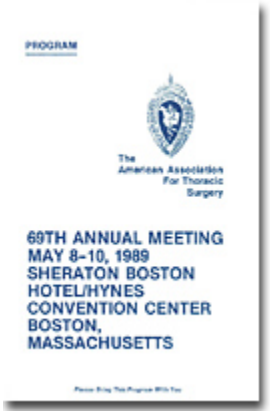
1989 ANNUAL MEETING PROGRAM



THE AMERICAN ASSOCIATION FOR THORACIC SURGERY
1988 – 1989

President W. Gerald Austen, *Boston, MA*
Vice-President F. Griffith Pearson, *Toronto, Ontario, Canada*
Secretary Martin F. McKneally, *Albany, NY*
Treasurer Floyd D. Loop, *Cleveland, Ohio*
Editor John W. Kirklin, *Birmingham, AL*

Councilors Paul A. Ebert (1989), *Chicago, IL*
L. Henry Edmunds, Jr. (1992), *Philadelphia, PA*
John R. Benfield (1989), *Sacramento, CA*
Nicholas T. Kouchoukos (1991), *St. Louis,* MO
John A. Waldhausen (1990), *Hershey, PA*

Membership Committee Randall B. Griepp, *Chairman, New York, NY*
James A. Alexander, *Gainesville, FL*
Alden H. Harken, *Denver, CO*
Alex G. Little, *Chicago, IL*
Ashby C. Moncure, *Boston, MA*
Kevin Turley, San *Francisco, CA*
William G. Williams, *Toronto, Ontario, Canada*

Association Representatives, The American Board of Thoracic Surgery
Harvey W. Bender, Jr., *Nashville, TN*
Richard J. Cleveland, *Boston, MA*
James V. Maloney, Jr., *Los Angeles, CA*
Benson R. Wilcox, *Chapel Hill, NC*

Board of Governors, American College of Surgeons
Robert B. Wallace (1989), *Washington, DC*
Anthony R. C. Dobell (1990), *Montreal, Quebec, Canada*

THE AMERICAN ASSOCIATION FOR THORACIC SURGERY
1989 Annual Meeting
COMMITTEES

LOCAL ARRANGEMENTS

Mortimer J. Buckley, *Chairman*
Cary W. Akins
W. Gerald Austen
Aldo Castaneda
Richard J. Cleveland
Lawrence H. Cohn
John J. Collins Alan D. Hilgenberg
Ashby Moncure
J. Gordon Scannell
Richard J. Shemin
Robert L. Thurer

LADIES' HOSPITALITY COMMITTEE

Mrs. Mortimer J. Buckley, (Marilyn) *Chairman*
Mrs. Cary W. Akins (Barbara)
Mrs. W. Gerald Austen (Patty)
Mrs. Aldo Castaneda (Arcely)
Mrs. Richard J. Cleveland (Carol)
Mrs. Lawrence H. Cohn (Roberta)
Mrs. John J. Collins (Mary)
Mrs. Alan D. Hilgenberg (Joanna)
Mrs. Ashby Moncure (Pat)
Mrs. J. Gordon Scannell (Helen)
Mrs. Richard J. Shemin (Susan)
Mrs. Robert L. Thurer (Shari)

PROGRAM COMMITTEE

Chairman, W. Gerald Austen, M.D..... Boston, Massachusetts
Lawrence H. Cohn, M.D..... Boston, Massachusetts
John W. Kirklin, M.D..... Birmingham, Alabama
Martin F. McKneally, M.D..... Albany, New York
F. Griffith Pearson, M.D..... Toronto, Ontario, Canada
Benson R. Wilcox, M.D..... Chapel Hill, North Carolina

EVARTS A. GRAHAM MEMORIAL TRAVELING FELLOWSHIP COMMITTEE

Chairman, Bruce A. Reitz, M.D..... Baltimore, Maryland
W. Gerald Austen, M.D..... Boston, Massachusetts
Bartley P. Griffith, M.D..... Pittsburgh, Pennsylvania
Floyd D. Loop, M.D..... Cleveland, Ohio
Martin F. McKneally, M.D..... Albany, New York
Mark B. Orringer, M.D..... Ann Arbor, Michigan

ETHICS COMMITTEE

Chairman, Willard M. Daggett, M.D..... Boston, Massachusetts
Anthony R.C. Dobell, M.D..... Montreal, Quebec, Canada
Lucius D. Hill, III, M.D..... Seattle, Washington
Will C. Sealy, M.D..... Macon, Georgia

Watts R. Webb, M.D..... New Orleans, Louisiana

LIAISON COMMITTEE FOR THORACIC SURGERY
Chairman, Mark B. Orringer, M.D..... Ann Arbor, Michigan
Joel D. Cooper, M.D..... St. Louis, Missouri
George J. Magovern, M.D..... Pittsburgh, Pennsylvania
James B. D. Mark, M.D..... Stanford, California
Martin F. McKneally, M.D..... Albany, New York
Watts R. Webb, M.D..... New Orleans, Louisiana

GOVERNMENT RELATIONS COMMITTEE (AATS/STS)
Chairman, John E. Albers, M.D..... Cincinnati, Ohio
Benjamin L. Aaron, M.D..... Washington, DC (1991)
Arthur E. Baue, M.D..... St. Louis, Missouri (1990)
Mortimer J. Buckley, M.D..... Boston, Massachusetts (1991)
Willard A. Fry, M.D..... Evanston, Illinois (1990)
Robert W. Jamplis, M.D..... Palo Alto, California (1991)
Joseph S. McLaughlin, M.D..... Baltimore, Maryland (1989)
Lawrence J. Michaelis, M.D..... Chicago, Illinois (1989)
George E. Miller, Jr., M.D..... Sacramento, California (1991)
Noel L. Mills, M.D..... New Orleans, Louisiana (1989)
Robert B. Wallace, M.D..... Washington, DC (1990)

AMERICAN ASSOCIATION FOR THORACIC SURGERY
13 Elm Street
Manchester, Massachusetts 01944
(508) 526-8330

AMERICAN COLLEGE OF SURGEONS
ADVISORY COUNCIL FOR CARDIOTHORACIC SURGERY
Floyd D. Loop, M.D., Cleveland, Ohio (1991)
John A. Waldhausen, M.D., Hershey, Pennsylvania (1992)

AMERICAN HEART ASSOCIATION
COUNCIL OF CARDIOPULMONARY DISEASE
Melvin R. Platt, M.D., Dallas, Texas (1989)

AMERICAN MEDICAL ASSOCIATION HOUSE OF DELEGATES
Thomas D. Hartley, M.D., Pueblo West, Colorado (1989)

AMERICAN MEDICAL ASSOCIATION
CPT-4 ADVISORY COMMITTEE
Milton Weinberg, Jr., M.D., Chicago, Illinois

ASSOCIATION OF AMERICAN MEDICAL COLLEGES
COUNCIL OF ACADEMIC SOCIETIES
Thomas C. King, M.D., New York, New York - Representative (1989)
Vincent L. Gott, M.D., Baltimore, Maryland - Alternate (1989)

ASSOCIATION FOR THE ADVANCEMENT OF MEDICAL INSTRUMENTATION
Robert H. Bartlett, M.D., Ann Arbor, Michigan - Representative (1989)
D. Glenn Pennington, M.D., St. Louis, Missouri - Alternate (1989)

COMMITTEE FOR COORDINATING CONTINUING EDUCATION IN THORACIC SURGERY
Gordon F. Murray, M.D., Morgantown, West Virginia (1990)

Mark B. Orringer, M.D., Ann Arbor, Michigan (1994)
Henry M. Spotnitz, M.D., New York, New York (1992)

EXTRACORPOREAL PERFUSION (AmSECT, ABCPT AND CAHEA)
Richard P. Anderson, M.D., Seattle, Washington - Representative (1989)
Stanton P. Nolan, M.D., Charlottesville, Virginia - Representative (1989)
Richard G. Fosburg, M.D., La Jolla, California - Representative (1989)
Hendrick B. Barner, M.D., St. Louis, Missouri - Alternate (1989)

NATIONAL ASSOCIATION FOR BIOMEDICAL RESEARCH
Timothy J. Gardner, M.D., Baltimore, Maryland (1989)

American Association for Thoracic Surgery
69TH ANNUAL MEETING
MAY 8-10, 1989
Scientific Program

MONDAY MORNING, May 8, 1989

8:30 a.m. BUSINESS SESSION (Limited to Members)

8:45 a.m. BASIC SCIENCE LECTURER

THE PATHOGENESIS OF ATHEROSCLEROSIS
Russell Ross, Seattle, Washington

9:30 a.m. SCIENTIFIC SESSION - HYNES BALLROOM

1. Ascending and Aortic Arch Replacement: Factors Influencing Early and Late Survival

E. STANLEY CRAWFORD, LARS G. SVENSSON,*

JOSEPH S. COSELLI, HAZIM J. SAFI* and*

*KENNETH R. HESS**

Houston, Texas

Ascending and/or aortic arch reconstruction by composite valve graft (N = 271), separate valve graft (N = 107), graft only (N = 233), and other procedures (N = 57) was employed from Jan. 1980 to Oct. 1988 in 668 patients for trauma (N = 5), infection (N = 19), aortitis (N = 44), Marfan (N = 95), non-Marfan dissection (N = 191), and medial degeneration (N = 314). Of these, 139 were redo operations in patients who had either previous heart or aortic operations. The 30-day survival was 91%. Factors favoring increased survival ($p<0.05$) included asymptomatic aneurysm (276/291, 95%) and age less than 50 (190/201, 95%). Survival in those with ascending was 92% (278/301, ascending and arch 89% (242/272), and arch 91% (86/95) and was similar regardless of operative technique. The independent determinants predictive of 30-day mortality ($p<0.03$) were increasing age, severity of symptoms, diabetes, previous operation, cardiac, and neurologic complications. After a total of 1057 operations, the entire aorta was replaced in 73, near total aorta in 30, and the entire thoracic aorta in 117; regardless, late survival (Kaplan-Meier) was 66% and 56% at 5 and 7 years. Survival of 30-day survivors according to age was 10-49 years, 78%, 50-74, 71%, and 75-

