenogram and autopsy. Seventy-five percent of tumors were localized within 2.5 months following positive sputum cytology (range 1 day to 49 months). Six patients (10%) had 2 simultaneous occult neoplasms. All 65 localized tumors were squamous cell bronchogenic carcinoma. Nine patients did not undergo pulmonary resection; 6 had evidence of distant metastases, 1 refused operation, 1 had severe coronary artery disease precluding operation, and 1 died of an acute myocardial infarct prior to treatment. Curative pulmonary resections were performed in the remaining 50 patients (83%): lobectomy in 34, pneumonectomy in 11, bilobectomy in 4, and segmentectomy in 1. Forty-five patients were classified post-surgically as stage I (41, T1 NO MO; 3, T2 NO MO, 1, T1 N1 MO), 4 as stage II, and 1 as stage III.

Follow-up ranged from 16 to 115 months (mean 72 months). Overall five-year actuarial survival (lung cancer death only) for all 60 patients was 81% (Kaplan-Meier). Five-year survival for the 41 patients with T1 NO MO neoplasms was 91%. Currently 28 patients have died, but only 13 (22%) from lung cancer. Subsequent lung cancer developed in 20 patients (33%). Eleven of these patients had a second primary lung cancer, 4 of which were occult. We conclude that occult lung cancer has a strong likelihood of long-term survival. Close surveillance is indicated because of the high incidence of a second primary lung cancer.

*By Invitation

11. Prognostic Significance of N1 Disease in Carcinoma of the Lung

NAEL MARTINI, FUMIO NAGASAKI,*

*and BETTY J. FLEHINGER**

New York and Yorktown Heights, New York; Chiba, Japan

From 1973 to 1981, 75 patients with T1N1M0 and T2N1M0 disease had a complete potentially curative resection with mediastinal lymph node dissection. There were 38 adenocarcinomas, 36 epidermoid cancers and 1 large cell carcinoma. Surgical treatment consisted of lobectomy in 54, sleeve lobectomy in 3 and pneumonectomy in 18. Two patients died postoperatively.

Of 17 patients with T1N1 disease, 13 had no further treatment and 4 received postoperative radiation and/or chemotherapy. There was no local or regional recurrence, but 8 patients had distant metastases and 1 developed another cancer. Eight are now alive and well and one is alive with recurrence. The 5-year cumulative survival of these patients was 51%.

There were 58 patients with T2N1 disease. Forty-two had no further treatment and 14 received postoperative radiation and/or chemotherapy. Twenty-six patients had no recurrence, 9 developed local and regional recurrence and 21 had distant metastases. The 5-year cumulative survival of these patients was 52%.

Factors influencing recurrence were the size of the tumor and proximity to the nilum. The specific location of nodal involvement, the number of nodes affected and the extent of involvement within the nodes had no observed effect on survival.

The incidence of local and regional recurrence was low. This we believe is due to the fact that systematic mediastinal lymph node evaluation reduced the margin of error in staging these patients. We conclude that patients with correctly staged N1 disease have a favorable prognosis.

*By Invitation

12. Modern Postoperative Mortality of Surgical Resections for Lung Cancer

F. GRIFFITH PEARSON, ROBERT J. GINSBERG,

LUCIUS D. HILL, ROBERT T. BAGAN,*

PAUL THOMAS, WILLARD FRY, RALPH BUTZ,*

PAUL F. WATERS, MELVYN GOLDBERG*,*

DONALD P. JONES and THE LUNG CANCER STUDY GROUP*

Toronto, Ontario; Seattle, Washington; Rochester,

Minnesota; Chicago, Evanston and Hines, Illinois

Modern postoperative mortality rates for resectional surgery for lung cancer is not readily available. In recent publications estimating the risk factors for surgical resection, mortality rates of 10 - 15% for pneumonectomy and 5-7% for lobectomy are frequently quoted.

In order to determine modern operative mortality rates (up to 30 days postoperative), the Lung Cancer Study Group analyzed the surgical mortality rates of the various participating centers during the years 1977 to 1982. Fourteen hundred and ten resections for lung cancer were available for analysis. Not all participating centers were able to report their data, nor could every contributing institution from the various centers. However, each reporting institution included all resections performed during their assessment period.

Of the 1410 resections performed, 75% were lobectomies, 20% were pneumonectomies and 5% were lesser resections (segmental or wedge). There was no major difference in these proportions among the contributing centers or institutions within these centers.

Fifty postoperative deaths occurred among the 1410 resections (3.6%). The mortality rate for pneumonectomy was 5.5% and lobectomy 2.6%. Lesser resections carried a 3.5% mortality rate, probably reflecting a higher risk group. There was no significant difference in any of these results among the centers and institutions.

In one institution, data was available for mortality rates adjusted for age (315 resections). Between ages of 50-59, the mortality rate was 0.9%, 60-69 - 3.3%, and 70-79 - 6.1%.

The striking similarity of postoperative mortality rates for resectional surgery for lung cancer among the various centers of the Lung Cancer Study Group and among the various institutions within these centers suggest that this data is a reasonably accurate analysis of modern surgical mortality in the treatment of lung cancer.
*By Invitation

13. Human Trials of the Automatic Implantable Defibrillator: A Two Year Program Report

LEVI WATKINS, JR., MARTINM. MOWER*,*

M. MIROWSKI, PHILIP R. REID*,*

LAWRENCE S.C. GRIFFITH and EDWARD PLATIA**

Baltimore, Maryland

Sponsored by: VINCENT L. GOTT, Baltimore, Maryland

Since February 1980, 53 survivors of multiple arrhythmic cardiac arrests unresponsive to therapy underwent implantation of the automatic defibrillator (AD). In 30 patients (pts) (Group I), AD implantation alone was performed by sub-xiphoid insertion (15 pts) and thoracotomy (15 pts). In 23 pts (Group II), implantation was combined with definitive open-heart procedures. Coronary artery bypass (CAB) was performed in 7 pts, CAB and mitral valve replacement in 4 pts and left ventricular aneurysmectomy with endocardial resection in 12 pts. There was no surgical mortality in Group I, three operative deaths occurred in Group II.

The longest follow-up was 31 months; the average is 14 months. Following hospital discharge, 20 episodes of automatic out-of-hospital resuscitations were observed in 10 Group I pts. Similarly 2 resuscitations were observed in 2 Group II pts. Kaplan-Meier survival curves based on the assumption that out-of-hospital resuscitations would otherwise have been lethal indicated a one-year mortality expected to be 45 percent in Group I. Eight late deaths were observed for an actual mortality of 25 percent. One late death was observed in Group II for an actual late mortality of 5 percent. In view of two successful automatic resuscitations, the expected late mortality was calculated to be 15 percent.

While surgery markedly reduced the number of automatic resuscitations, the automatic defibrillator appears to increase survival when implanted alone or in combination with open-heart surgery.

Invited Commentary - ALDEN H. HARKEN, Wynnewood, Pennsylvania

3:45 p.m. Intermission - Visit Exhibits - Lower Level (Galleria) - Complimentary Coffee

*By Invitation

4:30 p.m. Forum Session
Grand Ballroom

14. Improved Myocardial Preservation During Global Ischemia by Continuous Retrograde Coronary Sinus Perfusion

STEVEN F. BOLLING, JOHN T. FLAHERTY*,*

VINCENT L. GOTT and TIMOTHY J. GARDNER
Baltimore, Maryland

In an attempt to avoid poor cardioplegia delivery and inadequate myocardial cooling in a model of regional coronary obstructions, continuous retrograde low pressure coronary sinus perfusion was studied during prolonged global ischemia. Forty-one isolated *in situ,* blood perfused canine heart preparations were divided into four study groups. All hearts had reversible occlusion of the proximal circumflex coronary artery at the onset of 90 min of global ischemia. Group I hearts (n = 11) then received 250 ml of a crystalloid hyperkalemic (25mM) cardioplegic solution at 4°C via aortic root perfusion. Group II (n = 10) received the same initial cardioplegic infusion and in addition had uniform topical myocardial cooling with cold saline to maintain myocardial temperature at or below 16°C. Using a balloon tipped catheter, Group III hearts (n= 10) received continuous retrograde coronary sinus perfusion of a crystalloid hyperkalemic (10 mM) solution at 15 mm Hg pressure and 15°C throughout the ischemic period, while Group IV (n= 10) had continuous coronary sinus perfusion with a similarly cooled (15 °C), oxygenated and hyperkalemic (10 mM) perfluorocarbon solution at 15 mm Hg perfusion pressure. Following 90 min of ischemia, the circumflex obstruction was released and all hearts were reperfused at 80 mm Hg for 60 min at 37 °C. Left ventricular (LV) developed pressure (DP, maximum positive dP/dt and end diastolic pressure (EDP) were measured isovolumically before ischemia and every 15 min during reflow. Intramyocardial gas tensions (PmO_2 and $PmCO_2$) were measured continuously during ischemia and reperfusion both in the obstructed circumflex and unobstructed left anterior descending regions. Myocardial water content (%H_2O) was determined in all hearts after reperfusion. Results below are expressed as mean ± SEM (*$p < 0.05$).

GROUP	Mean Myocardial Temp During Ischemia (°C)	Circumflex Area PmO_2 At End Ischemia (mmHg)	DP After 60 min Reperf (% control)	EDP After 60 min Reperf (mmHg)	% H_2O
I	22.9 ± .6*	13 ± 3	36 ± 4	16 ± 3	80.0 ± 0.6
II	15.7 ± .5	18 + 4	41 ± 5	17 ± 2	80.4 ± 0.3
III	17.4 ± .4	39 ± 6*	78 ± 5*	12 ± 2*	81.1 ± 1.0
IV	16.0 ± .4	49 ± 6*	73 ± 5*	13 ± 1*	80.3 ± 0.4

Not only did intramyocardial oxygen and carbon dioxide tensions remain at or near normal levels throughout ischemis, even in the area of the obstructed coronary artery, but LV functional recovery was significantly better in the two groups receiving retrograde coronary sinus perfusion.

In addition, LVEDP remained low and there was no increase in myocardial edema formation in these same hearts. These results clearly demonstrate enhanced myocardial protection with low pressure coronary sinus perfusion during prolonged ischemia in hearts with severe coronary arterial obstructions.

*By Invitation

15. Prevention of Reperfusion Injury by Verapamil After Hypothermic Cardioplegia

W.R. ERIC JAMIESON, HARTMUT HENNING*,*

HILTON LING, CHERYL A. DA VIES*,*

BRENDA UHRYNUK and DONALD M. LYSTER**

Vancouver, British Columbia

Sponsored by: G. FRANK O. TYERS,

Vancouver, British Columbia

The effect of Verapamil (V) on myocardial protection was assessed in two techniques of administration and compared to hypothermic cardioplegia (HC). Three groups of seven canine experiments were compared-Group I (HC-370m Osm/L; K 25 meq/L; Ca 2 meq/L; Mg 3 meq/L; Group II HC with V (HC-V Img/L as intermittent infusion); and Group III-HC with single dose V (HC-SDV 0.5 mg in 500 cc infusion 20 mins prior to reperfusion). Myocardial temperatures were maintained between 15-19°C for two hours of ischemia during cardiopulmonary bypass (CPB). Left ventricular global function was measured by ultrasound as LV internal diameter shortening (AL) and velocity of fiber shortening (Vcf), and regional function as LV wall thickening (%Ah). At 30 min after CPB AL declined by 14 ± 8% in I (HC) and increased by 23 ± 21% in II (HC-V) and by 15 ± 14% in III (HC-SDV), (I vs II and III $p<0.01$, analysis of variance, II vs III pNS). The mean Vcf declined by 14.7 ± 12<% in I, but increased by 25.7 ± 26 in II and by 21 ± 16 in III (I vs II and III $p<0.001$; II vs III pNS). The mean Ah decreased by 41 ± 6% (I), 11 ± 6% (II), and 15 ± 8% (III) (I vs II and III $p<0.001$; II vs III pNS). Systemic vascular resistances were not significantly changed after CPB and after V and not different between groups (pNS). We conclude that Verapamil combined with HC provides protection of LV function in the early postop period, both by intermittent infusion during CPB and single bolus infusion at the end of CPB. Our data indicate that LV function impairment is caused by reperfusion after HC rather than during HC. The three methods of protection do not change myocardial perfusion and energy contents during CPB.
*By Invitation

16. Warm-Glutamate Blood Cardioplegic Induction in Inotrope, Intraaortic Balloon Dependent Coronary Patients in Cardiogenic Shock: Initial Experience.

ELIOT R. ROSENKRANZ, GERALD D. BUCKBERG,*

HILLEL LAKS and DONALD G. MULDER

Los Angeles, California

This report reviews the initial clinical application of our experimental studies of warm (37°C)-glutamate blood cardioplegic induction in ischemically damaged hearts. Over the past 14 months, 23 consecutive coronary patients requiring pre-operative intraaortic balloon and inotropic drug

support for cardiogenic shock underwent emergency operation. Twelve patients received warm-glutamate blood cardioplegic induction during the first 5 minutes of aortic clamping before multidose cold (4°C) glutamate blood cardioplegia; 11 patients received standard multidose cold blood cardioplegia without glutamate.

All patients had comparably depressed preoperative LV performance despite maximal inotropic and balloon support and showed evidence of extending myocardial infarction. They did not differ in the number of grafts placed (3.7 ± 0.2), associated valve and aneurysm procedures (7 patients) or cross-clamp time (89 ± 6 min). All patients received warm blood cardioplegic reperfusion before aortic unclamping.

The overall mortality was 9%(2/23); both patients who died received cold blood cardioplegia without glutamate. In addition to lower mortality, patients receiving warm glutamate blood cardioplegia exhibited better hemodynamics, allowing earlier discontinuation of inotropic and balloon support.

Blood Cardioplegic Induction	CI** (1/min/m^2)	LAP (mmHg)	IABP (days)	Inotrope (days)	Deaths (%)
Warm Glutamate (n = 12)	2.6 ± 0.1	16 ± 1*	1.2 ± 0.2*	1.3 ± 0.5	0
Cold (n = 11)	2.4 ± 0.2	22 ± 1	3.6 ± 0.5	2.7 ± 0.8	18

*p<0.05 SEM, **Recovery Room

Management principles evolving from this early experience include 1) warm blood cardioplegic induction; 2) glutamate enrichment; 3) meticulous attention to cardioplegic distribution and grafting sequence and 4) warm cardioplegic reperfusion before unclamping. Hopefully, further application of these techniques will improve results in these extremely high risk coronary patients requiring operation.

*By Invitation

17. Creatine Phosphate: An Additive Myocardial Protective Agent in Cardioplegia

LARY A. ROBINSON, DAVID J. HEARSE**

*and MARK V. BRAIMBRIDGE**

Durham, North Carolina; London, England

Sponsored by: ROBERT H. JONES, Durham, North Carolina

The potential for enhanced myocardial protection by the addition of high energy phosphates to cardioplegic solutions was investigated using the isolated working rat heart model of cardiopulmonary bypass and ischemic cardiac arrest. Creatine phosphate (CP) was chosen for evaluation as an additional protective agent in the St. Thomas' Cardioplegic Solution (NaCl 110 mM/l, KCl 16 mM/l, $CaCl_2$ 1.2 mM/l, $MgCl_2$ 16 mM/l, $NaHCO_3$ 10 mM/l, pH 7.8). Dose response studies (CP 0-50 mM/l) revealed 10 mM/1 as the optimal additive concentration, such that its inclusion in the St. Thomas' Cardioplegic Solution during a 3 min period of pre-ischemic cardioplegic infusion improved the recovery of aortic flow (AF) and cardiac output (CO) upon reperfusion after a 40 min period of normothermic (37°C) ischemic arrest from 21.1 ± 5.4% and 32.8 ± 4.6% in the CP-free control group to 82.5 ± 3.7% and 82.6 ± 4.2% ($p<0.001$) respectively. Creatine kinase (CK) leakage was reduced by 68.7% ($p<0.001$) in the CP group. With hypothermic

(20°C) ischemia (240 min) and multidose (every 30 min) car-dioplegia, recoveries of AF and CO were improved from 33.1 ± 8.3% and 42.2 ± 7.7% in the CP-free control group to 77.9 ± 4.2% and 79.6 ± 4.3% ($p<0.001$) respectively in the drug group. In addition to improving various indices of function and decreasing CK release, CP reduced reperfusion arrhythmias. The drug significantly decreased the time between cross clamp removal and the return of regular rhythm and also completely obviated the need for electrical defibrillation. In studies with ^{51}Cr-EDTA (extracellular space marker), disappearance of CP from the cardioplegic solution during its stasis in the heart was assessed. Upon reperfusion, two thirds of the infused dose appeared unchanged in the coronary effluent; the remainder was either degraded or accumulated by the myocardium. In conclusion, despite its alleged inability to enter the myocardial cell, exogenous CP exerts potent protective and antiarrhythmic effects when added to St. Thomas' Cardioplegic Solution. Although the mechanism of action remains to be elucidated, it may involve binding or uptake of the drug.

*By Invitation

18. First Report of On-Line Monitoring of Intramyocardial pH in Man

SHUKRI F. KHURI, MIGUEL JOSE*,*

NINA S. BRAUNWALD and ERNEST M. BARSAMIAN

Boston, Massachusetts

Except for myocardial temperature (T) measurement, there has not been an on-line intraoperative technique to monitor adequacy of myocardial preservation in man. This report comprises the first 20 patients undergoing cardiopulmonary bypass (CPB) in whom a new glass electrode system was utilized intraoperatively to measure septal intramyocardial pH (MpH) and T before and throughout CPB. All patients had LAD disease and underwent bypass grafting of the LAD amongst other procedures. Periods of aortic clamping (AC) ranged from 32-130 minutes. The myocardium was protected with a cold K+ cardioplegic solution (CKCP) having a pH of 7.8. There were no operative deaths. Intraoperative need for inotropic support or intraaortic balloon, and postoperative ECG, enzymatic or radionuclide evidence of septal ischemic injury, constituted the criteria according to which the patients were divided into Group I (n = 14) with optimal protection, and Group II (n = 6) with suboptimal protection. Results are listed below as means ±SEM.

	Before CPB		On CPB Prior to AC		Integrated Mean Values During AC		At End of CPB	
	MpH	**T**	**MpH**	**T**	**MpH**	**T**	**MpH**	**T**
Group I	**6.84** ± 0.3	35.4 ± 0.6	7.04 ± 0.2	25.7 ± 0.7	7.29 ± 0.07	16.4 ± 0.6	7.01 ± 0.03	37.5 ± 3.3
Group II	6.81 ± 0.2	36.2 ± 1	24.2 ± 0.1	24.2 ± 1.5	6.85 ± 0.1	14.5 ± 1.6	6.89 ± 0.1	38.3 ± 1.0
p*	N.S.	N.S.	<0.02	N.S.	<0.005	N.S.	N.S.	N.S.

*Group I vs. Group II

Changes in T were not significantly different in both groups. A significant *rise* in MpH occurred with the administration of CKCP in every patient ($P<0.0001$), but it was significantly more marked in Group I than Group II ($P<0.005$). All 9 patients with an integrated mean MpH during AC>7.2 belonged to Group I, while 6 of 11 patients (54%) with an integrated mean MpH<7.2 belonged to Group II ($P<0.001$). These data confirm our recent animal experiments and prompt us to conclude that: (1) On-line intraoperative monitoring of MpH in patients is feasible, practical, and reproducible. (2) Cold K + cardioplegia results in a marked rise in MpH, suggesting

that a hallmark of intraoperative myocardial preservation may be the avoidance of tissue acidosis. (3) Monitoring both MpH and T is a significantly more sensitive indicator of the adequacy of myocardial preservation than monitoring T alone.
*By Invitation

TUESDAY MORNING, April 26, 1983

6:45-8:15 a.m. Simultaneous Breakfast Sessions**

A. Interventional Therapy (Thrombolytic/Angioplasty) for Acute Myocardial Ischemia and Infarction

MODERATOR: Ellis L. Jones, Atlanta, Georgia

B. Surgical Problems of Aortic Arch

MODERATOR: E. Stanley Crawford, Houston, Texas

C. Controversies in the Surgical Management of Advanced Non-Small Cell and Limited Small Cell Carcinoma of the Lung

MODERATOR: E. Carmack Holmes, Los Angeles, California

8:30 a.m. Scientific Session
Grand Ballroom

19. Immediate Coronary Artery Re-Perfusion for Evolving Myocardial Infarction

STEVEN J. PHILLIPS,*

CHAMNAHN KONGTAHWORN, JAMES R. SKINNER**

*and ROBERT H. ZEFF**

Des Moines, Iowa

Sponsored by: RALPH A. DORNER, Des Moines, Iowa

Between 1975 and 1981, 339 patients underwent emergency coronary artery re-perfusion (CR) for treatment of evolving myocardial infarction (EMI). Group I (181 patients) had saphenous vein bypass graft (SVBG). Group II (112 patients) had CR with intercoronary streptokinase (SK). Group III (46 patients) had re-perfusion with a combination of intercoronary SK and percutaneous transluminal coronary angioplasty (PTCA). Twenty Group II patients and one Group III patient had emergency SVBG as SK and PTCA were unsuccessful and significant myocardium remained at risk due to residual stenosis in the EMI artery. Group I had successful thrombectomy of the infarcted artery in 79% of cases, and 17% of these patients had no observable lesion on restudy. There were eight early, and two late deaths in the surgical patients. Group II had four deaths, and Group III patients had no deaths. Patients with an EMI should be treated via re-perfusion of the EMI vessel by one of the above mentioned techniques. With single vessel involvement, streptokinase lysis of the intercoronary thrombosis should be attempted. If this is successful and there is a significant residual stenotic lesion, it should undergo balloon angioplasty at that time. If PTCA is unsuccessful, then SVBG should be done. When significant multiple vessel disease exists in conjunction with an acute myocardial infarction, the patient should have emergency saphenous vein bypass graft as the treatment of choice.

*By Invitation

**No advance registration. Attendance by ticket only. Tickets must be purchased at registration desk by 2:00 p.m. on Monday, April 26. Price of ticket covers attendance at session and breakfast.

20. Combined Carotid Coronary Surgery - When is it Necessary

ELLIS L. JONES, RICHARD E. MICHALIK,*

JOE M. GRAVER, ROBERT A. GUYTON,*

CHARLES R. HATCHER, JR. and

*NORMAN REICHWALD**

Atlanta, Georgia

Numerous centers have reported that simultaneous carotid-coronary surgery (C/CS) can be performed safely; few have discussed whether it is always necessary. To answer this question three groups of patients were analyzed: Group I (N = 111) having C/CS, Group II (N = 51) having postop stroke (POS) after isolated coronary artery bypass (CAB) and Group III (N = 169) having CAB alone, but with preop findings of either asymptomatic cervical bruit (ACB), positive ischemic neurological history (NH) or prior carotid endartectomy (CE). There was no significant difference between Group I patients and a control group having CAB alone with regard to anginal pattern, vessel disease, ventricular functin, grafts performed or peri-operative complications (hospital mortality 2.7% vs. 0, inotropic need 8% vs 7%,IABP 1.8% vs. 1.5%, and POS 2% vs. 0%, respectively). The incidence of POS in Group II was 0.9% (51/5676). Group II patients were characterized by: diabetes (27%), + neurologic history (20%), carotid bruit (20%) and diseased ascending aorta (33%). There were no differences in extra-cranial arteriographic findings (ECAF) for patients in Group III with ACB, + NH (TIA/stroke) or prior CE. In patients with ACB (N = 60), ECAF were normal in 38% and revealed significant unilateral or bilateral carotid stenosis in 41%. POS rate in patients with ACB, + NH and CE (Group III) was 3.3%, 8.6% and 5.1%, respectfully compared to 2% in patients having C/CS (p = NS). This study suggests that although C/CS can be performed safely under ideal conditions, the combined approach is not mandatory, and in patients with unstable angina the ACB or positive NH can be ignored and CAB performed alone with acceptable incidence of POS. It is probable that POS after isolated CAB is most often caused by conditions other than known or occult carotid vascular obstruction.

*By Invitation

21. Coronary Artery Bypass Grafting in Patients with Ejection Fractions Below 40%: Early and Late Results in 466 Patients

MARK S. HOCHBERG. VICTOR PARSONNET,*

ISAAC GIELCHINSKY, RONALD M. ABEL and

S. *MANSOOR HUSSAIN**

Newark, New Jersey

The outcome of patients undergoing coronary artery bypass grafting with preoperative ejection fractions (E.F.) below 40% was evaluated to determine if a specific level of ventricular dysfunction

resulted in an unacceptably low short-term or long-term result. Ejection fractions were broken down into groups of five percentage points starting with 35-39% and progressing down to 10-14%. In evaluating the six ejection fraction groupings between 10 and 39%, there was no significant difference between the groups in the number of vessels bypassed, the number of previous myocardial infarctions, age, preoperative NYHA class, or the length of time of intra-operative ischemic arrest. From 1976 through 1981, 466 patients were distributed among these groups, all having ejection fractions below 40% (average 30 ± .32).

There was a significant difference (p= .001) in the hospital and long-term survival of patients with pre-operative ejection fractions from 20-39% (425 patients) as compared to those with pre-operative ejection fractions from 10-19% (41 patients). Hospital survival was 89% for patients with ejection fractions from 20%-39%, but only 63% for patients with ejection fractions below 20%. At three years, patients with ejection fractions of 20-39% had an average survival of 60% as compared to an average survival of 15% for those with ejection fractions below 20%. The pre-operative LVEDP did not significantly predict the survival except at the lowest ejection fraction (10-14%). NYHA class decreased an average of 3.00 to 1.25 in surviving cases following CABG.

It is concluded that ejection fraction is an excellent predictor of short-term and long-term survival following coronary artery bypass grafting. Patients with ejection fractions of 10-14% and 15-19% have a significantly reduced short- and long-term survival as compared to patients with ejection fractions ‰¥20%.

*By Invitation

22. Technique and Results of Operative Transluminal Angioplasty in 60 Consecutive Patients

NOEL MILLS, JOHN L. OCHSNER, DANIEL P. DOYLE and WILLIAM P. KALCHOFF**

New Orleans, Louisiana; Montreal, Quebec;

Houston, Texas

Sixty consecutive patients with distal multivessel coronary artery disease had attempted operative transluminal angioplasty at the time of coronary bypass surgery. Lesions chosen for angioplasty were those in coronary arteries that otherwise would not be bypassed because of size and/or location. A guidewire-tipped catheter with a 2 mm. balloon was found to be most satisfactory of the two devices used. Seventy lesions in 60 patients had attempted dilatation. Thirty-eight lesions were in primary coronary arteries with distal disease, 16 lesions were obstructing flow to branches not large enough for grafting, and 16 lesions were tandem lesions that otherwise would not warrant two grafts. The distal left anterior descending lesion was the most common attempted (55%). A dilatation was classified "successful" when a 1.5 or 2 mm. dilator could be passed across the lesion postdilatation. This was achieved with 58 lesions (83%). Ten unsuccessful dilatations occurred due to inability to traverse the lesions with the catheter.

Postoperative angiography performed in 21 patients to study 23 lesions was carried out 10 days to 6 months postoperatively. In 16 of 21 successfully dilated lesions (76%), the stenoses were completely alleviated. Three lesions were found unimproved and in one the coronary artery was occluded distally. Two bypass grafts were closed involving two lesions with extensive dilatation. One patient suffered an asymptomatic perioperative myocardial infarction and there were no deaths in this series. Calcification of lesions did not bear upon operative or late angiographic success, whereas length of the lesion was indirectly proportional to a successful dilatation. Operative dilatation of short coronary distal lesions is safe, has a high percentage of success, and offers a larger distal runoff for coronary bypass grafts. Areas of normal coronary arteries should not be dilated. Careful attention to detail, and proper selection of lesions to be dilated is required. The technique should be used only to dilate arteries that otherwise would not accept a bypass graft.

10:00 a.m. Intermission - Visit Exhibits - Lower Level (Galleria) - Complimentary Coffee

*By Invitation

10:45 a.m. Scientific Session
Grand Ballroom

23. The Use of Transluminal Coronary Angioplasty in the Patient with Prior Bypass Surgery

GERALD DORROS, W. DUDLEY JOHNSON,*

*ALFRED TECTOR and LYNNE JANKE**

Milwaukee, Wisconsin

Transluminal coronary angioplasty (TCA) has proved successful in treating patients (pts) with single vessel disease (SVD). TCA has been used in prior coronary bypass surgery (CABG) pts with a saphenous vein graft (SVG) and/or a native arterial (NA) stenosis. During 47 months, 50 pts (37 males, 13 females) underwent TCA with 84 attempts and 73 (87%) primary successes (PS). A PS was determined by a ‰¥20% decrease in the percent diameter stenoses coupled with an improved clinical response. 43 pts had 1 prior CABG; and 7 pts had 2 or more prior CABG's. SVD was present in 5 pts (10%), and multivessel disease (MVD) in 45 pts (90%). A SVG stenosis was dilated in 33 cases with a PS achieved in 27 (82%): with 20/25 PS's at an anastomotic site (80%) and 7/8 PS's in the body of the graft (88%). A NA stenosis was dilated in 51 cases with a PS achieved in 46 (90%) with 12/15 PS's in the left anterior descending (80%); 8/13 (62%), in the circumflex; 21/23 (91%), in the right coronary; and 5/5 (100%), in the left main. Complications included: emergency CABG in 1 pt (2.0%); a myocardial infarction (MI), in 2 pts (4.0%); and no related mortalities. Two pts died of arrhythmias, awaiting EL CABG. There were 11 failures with 8 pts having EL CABG, and 3 pts managed medically. There were 39 successful pts of which 15 had restenoses 5 of these had EL CABG, 9 had a second TCA (6 remained well, 3 restenosed and underwent EL CABG). There was one late death and one late MI. A restenosis occurred in 8 NA's (16%) and 10 (30%) SVG's: 5 NA and 4 SVG restenoses underwent a successful second TCA. Thus, there were 29 pts (58%) with late clinical success of which 28 pts (97%) had no or improved angina, and all had improved exercise treadmills. Thus, TCA is technically feasible in selected pts with prior CABG and can result in the avoidance of a subsequent higher risk surgical procedure in the majority of selected pts.
*By Invitation

24. Intracoronary 201-Thallium Scintigraphy - An Immediate Predictor of Salvaged Myocardium Following Intracoronary Thrombolysis

HANS J. KREBBER, JOCHEN SCHOFER*,*

DETLEF MATHEY, RICHARD MONTZ*,*

PETER KALMAR and GEORG RODEWALD**

Hamburg, Federal Republic of Germany

Sponsored by: J. DONALD HILL, San Francisco, California

Since February of 1980, 140 patients having the symptoms of acute myocardial infarction for less than 3 hours, underwent intracoronary lysis (ICL). Thirty-eight patients required early aortocoronary revascularization. Surgery, however, was felt to be indicated only when ICL was successful and myocardium was salvaged. As left ventricular angiography proved unreliable in the assessment of the viability of the myocardium in the acute stage, we therefore from March of 1981, obtained intracoronary 201-Thallium scintigrams (i.e. TL 201) in 23 patients, before and after ICL. Patients who showed significant reduction (>50%) in their initial 201 TL defect (n = 12) were considered ideal candidates for surgery (Group 1). Patients with poor or unimproved 102 TL uptake after successful ICL (n = 6) were treated medically (Group 2), as were patients whose ICL had been unsuccessful (n = 5, Group 3). In order to validate this new approach we compared the change in the regional wall motion of the "infarcted area" as shown in the acute and follow-up left ventricular angiograms in all 3 groups. In the acute stage the mean regional EF was 20% in Group 1, 19% in Group 2 and 20% in Group 3. Only in Group 1 was there a significant increase in regional EF to a mean of 51%. The mean EF obtained at follow-up in Groups 2 and 3 was 17%.

Conclusion: 201-Thallium scintigraphy is a valuable predictor of the salvagability of myocardium immediately following ICL and, has been to date, the most valuable tool in assessing those patients suitable for early coronary revascularization.

11:30 a.m. Address by Honored Speaker
ALAIN CARPENTIER, Paris, France Valve Surgery: "The French Correction"

12:15 p.m. Adjourn for Lunch - Visit Exhibits

12:15 p.m. Cardiothoracic Residents' Luncheon
Dusseldorf & Lisbon Rooms

*By Invitation

TUESDAY AFTERNOON, April 26, 1983

2:00 p.m. Forum Session
Grand Ballroom

25. Atypical Carcinoids of the Lung

RAO R. PALADUGU, LOUIS F. DECARO*, RAYMOND L. TEPLITZ*, HYUN Y. PAK*, LOVISATTI LEONARDO* and JOHN R. BENFIELD*
Duarte, California; Verona, Italy

Biological behavior and morphology of pulmonary carcinoids is not constant; management is controversial. We shall define characteristics of car-cinoid (C) and atypical carcinoid (AC) tumors, based on new knowledge that increasing nuclear DNA content correlates well with progressive steps of lung carcinogenesis, and on electron microscopic comparative studies.

Findings in 24 patients (17 or 71% C; 7 or 29% AC) were reviewed:

Microscopic similarities between AC and small (oat) cell cancers were observed.

Nuclear DNA measurements by image analysis were made on 2609 cells, including 216 normal bronchial cells, 1645 oat cells, 399 cells from C and 344 AC cells. Normal bronchial cells were diploid (n = 1.15 ± 0.24) as were C cells (n = 1.12 ± 0.19). AC cells had 1.27 ± 0.73 DNA units

and oat cells were essentially tetraploid (n = 1.94 ± 0.73). The DNA content of AC was greater than C ($p<0.001$). The DNA content of AC was less than small undifferentiated (oat) cell cancers ($p<0.001$).

We conclude that AC behaves like common bronchogenic cancers, and the morphology of AC has features of oat cell cancers. Nuclear DNA content is a clinically relevant measurement with the potential to help differentiate AC from other carcinoids and from oat cell cancers based on preoperative percutaneous needle aspirates.