88, 69% (p = 0.0325). Independent predictors of late mortality (p<0.03) were heart disease, COPD, extent replaced, distal aneurysm, and neurologic events. Of survivors, 97% wen: in NYHA Class I or II and 93% were free of ascending and/or arch reoperation at 5 years. Thus, early operation appears justified before development of symptoms, rupture, or dissection.
*By Invitation

2. Atherosclerosis of the Ascending Aorta and Coronary Artery Bypass. Pathology, Clinical Correlates and Operative Management

NOEL L. MILLS, CHARLES T. EVERSON,*

CARL S. RIGBY and ANDREW M. SCHWARTZ**
Marrero, Louisiana

Analysis of 1735 patients undergoing CABG from January 1981 through July 1988 revealed 152 patients (8.8%) with mild (79 patients, 4.5%), moderate (39 patients, 2.2%) or severe (34 patients, 1.9%) ascending aortic atherosclerosis (As Ao Ascl). Distinct pathological patterns are: I. a lattice of circumferential medial calcification (porcelain aorta); II. grumous, liquid ASCL in the aortic wall; III. ragged, friable ulcerated intraluminal disease. A high incidence of stroke in CABG patients with the severe type of As Ao Ascl prompted development of a new operative technique that has been used in 12 patients (10 males, 2 females). Ages ranged 53 to 80 years. The "no touch" technique involves no ascending aortic cannulation or clamping, low flow hypothermic cardiac fibrillation with or without circulatory arrest, and all vein graft anastomoses placed end to side to IMA(s). The 12 patients had 37 distal IMA and SVG anastomoses and 17 proximal SVG end to side IMA anastomoses. IMA free flows ranged 130-300 cc/min. There have been no early or late CVA's or recurrence of angina. Two hospital deaths (drug error and ruptured aneurysm) were unrelated to the technique. One late death secondary to ruptured abdominal aneurysm six months postoperatively prompted a review of patients with severe As Ao Ascl. An inordinately high incidence of significant carotid disease and abdominal aortic disease (aneurysm and/or occlusive disease) was discovered. As Ao Ascl must be suspected in (1) all CABG patients with significant carotid and abdominal aortic disease, (2) aortic wall irregularity on coronary angiography (3) adhesions between the ascending aorta and its adventitia, (4) pale appearance of the ascending aorta, and (5) when an aortic stab wound for cannulation does not bleed appropriately. Diagnosis is confirmed by aortic palpation during intermittent cava! occlusion prior to aortic cannulation and by operative echocardiography.

Conclusion: A "no touch" technique that avoids any manipulation of the ascending aorta and that utilizes the IMA(s) as the source of blood supply for coronary bypass is an effective method to prevent aortic clamp injury, trash heart or stroke from severe As Ao Ascl.

*By Invitation

3. Influence of Oxygenator Type on the Incidence and Extent of Microembolic Retinal Ischemia During Cardiopulmonary Bypass. Assessment by Digital Image Analysis

CHRISTOPHER I. BLAUTH, PETER L. SMITH*,*

JOHN V. ARNOLD, J. ROGER JACOB*,*

RICHARD WOOTTON and KENNETH M. TAYLOR**

London, England Sponsored by: Floyd D. Loop, Cleveland, Ohio

We have previously reported the occurrence of microembolic ischemia in the retina during cardiopulmonary bypass, as revealed by fluorescein angiography. This method has been extended by digital image analysis to include quantification of the extent of retinal ischemia, and applied to a prospective comparative study of 64 patients undergoing elective coronary operations using either a bubble or a membrane oxygenator. Patients with diabetes or clinically evident cerebrovascular disease were excluded. Bypass procedures were standardised in all cases with pulsatile flow and a 40 micron arterial line filter (Pall EC Plus). 30 patients had bypass with a bubble oxygenator (Harvey H1700) and 34 patients had bypass using a flat sheet membrane oxygenator (Cobe CML). In each case retinal fluorescein angiograms were obtained preoperatively and 5 minutes before the end of bypass, and processed using a digital image analyser (Context Vision GOP-302). Microembolic perfusion defects were identified by digital subtraction of preoperative and end-bypass angiograms and their total area was computed.

Results. In the bubble oxygenator group retinal perfusion defects indicative of microembolism occurred in 30/30 (100%) patients. In contrast 19/34 (56%)patients in the membrane oxygenator group had normal retinal perfusion, and the incidence of perfusion defects (44%; 70% confidence limits (CL 34%-54%) was significantly reduced compared to the bubble group ($p<0.001$). In addition, those patients in the membrane group with retinal perfusion defects ($n = 15$) had significantly fewer lesions (median 1; 70% CL 1-2) than patients in the bubble group (median 2; 70% CL 2-2; $p<0.04$), and also had significantly small total areas of retinal ischemia (median $0.13mm^2$; 70% CL $0.10mm^2$-$0.25mm^2$) compared to the bubble group (median $0.22mm^2$; 70% CL $0.21mm^2$-$0.27mm^2$; $p<0.05$). There was no relationship between the extent of retinal ischemia and bypass time, arterial blood gas concentrations, volume of cardiotomy suction or donor blood returned to the pump, or recent medication with aspirin.

Inferences. Digital image analysis of retinal fluorescein angiograms may provide a method of quantifying microembolic ischemia in the central nervous system during oardiopulmonary bypass. Flat sheet membrane oxygena-tion appears to provide significant protection against microembolic ishcemia compared to bubble oxygenation.

10:30 a.m. Intermission - Visit Exhibits

*By Invitation

11:00 a.m. SCIENTIFIC SESSION - HYNES BALLROOM

4. Reoperative Coronary Surgery: Comparative Analysis of 6840 Primary and 502 Reoperative Coronary Artery Bypass Patients (CAB)

NEAL W. SALOMON, J. EDWARD OKIES,*

ALBERTH. KRAUSE, JOHN C. BIGELOW,*

U. SCOTT PAGE and MARK T. METZDORFF**

Portland, Oregon

During an eighteen year period a consecutive series of 6271 patients (pts) underwent primary and 475 pts underwent reoperative (reop) CAB. Results were analyzed to determine comparative risk factors for morbidity as well as early and late survival. The mean patient age for the reop group was identical to the primary group, 59.2 years (yrs); however mean age at initial operation for reop patients was 55.2 yrs. Mammary grafts were done at initial operation in 59% of patients who have had one operation vs only 45% of patients who subsequently came to reop ($p<.01$). Overall operative mortality was 2% (135/6271) for primary pts versus 6.3% (30/475) for reop pts ($p<.01$). Patients with a reoperative interval less than ten yrs had a 4.7% mortality vs 22% if greater than ten

yrs between operations (10/46 pts) (p<.01). Reop pts had a higher incidence of male gender, ventricular arrhythmias, excessive bleeding, neurologic complications, prolonged ventilatory support, and intra-aortic balloon pump insertion (all p<.01). Mean peak CPK-MB was 31 for reop vs 17 for primary pts (p<.01). EKG changes of infarction were present in 6.9% of reops versus 3.0% of primary pts (p<.01). The presence of a patent mammary graft from the initial operation did not affect reoperative survival. Actuarial survival was 80% vs 90% at five yrs and 65% vs 75% at ten yrs for reop vs primary CAB pts, respectively (p<.01). The probability of undergoing reop within five and ten yrs was .020 +/- .003 and .027 +/- .004, respectively. At five yrs postop moderate to severe angina had recurred in 24% of reop pts but only 13% of primary pts (p<.01). Patients undergoing reop CAB represent a substantially higher risk sub-group than pts undergoing initial operation in terms of perioperative morbidity, mortality, decreased long-term survival, and decreased relief of anginal symptoms.
*By Invitation

5. Favorable Results of Coronary Artery Bypass Grafting in Patients Older Than 75 Years

KEITH A. HORVATH, VERDI J. DiSESA *,*

PAMELA S. PEIGH, GREGORY S. COUPER*,*

JOHN J. COLLINS, JR. and LA WRENCE H. COHN

Boston, Massachusetts

There is controversy whether the short and long term results of coronary artery bypass grafting in elderly patients justifies performing the procedure. Between January, 1977 and December, 1986, 4580 patients underwent coronary artery bypass grafting of whom 222 (4.9%) were 75 years old or older (mean 77). There were 143 males and 79 females and 139 (63%) were in New York Heart Association Class IV. 146 patients (66%) had suffered at least one preoperative myocardial infarction. Myocardial revascularization was performed under emergency conditions in 17 patients (7.7%). The mammary artery was used in 43% of cases and 96% of the patients received two or more grafts (mean numbers of grafts was 2.7). The hospital mortality was 7.1% (17/222) compared to an operative mortality of 1.4% in the 4,358 patients less than 75 years old. 198 patients discharged from the hospital survived for a mean of 88 months. Actuarial probability of survival was 75% at 48 months and was the same in patients with ejection fractions less than or greater than 0.40. Post-operatively 70% of patients were in NYHA Class I or II, and only 21% were rehospitalized for cardiac related problems. 77% of the patients were free from angina during the entire follow-up and of those experiencing angina the mean time from operation to their first episode was 75 months. While elderly patients have a somewhat increased operative mortality, long term survival and freedom from angina are excellent and justify continued performance of coronary bypass grafting in well selected patients over 75 years of age.
*By Invitation