*By Invitation

26. Neovascularity of a Tracheal Prosthesis/Tissue Complex

RONALD J. NELSON, LISE GOLDBERG, RODNEY A. WHITE*, EDWIN SHORS* and FRANK M. HIROSE**
Torrance, California; Seattle, Washington

Permanent bioincorporation of a microporous tracheal prosthesis will require a stable blood supply to connective tissue supporting an epithelial surface. In experience with over 80 tracheal implants in dogs we have observed that 1) end-on ingrowth and epithelialization does not occur in the absence of lateral ingrowth, 2) epithelialization is marked by the appearance of a subepithelial network of vessels, and 3) this process must be well advanced by 6-8 wks. for long term stability. These observations were extended using microangiography to delineate the blood supply of the prosthesis/tissue complex.

Six implants of bioelectric polyurethane with 10% Gentamicin (3 cm. length, 2 cm dia., 1-1.25 mm. wall thickness, 60-120 μmicropore dia.) were interposed in the dog thoracic trachea and wrapped with an omental pedicle. The aorta was perfused with a barium suspension at elective sacrifice between 10 wks. and 22 mos. Radiographs of specimens were correlated with bronchoscopic, gross and histopathologic findings.

Neovascularity to the prosthesis/tissue complex can be described in three categories: outer capsule (OC), prosthetic wall (PW), and inner lining (IL). OC vessels were oriented circumferentially immediately adjacent to the prosthetic wall and extended up to the length of 180° cross-sections. They resembled arteries up to 75 μ dia. on microscopy and appeared to originate in the omentum with connections developing to the bronchial circulation. PW vessels up to 75 μdia. with thin muscular walls were noted to traverse the porous wall. The IL had subepithelial longitudinal vessels up to 120 μdia. linking tracheal and prosthetic networks across the anastomoses. We conclude the omentum provides an immediate blood supply oriented circumferentially adjacent to the wall and a base for early connective tissue ingrowth. Epithelialization occurs as early as 3 wks. on this favorable bed accompanied by vascular connections to the existing lamina propria tracheal vessels. This dual organization is probably important to long term stability.

*By Invitation

27. Single Lung Transplantation with Cyclosporine Immutio-suppression

STEPHAN L. KAMHOLZ, FRANK J. VEITH, FRED P. MOLLENKOPF*, KENNETH L. PINSKER*, RONALD R. KALEYA*, ALLEN J. NORIN*, MARVIN L. GLIEDMAN*. EUGENE E. EMESON*, AVRAHAM D. MERAV*, RICHARD BRODMAN*, STANLEY C. FELL and CHERYL M. MONTEFUSCO**
New York, New York

Cyclosporine (Cy), a potent new immunosuppressive agent, was used (alone or in combination with other drugs) in 28 canine single lung allograft (SLA) recipients. Mean recipient survival with good SLA function was 144 days with Cy and far exceeded that obtained in previous SLA recipients treated with standard immunosuppression (11 days). The results of these experiments showed that: (1) 20% of the recipient animals exhibited no evidence of rejection whatsoever, one of these

animals survives over 2½ years with normal allograft function and morphology; (2) 75%of the animals exhibited some evidence of rejection that was easily reversed in 76% of instances with corticosteroids. Over 50% of these animals exhibited good lung allograft function 6 months or more after surgery. (3) Rejection in these animals was characterized by more prominent vascular involvement (demonstrated on open lung biopsy and by decreased blood flow on nuclear perfusion scan) than that which occurred in SLA recipients treated with standard immunosuppression. (4) Diagnosis of rejection in Cy treated lung allograft recipients was made by microscopic analysis of sputum and analysis of the cellular content of bronchoalveolar lavage samples coupled with deterioration of pulmonary hemodynamics and decreased perfusion on ^{99}Tc lung scan. (5) Successful and complete healing without stenosis of the bronchial anastomosis occurred in 80% of the animals studied.

These findings have been corroborated in two human Cy treated SLA recipients, one of whom survives 4 weeks after transplantation and 2 weeks after contralateral pneumonectomy. This overall experience indicates that Cy, although not a perfect immunosuppressive agent, increases the likelihood of success with therapeutic single lung transplantation.

*By Invitation

Scientific Session - Grand Ballroom

28. Powerful but Limited Immune Suppression for Cardiac Transplantation with Cyclosporin A and Low Dose Steroid

HARTLEY P. GRIFFITH, ROBERT L. HARDESTY* and HENRY T. BAHNSON*
Pittsburgh, Pennsylvania

Seventeen of thirty patients have survived a new trial of cardiac transplantation to evaluate Cyclosporin A and prednisone (15 mg.) alone for immune suppression (range 1 to 19 months). The low dose of prednisone was chosen to lessen steroid morbidity, and other immunosuppressive drugs (ATG, azathioprine) were avoided to reduce the risk of infection and lymphoma. Rejection was graded as moderate with focal myocyte necrosis and as severe with general myocyte necrosis. Both moderate and severe rejection were treated with 1 gm. pulses of cortisone, usually for 3 to 5 days without alteration of the prednisone dose.

Death occurred in 2 patients with mild rejection (1 stroke, 1 pneumonia); 3 with moderate rejection (2 stroke, 1 abdominal infection); and in all 7 patients with severe rejection (2 patients with acute rejection and 1 with acute rejection and infection less than 5 weeks postoperatively, and 4 with chronic rejection and restrictive cardiomyopathy). Two of the four patients with restrictive cardiomyopathy succumbed from coronary atherosclerosis.

Seventy-seven percent of all patients treated have been judged to have adequate immune suppression. Pulse therapy with cortisone was effective in all but those with general myocyte necrosis. Cardiac catheterization in 9 patients surviving greater than 10 months has documented adequate graft function (cardiac index 2.5, ejection fraction 52%). Infections have been rare and side effects of steroids minimal; one patient died following the development of a polyclonal Epstein-Barr lymphoma.

This trial suggests that Cyclosporin A and prednisone is superior immune suppression for cardiac transplantation, but patients with advanced stages of rejection should be treated with an additional immunosuppressive agent. Subsequent to this analysis, 5 patients have been treated during rejection episodes with ATG, and in all further myocyte necrosis was eliminated.

2:45 p.m. Intermission - Visit Exhibits - Lower Level (Galleria) - Complimentary Coffee

*By Invitation

3:30 p.m. Scientific Session
Grand Ballroom

29. Reoperation on Prosthetic Heart Valves

DAVID G. HUSEBYE, JAMES R. PLUTH,*
JEFFREY M. PIEHLER, HARTZELL V. SCHAFF*,*
THOMAS A. ORSZULAK, FRANCISCO J. PUGA**
and GORDON K. DANIELSON
Rochester, Minnesota

Five-hundred-and-twenty-nine patients underwent reoperation for prosthetic valve dysfunction during the years 1961-1980. Included were 391 patients with aortic valves, 107 with mitral valves, 12 with tricuspid and 19 with double valve reoperations. Surgery was elective in 235, urgent in 263, and emergent in 31. At the time of reoperation, 82 patients were Class I (NYHA), 156 Class II, 200 Class III, and 91 Class IV. Ten percent of patients with heterografts or disk valves required emergency reoperation as compared to 5.4%with ball valves and 0%with homograft valves. Thirty percent of patients with disk valves were Class IV compared to 20% for heterografts, 14% for homografts and 7.5% for ball valves. Twenty-three patients had complications related to sternal reopening including lacerations of the right atrium (5), right ventricle (7), aorta (9), and division of a previous coronary graft in 2. Two deaths resulted from these complications. The risk of reoperation for aortic valve was 1.3% for Class I patients, 1.6% for Class II, 6.3% for Class III and 20.8% for Class IV. For mitral valves, the risk was 4% for Class II, 9.3% for Class III, and 41% for Class IV. Based on urgency of operation, the operative risk for elective aortic valve replacement was 1%, urgent 8%, and emergency 37.5%. Corresponding figures for the mitral valve were 0%, 20%, and 55%. Risk was highest for infective endocarditis and for patients undergoing disc or heterograft valve replacement. At five years following valve reoperation, 75% of the patients were surviving. Survivorship of patients who were Class I or II at the time of second operation was 85% and identical to patients undergoing initial valve replacement. Survivorship for Class III and IV patients was significantly decreased. This study indicates that if valve repair or replacement is necessary, it should be done early and electively before functional cardiac deterioration occurs.
*By Invitation

30. Mitral Reconstructive Surgery: A Series of 130 Consecutive Cases

*ARRIGO LESSANA *, TU TRAN VIET*,*
FRANCOIS ADES, SAID MOSTEFA KARA*,*
*ABDERRAHMAN AMEUR *, FRANCOISE HERREMAN**
*and MICHEL DEGEORGES**
Paris, France and Oran, Algeria
Sponsored by: R.W.M. PRATER, Bronx, New York

Between January 1975 and January 1982, 130 patients (pts) underwent mitral valvuloplasty (MV) for pure or predominant mitral insufficiency (MI). Mean age at operation was 30 ± 17 years. 25 pts were under 15 years of age. MI was mainly (111/130) due to rheumatic disease. 59 pts (45,4%) had another valve disease which necessitated a surgical correction (tricuspid 36, aortic 23).

Surgical technique for MV varied according to the lesions. 2 pts only had an isolated annuloplasty (AN). The remaining 128 (with 125 AN) can be divided in 3 groups. Group I (35 pts) in which MI was due to restricted amplitude of valve motion, was treated by mobilisation of the leaflets by resection of basal chordae, with or without fenestration of the chordae and/or commissurotomy. In group II (48 pts) in which MI was due to chordal elongation and valve prolapse, correction of MI consisted in chordal shortening, and/or partial resection of the prolapsed leaflet. In type III (45 pts), both mechanisms were responsible for MI and both types of MV were associated according to the lesions.

Three pts died in the first month after surgery (2,3%). 5 pts are lost to follow-up. Mean follow-up period for the 122 remaining pts is 38 months ± 27. 7 pts had to be reoperated and 2 of them died. An additional pt died without reoperation. Late mortality was therefore 3/122 (2,5%).

Almost all (117/119) the remaining pts are in class I (105) or II (12) of the NYHA. There is no residual murmur in 29 pts. A trivial systolic murmur is found in 61 pts. In only 6 pts was there a

loud residual systolic murmur. Mean cardio-thoracic ratio decreased from 60,6% ± 7,7 preoperatively to 53,7% ± 6,2 postoperatively ($p<0.001$). Thromboembolic episodes were noted in 4 pts, all of them in atrial fibrillation. Actuarial curves including hospital mortality show a 91,3% survival rate at 7 years, an 87,4% embolus-free rate at 7 years and a 92,5% absence of reoperation at 6 years. There was no significant difference between the results of the groups I, II and III. Although the mean follow-up period of this series is only of 38 months ± 27, these results show that even complex procedures of MV can provide stable functional results, low surgical and late mortality, together with an acceptable rate of reoperations.
*By Invitation

31. The Hancock Conduit: A Dichotomy Between the Late Clinical Results and the Late Catheterization Findings

SCOTT STEWART, PETER HARRIS,*
JAMES MANNING and CHLOE ALEXSON**
Rochester, New York

Eighteen patients received a Hancock valved conduit as part of their corrective operation for complex congenital heart disease between 1974 and 1977. Seventeen patients survived operation. We had concern for the long-term durability of the conduit. Therefore, a postoperative protocol was established to evaluate each patient clinically every 6 to 12 months and, in addition, to perform a follow-up catheterization study at both 1 and 5 years after operation. Fifteen patients have had a good to excellent longterm clinical result while only two have had a poor result.

Each surviving patient has undergone several routine serial follow-up catheterization studies. The results of these studies are in direct contrast to the excellent clinical results. One year after operation the mean trans-conduit gradient had increased from 16 mm Hg. at the time of operation to 25 mm Hg. Two of 16 patients (12%) had a gradient exceeding 50 mm Hg. Five years after operation the mean gradient had increased to 40 mm Hg. and 6 of 15 patients (40%) had a gradient in excess of 50 mm Hg. Each of those patients with a significant conduit gradient were considered to have had a very satisfactory result by clinical evaluation.

Five patients (30% of all those followed 5-8 years) have undergone replacement of an obstructed conduit without mortality and with satisfactory resolution of the gradient. Each of the excised conduits have shown varying degrees of internal fibrous peel formation and valve degeneration.

This experience emphasizes the dichotomy between an apparent good clinical result with the Hancock conduit and the tendency for a significant late pressure gradient to develop across it. Late catheterization studies must be performed in all patients with this conduit (not just in those in whom a pressure gradient is clinically suspected) since obstruction of the conduit does occur in the presence of a clinically good result.
*By Invitation

32. Early Experience with the Ionescu-Shiley Pericardial Xenograft Valve-Accelerated Calcification in Children

*WILLIAM E. WALKER *, DAVID A. OTT*,*
JAMES J. LIVESAY, GEORGE J. REUL*,*
J. MICHAEL DUNCAN and DENTON A. COOLEY*
Houston, Texas

Current selection of a valve prosthesis depends on a choice between the durability of mechanical valves and the low incidence of embolic problems with tissue valves. Accepting the IS valve as suitable for adults, we hoped that it would also be good for children, who have difficulties with anticoagulation and who have shown a tendency to early calcification of porcine valves. This review outlines our disappointing experience in children.

Over the period 1978-1982, 2167 consecutive patients survived placement of 2372 IS valves in the left heart. Of 30 valves implanted in children 16 years of age and younger, 6 (20%) have already required replacement because of severe calcification. There has been no known embolic episode in children, no valve has become infected, and there has been no valve leaflet disruption, a

complication we have seen with three mitral implantations in adults. No teenager over sixteen years of age has developed significant calcification so far, and only two adults have had their prosthesis replaced because of calcification.

While the Ionescu-Shiley valve has good hemodynamics in small sizes, and a low incidence of embolic complications, it appears to have an unacceptably high incidence of calcification in children. We believe this is more frequent in post-pubertal than in pre-pubertal children, and we have seen no calcification in children over sixteen years at the time of implantation, but it is too early to say that this valve is satisfactory in some children, and we again use mechanical valves in childhood.

4:45 p.m. Executive Session (Members Only)
Grand Ballroom

7:00 p.m. President's Reception
Empire Room - Twin Towers Complex

*By Invitation

WEDNESDAY MORNING, April 27, 1983

8:30 a.m. Scientific Session
Grand Ballroom

33. Intrathoracic Transposition of Extrathoracic Skeletal Muscle

PETER C. PAIROLERO, PHILLIP G. ARNOLD* and*

*JEFFREY M. PIEHLER**

Rochester, Minnesota

Sponsored by: DWIGHT C. MCGOON, Rochester, Minnesota

During the past five years, 25 patients (16 male and 9 female) with life-threatening intrathoracic infections were treated by transposing an extrathoracic skeletal muscle into the pleural cavity or mediastinum. Indications for muscle transposition included bronchopleural fistula (BPF) following irradiation and/or infection (11 patients), postpneumonectomy empyema with BPF (8 patients), infected false aneurysm of the heart and great vessels (4 patients), and esophageal perforation (2 patients). Ages ranged from 16 to 79 years with a mean of 57.6 years. Thirty-seven muscles were transposed, including 16 latissimus dorsi, 15 pectoralis major, 3 pectoralis minor, 2 serratus anterior, and 1 rectus abdominis. Eleven patients had multiple muscle transpositions (7 concurrently and 4 staged). Thirty-day operative mortality was 12% (3 patients). Follow-up ranged from 2 to 66 months with a mean of 17 months. Eighteen patients had no further signs or symptoms of the original infection. Infection recurred in 4 patients, resulting in death in 3 (BPF in 2 and tracheoaortic fistula in 1). The fourth patient had a recurrent BPF which was successfully treated with further muscle transposition.

Nineteen patients (76%) were successful long-term survivors. These included 88% of patients (7/8) with postpenumonectomy empyema and BPF, 75% of patients (3/4) with infections of the heart and great vessels, 73% of patients (8/11) with BPF, and 50% (1/2) with perforated esophagus. Three patients eventually died from other causes (myocardial infarction in 2 and recurrent cancer in 1). We conclude that intrathoracic transposition of an extrathoracic skeletal muscle is an excellent method of treatment of persistent, life-threatening intrathoracic infection.

*By Invitation

34. Open Window Thoracostomy in the Management of Post-Pneumonectomy Empyema With or Without Bronchopleural Fistula

FARID M. SHAMJI, ROBERT J. GINSBERG,*

JOEL D. COOPER, ERNEST H. SPRATT,*

MELVYN GOLDBERG, PAUL F. WATERS*,*

RIIVO ILVES, THOMAS R. TODD* and*

E. GRIFFITH PEARSON

Toronto, Ontario

The management of chronic post-pheumonectomy empyema with or without bronchopleural fistula (BPF) continues to plague thoracic surgeons. Total thoracoplasty is successful but mutilating. Large myovascular bundles are useful if the empyema cavity is relatively small. Clagett introduced the concept of open window thoracostomy, daily irrigation with an antiseptic solution and eventual closure of the window as an alternative. This method could be utilized with empyemas associated with BPF only if these fistulae were to close spontaneously or by operative intervention.