6. Surgical Angioplasty of the Left Main Coronary Artery

ROBERT A.E. DION, ROBERT VERHELST*,*

*AMIN MATTA *, MICHEL ROUSSEAU* and*

*CHARLES H. CHALANT**

Brussels, Belgium

Sponsored by: Mark Braimbridge, London, England

Critical isolated stenosis of the left main coronary artery (LM) is currently treated by conventional bypass surgery. However, this invariably leads to the definitive occlusion of LM, restores only a retrograde perfusion to a rather extensive myocardial area when a single bypass graft is constructed, and consumes a non-negligible length of bypass material. As from June 1985, we performed 20 LM patch plasties in 19 patients. 16 patients were male, age averaged 51 years (38-76 years). LM was approached either from behind after a curved right-sided aortotomy (10 cases), either anteriorly (10 cases) after retraction to the left or division (1 case) of the main pulmonary artery. LM was divided longitudinally across the stenosis, the incision being extended for 2 cms onto the aortic wall: a venous (16 cases) or pericardial (4 cases) inlay patch was used to close the defect so as to give the LM a funnel shape. In 4 of the 5 patients aged 60 years or more, LM plasty failed because of an underestimated local calcifications, and a conventional bypass graft was needed. One of these patients, a 61-year-old female, developed early graft failure and underwent 8 months later a successful repeat patch plasty pericardial using a transpulmonary anterior approach. A 76-year-old male, suffering from impending infarction, died at surgery: LM plasty had been attempted in view of the extremely poor quality of the saphenous veins. There were 2 perioperative myocardial infarctions in the "failure" group. The 16 patients having undergone a successful LM plasty are asymptomatic and all have resumed a normal activity. The follow-up averages 20 months (4-41 months). A maximal stress-test combined with a Thallium scintigraphy, performed in all of them 6 months after the operation, showed no residual ischemia and a normalized physical capacity. 13 patients consented to a 6 months postoperative angiographic control, demonstrating an excellent LM patency in all of them. Surgical patch plasty of LM restores a physiological perfusion of the left coronary tree, allows subsequent percutaneous coronary angioplasty of the distal left coronary tree, saves bypass material and can be performed safely. It should not be attempted if calcifications can be seen on the preoperative angiogram, in patients above 65 years of age and when the stenosis involves the distal bifurcation of the LM.

12:00 p.m. Adjourn for Lunch - Visit Exhibits

*By Invitation

MONDAY AFTERNOON, MAY 8, 1989

1:30 p.m. SCIENTIFIC SESSION - HYNES BALLROOM

7. Clinical Experience With the Silicone Tracheal Prosthesis

*WILLIAM E. NEVILLE, PAUL J.P. BOLANOWSKI**

*AND GODSON KOTIA**

Newark, New Jersey

When extensive pathology precludes a primary anastomosis of the trachea an alternate method is mandatory to reconstitute a suitable airway. After several years of animal research we established to our satisfaction that a molded silicone tubular prosthesis was applicable in selected cases. In the past 17 years, 62 patients with benign and malignant tracheal stenosis have had airway reconstruction with this type of tube. A straight prosthesis was used in 48 patients. Twenty-nine had strictures, 2 TE fistula and stricture, 5 tracheal malacia and 14 malignancy. Either an end to end anastomosis of the graft to the resected tracheal margins was performed or the prosthesis was used as a permanent intraluminal stem. Eight individuals had non obstructive postoperative distal suture line granulomas, 2 had subglottic granulomas and one had dehiscence of the proximal anastomosis. Lazar excision was used to remove the granulomas and the dehiscence reattached. Eight patients died 1 to 3 years after surgery. In 14 patients with malignancy, 5 are alive 1-6 years,

and 2 of 6 with an intraluminal stent, are living at 10 to 16 months. A palliative bifurcated intratracheal stent was used for palliation in 6 cases, 8 had a carinal resection - 4 are living 2-5 years.

Graft disruption, mediastinal infection, intraluminal prosthesis mucus encrustations and impedence of pulmonary secretions across long tubular segments had not occurred.

These silicone tubes are well tolerated and function satisfactorily as a permanent airway. From our observations these would seem to be a reasonable approach to the problem of complicated airway reconstruction.

*By Invitation

8. Airway Complications Following Double Lung Transplantation

G. ALEXANDER PATTERSON, THOMAS R. TODD,*

JOEL D. COOPER, F. GRIFFITH PEARSON,

TIMOTHY L. WINTON*

Toronto, Ontario, Canada and St. Louis, Missouri

En bloc Double Lung Transplantation (DLTX) is a therapeutic option to combined heart/lung transplantation in selected patients with bilateral end stage lung disease. While DLTX preserves the native heart, the donor airway, devoid of systemic arterial circulation is at risk of ischemia.

In the past 2 years, 14 DLTX have been performed in our centre. Seven recipients had emphysema and 3 patients had bronchiectasis. There was one patient with cystic fibrosis, 1 with bronchiolitis obliterans, 1 with pulmonary hypertension and 1 patient had eosinophilic granulomatosis. There have been 3 operative deaths and no late deaths. Major airway complications have occurred in 7 patients. Three patients developed fatal ischemic necrosis of the trachea and main bronchi. One patient underwent DLTX with bilateral main bronchial anastomoses, developed ischemic necrosis of the right bronchus and required retransplantation of the right lung. One patient developed partial donor tracheal necrosis which healed secondarily, leaving an anastomotic stricture. Two additional patients had satisfactory early airway healing, but developed late tracheal anastomotic and proximal left main broixchial strictures 2 months postoperatively. These latter 3 patients were treated by repeated bronchoscopic dilatations and transbronchoscopic placement of silastic bifurcation stents. These patients remain well up to 18 months with stents in place. Seven of 14 patients had excellent airway healing.

Patients with early airway necrosis had other postoperative complications (hemorrhage requiring re-exploration, 2 patients; rejection, 1 patient; bilateral pulmonary sepsis, 1 patient) which might decrease the pulmonary artery/bronchial artery collateral circulation to the donor airway. Notwithstanding the airway complications observed, 11 of 14 patients are alive and functioning normally following DLTX. By permitting separate extraction of heart and lung grafts for use in two recipients, DLTX has facilitated application of lung transplantation in our centre. We continue to employ this procedure in selected patients while seeking methods to achieve more reliable airway healing.

*By Invitation

9. Improved Survival Following Heart-Lung Transplant

PATRICK M. MCCARTHY, VAUGHN A. STARNES*,*

EDWARD B. STINSON, PHILIP E. OYER and

NORMAN E. SHUMWAY

Stanford, California

Fifty-five patients underwent heart-lung transplant (HLT) before 10/88. Thirty of these patients (operated on 3/81-2/86) were immunosuppressed using Cyclosporin A (CyA) and prednisone (P). These 30 patients (Group 1) are compared to the 25 most recent patients (Group 2) immunosuppressed using Cya, P, and Azathioprine (Aza). Patient characteristics (Group 1 vs. Group 2) were similar, including age (mean 32.4 years vs. 29.2 years) and indication for surgery (50% vs. 44% primary pulmonary hypertension, 47% vs. 48% Eisenmenger's complex, 3% vs. 8% other). Perioperative (in-hospital) mortality was 36.7% (11/30) in Group 1 vs. 8% (2/25) in Group 2 ($p < 0.05$). The linearized rejection rate (per 100 patient days) was similar in the first month after surgery (1.76 vs. 1.83). However, the linearized infection rate (per 100 patient days) in the first month was lower for the more recent patients (3.25 vs. 2.10, $p< 0.001$). Of the 19 hospital survivors in Group 1, 12 (63.1%) developed obliterative bronchiolitis (OB) from 44 to 1,461 days post-operatively (mean 361 days). Three (13.0%) of the 23 hospital survivors in Group 2 have developed OB, with a mean follow-up of 391 days (10-920) range). Overall, 23 of the 30 patients died in Group 1 (infection - 6, OB - 5, hemorrhage - 4). Four of the 25 patients in Group 2 died, all from infection. Survival for Group 1 patients was 60.0% one-year, 50.0% two-year, and 10.0% five-year versus 84.6% one-year and 84.6% two-year for Group 2 ($p < 0.05$).

A combination of improved immunosuppression with lower perioperative mortality has led to better early survival in our more recent HLT experience. Obliterative bronchiolitis has decreased in incidence and severity.