We report our experience with thoracostomy window for the mangement of post-pneumonectomy empyema with or without BPF in 31 patients. The pneumonectomies were performed for primary bronchogenic carcinoma (21), inflammatory conditions (9) and invasive, malignant thymoma (1). Empyema with BPF occurred in 24 cases and empyema alone in 7 cases.

In all cases, initial treatment was tube thoracostomy followed by open window thoracostomy by the method of Clagett. Daily irrigations were begun, awaiting bronchopleural fistula closure if present, hoping to attempt eventual closure of the thoracostomy window.

In the 7 patients with empyema alone, 3 completed the total Clagett procedure. In 2 cases, it was successful without re-infection. In 3 patients, death from metastatic disease ensued prior to closure of the window. There are 2 patients alive with the window present.

In those patients with associated BPF, the fistula healed spontaneously in 8, required operative closure in 9, and did not close before their demise from metastatic disease in 7. Of 17 patients with closed fisulae, 5 completed the Clagett procedure, and only 1 was successful. Re-infection occurred in the 4 other attempts, necessitating re-opening of the window. In 5 further patients, the persisting space was closed either by limited thoracoplasty (2) or myovascular bundle (3). The remaining 7 patients are alive up to 7 years with persisting open thoracostomies, and tolerating them well.

We conclude that open thoracostomy window provides adequate drainage and excellent interim treatment of an empyema space. In those patients with associated BPF, spontaneous healing of the fisula can occur (8 of 24). In our hands, successful completion of the Clagett procedure is rare when associated with BPF. However, with open thoracostomy, the residual cavity does reduce markedly in size and allows for the use of a myovascular bundle or a more limited thoracoplasty, to close the space.

*By Invitation

35. Aortic Valve Homografts in the Surgical Treatment of Complex Cardiac Malformations

FRANCIS M. FONTAN, ALAIN CHOUSSAT,*

CLAUDE DEVILLE, CHRISTIAN DOUTREMEPUICH*,*

JOSEPH COUPILLAUD and CARLO VOSA **

Bordeaux, France

From April 1968 to September 1982, the surgical treatment of complex congenital cardiac malformations requiring a conduit for their correction was performed with Aortic Valve Homograft (AVH) or Aortic Valved Homograft Conduits (AVHC), prepared sterilized and preserved in our hospital. Our experience concerns 91 patients (pts) in whom a total of 101 AVH with a diameter ranging from 14 to 26 mm were implanted. Since March 1981, it was possible to match recipient and donor ABO Blood group and Rhesus in 10 pts. The pts ranged in age from 5 months to 37 years (mean 12.1 years). The AVH or the AVHC were implanted according to one of the five following situations:

1. AVHC between Right Ventricle (RV) and Pulmonary Artery (PA) in 32 pts: Pulmonary Atresia 14, extreme Fallot 7, Transposition of the Great Vessels 8, Truncus Arteriosus 1, Double Outlet Right Ventricle 2.
2. AVH between right atrium (RA) and PA in 21 cases of Tricuspid Atresia (TA) and in 19 cases of Single Ventricle.
3. AVH between RA and RV - Outlet Chamber - in 9 cases of T.A. and 2 cases of hypoplastic right heart.
4. AVH in the pulmonary valve site in 4 pts: pulmonary valve agenesia in 2, severe pulmonary valve incompetence in 2.
5. AVH for vena cava valvulation in 12 pts: Inferior vena cava in 11 pts and both venae cavae in one.

There was a hospital mortality of 21 pts (23%) ranging from 0 to 100% and a late mortality of 6 pts (6.6%), in none the death can be related to the AVH. The clinical follow-up of the survivors (1 to 172 months, mean 55.1) evidenced neither dysfunction of the AVH, nor thromboembolism or hemolysis. Control cardiac catheterization was performed postoperatively in 42 pts at a mean delay of 8.1. months and a second control in 5 of them at a mean delay of 62 months. A gradient was found in 10 pts with a AVHC between RV and PA. In 4 of them it was lower than 5 mm Hg and in the others respectively 6, 9, 11, 15, 15 and 30 mm Hg. It was not possible to locate the precise level of the gradient: AVH or proximal or distal anastomosis. These results lead us to conclude that at the present time AVH is the best material for the surgical treatment of complex cardiac congenital malformations requiring valved connexions or valved conduits.

*By Invitation

36. Systemic-Pulmonary Shunts in Infants and Children: Early and Late Results

JOHN J. LAMBERTI, JAMES R. CARLISLE,*

FREDERICK A. LODGE and J. DEANE WALDMAN**

San Diego, California

We reviewed our recent experience with systemic to pulmonary artery (PA) shunts to compare four techniques currently in use. From September 1978 to September 1982, 53 shunts were performed in 41 consecutive patients. Age ranged from 18 hours to 4 years, mean age = 0.66 years,

with 25 patients (25/53=47%) ‰¤1 month at operation. Weight ranged from 1.7 kg to 13.2 kg, mean = 5.7 kg, with 25 patients (25/53 = 47%) ‰¤4.0 kg at operation. There were 14 classical Blalock-Taussig (BT), 5 central polytetrafluoroethylene prosthetic (PTFE), and 34 BT type interposition PTFE shunts (8 end of subclavian artery to end of PTFE [IBT], 26 side of subclavian artery to end of PTFE "Great Ormond Street" [GOBT]; all 34 were distal end of PTFE to side of PA). No direct aorta to PA anastomoses were performed. There was one operative death (1/53= 1.9%) (GOBT) in a 1.7 kg neonate with a patent shunt. Three patients required reoperation for shunt revision (one banding of a GOBT, one kinked but patent IBT, and one patent BT anastomosed to ductal tissue in an interrupted PA patient) and there was one reoperation for conversion of a clotted central shunt to an IBT.

Ten of 53 procedures represented a second palliative procedure 12 to 36 months after initial operation. Initial shunts in these 10 cases were: 4 BT, 4 IBT, 1 central, and 1 GOBT. Two late deaths were possibly shunt related, (1 BT; 1 central). All survivors had shunt patency confirmed by angiography or 2-D echo with Doppler plus clinical criteria. Excellent palliation was afforded by all four types of shunt for 12 to 36 months.

We conclude that the GOBT is the optimal shunt due to low operative risk, ease of performance, predictable patency (100% in this series), non-distortion of PA, and ease of takedown. These advantages far outweigh the minor disadvantage of the need for complete repair or a second shunt at 12-18 months in some patients.

10:00 a.m. Intermission - Visit Exhibits - Lower Level (Galleria) - Complimentary Coffee

*By Invitation

10:45 a.m. Scientific Sessions - Grand Ballroom

37. Intelligence Quotient and Development Following Use of Profound Hypothermia and Circulatory Arrest for the Repair of Congential Heart Defects in Infants and Young Children

CHRISTOPHER LINCOLN, FRANK WELLS*,*

STEPHEN COGHILL and DAVID NAFTEL**

London, England

Sponsored by: JOHN W. KIRKLIN, Birmingham, Alabama

Published works on intelligence quotient (IQ) and development following the use of profound hypothermia and circulatory arrest (PHCA) suggests little or no impairment when used to repair congenital heart defects (CHD) in infants and young children. IQ and other development aspects, cognitive, memory, perceptual, quantitative and verbal, (McCarthy's scale of children's abilities, mean score 100, SD 16) were measured *5* years following surgery which was performed between 1973-1976, in 31 patients using PHCA. These patients were compared with three control groups. (1) 19 patients (pts) with similar defects but using moderate hypothermia and continuous cardiopulmonary bypass (CPB), (2) The siblings of those PHCA pts-16, and (3) Siblings of the CPB pts-14. The hypothermia temperature reached within each technique was closely clustered around 15°C in the PHCA group (GP) and 28°C in the CPB gp. PHCA time ranged from 22-71 minutes. Statistical analysis, T-test, Chi-square test of association and Wilcoxon Test showed the only base-line characteristic difference between the two pt gps in respect of preoperative and age variables, diagnosis, level of cyanosis, oxygen saturation, hemoglobin concentration, age at operation, was weight $p = 0.03$. The mean intelligence score 93 ± 5.7 (mean±SE) of the PHCA pt

gp was significantly lower (p = 0.002) than their siblings 106 ± 4.1. The CPB pts score 98 ± 6.0 was not demonstrably different than their siblings, 96 ± 5.9. Degree of cyanosis, oxygen saturation and hemoglobin concentration was not significantly related to the IQ score in either patient gps. The sibling and pt (PHCA) IQ differences are associated with length of arrest time in verbal p = 0.06, quantitative p = 0.07, and general cognitive p = 0.003 scores. A decrease of 0.53 points per minute of arrest time was estimated for the entire group of 31 pts; or, for the 19 with siblings, for each minute increase in circulatory arrest time the pts dropped 0.69 IQ points below their siblings. These and analysis of other published data do not support the generally accepted view that PHCA can be used entirely without penalty and questions the accepted safe estimates of duration of circulatory arrest of 60 minutes.

*By Invitation

38. Subclavian Flap Repair of Coarctation of the Aorta in Neonates - Realization of Growth Potential

ANTHONY L. MOULTON, JANET E. BURNS*,*

JOEL I. BRENNER and MICHAEL A. BERMAN**

Baltimore, Maryland

Sponsored by: JOSEPH S. MCLAUGHLIN, Baltimore, Maryland

The subclavian flap repair for coarctation of the aorta (CoA) allows potential for growth by utilizing autogenous tissue. Though well-documented in young children, its promise in the tiny neonate warrants further evaluation. Since 8/79, 27 patients have undergone subclavian flap repair. Nineteen were less than 1 month of age and 13 were less than 10 days. Weights ranged from 1.4 to 5 kg (means 3.0 kg). All had associated severe intracardiac defects and were in severe congestive failure. Fourteen received preoperative prostaglandin.

There was 1 intraoperative death among the 6 patients who underwent simultaneous pulmonary artery banding (PAB). One patient with a small VSD and severe mitral stenosis, acidotic and seizuring preop, died in low output 36 hours after repair and PAB. Another premature baby with mid-septal VSD's died from pneumonia and sepsis 2 weeks after CoA repair and PAB. One newborn whose CoA was repaired at 2 days of age underwent emergency open aortic valvotomy at 5 days, had a smooth immediate postop course and died suddenly 1 day after the open procedure. There was 1 late death, 6 months after satisfactory CoA repair, in a baby with a hypoplastic left ventricle.

Twenty-two survivors continue to do well up to 3 years postop. All have normal weight gain, but no patient has a measured arm-to-leg gradient greater than 10 mmHg. In 1 patient the VSD spontaneously closed, and open VSD closure was uneventfully performed in 2 others at 1 and 5 months of age. Another underwent a successful Mustard procedure at 3 months. Five patients have undergone repeat catheterization, and all demonstrated satisfactory growth of the subclavian flap segment of repair. We therefore continue to utilize this technique for the treatment of CoA even in tiny neonates.

*By Invitation

39. Reparative Surgery for Interrupted Aortic Arch

WILLIAM I. NORWOOD, ALDO R. CASTANEDA,

FLORENS VERSTEEGH and THOMAS J. HOUGEN**

Boston, Massachusetts

From January 1975 through September 1982, 24 infants underwent primary or staged repair of interrupted aortic arch (IAA) with ventricular septal defect (VSD). Excluded are 10 infants with associated truncus arteriosus (5), single ventricle (3), Taussig-Bing anomaly (1), or aortic atresia (1). Seven of 24 had Type A and 17 had Type B IAA. Eleven infants, median age 5 days, underwent staged surgery while 13, median age 6 days, underwent primary repair. Palliation was by tube graft interposition (6), subclavian aortic anastomosis (3), left carotid aortic anastomosis (1), and end-to-side aortic anastomosis (1) combined with pulmonary artery banding (8) or early ventricular septal defect closure (3). There were 3/11 (27%) early and 1/8 (13%) late deaths with palliation. Delayed repair at 5 days to 14 months (median 7 months) incurred 3/7 (43%) early and no late deaths. Primary repair consisted of ventricular septal defect closure combined with graft interposition (12) or end-to-side aortic anastomosis (1) with 3/13 (23%) early and no late deaths. Four (17%) patients had or developed severe subaortic stenosis. In 3 with long segment obstruction, a ventricular apico-aortic conduit was placed at 5 and 7 days and at 15 months with no mortality. The other had resection of fibrous subaortic stenosis at age 3 years. Of note, 9 (38%) infants had perioperative hypocalcemia and of these, 3 had DiGeorge's syndrome diagnosed by absence of T-cells.

Nine of 14 survivors had hemodynamic evaluation by catheterization 1 to 3 years following repair. None had residual VSD or pressure gradients between the ascending and thoracic aorta. Six had subaortic stenosis; 4 mild (gradient < 20 mm Hg) and 2 severe (gradient > 70 mm Hg) requiring surgery.

Results of surgery in neonates with interrupted aortic arch continue to improve. Essential in management is an awareness that subaortic stenosis and hypocalcemia may be accompaniments of this anomaly. Based on these data, we prefer primary repair for interrupted aortic arch with VSD.

*By Invitation

40. Individualized Surgical Management of Complete Atrioventricular Canal

WILLIS H. WILLIAMS, ROBERT A. GUYTON*,*

RICHARD E. MICHALIK, ELLIS L. JONES,*

KYOO H. RHEE, WILLIAM H. PLAUTH, JR.**

and CHARLES R. HATCHER, JR.

Atlanta, Georgia

In a five year interval (10/1/77-9/31/82), 51 children with complete atrioventricular canal required operation. Group A (31 children) underwent correction; Group B (20 children) received surgical palliation.

Group A ranged in age from 4 months to 14.6 years (mean 4.2 years), 14 being less than one year old. Weights ranged from 4.1 kg to 39 kg (mean 13.6 kg), 14 weighing less than 10 kg. Ten had undergone previous surgical palliation (7 by pulmonary arterial band; 3 by systemic-to-pulmonary arterial shunt). There were no early deaths and one late death. One infant required mitral

valve replacement at total correction; two required subsequent mitral valve replacement, successful in both cases.

Group B ranged in age from 8 days to 4.5 years (mean 8.4 months), 17 being less than one year old. Weights ranged from 2.5 kg to 11.2 kg (mean 4.8 kg), 19 weighing less than 10 kg. Operations included pulmonary arterial banding in 14 and shunt creation in 5. One child underwent pericardial enlargement of the right ventricular outflow tract without closure of VSD to promote growth of small pulmonary arteries. Subsequent correction was successful. One death occurred five days after operation in an infant with unrecognized coarctation. One late death occurred several months after the creation of a second shunt in a child with severe associated tetralogy of Fallot.

Of the 51 children, 48 (94%) are alive now. Postoperative cardiac catheterizations and clinical courses will be described.

Even though universal primary total correction of complete atrioventricular canal in infancy is now widely advised, we believe the outcome in these 51 patients managed by selective palliation and/or total correction supports individualized choice of initial operation based upon clinical condition, weight, pathophysiology, associated anomalies, and intracardiac anatomic details.

12:15 p.m. Adjourn for Lunch - Visit Exhibits

*By Invitation

WEDNESDAY AFTERNOON, April 27, 1983

2:00 p.m. Scientific Sessions
Grand Ballroom

41. Complement and the Possibly Damaging Effects of Cardio-pulmonary Bypass

JAMES K. KIRKLIN, EUGENE H. BLACKSTONE*,*
JOHN W. KIRKLIN, DENNIS E. CHENOWETH,*
*ALBERTO. PACIFICO and STEVE WESTABY**
Birmingham, Alabama; LaJolla, California; London, England

The existence of a damaging effect from cardiopulmonary bypass (CPB) *per se,* especially in infants, and its possible mechanisms and prevention remain controversial. Therefore, a prospective clinical study was made of the blood levels of C3a (a product of complement activation), the usual clinical laboratory tests, and a number of clinical events which were carefully recorded and categorized for severity, in all patients undergoing open (n = 116) and closed (n = 12) cardiac operations on two surgical services in a 6 week period. C3a levels were normal (160-220 ng/ml) in the closed cases, and in the open cases were 1170 ng/ml (± 660-2050, p for difference from closed cases <0.0001), 870 (± 500-1510, p<0.0001), and 280 (± 160-490, p = 0.4) at the end of CPB, 3 hours later, and 20 hrs. later, respectively. By multivariate analysis, only the C3a levels 3 hours after CPB, young age, and longer CPB times increased the probability of postoperative bleeding diathesis (p = 0.08, 0.06, 0.004 respectively), importantly impaired cardiac performance (p = 0.02, <0.0001, 0.02) important pulmonary dysfunction (p = 0.01, <0.0001, 0.03), and important renal dysfunction (p = 0.02, <0.0001, >0.2). Only the same three variables were related to the probability of important morbidity (all of the above events combined, p = 0.008, <0.0001, 0.0002). The nature of the relations were such that C3a levels above 1750 ng/ml tended to be associated with a high probability (‰¥ 30%) of morbidity, the tendency increasing exponentially with younger ages (particularly rapidly <1 yr) and longer CPB times (particularly >120 minutes). The hypothesis that

results is that a damaging effect in the form of a whole body inflammatory reaction, with complement activation, results from even short periods of CPB; that very small patients are particularly vulnerable; and that long CPB times add further damage. Many techniques allow considerable suppression of these risk factors and good results, but more knowledge is required for their complete neutralization.
*By Invitation

42. Management of Intracardiac Fungal Masses in Premature Infants

JOHN E. FOKER, THEODORER. THOMPSON*,*
JOHN L. BASS, JOHN A. TILLELI**
*and DANA E. JOHNSON**
Minneapolis, Minnesota
Sponsored by: ROBERT W. ANDERSON,
Minneapolis, Minnesota

Intracardiac fungal masses can develop following episodes of candidemia in premature infants with indwelling right atrial lines. Previously, this diagnosis had only been made at autopsy in the cases described in the literature. We report here the diagnosis and first successful surgical removal of Candida-containing intracardiac masses in three premature infants. Over the past four years, six premature newborns developed condidemia despite oral nystatin prophylaxis. The right atrial lines were removed and the infants treated with amphotericin B and 5-flucytosine. Echocardiograms revealed that three (50%) had developed intracardiac masses. In two, a pedunculated solitary mass was found within the right atrium. In these two patients the antifungal therapy over the next 21-42 days controlled the Candida sepsis. By echocardiography, however, the intracardiac masses did not decrease in size and became quite mobile. In the third infant, masses were present from the right atrium to the main pulmonary artery and surgical removal was recommended four days after beginning antifungal therapy. In all three patients, the masses were nearly the size of the main pulmonary artery and presumably contained viable organisms. Because of concern for the risk of pulmonary embolism, surgical removal was undertaken and two were removed using cardiopulmonary bypass (CPB). One infant, however, weighed only 1300 grams and CPB seemed too hazardous. In the latter case, the intraatrial mass was easily removed using inflow stasis of 30 seconds duration. In all three patients removal was grossly complete and the masses were filled with Candida mycelia. All patients tolerated the operation well and have been discharged home with a minimum follow-up of six months without evidence of recurrent Candida infection. In summary, this report documents that: (1) Echocardiography provides a noninvasive method of diagnosing the development of intracardiac fungal masses and should be performed in all infants who have had condidemia and a central venous line. (2) The intracardiac masses do not appear to resolve even with prolonged systemic antifungal therapy. (3) The masses can be safely removed even in the premature infant, using either inflow stasis or CPB. (4) Surgical removal seems to be an effective component of the management of these infants by eliminating the acute threat of a large septic embolism and contributing to the elimination of the focus of infection.
*By Invitation

43. Prospective Evaluation of Patients with Chronic Respiratory Signs and Symptoms for the Presence of Occult Esophageal Disease

*TOM R. DEMEESTER, CLEMENTE IASCONE**
and DAVID B. SKINNER
Chicago, Illinois

Seventy-seven patients with chronic respiratory signs and symptoms of unknown etiology (18 chronic cough, 20 recurrent pneumonia, 10 pulmonary fibrosis and 29 wheezing) were evaluated with a careful clinical history, esophageal manometry and 24-hour esophageal pH monitoring for the presence of esophageal disease. Symptoms of heartburn, regurgitation or dysphagia were mild or absent in 90% of the patients. Fifty-four of the 77 patients had abnormal esophageal acid exposure on 24-hour pH monitoring and the severity of acid exposure was similar in all groups with the exception of those who wheezed. They had less acid exposure, mainly in the supine

position ($p<.05$). The lower esophageal sphincter pressure and length of abdominal esophagus were similar in each group, but significantly less than normal. The 54 patients who refluxed were divided into four groups depending on the severity of their respiratory symptoms as manifested by the presence of one or more of the following complaints: morning cough, nocturnal cough, recurrent pneumonia and wheezing spells. Group I had only one symptom; group II had two symptoms, etc. Both the incidence of dysmotility of the body of the esophagus and the occurrence of a respiratory symptom immediately preceeding or following a reflux episode increased as the severity of their respiratory complaints increased, suggesting that dysmotility may predispose to aspiration in those who have abnormal acid exposure (Table I). Twelve patients were suspected aspirators on the basis of respiratory symptoms occurring during or following an reflux episode. This was commonly seen in patients complaining of only one respiratory symptom suggesting that the esophageal disease was primary. Patients with wheezing during or after a reflux episode had less acid exposure suggesting that wheezing spells were not due to aspiration, but to a reflex bronchial constriction triggered by the reflux episode. Seventeen patients were operated on, and the results are shown on Table II.

Table 1

Severity of Respiratory Symptoms:
Relationship with Esophageal Body Dysmotility and Reflux Episodes

Severity of Respiratory symptoms		Dysmotility		Reflux Episodes Following or preceding Respiratory symptoms	
# patients		# patients	%	# patients	%
Group 1	19	6	31.6	5	26.3
Group 2	12	5	41.7	5	41.7
Group 3	13	7	53.7	7	53.7
Group 4	10	7	70	6	60

Table 2

Results of Anti-Reflux Surgery For Respiratory Symptoms

	Pre-Op Presence	Post-Op Improvement
Recurrent Pneumonia	9/17	7/9 78%
Wheezing	12/17	6/12 50%
Nocturnal Cough	14/17	12/14 86%
Morning Cough	10/17	8/10 80%

*By invitation

44. Treatment of Malignant Disease in Trachea and Main Stem Bronchi by Carbon Dioxide Laser

*RICHARD B. MCELVEIN and GEORGE ZORN**
Birmingham, Alabama

The use of a carbon dioxide laser to erradicate intralumenal malignant lesions of the trachea and main stem bronchi is described.

Forty-three patients, 28 males and 15 females ranging from 36 to 78 years have received from one to five laser treatments to provide an improved airway with relief of major respiratory tract obstruction. There has been one in-traoperative death and two immediate postoperative deaths.

Improvement in respiratory status has been accomplished in all surviving patients persisting from 1 to 14 months. The carbon dioxide laser treatment does not cure cancer but does provide an improved airway with low risk so other treatment can be used.

A major advantage of this form of treatment is decreased bleeding and the ability to provide an improved airway. The disadvantages are the necessity for general anesthesia and expense of the equipment.

45. Thymoma - Ten Year Experience

JOEL D. COOPER, FARID SHAMJI,*
FREDERICK G. PEARSON, THOMAS R.J. TODD,*
RIIVO ILVES and ROBERT J. GINSBERG*
Toronto, Ontario

Between 1970 and 1980, 53 patients with a thymoma were seen at the Toronto General Hospital. Two patients were inoperable at the time of admission and the remaining 51 patients underwent surgical resection. The results in these 51 patients, all of whom had a complete resection, forms the basis of this report. Treatment consisted of resection alone in 30, resection with preoperative radiation in 11, resection with postoperative radiation in 8, and resection combined with preoperative and postoperative radiation in 2.

Twenty-three patients had invasive tumours and 28 had non-invasive tumours. Myasthenia gravis was present in 25 patients (10 with invasive tumours and 15 with non-invasive tumours). Current information is available on 46 of the 51 patients with a mean follow-up of 5.4 years.

Results

Of the 23 patients with invasive tumour, 7 patients are dead, 3 of unrelated causes, 2 from tumour recurrence, and 2 from complications associated with radiotherapy. In addition 2 patients are alive with tumour recurrence (2 years and 10 years). One patient is lost to follow-up.

Of the 21 patients with non-invasive tumours, 3 patients are dead, all of unrelated causes. No patient has developed recurrent tumour, 4 patients are lost to follow-up.

Contrary to earlier experience, the presence of myasthenia gravis did not alter the prognosis: of 23 patients with invasive tumours, there were 3 deaths in the 10 patients **with** myasthenia gravis and 4 deaths in the 13 patients **without** myasthenia gravis. The mean survival time was not affected by the presence of myasthenia gravis and no patient had died of myasthenia.

We conclude that surgical excision for thymoma is associated with a very favourable prognosis and that the long term survival is not significantly affected by the presence of myasthenia gravis.

*By Invitation

46. Surgical Treatment of Aneurysm of the Ascending Aorta with Aortic Insufficiency - A Selective Approach

DOUGLAS P. GREY, DAVID A. OTT**
and DENTON A. COOLEY
Jacksonville, Florida; Houston, Texas

The selection of an appropriate surgical technique for repair of aneurysm of the ascending aorta with aortic insufficiency (AATA-AVR) is unsettled. Placement of a supra-coronary graft is a compromise if the coronary ostia are displaced cephalad by the aneurysm; whereas, insertion of a valved conduit is difficult and unnecessary if the coronary ostia are normally placed.

From June 1979 to May 1982, 123 patients underwent repair of AATA-AVR. Mean age was 46.4 years. Annuloaortic ectasia was the most common indication for repair (44/123, 35.8%), followed by acute and chronic dissection (39/123, 31.7%). Twelve patients had previous operations on the ascending aorta or aortic valve, and five patients had undergone prior AATA-AVR using conventional methods (separate valve and supra-coronary graft). Seventy-two patients (58.5%) underwent composite replacement with coronary reimplantation, and fifty-one patients had repair using the conventional technique. Cardiopulmonary bypass methods, times, and postoperative complications were comparable between the two groups.

Hospital mortality for the whole series was 7.4% (9/122), with 4.2% (3/71) in patients having composite replacement and 11.8% (6/51) in patients having a conventional repair (p = N.S.). Five Jehovah's Witness patients survived. One patient committed suicide on the third post-operative day and was eliminated from subsequent calculations. Of sixty patients who have been followed locally for a total of 1183 patient-months, three died: two new dissections (6 weeks, 14 months) and one death without autopsy (2 months). Notably, no patient has required reoperation for conduit malfunction or required repair of aneurysm below a supra-coronary graft. Clinical anatomic

assessment at operation should determine the technique of repair employed, based on the degree of displacement of the coronary ostia relative to the aortic annulus.

4:00 p.m. Adjournment

*By Invitation

American Association for Thoracic Surgery, 1982-1983

(Listed by Countries, States, Provinces and Cities)

Geographical - UNITED STATES

ALABAMA

BIRMINGHAM
- Kahn, Donald R.
- Karp, Robert B.
- Kessler, Charles R.
- Kirklin, John W.
- Kouchoukos, Nicholas T.
- McElvein, Richard B.
- Pacifico, Albert D.

MONTGOMERY
- Simmons, Earl M.

OPELIKA
- Le Beck, Martin

ALASKA

ANCHORAGE
- Phillips, Francis J.

ARIZONA

PHOENIX
- Brown, Lee B.
- Cornell, William P.
- Melick, Dermont W.
- Nelson, Arthur R.

SUN CITY
- Read, C. Thomas

TUCSON
- Burbank, Benjamin
- Sanderson, Richard G.

ARKANSAS

JASPER
- Hudson, W. A.

LITTLE ROCK
- Campbell, Gilbert S.
- Read, Raymond C.
- Williams, G. Doyne

CALIFORNIA

ANAHEIM
- Main, F. Beachley

ARCADIA
- Silver, Arthur W.

CARMEL
- Daniels, Albert C.

COVINA
- Carter, P. Richard

EL MACERO
- Andrews, Neil C.

ESCONDIDO
- Mannix, Edgar P., Jr.

FRESNO
- Evans, Byron H.

HEMET
- Hewlett, Thomas H.

INGLEWOOD
- Carey, Joseph S.

IRVINE
- Connolly, John E.
- Miller, Don R.
- Wakabayashi, Akio

LA CANADA
- Aronstam, Elmore M.

LA JOLLA
- Fosburg, Richard G.
- Hutchin, Peter

LA MESA
- Long, David M., Jr.

LOMA LINDA
- Bailey, Leonard L.
- Wareham, Ellsworth E.

LONG BEACH
- Bloomer, William E.
- Carlson, Herbert A.
- Stemmer, Edward A.

LOS ANGELES
- Baisch, Bruce F.
- Benfield, John R.
- Buckberg, Gerald D.
- Davis, Lowell L.
- Fonkalsrud, Eric W.
- Holmes, E. Carmack
- Hughes, Richard K.
- Kay, Jerome Harold
- Lindesmith, George G.
- Longmire, William, Jr.
- Maloney, James V., Jr.
- Matloff, Jack M.
- Meyer, Bert W.
- Morton, Donald L.
- Mulder, Donald G.
- Stiles, Quentin R.

MARTINEZ
- Guernsey, James M.

MONTEBELLO
- Lui, Alfred H. F.

OAKLAND
- Dugan, David J.
- Ecker, Roger R.
- Leeds, Sanford E.
- McEnany, M. Terry
- Richards, Victor
- Roe, Benson B.
- Rogers, W. L.

May, Ivan A.
ORANGE
Mason, G. Robert
Salyer, John M.
ORINDA
Stephens, H. Brodie
PACIFIC PALISADES
Weinberg, Joseph A.
PALM SPRINGS
Goldman, Alfred
PALM DESERT
Julian, Ormand C.
PALO ALTO
Cohn, Roy B.
Gonzalez-Lavin, Lorenzo
Jamplis, Robert W.
PASADENA
Ingram, Ivan N.
Penido, John R. F.
S. LACUNA
Oatway, William H., Jr.
S. PASADENA
Brewer, Lyman A., III
SACRAMENTO
Harlan, Bradley J.
Hurley, Edward J.
Miller, George E., Jr.
Smeloff, Edward A.
Tyson, Kenneth R. T.
SAN BERNADINO
Flynn, Pierce J.
SAN DIEGO
Baronofsky, Ivan D.
Chambers, John S., Jr.
Daily, Pat O.
Lamberti, John J., Jr.
Peters, Richard M.
Trummer, Max J.
Utley, Joe R.
SAN FRANCISCO
Culiner, Morris M.
Ebert, Paul A.
Ellis, Robert J.
Fishman, Noel H.
Gardner, Richard E.
Gerbode, Frank
Grimes, Orville F.
Hill, J. Donald
Kerth, William J.
Thomas, Arthur N.
Ullyot, Daniel J.
SAN JOSE
Angell, William W.
SANTA ANA
Gazzaniga, Alan B.
Pratt, Lawrence A.
SANTA BARBARA
Higginson, John F.
Jahnke, Edward J., Jr.
Lewis, F. John
Love, Jack W.
SANTA MONICA
Ramsay, Beatty H.
STANFORD
Mark, James B. D.
Miller, D. Craig
Shumway, Norman E.
Wilson, John L.
THOUSAND OAKS
Tsuji, Harold K.
TORRANCE
Moore, Thomas C.
Nelson, Ronald J.
State, David
VENTURA
Dart, Charles H., Jr.

COLORADO

DENVER
Blair, Emil
Brown, Robert K.
Burrington, John D.
Condon, William B.
Eiseman, Ben
Grow, John B.
Harper, Frederick R.
Hopeman, Alan R.
Kovarik, Joseph L.
Newman, Melvin M.
Pappas, George
Paton, Bruce C.
Pomerantz, Marvin
Rainer, W. Gerald
Van Way, Charles W., III
Waddell, William R.
Wright, George W.
LAKEWOOD
Swan, Henry, II

CONNECTICUT

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Kemler, R. Leonard
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Laks, Hillel
Lindskog, Gustaf E.
Stansel, Horace C., Jr.
Stern, Harold
NORTHFORD
Amberson, J. B.
NORWICH
Kelley, Winfield O.
SHARON
Wylie, Robert H.
JACKSONVILLE
Stephenson, Sam, Jr.
LAKELAND
Brown, Ivan W., Jr.
MIAMI
Bolooki, Hooshang
Center, Sol
Daughtry, Dewitt C.
Gentsch, Thomas O.
Jude, James R.
Kaiser, Gerard A.
MacGregor, David C.
Papper, Emanuel M.
Reis, Robert L.
Ripstein, Charles B.
Stanford, William
Thurer, Richard J.
MIAMI BEACH
Greenberg, Jack J.

WILTON
Pool, John L.

DELAWARE

WILMINGTON
Pecora, David V.

DISTRICT OF COLUMBIA

WASHINGTON
Aaron, Benjamin L.
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Midgley, Frank M.
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Smyth, Nicholas P. D.
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FLORIDA

BELLEAIRE
Lasley, Charles H.
BOCA RATON
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CORAL GABLES
Cooke, Francis N.
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Geary, Paul
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NO. PALM BEACH
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Logan, William D., Jr.
Mansour, Kamal A.
Miller, Joseph I.
Rivkin, Laurence M.
Symbas, Panagiotis
AUGUSTA
Ellison, Robert G.
Rubin, Joseph W.
BRUNSWICK
Collins, Harold A.
MACON
Van De Water, Joseph
SAVANNAH
Langston, Hiram T.
Yeh, Thomas J.

HAWAII

HONOLULU
Ching, Nathaniel P.
Gebauer, Paul W.
McNamara, Joseph J.
KAILUA KONA
Fell, Egbert H.

IDAHO

BOISE
Ashbaugh, David G.
Herr, Rodney H.