2:30 p.m. PRESIDENTIAL ADDRESS

W. Gerald Austen, Boston, Massachusetts

3:15 p.m. Intermission - Visit Exhibits

*By Invitation

3:45 p.m. SCIENTIFIC SESSION - HYNES BALLROOM

10. Evaluation of Heart-Lung Transplant Recipients with Prospective, Serial Transbronchial Biopsies and Pulmonary Function Studies

VAUGHN A. STARNES, JAMES THEODORE*,*

PHILIPE. OYER, MARGARETE. BILLINGHAM,*

RICHARD K. SIBLEY*, NORMAN E. SHUMWAY
and EDWARD B. STINSON

Stanford, California

The insidious development of obstructive airway disease (OB) following heart-lung (H-L) transplantation is thought to be secondary to rejection and possibly infection (CMV). To evaluate further, we studied prospectively the last 10 consecutive H-L transplants with serial transbronchial biopsies with lavage (TBXL) and pulmonary function studies (PFT's) as part of a surveillance

protocol or as dictated by clinical presentation. Seventy TBSL's were performed, 40 for clinical indications (Group I) and 30 for surveillance (Group II). Twenty-nine (72.5%) of Group I biopsies were positive for rejection or infection. Five (16.7%) of Group II biopsies were positive for rejection or infection. Fifteen biopsies were positive for rejection (13 in Group I, 2 in Group II) characterized by perivascular mononuclear infiltrates, lym-phocytic bronchiolitis, and alveolar septal mononuclear infiltrates. Forty-four serial PFT's were performed. The forced vital capacity (FVC), $FEF_{25-75)}$ and PaO_2 in Group I were significantly lower that Group II and correlated with positive biopsies: FVC - 51.4 ± 2.3 vs. 64.4 ± 3.6 (p= .003), FEF_{25-75} -70 ± 6.3 vs. 98.2 ± 7.3 (p = .006), $Pa0_2$ - 74.7 ± 2.8 vs. 84.8 ± 2.1 (p = .007).

The most significant fall in PFT's (FEF_{25-75}, in particular) occurred in 6 patients with rejection and was reversed with treatment. Two patients developed OB with a history of continuing rejection and CMV pneumonitis.

Serial TBXL, as dictated by PFT's and clinical status, has guided early and more specific therapy directed against rejection and infection. With early detection, airway disease has been reversible.

*By Invitation

11. Multiple Primary Lung Cancers: Results of Surgical Management

CLAUDE DESCHAMPS, PETER C. PAIROLERO,*

VICTOR F. TRASTEK and W. SPENCER PAYNE*

Rochester, Minnesota

From July 1970 to October 1983, 117 consecutive patients were diagnosed as having multiple primary cancers of the lung. Eighty patients (63 men, 17 women) underwent curative pulmonary resection (PR) for at least 2 cancers. Forty-four of these 80 patients (age 39-81 years; median, 61 years) presented with metachronous cancers. The interval between diagnoses ranged from 4 to 90 months (median, 24 months). The initial PR was wedge or segmentectomy in 4 patients, lobectomy in 36, bilobectomy in 3, and pneumonectomy in 1. The cancer was post-surgical stage I in 41 patients and stage II in 3. There were no operative deaths. The second PR was wedge or segmentectomy in 19 patients, lobectory in 16, bilobectomy in 2, and completion pneumonectomy in 7. There were two 30-day operative deaths (mortality, 4.5%). Eleven patients developed a third cancer; 6 of whom had a third PR. Actuarial 5 and 10 year survival following the first PR was 55.2% and 27% respectively. Five year survival following the second PR was 33.8%.

The remaining 36 patients, (age 40-84; median, 67.5 years) presented with synchronous cancers. Three patients underwent staged bilateral PR. The PR was wedge or segmentectomy in 8, lobectomy in 18, bilobectomy in 3, and pneumonectomy in 10. The cancer was postsurgical stage I in 24 patients, stage II in 7, stage IIIA in 4, and not staged in 1. There were two 30-day operative deaths (mortality 5.5%). Six patients later developed a third lung cancer, and all underwent another PR. Actuarial 5 year survival after PR was 15.7% which was significantly less than the survival observed after resection of the second cancer in the metachronous group ($P<.05$).

We conclude that an aggressive surgical approach is safe and warranted in most patients with multiple primary lung cancers and that the finding of synchronous primary cancers is an ominous event.

*By Invitation

12. Primary Mediastinal Nonseminomatous Germ Cell Tumors: Results of a Multimodality Approach

CAMERON D. WRIGHT, KENNETH A. KESLER*,*

CRAIG R. NICHOLS, YOUSUF MAHOMED*,*

LAWRENCE H. EINHORN*, MICHAEL E. MILLER*
and JOHN W. BROWN

Indianapolis, Indiana

Prior to cisplatin-based chemotherapy (CTX), long-term survival following resection of primary mediastinal nonseminomatous germ cell tumors (PMGCT) was unusual. We reviewed 48 patients with PMGCT who underwent an integrated treatment program including CTX, serial serum tumor marker (STM) assays, and surgery. All patients were males ranging from 14 to 46 years of age. Forty-four patients (92%) had either one or both STM elevated at the time of diagnosis. Five patients had choriocarcinoma, 4 embryonal carcinoma, 11 yolk sac carcinoma, 4 teratocarcinoma, 22 mixed cell type, and 2 had an unclassified PMGCT. Twenty-six patients had a complete response to treatment as defined by both normalization of STM and absence of residual tumor. In this group, 20 patients obtained a complete response by CTX and subsequent surgical resection, 4 with either total or near total resection followed by CTX, and 2 with CTX alone. Incomplete responders included 17 patients who failed to normalize STM after CTX, 2 with incomplete resections, and 3 with progressive disease during CTX. There was no operative mortality or significant morbidity. Overall actuarial survival was 32% at 5 years with a mean follow-up of 50 months. Five year actuarial survival was 64% if a complete response was obtained in contrast to 0% ($p<.0001$) if not. An 89% 5 year survival was achieved in 13 complete response patients with only mature teratoma found in the surgical specimen. Other favorable prognostic factors include presence of teratoma elements before CTX ($p<.001$), absence of persistent PMGCT or non-germ cell tumor after CTX ($p<.002$), absence of pulmonary metastasis at diagnosis ($p<.002$), and normalization of STM after CTX ($p<.001$). A multimodality approach to PMGCT including aggressive surgical resection and cisplatin-based chemotherapy now offers survival to a significant number of patients.
*By Invitation

13. Chemo-Radiation Therapy and Resection for Carcinoma of the Esophagus: Long-Term Results

EDWARD F. PARKER, CAROLYN E. REED,*

RICHARD D. MARKS, JOHN M. KRATZ* and*

*MARY CONNOLLY**

Charleston, South Carolina

From May, 1980 - 1984, preoperative chemotherapy (Mitomycin C and 5-FU) was added to radiation therapy in potentially operable patients with squamous cell carcinoma of the esophagus. Of 129 patients observed, only 33 were able to complete preoperative chemotherapy and radiation and undergo resection. There were 28 men and 5 women, ranging in ages from 42 to 81 yrs. (ave 60). Twenty-two patients were Black and 11 White. The location of the tomor was in the middle third in 70% of the cases. Clinical TNM classification was as follows: 3 cases TiN_0Mo, 27 cases $T_2N_0M_0$, 2 casesTiN.Mo, 1 case $T_3N_0M_0$. The length of the lesions, where measurable in the absence of complete obstruction, varied from 3 to 17 cms (ave 7 cms). Operative mortality in this group was 12% (4/33). There was no residual tumor in the surgical specimen of the esophagus in

33% (11/33) of those patients completing triple therapy. However, in two of these patients celiac nodes contained tumor and in one there was a minute esophageal perforation. The two-year survival rate was 33% (11/33), and the 5-year survival rate 15.4% (5/33). Of the 11 cases having 2-year survival, the surgical specimen was negative in 6 and positive in 5. Of the 5 cases having 5-year survival, the surgical specimen was negative in 3 and positive in 2. The absence of tumor in the surgical specimen did not appear to confer any better chance for long-term survival. Data was compared to our 1967-75 series of 75 patients receiving only preoperative radiation and resection. There was no significant difference in survival at two years [20% (1975) vs 33% (1984), p = .2118] or at five years [10% (1975) vs 15.4% (1984), p = .5796]. The addition of preoperative chemotherapy as an adjunct did not result in a statistically significant increase in 2-year or 5-year survival.

5:05 p.m. ADJOURN

*By Invitation

TUESDAY MORNING, MAY 9, 1989

7:30 a.m. FORUM SESSION - HYNES BALLROOM

F1. Selective Annuloplasty of the Tricuspid Valve: Two-Year Experience

CARMINE MINALE, HEINRICH LAMBERTZ*,*

SIGRID NIKOL and BRUNO JOSEF MESSMER*

Aachen, West Germany

Between June 1986 and October 1988, 40 patients with multiple-valve disease underwent tricuspid valve repair by a new technique. This consists of three steps: separation of the anterior and posterior leaflets from the anulus over a length averaging 4.6 + /- 1.2 cm (2.5 - 8.0 cm) to allow coaptation of the three leaflets in the middle; exclusion of ^{2}A of the isolated anulus (average: 3.4 + /- 1.0 cm) with a continuous 3-0 Tycron suture; repositioning of the leaflets to the shortened anulus by a 4-0 Prolene suture. Patients age averaged 60 yrs (29 - 73). Preoperatively 16 of them were class III and 24 class IV of the NYHA. 22 Pts had a previous valve operation. Three patients died in hospital because of respiratory failure (2.5%) and cardiac failure (5.0%), respectively. After a mean follow-up time of 12 months, there were neither late mortality nor valve related complications. Overall late survival was 92.5%. All Pts whose follow-up period lasted 6 or more months improved their functional status to class I or II (NYHA). Echocardiographic evaluation of the tricuspid valve in four chamber view showed a maximal anulus diameter averaging 26.5 +/- 6.0 mm/m^{-2} BSA preoperatively and 19.6 + /- 1.7 $mm/m"^{2}$ BSA postoperatively (p = 0.005). Postoperatively the shortening fraction of tricuspid anulus during systole averaged 11 + / 5.0%; trivial (1/3) and moderate (2/3) regurgitation were evidenced in two and one patient, respectively. Eighteen patients underwent postoperative hemodynamic investigation. Over the tricuspid valve there was a median gradient of 1.3 +/- 1.0 mmHg. Mean right atrial pressure dropped from an average of 18 +/- 8.0 mmHg preoperatively to 12 +/- 8.0 mmHg postoperatively (p = 0.03). The medium-term results with the present method show a high survival rate compared with the current methods of tricuspid valve surgery. In addition clinical and hemodynamic improvements are striking in almost all patients. Further advantage of the present method is the simplicity of restoring normal anatomic relationship of the tricuspid ap-parate without using rigid and/or prosthetic materials.

*By Invitation

F2. Electrically Conditioned Skeletal Muscle for Augmentation of Cardiac Function

RACE L. KAO, IGNACIO Y. CHRISTLIEB*,*

GEORGE J. MAGOVERN, SANG B. PARK and*

*GEORGE J. MAGOVERN, JR.**

Pittsburgh, Pennsylvania

Since skeletal muscle and heart muscle are composed mainly of contractile proteins, utilization of autogenous skeletal muscle to correct a myocardial defect and augment ventricular contraction are logical approaches. However, all the early attempts to replace or assist cardiac function with skeletal muscle have been plagued by rapid skeletal muscle fatigue. Recently, electric conditioning has made skeletal muscle capable of continuous repetitive contraction, and clinical application of dynamic cardiomyoplasty has become a reality. Skeletal muscle contracts mainly in the direction of its fiber orientation. How to utilize the skeletal muscle contractile force to maximize cardiac output in an ailing heart is the goal of this study.

Dogs were anesthetized and prepared for sterile surgical procedure. The left latissimus dorsi muscle was freed with intact neurovascular supply before being iternalized into the thoracic cavity and wrapped around the ventricles at different muscle fiber orientations with fixation stitches. After three weeks of recovery and revascularization, the muscle was conditioned over a six week period by a pulse generator. At 6, 12 and 24 weeks after the conditioning, hemodynamic evaluation after propranolol infusion (3mg/kg) was performed. Propranolol decreased the cardiac output, contractility, stroke work index, and blood pressure 40% to 60% for several hours, thus providing an ideal condition to study the improvement of cardiac function by synchronously stimulating the latissimus dorsi muscle to contract with the heart. When the muscle fibers were oriented in an ideal direction, a significant increase in cardiac output (44%), stroke work index (126%), and arterial systolic pressure (57%) were observed. These results clearly documented the augmentation of depressed ventricular function by stimulating a conditioned skeletal muscle with proper fiber orientation over the heart.

*By Invitation

F3. Oxygen Utilization in Postischemic "Stunned" Myocardium

JOSEPH E. BAVARIA, SATOSHI FURUKAWA*,*

GERHARD KREINER, MARK B. RATCLIFFE*,*

DANIEL K. BOGEN and L. HENRY EDMUNDS, JR.*

Philadelphia, Pennsylvania

We tested the hypothesis that oxygen (0_2) consumption increases after reversible myocardial ischemia. Left ventricular (LV) 0_2 consumption (LV02) before and after 20 minutes of warm (37°C) ischemia was related to the integral of LV systolic wall stress (SSI) at different afterloads in 16 sheep. LV coronary blood flow (CBF) was measured by ultrasonic Doppler and coronary sinus 0_2 saturation by fiberoptic oximetry. LV pressure was measured directly by Millar catheter; LV volume and wall thickness was calculated from sonomicrometry measurements (4 axes) using

an ellipsoid model. Afterload was varied by partial inflation of a descending thoracic aortic balloon. Animals were cannulated for cardiopulmonary bypass; preishemic measurements were obtained (n=129); bypass was started; the aorta was clamped 20 min. (CBF = 0); and 89 measurements were obtained 45-90 minutes after release of the aortic clamp. LV wall stress throughout systole (SSI) was calculated and integrated from continuous computer generated LV pressure and volume measurements (mmHg*sec). LV02 (ul/100gm/beat) was olotted as a function of SSI for ore- and DOstischemic hearts.

	Regression Equation	**n**	**r-value**
Preischemic:	LV02 = 1.15 (SS) + 18.3	129	0.78
Postischemic:	**LV02 = 4.35 (SSI) + 5.6**	89	0.65

LV02 is linearly related to SSI in both pre- and postischemic hearts. However, the 378% increase in slope (p = .005) indicates a massive increase in 0_2 consumption of postischemic "stunned" myocardium, severe impairment of 0_2 utilization efficiency and increased vulnerability to ischemic necrosis if coronary vessels are diseased.

*By Invitation

F4. Augmenting Intracellular Adenosine Improves Postischemic Myocardial Recovery

STEVEN F. BOLLING, LARRY E. BIES*,*

KIM P. GALLAGHER and EDWARD L. BOVE*

Ann Arbor, Michigan

The use of cardioplegia during surgically-induced ischemia greatly reduces myocardial metabolic requirements. However, adenosine triphosphate (ATP) depletion may occur, resulting in poor functional recovery after ischemia. This study investigated if augmenting intracellular adenosine by delivering exogenous adenosine or by inhibiting adenosine degradation with 2-deoxycoformycin (DCF, a non-competitive inhibitor of adenosine deaminase), during cardioplegic arrest, could enhance myocardial functional and metabolic recovery following ischemia. Isolated, perfused rabbit hearts were subjected to 120 minutes of hypothermic (34 °C) cardioplegic-induced ischemia. Controls received St. Thomas cardioplegia (CTL); remaining hearts received cardioplegia containing 200 mM adenosine (ADO), or 1 mM DCF or combined ADO/DCF. Functional results are 45 min after reperfu-sion, (mean ± SEM,*p < .05 vs CTL):

	N	**(%DP)**	**Î”EDP/Î”EDV**	**CK-loss (IU/L)**
CTL	23	38 ± 4	74 ± 10	424 ± 11
ADO	10	66 ± 7*	41 ± 6*	105 ± 11*
DCF	8	59 ± 2*	52 ± 4*	188 ± 10*
ADO/DCF	10	75 ± 2*	31 ± 1*	104 ± 9*

Following ischemia and reperfusion, recovery of developed pressure (%DP) and post ischemic diastolic stiffness (AEDP/AEDV, the slope of linear end-diastolic pressure-volume curves) was significantly better in treated hearts when compared with control. Creatine kinase (CK) loss, a reflection of ATP wastage, was less in all treated hearts. To determine if ADO or DCF minimized depletion of ATP during ischemia or accelerated repletion of ATP in the postischemic period, Nucleotide levels were obtained before, during and after ischemia. Metabolic results show myocardial adenosine (AD) and ATP as mM/mg protein, (mean ± SEM,*p<.05 vs. CTL).

	Baseline		Ischemia		1 min p reflow		15 min p reflow	
	AD	**ATP**	**AD**	**ATP**	**AD**	**ATP**	**AD**	**ATP**
CTL	2.2 ± .04	2. 3 ± .01	2.0 ± .12	0.8 ± .01	1 .9 ± .05	1.8.05	1 .5 ± .07	1.3 + .19
ADO	2.4 ± .06	2.6 ± .11	5.6 ± .17*	0.8 ± .04	5.9 ± .09*	5.9 ± .05*	4.0 ± .11*	6.9 ± .07*
DCF	2.0 ± .13	2.2 ± .09	2.9 ± .09*	0.8 ± .01	3.1 ± .06*	2.4 ± .06*	2.9 ± .06*	2. 7 ± .05*

During ischemia, ATP fell equally in all groups, indicating that ADO and DCF did not alter ischemia-induced depletion of ATP. However, intracellular adenosine was augmented in treated hearts. Consequently, during reperfusion ADO and DCF hearts had significantly enhanced ATP levels suggesting that augmenting myocardial adenosine accelerated repletion of ATP postischemia. In conclusion, adenosine and DCF augment intracellular adenosine and allow better metabolic ATP repletion following ischemia, improving post ischemic myocardial functional recovery.
*By Invitation

F5. Increased Tolerance of the Immature Myocardium to Hypoxia and Ischemia by Intravenous Metabolic Support

PIERRE L. JULIA, EDWARD R. KOFSKY*,*

*GERALD D. BUCKBERG, HELAN I. BUGYI**

*and HELEN YOUNG**

Los Angeles, California

Hypothesis: A substrate enriched intravenous solution increases tolerance of the immature myocardium to acute hypoxia and allows a better recovery after subsequent ischemia.

Methods: Thirteen neonatal puppies (2-4 kg) underwent one hour of acute hypoxia (pO_2 = 25 to 30 mmHg), followed by 45 minutes of normothermic global ischemia on total vented bypass and normal blood reperfusion. Ventricular function was assessed by inscribing Starling function curves and measuring stroke work indices (SWI) before hypoxia and after reperfusion. Seven puppies (control), received normal saline infusion at 4 cc/kg/hour. Six other puppies received a 4 cc/kg/hour intravenous infusion of Glutamate/ Aspartate, Glucose Insulin Potassium, Mercaptopropionylglycine (MFC), Carnitine and Catalase, during hypoxia and reperfusion.