ILLINOIS

CHICAGO
Anagnostopoulos, C.
Barker, Walter L.
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Shields, Thomas W.
Skinner, David B.
Thomas, Paul A., Jr.
Vanecko, Robert M.
Weinberg, Milton, Jr.
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Fry, Willard A.
Tatooles, Constantine
GLENCOE
Rubenstein, L. H.
MAYWOOD
Keeley, John L.
Pifarre, Roque
OAK BROOK
Nigro, Salvatore L.
PEORIA
DeBord, Robert A.
SKOKIE
Baffes, Thomas G.
WINNETKA
Mackler, S. Allen

INDIANA

INDIANAPOLIS
Battersby, James S.
King, Harold
King, Robert D.
Mandelbaum, Isidore
Siderys, Harry
SOUTH BEND
Van Fleit, William E.

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CEDAR RAPIDS
Lawrence, Montague S.
DES MOINES
Dorner, Ralph A.

DeMeester, Tom R.
Faber, L. Penfield
Hanlon, C. Rollins
Head, Louis R.
Hudson, Theodore R.
Hunter, James A.
Idriss, Farouk S.
Javid, Hushang
Jensik, Robert J.
Kittle, C. Frederick
Leininger, Bernard J.
Levitsky, Sidney
Michaelis, Lawrence
Midell, Allen I.
Moran, John M.
Najafi, Hassan

KENTUCKY

LEXINGTON
Crutcher, Richard R.
Dillon, Marcus L., Jr.
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Mahaffey, Daniel E.
Ransdell, Herbert, Jr.

LOUISIANA

ALEXANDRIA
Knoepp, Louis F.
BATON ROUGE
Beskin, Charles A.
METAIRIE
Ochsner, Alton, Jr.
NEW ORLEANS
Blalock, John B.
DeCamp, Paul T.
Hewitt, Robert L.
Lindsey, Edward S.
Mills, Noel L.
Moulder, Peter V.
Ochsner, John L.
Pearce, Charles W.
Rosenberg, Dennis M.
Schramel, Robert J.
Strug, Lawrence H.
Webb, Watts R.

MAINE

KENNEBUNK
Hurwitz, Alfred
PORTLAND
Drake, Emerson H.
Hiebert, Clement
ROCKPORT
Swenson, Orvar

MARYLAND

BALTIMORE
Attar, Safuh M.A.
Baker, R. Robinson
Brawley, Robert K.
Cowley, R. Adams
Gardner, Timothy J.
Gott, Vincent L.
Haller, J. Alex, Jr.
Hankins, John R.
McLaughlin, Joseph S.
Michelson, Elliott
Turney, Stephen Z.
BETHESDA
Iovine, Vincent M.
Shumacker, Harris, Jr.
Watkins, David H.
IOWA CITY
Doty, Donald B.
Ehrenhaft, Johann L.
Rossi, Nicholas P.

KANSAS

CUNNINGHAM
Allbritten, F. F., Jr.
KANSAS CITY
Friesen, Stanley R.
OVERLAND PARK
Barnhorst, Donald A.
WICHITA
Tocker, Alfred M.
WINFIELD
Snyder, Howard E.

WORTON
Walkup, Harry E.

MASSACHUSETTS

BOSTON
Austen, W. Gerald
Barsamian, Ernest M.
Berger, Robert L.
Bernhard, William F.
Bougas, James A.
Braunwald, Nina S.
Buckley, Mortimer J.
Burke, John F.
Castaneda, Aldo R.
Cleveland, Richard J.
Clowes, George, Jr.
Cohn, Lawrence H.
Collins, John J.
Daggett, Willard M.
Deterling, Ralph, Jr.
Frank, Howard A.
Gaensler, Edward A.
Grillo, Hermes C.
Moncure, Ashby C.
Neptune, Wilford B.
Norwood, William I.
Overholt, Richard H.
Rheinlander, Harold
Russell, Paul S.
Scannell, J. Gordon
Schuster, Samuel R.
Starkey, George W.
Weintraub, Ronald
Wilkins, Earle W., Jr.
BROOKLINE
Madoff, Irving M.
BURLINGTON
Boyd, David P.
Ellis, F. Henry, Jr.
Watkins, Elton, Jr.
CAMBRIDGE
Harken, Dwight E.
CONCORD
Soutter, Lamar
LYNNFIELD
Wesolow, Adam
MEDFORD
Boyd, Thomas F.
Desforges, Gerard
METHUEN
Wilson, Norman J.
N. ANDOVER
Cook, William A.

POTOMAC
Zajtchuk, Rostik

NANTUCKET
Mahoney, Earle B.
NEWTON LOWER FALLS
Laforet, Eugene G.
Strieder, John W.
S. WEYMOUTH
Malcolm, John A.
SPRINGFIELD
Engelman, Richard M.
STOUGHTON
Black, Harrison
WESTPORT HARBOR
Findlay, Charles, Jr.
WINCHESTER
Taylor, Warren J.

MICHIGAN

ANN ARBOR
Bartlett, Robert H.
Behrendt, Douglas M.
Gago, Otto
Kirsh, Marvin M.
Morris, Joe D.
Orringer, Mark B.
Sloan, Herbert
BIRMINGHAM
Dodrill, Forest D.
Levine, Frederick H.
DETROIT
Arbulu, Augustin
Arciniegas, Eduardo
Day, J. Claude
Lam, Conrad R.
Magilligan, D. J., Jr.
Wilson, Robert F.
GRAND RAPIDS
Harrison, Robert W.
Meade, Richard H.
Rasmussen, Richard A.
Tomatis, Luis A.
GROSSE POINTE
Benson, Clifford D.
Gerbasi, Francis S.
Taber, Rodman E.
KALAMAZOO
Neerken, A. John
ROYAL OAK
Timmis, Hilary H.

MINNESOTA

CROOKSTON
Deniord, Richard N.
DULUTH
Fuller, Josiah

Roper, Charles L.
Weldon, Clarence S.
Willman, V. L.
ST. PAUL
Codd, John E.

NEBRASKA

OMAHA
Fleming, William H.
Malette, William G.

MINNEAPOLIS
Anderson, Robert W.
Arom, Kit V.
Gannon, Paul G.
Garamella, Joseph J.
Helseth, Hovald K.
Humphrey, Edward W,
Johnson, Frank E.
Kiser, Joseph C.
Molina, J. Ernesto
Nicoloff, Demetre M.
ROCHESTER
Bernatz, Philip E.
Clagett, O. Theron
Danielson, G. K.
Kaye, Michael P.
McGoon, Dwight C.
Olsen, Arthur M.
Payne, W. Spencer
Pluth, James R.
ST. PAUL
Leven, N. Logan
Lillehei, C. Walton
Miller, Fletcher A.
Perry, John F., Jr.

MISSISSIPPI

JACKSON
Hardy, James D.
Johnston, J. H., Jr.
Neely, William A.
Netterville, Rush E.

MISSOURI

COLUMBIA
Silver, Donald
KANSAS CITY
Adelman, Arthur
Ashcraft, Keith W.
Benoit, Hector W., Jr.
Holder, Thomas M.
Killen, Duncan A.
Mayer, John H., Jr.
Padula, Richard T.
Reed, William A.
MT. VERNON
Campbell, Daniel C., Jr.
ST. LOUIS
Earner, Hendrick B.
Bergmann, Martin
Clark, Richard E.
Connors, John P.
Ferguson, Thomas B.
Kaiser, George C.
Lewis, J. Eugene, Jr.
Pennington, D. Glenn

NEW MEXICO

ALBUQUERQUE
Edwards, W. Sterling
ARTESIA
Glass, Bertram A.
LAS VEGAS
Thai, Alan P.

NEW YORK

ALBANY

Sellers, Robert D.

NEW HAMPSHIRE

HANOVER
Crandell, Walter, B.
PETERBOROUGH
Woods, Francis M.
WHITE RIVER JUNCTION
Tyson, M. Dawson

NEW JERSEY

BELLVILLE
Gerard, Franklyn P.
BELMAR
Bailey, Charles P.
BROWNS MILL
Fernandez, Javier
CAMDEN
Camishion, Rudolph C.
EAST ORANGE
Auerbach, Oscar
MILLBURN
Parsonnet, Victor
MOORESTOWN
Morse, Dryden P.
N. CALDWELL
Wychulis, Adam R.
NEW BRUNSWICK
Kunderman, Philip J.
MacKenzie, James W.
NEWARK
Abel, Ronald M.
Amato, Joseph J.
Gielchinsky, Isaac
Neville, William E.
PATTERSON
Bregman, David
PENNSAUKEN
Pierucci, Louis, Jr.
SHORT HILLS
Demos, Nicholas J.
Timmes, Joseph L.
TENAFLY
Gerst, Paul H.

Alley, Ralph D.
Kausel, Harvey W.
McKneally, Martin F.
BAY SHORE
Ryan, Bernard J.
BRONX
Becker, Ronald M.
Fell, Stanley C.
Frater, Robert W. M.
Hirose, Teruo
Robinson, George
BROOKLYN
Griepp, Randall B.
Levowitz, Bernard S.
Sawyer, Philip N.
BUFFALO
Adler, Richard H.
Andersen, Murray N.
Bhayana, Joginder
Lajos, Thomas Z.
MacManus, Joseph E.
Subramanian, S.
COOPERSTOWN
Blumenstock, David A.
FAYETTEVILLE
Bugden, Walter F.
FLORAL PARK
Crastnopol, Philip
IRVINGTON
Altai, Lari A.
NEW PALTZ
Johnson, Elgie K.
NEW YORK
Beattie, Edward, Jr.
Bloomberg, Allan E.
Bowman, Frederick, Jr.
Boyd, Arthur D.
Cahan, William G.
Clauss, Roy H.
Conklin, Edward F.
Cournand, Andre
Cracovaner, Arthur J.
Cunningham, J. N., Jr.
Davidson, Louis R.

Ford, Joseph M.
Friedlander, Ralph
Gay, William A., Jr.
Giannelli, Stanley, Jr.
Green, George E.
Holman, Cranston W.
Holswade, George R.
Hood, R. Maurice
Humphreys, G. H., II
Hutchinson, John E., Ill
Isom, O. Wayne
Jaretzki, Alfred, III
King, Thomas C.
Kirschner, Paul A.
Lambert, Adrian
Litwak, Robert S.
Maier, Herbert C.
Malm, James R.
Martini, Nael
Nealon, Thomas F., Jr.
Okinaka, Arthur J.
Redo, S. Frank
Reemtsma, Keith
Rubin, Morris

SOUTHAMPTON
Heroy, William W.
STATEN ISLAND
Garzon, Antonio A.
STONY BROOK
Dennis, Clarence
Soroff, Harry S.
SYRACUSE
Bredenberg, Carl E.
Effler, Donald B.
Meyer, John A.
Parker, Frederick, Jr.
VALHALLA
Reed, George E.
W. HAMPTON BEACH
Sarot, Irving A.

NORTH CAROLINA

ASHEVILLE
Belts, Reeve H.
Scott, Stewart M.
Sethi, Gulshan K.
Takaro, Timothy
CHAPEL HILL
Murray, Gordon F.

Spencer, Frank C.
Spotnitz, Henry M.
Steichen, Felicien M.
Subramanian, V. A.
Tice, David
Veith, Frank J.
Wallsh, Eugene
Wichern, Walter, Jr.
Wolff, William I.

CLEAN
Douglass, Richmond

PATCHOGUE
Finnerty, James

PLATTSBURG
Potter, Robert T.

ROCHESTER
DeWeese, James A.
Emerson, George L.
Schwartz, Seymour I.
Stewart, Scott
Zaroff, Lawrence I.

ROSLYN
Thomson, Norman, Jr.
Wisoff, B. George

SARANAC LAKE
Decker, Alfred M., Jr.
Merkel, Carl G.

SEAFORD
Mangiardi, Joseph L.

OHIO

AKRON
Falor, William H.

CHARDON
Mautz, Frederick R.

CINCINNATI
Carter, B. Noland
Gonzalez, Luis L.
Helmsworth, James A.
Rosenkrantz, Jens G.
Wright, Creighton B.

CLEVELAND
Ankeney, Jay L.
Cosgrove, Delos M.
Cross, Frederick S.
Groves, Laurence K.
Kay, Earle B.
Loop, Floyd

COLUMBUS
Clatworthy, H. W., Jr.
Kilman, James W.
Meckstroth, Charles
Vasko, John S.
Williams, Thomas, Jr.

DAYTON
Dewall, Richard A.

PEPPER PIKE
Mendelsohn, Harvey J.

TOLEDO
Blakemore, William S.

OKLAHOMA

OKLAHOMA CITY
Elkins, Ronald C.
Felton, Warren L., II
Fisher, R. Darryl
Greer, Allen E.
Munnell, Edward R.
Wilder, Robert J.
Williams, G. Rainey

Starek, Peter J. K.
Wilcox, Benson R.

CHARLOTTE
Robicsek, Francis
Taylor, Frederick H.

DURHAM
Jones, Robert H.
Oldham, H. N., Jr.
Sabiston, David C.
Sealy, Will C.
Smith, David T.
Wechsler, Andrew S.
Wolfe, Walter G.
Young, W. Glenn, jr.

ORIENTAL
Deaton, W. Ralph, Jr.

PINEHURST
Fischer, Walter W.

TRYON
Wilson, Julius L.

WINSTON-SALEM
Cordell, A. Robert
Hudspeth, Allen S.
Johnston, Frank R.
Meredith, Jesse H.

NORTH DAKOTA

GRAND FORKS
James, Edwin C.

PENNSYLVANIA

ABINGTON
Frobese, Alfred S.

BETHLEHEM
Snyder, John M.

BUCK HILL FALLS
Thompson, Samuel A.

DARBY
McKeown, John J., Jr.

FAIRFIELD
McClenathan, James E.

GLADWYNE
Johnson, Julian

HAMBURG
Judd, Archibald R.

HAVERTOWN
Chodoff, Richard J.

HERSHEY
Demuth, William, Jr.
O'Neill, Martin J., Jr.
Pierce, William S.
Waldhausen, John A.

LANCASTER
Witmer, Robert H.

LUMBERVILLE
O'Neill, Thomas J. E.

PHILADELPHIA
Brockman, Stanley K.
Donahoo, James
Edie, Richard N.
Edmunds, L. Henry, Jr.
Fineberg, Charles
Haupt, George J.
Lemmon, William M.
Lemole, Gerald M.
MacVaugh, Horace, III
Mendelssohn, Edwin
Mundth, Eldred D.
Nemir, Paul, Jr.

Wilson, Hugh E., III
Zuhdi, M. Nazih
TULSA
McPhail, Jasper L.

OREGON

DAYS CREEK
Miller, Arthur C.
PORTLAND
Lawrence, G. Hugh
Okies, J. Edward
Poppe, J. Karl
Starr, Albert
Parr, Grant V. S.
Rosemond, George P.
Stephenson, Larry W.
Templeton, John, III
Wallace, Herbert W.
PITTSBURGH
Bahnson, Henry T.
Ford, William B.
Magovern, George J.
Pontius, Robert G.
Rams, James J.
Ravitch, Mark M.
SAYRE
Sewell, William H.

WYNNEWOOD
Harken, Alden H.
YARDLEY
Sommer, George N., Jr.

RHODE ISLAND

PROVIDENCE
Karlson, Karl E.
Simeone, Fiorindo A.

SOUTH CAROLINA

CHARLESTON
Bradham, Randolph R.
Hairston, Peter
Parker, Edward F.
Sade, Robert M.
COLUMBIA
Almond, Carl H.
Tyras, Denis H.
LANDRUM
Stayman, Joseph W.
STATE PARK
Ryan, Thomas

TENNESSEE

CHATTANOOGA
Adams, Jesse E., Jr.
Hall, David P.
JACKSON
Chandler, John H.
JOHNSON CITY
Bryant, Lester R.
Lefemine, Armand A.
KNOXVILLE
Blake, Hu Al
Brott, Walter H.
Domm, Sheldon E.
MEMPHIS
Cole, Francis H.
Eastridge, Charles E.
Garrett, H. Edward
Howard, Hector S., Jr.
Hughes, Felix A., Jr.
McBurney, Robert P.
Pate, James W.
Robbins, S. Gwin, Sr.
Rosensweig, Jacob
Skinner, Edward F.
NASHVILLE
Alford, William, Jr.
Bender, Harvey W., Jr.
Dale, W. Andrew
Foster, John H.
Gobbel, Walter G., Jr.
Johnson, Hollis E.
Sawyers, John L.
Scott, Henry W., Jr.
Stoney, William S.
Thomas, Clarence, Jr.
SEWANEE
Thrower, Wendell B.