Results: In control hearts, acute hypoxia depleted myocardial glutamate and Aspartate by 52%* and 48%* caused severe hemodynamic deterioration (55% decrease of SWI)*; three of seven (43%) required premature institution of bypass. Post-ischemic LV function recovered to only 40% of control levels*. In contrast, IV metabolic infusions maintained tissue Glutamate** and Aspartate** in treated hearts during hypoxia, and allowed cardiac index to raise 20%*; all treated hearts tolerated 1 hour of hypoxia, and stroke work recovered 70%** of SWI after subsequent ischemia.

Conclusions: Imparied tolerance of immature hearts to acute hypoxia and subsequent ischemia is due to substrate depletion. This impairment can be reduced by intravenous metabolic support during hypoxia and reperfusion and leads to improved recovery of post-ischemic function.

*$p<0.05$ vs pre-hypoxia,**$p<0.05$ vs control group

*By Invitation

F6. Leukocyte Depletion Ameliorates Free-Radical Mediated Lung Injury Following Cardiopulmonary Bypass

KO BANDO, RAVI PILLAI*, DUKE E. CAMERON*,*

JEFFREY D. BRAWN, JERRY A. WINKELSTEIN*,*

GROVER M. HUTCHINS, BRUCE A. REITZ and WILLIAM A. BAUMGARTNER*

Baltimore, Maryland

Activated leukocytes and oxygen free radicals have been implicated in the pathogenesis of lung injury following Cardiopulmonary bypass (CPB). To determine whether leukocyte depletion could prevent this injury, a dog model simulating routine cardiac surgery was used. Mongrel dogs (11-17 kg) were placed on Cardiopulmonary bypass using a bubble oxygenator and cooled to 27°C. Following aortic cross-clamping and cardioplegic arrest for 90 min, animals were rewarmed, weaned from CPB, and stabilized for 90 min. Control animals (n = 5) were perfused on CPB with whole blood. Leukocyte-depleted (LD) animals (n = 5) had a leukocyte filter incorporated in the CPB circuit. Pre-CPB leukocyte counts (WBC) were similar in the groups. On CPB, WBC decreased by 64% in controls and by 97% in LD animals. After CPB, LD resulted in less intrapulmonary leukocyte sequestration, as determined by the difference in right and left atrial WBC counts. 90 min after CPB, WBC returned to pre-CPB levels in controls, but remained low in the LD group. Free radical activity was assayed by spectrophotometric measurement of plasma conjugated dienes and was significantly reduced by LD. Pulmonary function [paOj on $Fi0_2$=1, extravascular lung water (EVLW), pulmonary vascular resistance (PVR)] 90 minutes after CPB was better preserved in LD animals; only controls had histologic evidence of in-travascular leukocyte aggregation, perivascular hemorrhage, and focal alveolar injury.

	WBC				Lung Function			
	Pre-CPB	**On CPB**	**Posl-CPB**	**RA-LA**	**Free Rad Act**	**PaO_2**	**EVLW**	**PVR**
Control	6753 ± 638	2432 ± 197	6048 ± 817	1320 ± 217	287 ± 16	13.2 ± 1.0	607 ± 80	
LD	6534 ± 712	191 ± 76*	747 ± 160*	220 ± 45*	1.06 ± .06*	443 ± 23*	8.2 ± 0.5*	126 ± 33*

(Values are mean ± SEM;*p<0.05 compared to control; WBC in cells/mm^3; Free Radical Activity in absorption units @ 233nm 30 minutes after CPB; paO_2 in torr; EVLW in cc/kg; PVR in dynes sec cm^{-5})

These data suggest that circulating leukocytes contribute to lung injury during CPB and are associated with increased oxygen radical activity, pulmonary edema, and vasoconstriction. Leukocyte depletion substantially reduces the pulmonary injury seen after CPB.
*By Invitation

F7. Nuclear Scan Guided Rib Biopsy

DARROCH W.O. MOORES, BRUCE LINE*,*

STANLEY W. DZIUBAN and MARTIN F. McKNEALLY*

Albany, New York

The bone scan is a sensitive screening device which is frequently used to stage patients with known or suspected malignancy. An abnormal bone scan is associated with corresponding normal radiographs in approximately 50% of cases. Definitive tissue diagnosis of the bone lesion is often needed to determine optimal therapy, but localization of the lesion is imprecise unless it is palpable. Use of the nuclear scan to localize and mark the rib enhances the precision of the biopsy procedure.

Thirty-three consecutive cancer patients with suspicious rib abnormalities on bone scan underwent nuclear scan guided biopsy. Each patient had a repeat localizing scan with TC-99 MPD radionuclide on the day of the planned biopsy. The site of abnormality was marked with methylene blue injected into the skin overlying the lesion and down to the periosteum at the specific site. The patient was then taken to the operating room and underwent excision of the marked area, through a small incision.

Pathological abnormality was identified in all but one of the resected specimens, an accuracy of 97%. Despite a presumed or proven diagnosis of cancer in 33 patients, 16 specimens (48%) showed benign pathology. There were no complications associated with this technique which reduces the morbidity and increases the precision of rib biopsy.

*By Invitation

F8. Combination Immunotherapy Using Low Dose Interleukin-2 (IL-2) and Tumor Necrosis Factor-Alpha (TNF) **for Non-Small Cell Lung Cancer**

STEPHEN C. YANG, LAURIE OWEN-SCHAUB*,*

JERE LICCARDELLO, WAUN K. HONG*,*

ELIZABETH A. GRIMM and JACK A. ROTH**

Houston, Texas

Sponsored by: Clifton F. Mountain, Houston, Texas

The purpose of this study was to define alternative activation pathways for the induction of lymphokine-activated killer (LAK) activity against primary lung cancer cells. Single cell suspensions from 32 fresh surgical non-small cell lung cancer (NSCLC) specimens were tested for natural killer (NK) and LAK susceptibility in a 4-hr ^{51}Cr-release assay. All tumor samples were sensitive to LAK lysis induced with IL-2 alone. Using a combination of IL-2 and TNF, lytic activity was increased ($p<0.01$) a mean of 4-fold against 31 of the 32 specimens (range 0.7-16.3). All samples were resistant to lysis by NK cells and TNF alone. We initiated a clinical trial to test the toxicities, anti-tumor efficacy, and immunomodulatory effects of using combination low dose IL-2 and TNF in patients with Stage Illb and IV NSCLC. All patients received a continuous daily I.V. infusion of IL-2 at $1x10^6$ Cetus Units/m^2 on days 1-5. Seven patients were given a single daily I.M. dose of TNF at 25 /-ig/m^2 simultaneously with IL-2 on days 1-5 (Level I), with their TNF dose doubled after two cycles. Four patients were started at a TNF dose of 50 /-ig/m^2 with IL-2 (Level II). These doses of either agent alone were ineffective in previous studies. Treatment cycles were given every 3 weeks. All toxicities were reversible, and patients did not require ICU monitoring during therapy. Measurable tumor regression occurred in three patients (all at level I). In one patient, there was a major clinical response with complete resolution of pulmonary metastasis, which has lasted 7 months. Three patients had radiographic stabilization of disease (mean = 9 weeks) before progression. All patients had augmented LAK and NK activity while on therapy, assessed by *in vitro* cytolysis of Raji and K562 targets, respectively, with autologous lymphocytes.

Phenotypic analysis of these lymphocytes revealed a predominance of CD3+ cells during therapy, with varying levels of CD4 + and CD8 + populations. We conclude that IL-2 and TNF have synergistic efficacy in the treatment of NSCLC.

*By Invitation

F9. Angiogenic Factor: A Possible Mechanism for Neovascularization Produced by Omental Pedicles

RAYMOND CARTIER, ISABELLE BRUNETTE*,*

KAZUHIRO HASHIMOTO, WILLIAM M. BOURNE**

and HARTZELL V. SCHAFF Rochester, Minnesota

Recent success in single and double lung transplants may be credited to improved immunosuppressive regimens and bronchial omentopexy which reduces the incidence of early dehiscence and late stenosis. To determine a possible mechanism by which omental pedicles protect bronchial anastomoses from ischemia we studied the angiogenic potential of a lipid extract of omentum. A rabbit cornea model was used to quantify neovascularization produced by methanol-chloroform extract of homogenized autologous omentum (AO) or perirenal fat. In 22 anesthetized rabbits, 10 µlof omental lipid extract was injected in the cornea. In each animal the opposite eye was used as a control and was injected with a similar volume of extract prepared from perirenal fat. The side of injection of AO was randomized and was not known to the investigator assessing neovascularization on days 4, 7, 14, and 21 following injection. Neovascularization was measured by a point-counting method of microphotography and was expressed as the surface area (mm2), the relative density (point-count/mm2) and the absolute density (point-count/total surface) of new blood vessels. At day 4 after injection, neovascualrization in corneas injected with AO was significantly ($p<.05$) greater than control for all the parameters studied; absolute density of neovessel growth induced by AO was 3 times that of control. The angiogenic effect diminished with time, and by 21 days after injection corneal neovascularization was comparable for the two groups. Our results suggest that the lipid fraction of omentum has angiogenic activity which may stimulate neovascularization in ischemic tissues. Lack of sustained activity may be due to washout by neovessels or local tissue metabolism of angiogenic factor(s).

*By Invitation

FORUM ALTERNATE

Improved Myocardial Preservation During Cold Storage for Transplantation Using Substrate Enhancement

CONSTANCE HAAN, HAROLD L. LAZAR*,*

SAMUEL RIVERS, CHERYL COADY* and*

RICHARD J. SHEMIN
Boston, Massachusetts

Cold storage techniques used in cardiac transplantation may result in depressed ventricular function. Previous studies have shown that substrate enhancement with the amino acid L-Glutamate

minimizes ischemic damage when added to cardioplegic solutions during ischemic arrest. This study was therefore undertaken to determine whether substrate enhancement with L-Glutamate during periods of cold storage would improve ventricular function in transplanted hearts.

Sixteen rabbit hearts were rapidly excised and perfused with Krebs solution (37°C) on a Langendorff apparatus. Following control measurements of LV function and coronary blood flow, all hearts were arrested with hypothermic (4°C), crystalloid, potassium (28mEq/L) cardioplegia and stored at 3°C for 3 hours. They were then rewarmed and reperfused with Krebs solution for 60 minutes at which time final post-ischemic measurement were made. L-Glutamate (4mM) was added to both the cardioplegic and reperfusate solutions in 8 hearts while 8 others received no L-Glutamate. Results are Mean±SE;*p < .02; + LVEDV = .8 ml; + + MAP = 75 mmHg.

	Glutamate	**Non-Glutamate**
+dP/dt+ (% recovery)	87 ± 4*	64 ± 4
-dP/dt+ (% recovery)	94 ± 10*	73 ± 5
LVEDP + (mmHg) preischemia vs postischemia	34±3 vs 34 ±5	35 ± 4 vs 56 ± 5*
Coronary blood flow + + (ml/min)preischemia vs postischemia	53±4 vs 49±3	63 ± 2 vs 41 ± 2*

We conclude that the addition of L-Glutamate to hearts during periods of cold storage and subsequent reperfusion results in superior recovery of myocardial contractility, relaxation, compliance, and coronary blood flow. Substrate enhancement with L-Glutamate may become an important addition to cold storage techniques during cardiac transplantation.

*By Invitation

TUESDAY MORNING, May 9, 1989

9:00 a.m. SCIENTIFIC SESSION - HYNES BALLROOM

14. Phrenic Nerve Pacing of the Quadraplegic Patient

JOSEPH I. MILLER, JAMES A. FARMER,*

WILLIAM H. STUART and DAVID F. APPLE**

Atlanta, Georgia

Phrenic nerve pacing (PNP) is a modality that can be utilized to free a quadraplegic patient (pt) from ventilatory dependency. During a 4 year period (1984-1988), 23 pts with an age range of 17 to 63 years (mean 31 years) underwent implantation of a phrenic nerve (PN) pacemaker because of ventilatory dependency secondary to quadraplegia. Fourteen pts had a unilateral PN implant, and 9 pts had a bilateral PN implant. The time of injury to implant was 12 to 16 weeks. The site of implant was the cervical PN in 13 pts, and thoracic PN in 10 pts. During the past 24 months, only a transthoracic approach has been utilized. Indication for pacing was failure to be weaned from ventilatory support in all pts. Failure to stimulate the PN at implant was noted in 3 pts, despite preoperative testing indicating an acceptable response. There were no deaths, and minor complications developed in 4 pts. Follow-up is available in 21 of 23 pts. Eight pts are completely off the ventilator; 9 pts are markedly improved, but on the ventilator at night; 3 pts are moderately

improved; 3 pts showed no response at implant. Three pts required re-exploration for component failure from 6 weeks to 18 months after implant. A complete discussion of the surgical technique, PN testing, and PN training will be presented.

*By Invitation

15. Esophageal Reconstruction for Complex Benign Esophageal Disease

*F. HENRY ELLIS, JR. and S. PETER GIBB**
Burlington, Massachusetts

Conservative operations on the esophagus are currently preferred to radical resective procedures for benign esophageal disorders. However, our results after reoperative fundoplication and myotomy procedures are less good than those that follow primary procedures and our results after gastroplasty-fundoplication are suboptimal suggesting that a more radical approach be adopted for selected complex esophageal problems. We have therefore reviewed our experience with such operations to determine whether such an approach is warranted and which operation has the best results.

From January 1970 to October 1988, 32 reconstructive procedures for complex benign esophageal disease were performed representing 6.7% of all operations done for benign disorders of the distal esophagus. The procedures employed were esophagogastrostomy (6), colon interposition (7), and esophagogastrostomy, antrectomy and Roux-Y diversion (19). These 32 patients had undergone a total of 62 prior operations, an average of nearly two per patient. Pertinent associated disorders were achalasia in 11 patients, Barrett's esophagus in 3, one of whom also had scleroderma, and one patient each with scleroderma, lye stricture, diffuse esophageal spasm, and giant leiomyoma. Reconstruction was required because of severe gastroesophageal reflux disease in 26 patients, 20 of whom had an esophageal stricture. Other indications for reconstruction included three patients with esophageal perforation and mediastinitis requiring esophageal defunctioning and one patient each with an infarcted gastroplasty tube, lye stricture, and giant leiomyoma. Esophageal resection was required in 24 patients and two underwent cardiopksty. There was one hospital death and 9 (29%) postoperative complications.

Comparison of the results in the three surgical groups is difficult because of the small sample size. Even so, certain trends are apparent. Persistent reflux constitutes a potential hazard of simple esophagogastrectomy even when combined with fundoplication. Results after colon interposition are somewhat better but may be compromised by anastomotic leakage and resultant stricture formation. Successful relief of reflux and dysphagia coupled with the paucity of postoperative complications characterize the results of resection coupled with antrectomy and Roux-Y diversion. We currently prefer its use in properly selected cases of complex benign esophageal disease.

*By Invitation

16. Maintenance of Hemostasis by Aprotinin During Cardiopulmonary Bypass. High Continuous Versus Low Single Dosage Aprotinin

CH. R.H. WILDEVUUR L. EIJSMAN*,*
K. J. ROOZENDAAL and W. VAN OERVEREN**

Amsterdam and Groningen, Netherlands

Sponsored by: John W. Kirklin, Birmingham, Alabama

The efficacy of aprotinin to reduce postoperative bloodless after car-diopulmonary bypass (CPB) in routine coronary artery grafting and reopera-tions is well established. Aprotinin appears to preserve the platelet adhesive capacity and to inhibit the intrinsic clotting, kallikrein and fibrinolytic system. The capillary bleeding, which is the main cause of the increased postoperative bleeding after CPB is thought to be a platelet defect, which may be caused by blood-material contact and by proteolytic attack from activated plasmatic systems. These systems as well as platelet adhesive receptors alter mainly during the first 5 minutes of CPB. Therefore it seems of prime importance to overcome this period with protective measures. We studied the efficacy of aprotinin in three groups of patients. Group I received placebo, without aprotinin (n = 28). Group II received aprotinin during the whole operation (6.10^6 KIU) (n = 28). Group III received aprotinin in the pump prime only (2.10^6KIU) (n = 12).

Perioperative bloodless, determined by the loss of hemoglobin (Hb) in gauzes and suction system was 93 g Hb in Group I (placebo) and reduced in the aprotinin treated Groups II and III to 68 and 46 g Hb. respectively ($p<0.001$). Postoperative bloodloss was dramatically reduced by aprotinin treatment from around 40 g Hb in Group I to 21 and 16 g Hb in Group II and III, respectively ($p<0.001$). The mean bleeding time increased by 220 seconds in Group I and by 170 and 160 seconds in Group II and III, resp. A consequence of the reduced bloodloss by aprotinin treatment was that the volume and percentage postoperative blood transfusions were reduced by two third. Despite this, the aprotinin treated patients left the intensive care unit with a similar Hb as the placebo patients (6.5 mmol versus 6.4 mmol).

Since administration of a single bolus aprotinin in the prime solution has the same clinical benefit as the continuous infusion of the high dosis, a practical routine application of aprotinin in clinical CPB is envisaged.

10:00 a.m. Intermission - Visit Exhibits

*By Invitation

10:45 a.m. SCIENTIFIC SESSION - HYNES BALLROOM

17. Surgery for Infarct Related Ventricular Septal Defects Improved Early Results Combined with Analysis of Late-Functional Status

PETER D. SKILLINGTON, ROBERT H. DA VIES*,*

KEITH D. DAWKINS, NEVILLE CONWA Y*,*

ROBERT K. LAMB, DARRYL F. SHORE*,*

JAMES L. MONRO, KEITH ROSS and ANDREW J. LUFF**

Southampton, England

Sponsored by: Gary W. A kins, Boston, Massachusetts

101 patients (mean age 64.9 years) underwent surgical correction of post infarction ventricular septal defect (VSD) at this institution over a 15 year period (1973-1988). The overall early mortality

was 20.8%, although the most recent experience with 36 patients (January 1987 - October 1988) has seen this fall to 11.1%. Factors found to significantly influence early death were:

(1) Site of infarction: Anterior 12.1%, inferior 32.6% (p = .022);

(2) Time interval between infarction and surgery: Less than 1 week 34.1%, over 1 week 10.5% (p = .008);

(3) Cardiogenic shock: Present 38.1%, absent 8.5% (p = .001).

Non-significant variables included pre-operative renal function, age, and concomitant coronary artery bypass.