TEXAS

AMARILLO
Sutherland, R. Duncan
BURNET
Ross, Raleigh R.
DALLAS
Adam, Maurice
Davis, Milton V.
Holland, Robert H.
Kee, John L., Jr.
Lambert, Gary J.
Mills, Lawrence J.
Mitchel, Ben F., Jr.
Paulson, Donald L.
Platt, Melvin R.
Razzuk, Maruf A.
Seybold, William D.
Shaw, Robert R.
Sugg, Winfred L.
Urschel, Harold, Jr.
DILLEY
Hood, Richard H., Jr.
FORT SAM HOUSTON
Strevey, Tracy E., Jr.
Treasure, Robert L.
GALVESTON
Derrick, John R.
HOUSTON
Beall, Arthur C., Jr.
Burdette, Walter J.
Cooley, Denton A.
Crawford, E. Stanley
De Bakey, Michael E.
Hallman, Grady L., Jr.
Henly, Walter S.
Lawrie, Gerald M.
Mattox, Kenneth L.
Morris, George C., Jr.
Mountain, Clifton F.
Overstreet, John W.
Reul, George J., Jr.
Wukasch, Don C.
LUBBOCK
Bricker, Donald L.
Dalton, Martin L., Jr.
SAN ANTONIO
Dooley, Byron N.
French, Sanford, III

Grover, Frederick L.
Heaney, John P.
Nixon, James W.
Trinkle, J. Kent
TEMPLE
Brindley, G. V., Jr.
WOODVILLE
Harrison, Albert W.

UTAH

SALT LAKE CITY
Cutler, Preston R.
Johnson, Clive R.
Liddle, Harold V.
Mortensen, J. D.
Nelson, Russell M.
Wolcott, Mark W.

VERMONT

BRATTLEBORO
Gross, Robert E.
BURLINGTON
Coffin, Laurence H.
Miller, Donald B.
CHESTER DEPOT
Adams, Herbert D.

VIRGINIA

ARLINGTON
Conrad, Peter W.
Klepser, Roy G.
CHARLOTTESVILLE
Crosby, Ivan K.
Dammann, John F.
Minor, George R.
Muller, William, Jr.
Nolan, Stanton P.
Wellons, Harry A., Jr.
LYNCHBURG
Moore, Richmond L.
RICHMOND
Bosher, Lewis H., Jr.
Brooks, James W.
Cole, Dean B.
Greenfield, Lazar J.
Gwathmey, Owen
Johns, Thomas N. P.
Lower, Richard R.

WASHINGTON

BELLINGHAM
Varco, Richard L.
FRIDAY HARBOR
Fox, Robert T.
MERCER ISLAND
Mills, Waldo O.
SEATTLE
Anderson, Richard P.
Cantrell, James R.
Dillard, David H.
Hill, Lucius D., III
Jarvis, Fred J.
Jones, Thomas W.
Manilas, Dev R.
Mansfield, Peter B.
Miller, Donald W., Jr.
Pinkham, Roland D.
Rittenhouse, Edward
Sauvage, Lester
Thomas, George I.
SPOKANE
Berg, Ralph, Jr.

WEST VIRGINIA

E. CHARLESTON
Walker, James H.
HUNTINGTON
Littlefield, James B.
MORGANTOWN
Tarnay, Thomas J.
Warden, Herbert E.

WISCONSIN

LA CROSSE
Gundersen, Erik A.
MADISON
Berkoff, Herbert A.
Chopra, Paramjeet S.
Young, William P.
MARSHFIELD
Myers, William O.
Ray, Jefferson F., III
Sautter, Richard D.
MILWAUKEE
Bonchek, Lawrence I.
Flemma, Robert J.
Hausmann, Paul F.
Johnson, W. Dudley
Litwin, S. Bert
Mullen, Donald C.
Narodick, Benjamin
Olinger, Gordon N.
Tector, Alfred J.
WAUSAU
Davila, Julio C.
WEST BEND
Gardner, Robert J.

WYOMING

TETON VILLAGE
Kaunitz, Victor H.

CANADA

ALBERTA

CALGARY
Miller, George E.
EDMONTON
Callaghan, John C.
Meltzer, Herbert
Sterns, Laurence P.

BRITISH COLUMBIA

KELOWNA
Couves, Cecil M.
VANCOUVER
Allen, Peter
NOTTAWA
Key, James A.
OTTAWA
Keon, Wilbert J.
SUDBURY
Field, Paul
Walker, George R.
TORONTO
Baird, Ronald J.
Bigelow, Wilfred G.
Cooper, Joel D.
Ginsberg, Robert J.

Ashmore, Phillip G.
Harrison, Elliott
Tyers, G. Frank O.

VICTORIA
Stenstrom, John D.

W. VANCOUVER
Robertson, Ross

MANITOBA

WINNIPEG
Barwinsky, Jaroslaw
Cohen, Morley

NEWFOUNDLAND

ST. JOHNS
Brownrigg, Garrett M.

NOVA SCOTIA

HALIFAX
Murphy, David A.

KENTVILLE
Quinlan, John J.

MABOU
Thomas, Gordon W.

ONTARIO

DORSET
Mustard, William T.

HAMILTON
Sullivan, Herbert J.

KINGSTON
Charrette, Edward J. P.

LONDON
Heimbecker, Raymond
Goldman, Bernard S.
Henderson, Robert D.
Joynt, George H. C.
Lockwood, A. L.
Mclntosh, Clarence A.
Pearson, F. Griffith
Scully, Hugh E.
Trimble, Alan S.
Trusler, George A.
Weisel, Richard D.

WESTBROOK
Lynn, R. Beverley

WOODBRIDGE
Laird, Robert C.

QUEBEC

MONTREAL
Blundell, Peter E.
Bruneau, Jacques
Chiu, Chu-Jeng (Ray)
Dobell, Anthony R.
Grondin, Claude M.
Lepage, Cities
Mac Lean, Lloyd D.
Morin, Jean E.
Mulder, David S.
Scott, Henry J.

QUEBEC CITY
Gravel, Joffre-Andre

WESTMOUNT
Vineberg, Arthur M.

OTHER COUNTRIES

ARGENTINA

BUENOS AIRES
Favaloro, Rene G.

AUSTRALIA

NO. ADELAIDE
Sutherland, H. D'Arcy

BANGLADESH

DACCA DISTRICT
McCord, Colin W.

BRAZIL

SAO PAULO
Zerbini, E. J.

CHINA

BEIJING
Ying-Kai, Wu

ENGLAND

BATH
Belsey, Ronald

BUCKINGHAMSHIRE
Sellers, Sir Thomas

HEREFORD
Smith, Roger Abbey
Thompson, Vernon

LONDON
Kennedy, John H.
Lennox, Stuart C.
Ross, Donald N.

FRANCE

BORDEAUX
Fontan, Francis M.

PARIS
Binet, Jean-Paul
Carpentier, Alain F.
Dubost, Charles

GUATEMALA

GUATEMALA CITY

JAPAN

YAMAGUCHI
Mohri, Hitoshi

SAGA
Miyamoto, Alfonso T.

NETHERLANDS

LEIDEN
Brom, Gerard A.

NEW ZEALAND

AUCKLAND
Barratt-Boyes, Sir Brian

PORTUGAL

LISBON
Macedo, Manuel E. M.

SAUDI ARABIA

RIYADH
Merendino, K. Alvin

SCOTLAND

EDINBURGH
Logan, Andrew

SPAIN

SANTANDER
Duran, Carlos Gomez

SWEDEN

STOCKHOLM
Bjork, Viking O.
Crafoord, Clarence

SWITZERLAND

ZURICH
Senning, Ake

GENOLIER
Hahn, Charles J.

VENEZUELA

CARACAS
Tricerri, Fernando E.

WEST GERMANY

Herrera, Rodolfo

INDIA

RAIPUTANA
Van Allen, Chester M.

IRELAND

DUBLIN
O'Malley, Eoin

AACHEN
Messmer, Bruno J.

THE AMERICAN ASSOCIATION FOR THORACIC SURGERY
Charter Members
June 7, 1917

E. Wyllis Andrews
John Auer
Edward R. Baldwin
Walter M. Boothby
William Branower
Harlow Brooks
Lawrason Brown
Kenneth Bulkley
Alexis Carrel
Norman B. Carson
J. Frank Corbett
Armistead C. Crump
Charles N. Dowd
Kennon Dunham
Edmond Melchior Eberts
Max Einhorn
Herman Fischer
Albert H. Garvin
Nathan W. Green
John R. Hartwell
George J. Heuer
Chevalier Jackson
H. H. Janeway
James H. Kenyon
Adrian V. S. Lambert
Arthur A. Law
William Lerche
Howard Lilienthal
William H. Luckett
Morris Manges
Walton Martin
Rudolph Matas
E. S. McSweeney
Samuel J. Meltzer
Willy Meyer (Founder)
James Alexander Miller
Robert T. Miller
Fred J. Murphy
Leo S. Peterson
Eugene H. Pool
Walther I. Rathbun
Martin Rehling
B. Merrill Ricketts
Samuel Robinson
Charles I. Scudder
William H. Stewart
Franz Torek
Martin W. Ware
Abraham O. Wilensky
Sidney Yankauer

BY-LAWS OF

THE AMERICAN ASSOCIATION

FOR THORACIC SURGERY

ARTICLE I. Name

The name of this Corporation is The American Association for Thoracic Surgery (hereinafter the "Association").

ARTICLE II. Purposes

The purposes of the Association shall be:

To associate persons interested in, and carry on activities related to, the science and practice of thoracic surgery, the cure of thoracic disease and the related sciences.

To encourage and stimulate investigation and study that will increase the knowledge of intrathoracic physiology, pathology and therapy, and to correlate and disseminate such knowledge.

To hold scientific meetings featuring free discussion of problems and developments relating to thoracic surgery, and to sponsor a journal for the publication of scientific papers presented at such meetings and other suitable articles.

To succeed to, and continue to carry on the activities formerly conducted by, The American Association for Thoracic Surgery, an unincorporated association.

ARTICLE III. Membership

Section 1. There shall be four classes of members: Honorary, Senior, Active and, for a time, Associate. Admission to membership in the Association shall be by election. Membership shall be limited, the limits on the respective classes to be determined by these By-Laws. Only Active and Senior Members shall have the privilege of voting or holding office, except as provided by these By-Laws.

Section 2. Honorary Membership shall be reserved for such distinguished persons as may be deemed worthy of this honor by the Council with the concurrence of the Association.

Section 3. The number of Senior Members shall be unlimited. Active Members automatically advance to Senior Membership at the age of sixty years. In addition, a younger Active Member may be eligible for Senior Membership if incapacitated by disability, but for no other reason.

Section 4. Active Membership shall be limited to six hundred. A candidate to be eligible must be a citizen of the United States of America or Canada, unless in unusual cases this citizenship requirement shall have been waived by the Council. The candidate shall have achieved distinction in the thoracic field or shall have made a meritorious contribution to knowledge pertaining to thoracic disease or its surgical treatment.

Section 5. Election to Honorary, Senior or Active Membership shall be for life, subject lo the provisions of Section 9 following. There shall be no further additions to the Associate Membership. All new members shall be elected directly to Honorary or Active status.

Section 6. Associate Membership for those members elected after 1960 shall *be* limited to a five year period. During this limited period, an Associate Member, if properly qualified, may be elected to Active Membership. After the expiration of this limited period an Associate Member, if not yet qualified for Active Membership, must either be re-elected to an additional period of Associate Membership or dropped from the rolls of the Association.

Section 7. Candidates for membership in this Association must be formally nominated and seconded, in an approved manner, by not less than three Active or Senior Members. Such nomination must have been in the hands of the Membership Committee for not less than four months, and the name of the candidate must have been distributed to all members of the Association before final action may be taken on any new candidate for election to Active Membership. Provided the foregoing requirements have been met and the candidates have been approved by the Membership Committee and by the Council, their names shall be presented to the Association at a regularly convened annual meeting for final action. A three-fourths vote of those present and voting shall be required to elect. Any candidate for membership in this Association who has failed of election for three successive years shall automatically cease to be a candidate and may not be renominated until after a lapse of three years.

Section 8. The report of the Membership Committee shall be rendered at the second executive session of each annual meeting of the Association. Candidates shall be presented in groups in the following order: Candidates for Honorary Membership; retirement of Active Members to Senior Membership; Candidates for Active Membership, Associate Members for re-election; members dropped from the rolls of the Association.

Section 9. Membership may be voluntarily terminated at any time by members in good standing. The Council, acting as a Board of Censors, may recommend the expulsion of a member on the grounds of moral or professional delinquency, and submit his name, together with the grounds of complaint, to the Association as a whole at any of the regularly convened meetings, after giving such member ample opportunity to appear in his own behalf.

Section 10. The Council shall recommend that any Active or Associate Member whose dues are in arrears for two years, or who has been absent, without sufficient excuse, from three consecutive annual meetings, shall have his membership terminated.

Section 11. Notwithstanding Section 10, any member of the Association over 60 years of age is excused from the attendance requirement and upon his specific request may likewise be excused from the payment of dues.

ARTICLE IV. Board of Directors ("Council")

Section 1. The Board of Directors of the Association shall be called the Council and shall be composed of the President, Vice-President, Secretary, Treasurer and Editor of the Association, and five Councilors. All members of the Council must be Active or Senior Members of the Association, except that the Editor may be an Honorary Member.

Section 2. The Council shall be the governing body of the Association, and shall have full power to manage and act on all affairs of the Association, except

as follows:

a. It may not alter the initiation fees or annual dues, or levy any general assessments against the membership, except that it may, in individual cases, waive annual dues or assessments.
b. It may not change the Articles of Incorporation or By-Laws.

c. It may neither elect new members nor alter the status of existing members, other than to apply the provisions of Article III, Section 9.

d. It may not deplete the principal of the Endowment Fund.

Section 3. At the conclusion of the annual meeting, the retiring President shall automatically become a Councilor for a one-year term of office. One of the other four Councilors shall be elected at each annual meeting of the Association to serve for a four-year term of office in the place of the elected Councilor whose term expires at such meeting, but no Councilor may be reelected to succeed himself. Any Councilor so elected shall take office upon the conclusion of the annual meeting at which he is elected.

Section 4. Vacancies in the office of Councilor shall be temporarily filled by the Council subject to approval of the Association at the next annual meeting of the Association.

ARTICLE V. Officers

Section 1. The officers of the Association shall be a President, a Vice-President, a Secretary, and a Treasurer. All officers must be Active or Senior Members of the Association. Said officers shall be ex officio members of the Council of the Association.

Section 2. The Council may, for the purposes of Article IX, give status as officers of the Association to the individual members of any ad hoc Committee appointed by the Council.

Section 3. The President, Vice-President, Secretary and Treasurer shall be elected at the annual meeting of the Association and shall take office upon conclusion of the meeting. The President and the Vice-President shall be elected for a one-year term of office and neither may be reelected to succeed himself in the same office, unless such officer is filling the unexpired term of an officer previously elected to such office. The Secretary and the Treasurer shall be elected for a one-year term of office and may be reelected indefinitely.

Section 4. The President of the Association shall perform all duties customarily pertaining to the office of President. He shall preside at all meetings of the Association and at all meetings of the Council.

Section 5. The Vice-President of the Association shall perform all duties customarily pertaining to the office of the Vice-President, both as to the Association and the Council. In the event of a vacancy occurring in the office of President, the Council shall advance the Vice-President to the Presidency and appoint a new Vice-President.

Section 6. The Secretary of the Association shall perform all duties customarily pertaining to the office of Secretary. He shall serve as Secretary of the Association and as Secretary of the Council. When deemed appropriate, an Active or Senior Member may be elected to serve as an understudy to the Secretary in anticipation of the latter's retirement from office.

Section 7. The Treasurer of the Association shall perform all duties customarily pertaining to the office of Treasurer. He shall serve as Treasurer of the Association and shall also serve as custodian of the Endowment Fund.

Section 8. The Editor of the Association is not an officer of the Association. He shall be appointed by the Council at its annual meeting; provided, however, that such appointment shall not become effective until approved by the Association at the annual meeting of the Association. The Editor shall be appointed for a five-year term and may not be appointed to more than two successive terms; provided, however, that an Editor completing two years or less of the unexpired term of a previous Editor may be appointed for two successive five-year terms. The Editor shall serve as the Editor of the official Journal and shall be ex officio the Chairman of the Editorial Board and a member of the Council of the Association. Section 9. Vacancies occurring among the officers named in Section 1 or a vacancy in the position of Editor shall be temporarily filled by the Council, subject to approval of the Association at the next meeting of the Association.

ARTICLE VI. Committees

Section 1. The Council is empowered to appoint a Membership Committee, a Program Committee, a Necrology Committee and such other committees as may in its opinion be necessary or desirable. All such committees shall render their reports at an executive session of the Association, except that no ad hoc committee need report unless so directed by the Council.

Section 2. The Membership Committee shall consist of seven Active or Senior Members. The Council may appoint not more than one of its own members to serve on this Committee. The duties of the Membership Committee are to investigate all candidates for membership in the Association and to report its findings as expeditiously as possible to the Council through the Secretary of the Association. This Committee is also charged with searching the literature of this and other countries to the end that proper candidates may be presented to the Association for consideration. Appointment to this Committee shall be for a period of one year, and not more than five of the members may be reappointed to succeed themselves. This Committee is also charged with maintaining a record of membership attendance and participation in the scientific programs and reporting to the affected members and to the Council any deviations from the requirement of Article VIII, Section 4, of these By-Laws.

Section 3. The Program Committee shall consist of at least, six members: the President, the Vice President, the Secretary and the Editor of the Association, and at least two members-at-large appointed by the President. The duties of this

Committee shall be to arrange, in conformity with instructions from the Council, the scientific program for the annual meeting.

Section 4. The Necrology Committee shall consist of one or more Active or Senior Members. Appointments to this Committee shall be for a one-year term of office. Any or all members of this Committee may be reappointed to succeed themselves. The Council may, if it so desires, appoint one of its own members to serve as Chairman of this Committee. The duties of the Necrology Committee shall be to prepare suitable resolutions and memorials upon all deaths of members of the Association and to report such deaths at every annual meeting.

Section 5. The Nominating Committee shall consist of the five (5) immediate Past Presidents of the Association. The most senior Past President shall serve as Chairman. This Committee shall prepare a slate of nominees for Officers and Councilors upon instruction from the Council as to the vacancies which are to be filled by election and shall present its report at the Second Executive Session of the Annual Meeting.

Section 6. The Association as a whole may authorize the Council to appoint Scientific or Research Committees for the purpose of investigating thoracic problems and may further authorize the Council to support financially such committees to a limited degree. When Scientific or Research Committees are authorized by the Association, the Council shall appoint the Chairmen of these Committees, with power to organize their committees in any way best calculated to accomplish the desired object, subject only to the approval of the Council. Financial aid rendered to such Committees shall not exceed such annual or special appropriations as may be specifically voted for such purposes by the Association as a whole. Members are urged to cooperate with all Scientific or Research Committees of the Association.

Section 7. The Evarts A. Graham Memorial Traveling Fellowship Committee shall consist of six members: the President, Secretary, and Treasurer of the Association and three members-at-large, one member being appointed by the President each year to serve a term of three years. The Chairman shall be the member-at-large serving his third year. The duties of the committee shall be to recommend Fellowship candidates to the Graham Education and Research Foundation and to carry out other business pertaining to the Fellowship and the Fellows, past, present, and future.

Section 8. The Editorial Board shall be appointed by the Editor, subject only to the approval of the Council. The Editor shall be, ex officio, the chairman of this board and shall be privileged to appoint and indefinitely reappoint such members of the Association, regardless of class of membership, and such non-members of the Association as in his opinion may be best calculated to meet the editorial requirements of the Association.

Section 9. The Ethics Committee shall consist of five members appointed by the Council. No member shall serve more than four years. The Ethics Committee shall advise the Council concerning alleged breaches of ethics. Complaints regarding alleged breaches of ethics shall be received in writing by the Ethics Committee and shall be investigated by it. In addition, the Ethics Committee may investigate on its own initiative.

ARTICLE VII Finances

Section 1. The fiscal year of the Association shall begin on the first day of March and end on the last day of February each year.

Section 2. Members shall contribute to the financial maintenance of the Association through initiation fees, annual dues, and special assessments. The amount of the annual dues and the initiation fees shall be determined by these By-Laws. If, at the end of any fiscal year, there is a deficit in the current funds of the Association, the Council may send out notices to that effect and invite Active members to contribute the necessary amount so that no deficit is carried over from one fiscal year to another. The Association may, in any regularly convened meeting, vote a special assessment for any purpose consistent with the purposes of the Association, and such special assessment shall become an obligatory charge against the classes of members affected thereby.

Section 3. To meet the current expenses of the Association, there shall be available all revenue derived by the Association subject to the provisions of Section 4, following.

Section 4. Funds derived from the payment of initiation fees shall not be available for current expenses and shall be placed in a special fund, to be invested and reinvested in legal securities, to be held intact, and to be known as the Endowment Fund. The Council is responsible for the proper management of the Endowment Fund, and may divert any surplus in the current funds of the Association into this fund, but may not withdraw any of the principal of the Endowment Fund except in accordance with the provisions of Section 6, following.

Section 5. The income from the Endowment Fund shall be expended as the Council directs.

Section 6. The principal of the Endowment Fund maybe withdrawn, in whole or in part, under the following conditions only: The amount of principal to be withdrawn shall have been approved by the Council; it shall have been approved by a majority of the members present and voting at a regularly convened annual meeting; it shall have been tabled for one year; it shall have been finally passed by a three-fourths vote of the members present and voting at the next regularly convened annual meeting.

Section 7. In the event of the dissolution of the Association, the Endowment Fund shall be distributed among national institutions of the United States and Canada in a proportion equal to the then existing ratio between the numbers of citizens of the two nations who are members of the Association.

ARTICLE VIII. Meetings

Section 1. The time, place, duration, and procedure of the annual meeting of the Association shall be determined by the Council and the provisions of these By-Laws.

Section 2. Notice of any meeting of the Association shall be given to each member of the Association not less than five nor more than forty days prior to any annual meeting and not less than thirty nor more than forty days prior to any special meeting by written or printed notice delivered personally or by mail, by or at the direction of the Council, the President or the Secretary. Such notice shall state the place, day and hour of the meeting and in the case of a special meeting shall also state the purpose or purposes for which the meeting is called.

Section 3. A special meeting of the Association may be called by the Council or on the written request of fifteen members delivered to the Council, the President or the Secretary. The specific purposes of the meeting must be stated in the request.

Section 4. Attendance at annual meetings and participation in the scientific programs shall be optional for all Honorary and Senior Members, but it shall be expected from all Active and Associate Members.

Section 5. Each annual meeting shall have at least two executive sessions.

Section 6. When the Association convenes for its annual meeting, it shall immediately go into the first executive session, but the business at this session shall be limited to:

1. Appointment of necessary committees.

2. Miscellaneous business of an urgent nature.

Section 7. The second executive session of the Association shall be held during the afternoon of the second day of the meeting. The business at this session shall include, but is not limited to:
1. Reading or waiver of reading of the minutes of the preceding meetings of the Association and the Council.

2. Report of the Treasurer for the last fiscal year.

3. Audit Report.

4. Report of the Necrology Committee.

5. Report of the Program Committee.
6. Action on amendments to the Articles of Incorporation and By-Laws, if any.

7. Action on recommendations emanating from the Council.

8. Unfinished Business.

9. New Business.

10. Report of the Membership Committee.

11. Election of new members.

12. Report of the Nominating Committee.

13. Election of officers.

Section 8. Except where otherwise required by law or these By-Laws, all questions at a meeting of the members shall be decided by a majority vote of the members present in person and voting. Voting by proxy is not permitted.
Section 9. Fifty voting members present in person shall constitute a quorum at a meeting of members.

Section 10. While the scientific session of the annual meeting is held primarily for the benefit of the members of the Association, it may be open to non-members who are able to submit satisfactory credentials, who register in a specified manner, and who pay such registration fee as may be determined and published by the Council from year to year.

Section 11. There shall be an annual meeting of the Council held during the annual meeting of the Association. Additional meetings of the Council may be called on not less than seven days' prior written or telephonic notice by the President, the Secretary or any three members of the Council.

Section 12. Five members of the Council shall constitute a quorum for the conduct of business at any meeting of the Council, but a smaller number may adjourn any such meeting.

Section 13. Whenever any notice is required to be given to any member of the Council, a waiver thereof in writing, signed by the member of the Council entitled to such notice, whether before or after the time stated therein, shall be deemed equivalent thereto.

Section 14. Any action which may be or is required to be taken at a meeting of the Council may be taken without a meeting if a consent in writing, setting forth the action so taken, shall be signed by all of the members of the Council. Any such consent shall have the same force and effect as a unanimous vote at a duly called and constituted meeting.

ARTICLE IX. Indemnification of Directors and Officers

Section 1. The Association shall indemnify any and all of its Councilors (hereinafter in this Article referred to as "directors") or officers or former directors or officers, or any person who has served or shall serve at the Association's request or by its election as a director or officer of another corporation or association, against expenses actually and necessarily incurred by them in connection with the defense or settlement of any action, suit or proceeding in which they, or any of them, are made parties, or a party, by reason of being or having been directors or officers or a director or officer of the Association, or of such other corporation or association, provided, however, that the foregoing shall not apply to matters as to which any such director or officer or former director or officer or person shall be adjudged in such action, suit or proceeding to be liable for willful misconduct in the performance of duty or to such matters as shall be settled by agreement predicated on the existence of such liability.

Section 2. Upon specific authorization by the Council, the Association may purchase and maintain insurance on behalf of any and all of its directors or officers or former directors or officers, or any person who has served or shall serve at the Association's request or by its election as a director or officer of another corporation or association, against any liability, or settlement based on asserted liability, incurred by them by reason of being or having been directors or officers or a director or officer of the Association or of such other corporation or association, whether or not the Association would have the power to indemnify them against such liability or settlement under the provisions of Section 1.

ARTICLE X. Papers

Section 1. All papers read before the Association shall become the property of the Association. Authors shall leave original copies of their manuscripts with the Editor or reporter, at the time of presentation, for publication in the official Journal.

Section 2. When the number of papers makes it desirable, the Council may require authors to present their papers in abstract, and may set a time limit on discussions.

ARTICLE XI. Initiation Fees, Dues and Assessments

Section 1. Honorary Members of the Association are exempt from all initiation fees, dues, and assessments.

Section 2. Annual dues for Active Members shall be $75.00 and shall include a year's subscription to THE JOURNAL OF THORACIC AND CARDIOVASCULAR SURGERY.

Section 3. Annual dues for Associate Members shall be $75.00 and shall include a year's subscription to THE JOURNAL OF THORACIC AND CARDIOVASCULAR SURGERY.

Section 4. Senior Members are exempt from dues.

Section 5. The initiation fee for those elected directly to Active Membership shall be $15.00.

Section 6. If and when an Associate Member is elected to Active Membership, he shall pay an additional $5.00 initiation fee.

Section 7. Associate and Active Members must subscribe to THE JOURNAL OF THORACIC AND CARDIOVASCULAR SURGERY to retain their membership status.

Section 8. Subscription to THE JOURNAL OF THORACIC AND CARDIOVASCULAR SURGERY is optional for Senior Members.

Section 9. Bills for membership dues and for subscriptions to THE JOURNAL OF THORACIC AND CARDIOVASCULAR SURGERY will be mailed to members by the Treasurer after the annual meeting.

ARTICLE XII. Parliamentary Procedure

Except where otherwise provided in these By-Laws or by law, all parliamentary proceedings at the meetings of this Association and its Council and committees shall be governed by the then current Sturgis Standard Code of Parliamentary Procedure.

ARTICLE XIII. Amendments

Section 1. These By-Laws may be amended by a two-thirds vote of the members present and voting at an executive session of a properly convened annual or special meeting of the Association provided that the proposed amendment has been moved and seconded by not less than three members at a prior executive session of that meeting or a prior meeting of the Association.

Section 2. These By-Laws may be suspended in whole or in part for a period of not more than twelve hours by a unanimous vote of those present and voting at any regularly convened meeting of the Association.

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
1958-Boston..... President, Brian Blades
1959-Los Angeles..... President, Michael E. De Bakey
1960-Miami Beach..... President, William E. Adams
1961-Philadelphia..... President, John H. Gibbon, Jr.
1962-St. Louis..... President, Richard H. Sweet (Deceased 1-11-62)
..... President, O. Theron Clagett
1963-Houston..... President, Julian Johnson
1964-Montreal..... President, Robert E. Gross
1965-New Orleans..... President, John C. Jones
1966-Vancouver, B. C..... President, Herbert C. Maier
1967-New York..... President, Frederick G. Kergin
1968-Pittsburgh..... President, Paul C. Samson
1969-San Francisco..... President, Edward M. Kent
1970-Washington, D. C..... President, Hiram T. Langston
1971-Atlanta..... President, Thomas H. Burford
1974-Las Vegas..... President, Lyman A. Brewer, III
1975-New York..... President, Wilfred G. Bigelow
1976-Los Angeles..... President, David J. Dugan
1977-Toronto..... President, Henry T. Bahnson
1978-New Orleans..... President, J. Gordon Scannell
1979-Boston..... President, John W. Kirklin
1980-San Francisco..... President, Herbert Sloan
1981-Washington, D.C..... President, Donald L. Paulson
1982-Phoenix, Arizona..... President, Thomas B. Ferguson

EVARTS A. GRAHAM MEMORIAL TRAVELING FELLOWS

1st	1951-52	L. L. Whytehead, M.D., F.R.C.S. 790 Sherbrooke St., Winnipeg, Manitoba, R3A 1M3 CANADA
2nd	1953-54	W. B. Ferguson, M.B., F.R.C.S. Royal Victoria Infirmary, Newcastle-upon-tyne, ENGLAND
3rd	1954-55	Lance L. Bromley, M. Chir., F.R.C.S. St. Mary's Hospital, London, W.2, ENGLAND
4th	1955-56	Raymond L. Hurt, F.R.C.S. The White House, 8 Loom Lane, Radlett Herts, ENGLAND
5th	1956-57	Mathias Paneth, F.R.C.S. Brompton Hospital, London, S.W.3, ENGLAND
6th	1957-58	Peter L. Brunnen, F.R.C.S. Department of Thoracic Surgery, Woodend General Hospital Aberdeen, SCOTLAND
7th	1958-59	N. G. Meyne, M.D. University of Amsterdam, Wilhelmina-Gasthuis, Amsterdam, HOLLAND
8th	1960-61	Godrej S. Karai, M.D. Calcutta, INDIA
9th	1961-62	Fritz Helmer, M.D. Second Surgical Clinic, University of Vienna, Vienna, AUSTRIA
10th	1962-63	Theodor M. Scheinin, M.D. Oulun Laaninsairaala, Oulu, FINLAND
11th	1963-64	Masahiro Saigusa, M.D. National Nakano Chest Hospital, 3-14-20 Egata, Nakano-Ku, Tokyo 165, JAPAN
12th	1963-64	Adar J. Hallen, M.D. Department of Thoracic Surgery, University Hospital

		Uppsala, SWEDEN
13th	1964-65	Stuart C. Lennox, M.D. Marlowe House, 103 Dulwich Village, London, SE21 7B, ENGLAND
14th	1964-65	Elias Carapistolis, M.D., F.A.C.S. University Hospital A.H.E.P.A., Surgical Clinic Department Aristotelian University of Thessaloniki, Thessaloniki, GREECE
15th	1965-66	Gerhard Friehs, M.D. Chirugische University Klinik, Graz A-8036, AUSTRIA
16th	1965-66	Ary Blesovsky, M.D. London, ENGLAND
17th	1966-67	C. Peter Clarke, F.R.A.C.S. Ste. #4, 6th Floor, 55 Victoria Parade, Fitzroy 3065 AUSTRALIA
18th	1966-67	G. B. Parulkar, M.D. K.E.M. Hospital & Seth G.S., Medical College, Bombay 400 012, INDIA
19th	1967-68	Claus Jessen, M.D. Surg. Dept. D, Rigshospitalet, Blegdamsvej 9, Copenhagen, DENMARK
20th	1969-70	Peter Bruecke, M.D. AM Steinbruch 29 Linz-Puchenau, A-4020, AUSTRIA
21st	1970-71	Michel S. Slim, M.D. Department of Surgery, American University Hospital, Beirut, LEBANON
22nd	1971-72	Severi Pellervo Mattila, M.D. Department of Thoracic Surgery, Helsinki University Central Hospital, Helsinki 29, FINLAND
23rd	1972-73	Yasuyuki Fujiwara, M.D. Department of Cardiovascular Surgery, Tokyo Medical College Hospital, Shinjuku, Tokyo, JAPAN
24th	1973-74	Marc Roger deLeval, M.D. 8 Thornton Way, Hampstead Garden Suburb, London NW 11, ENGLAND
25th	1974-75	J. J. DeWet Lubbe, M.D. Dept. of Cardio-Thoracic Surgery, University of Stellenbosch P.O. Box 53, Bellville, REPUBLIC OF SOUTH AFRICA
26th	1975-76	Mieczyslaw Trenkner, M.D. c/o Dr. Sidney Levitsky, P.O. Box 6998, Chicago, IL 60680
27th	1976-77	Bum Koo Cho, M.D. Yonsei University, P.O. Box 71 Severance Hospital, Seoul, KOREA
28th	1977-78	Alan William Gale, M.D., FRACP, FRACS 171 Sutherland Paddington 2021 Sidney, AUSTRALIA
29th	1978-79	Eduardo Otero Goto, M.D. Servicio de Cirugia Cardiovascular Qudad Sanitaria "Le Fe" Valencia, SPAIN
30th	1981-82	Richard Firmin, M.D. The London Chest Hospital, Bonner Road London E2 9JX ENGLAND
31st	1981-82	Claudio A. Salles, M.D. Rua Niquel 237 Belo Horizonte MG, BRAZIL
32nd	1982-83	Yasuhisa Shimazaki, M.D. 1013 A Beacon Parkway, East Birmingham, Alabama 35209
33rd	1983-84	Georg S. Kobinia, M.D. I. Chirurgische Abteilung des Allgemeinen Krankenhauses d.Stadt Linz KrankenhausstraBe 9, A-4020, Linz, AUSTRIA