Of the 80 hospital survivors, eight (8) were subsequently found to have recurrence requiring re-operation with survival in seven (7). Late follow up is 99% complete and reveals an actuarial survival for all 101 patients of 70.3% at 5 years (95% confidence interval (60.6 - 80.0), and 40.0% at 10 years (95% confidence interval 21.7 - 58.4). The functional status of the surviving patients has been analyzed by a graded treadmill exercise protocol, whilst left ventricular functional assessment was by nuclear scan (ejection fraction calculated by M.U.G.A.), with additional information on miral and tricuspid valve function by echocardiogram. Colour Doppler has been used to determine the presence of residual V.S.D.

The findings are that most late survivors have limited exercise tolerance related to both cardiac and non-cadiac factors. Left ventricular function is moderately impaired (mean ejection fraction = 0.38). However, many patients are elderly and have adapted to their residual symptoms without significant changes to their lifestyle.

*By Invitation

18. Cardiogenic Shock: A Medical/Surgical Emergency

BRADLEY S. ALLEN, ELIOT R. ROSENKRANZ*,*

GERALD D. BVCKBERG, JAKOB DAVTYAN,*

*HILLEL LAKS and DAVIS C. DRINK WATER**

Los Angeles, California

Five years ago we reported our initial encouraging experience using warm (37°C) amino acid enriched blood cardioplegia induction in patients undergoing emergency CABG for cardiogenic shock. The use of aspartate/ glutamate blood cardioplegia allows for resuscitation of the heart with reversal of the LV power failure unlike medical therapy where hospital mortality is > 90% without operation. This report a) confirms these results in a larger population (78 patients) with up to 6 years follow-up, b) emphasizes operative strategy, and c) identifies predictive clinical characteristics of early and late mortality to improve patient selection and timing of operation in this otherwise fatal disease.

Seventy-eight consecutive patients on maximum inotropic and intra-aortic balloon support underwent emergency CABG (3.4 ± 1* days) post-infarction for severe LV power failure (SWI < 25, LAP > 20 mmHg). All received 37°C glutamate and aspartate blood cardioplegia (BCP) induction, multidose cold (r °C) BCP replenishment and warm BCP reperfusate. Viable areas were grafted first to ensure cardioplegic distribution.

LV power failure was reversed in 94% of patients; 73 of 78 patients had discontinuation of inotropes and intra-aortic balloon. Early mortality (< 30 days) was only 7% (3/45) with early operation (< 18 hours), and rose to 33%** (11/33) if operation was delayed > 18 hours. Six of 14 early deaths were due to progression of pre-op organ failure despite reversal of shock. Thirteen of

64 early survivors died 11 ± 5 months post-operatively of end-stage heart failure (13/78), 17% late mortality. Late mortality after early operation (< 18 hr) was 9.5% (4/42) vs 41% (9/22) after late operation (> 18 hr).** Non-survivors (early and late) had a higher incidence of extending vs acute evolving infarction (12/63 vs 1/15)**, b) longer delay from shock to operation (7/45 vs 20/33)**, more pre-op organ failure (6/27 vs 2/51)** and d) greater incidence of previous infarction (19/41 vs 8/37)**. Thirty-two of 51 late survivors (63%) remain physically active.

We conclude that cardiogenic shock should be considered a medical/ surgical emergency as early operation can reverse LV power failure in most patients. In order to accomplish this, a defined operative strategy using warm induction aspartate glutamate blood cardioplegia is necessary to resuscitate the myocardium. Post-operative mortality (early and late) is due principally to delay of operation leading to progression of preoperative organ failure or progression of underlying cardiac disease if infarction becomes established, "mean ± S.E.,**p<0.05

11:30 a.m. ADDRESS BY HONORED SPEAKER

TRANSPLANTATION OF KNOWLEDGE
Francis M. Fontan, Bordeaux-Pessac, France

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

12:15 p.m. Cardiothoracic Residents' Luncheon† - Independence Room, Sheraton

†Admission will be ticket only. Residents will be the guest of the Association.

*By Invitation

TUESDAY AFTERNOON, MAY 9, 1989

1:45 p.m. SCIENTIFIC SESSION - HYNES BALLROOM

19. Risk Factors for Pulmonary Thromboendarterectomy

PAT O. DAILY, WALTER P. DEMBITSKY,*

STEIN IVERSEN, KENNETH M. MOSER**

*and WILLIAM AUGER**

San Diego, California

Pulmonary thromboendarterectomy is emerging as an effective surgical procedure for incapacitating pulmonary hypertension secondary to chronic pulmonary embolism. However, patient-related risk factors and procedural complications associated with morbidity and mortality have not been described.

Since October 1, 1984, we have performed pulmonary thromboendarterectomy utilizing deep hypothermia and circulatory arrest in 103 consecutive patients (64% male, mean age 50 ± 16 (SD), range 19-81 years) in whom the exposure and dissection of the pulmonary arteries and methods for myocardial protection have been standardized. Ventilator dependency (‰¥5 days on respirator), the most common and severe postoperative complication, occurred in 27 patients. By univariate analysis, the occurrence of ventilator dependency was associated with long-standing symptoms (p<.01), severe right heart failure (p<.03), severely deranged pulmonary function tests (p<.02), and

longer cardiopulmonary bypass times (p<.03). Patients with postoperative neurological disorders (18) were found to have had prolonged total circulatory arrest times (69 ± 21 vs 53 ± 23 min, p<.02).

Hospital mortality was 11.7% (12/103). Causes of death were acute pulmonary hemorrhage or hemorrhagic edema (3), acute myocardial infarction (1), right heart failure (2), and acute respiratory (3) and secondary multi-organ failure (3). Factors associated with mortality were older age (61 ± 14 vs 49 ± 16 years, p<.03), prolonged cardiopulmonary bypass time (215 ± 42 vs 180 ± 47 min, p<.04), intraoperative pulmonary hemorrhage (16.7 vs 0%, p 0.01), and persistent elevated pulmonary vascular resistance (438 ± 141 vs 276 ± 142, p<.005).

Hospital mortality of 11.7% for pulmonary thromboendarterectomy is acceptable when compared to the approximately 25% rate for heart-lung transplantation which is the only therapeutic alternative. Factors associated with ventilator dependency were long standing symptoms, severe right heart failure, severely deranged pulmonary function tests, and longer cardiopulmonary bypass times while predictors of hospital mortality were older age, prolonged cardiopulmonary bypass times, intraoperative pulmonary hemorrhage, and persistent elevated pulmonary vascular resistance.

*By Invitation

20. Rupture of Thoracic Aorta Due to Blunt Trauma: A 15-Year Experience

R ADAMS COWLEY, STEPHEN Z. TURNEY,

JOHN R. HANKINS, AURELIO RODRIGUEZ,*

*SAFUH A TTAR and BELAVADI SHANKAR**

Baltimore, Maryland

Repair of rupture of the thoracic aorta from blunt trauma is associated with high mortality and major complications, especially paraplegia/paresis. This is the largest reported series of such cases. During the 15 years from 1971 through 1985, 114 patients with rupture of the thoracic aorta due to blunt trauma were admitted to a major trauma center. Mean age was 31.3 years (range, 15 to 80). Ninety were males and 24 were females, a 3.75:1 ratio. Of the 114, 88 (77.2%) survived initial resuscitation in the admitting area or operating rooms (AA/OR). Twenty-four of the 88 initial survivors (27.3%) died during or following surgical repair. Paraplegia occurred in 11 of the 88 survivors (12.5%).

Further analysis was done of the 83 cases admitted in the 10-year period from 1976 through 1985. Mean Injury Severity Score (ISS), excluding aortic injury, was 18.2. Twenty-six of the 83 (31.3%) died during resuscitation in the AA/OR. Six others died during surgical repair, and 12 died postoperatively leaving 39 survivors (39/83 or 47% of total admissions and 39/57 or 68.4% of survivors of resuscitation). Shunt/bypass adjunct was used to repair 36 injuries and 19 were repaired without adjunct. Paraplegia/paresis developed postoperatively in 10 of 51 survivors (19.6%), with 6 of 34 (17.6%) occurring in cases involving adjunct and 4 of 17 (23.5%) without adjunct (p N.S.). Hypotension during aortic cross-clamping occurred in 3 of 4 paraplegia cases done without adjunct and in 3 of 6 with adjunct (p N.S.). Other major complications occurred in 17 of the operative survivors, including 9 cases of adult respiratory distress syndrome, 3 cases of severe renal failure, 4 cases of severe sepsis, and 3 cases of pseudoaneurysm at graft-aorta anastomoses. Statistically significant risk of death or major complication was associated with higher ISS, larger presenting hemothorax, performance of other major surgery prior to aortic repair, longer aortic cross-clamp time, lower admission blood pressure, and less qualified surgeon.

There was no advantage in this series to using or not using shunt/bypass in preventing paraplegia. Mortality rates are realistic for a highly developed trauma system. Better techniques are needed for management of exsanguina-tion and to prevent paraplegia.

*By Invitation

21. Single Stage Management of Sternal Wound Infection

VALLUVAN JEEVANANDAM, CRAIG R. SMITH*,*

*ERIC A. ROSE, JAMES R. MALM, SEAN CAMPBELL**

*and NORMAN HUGO**

New York, New York

Median sternotomy wound infection is a significant source of morbidity following cardiac surgery. Accepted approaches in treating this complication include debridement, with either sternal closure over an irrigation system, or open dressings allowing for granulation and secondary closure. Muscle flaps are often used in subsequent procedures. In order to eliminate the need for multiple operations and to decrease hospital stay, a single stage procedure was developed and is compared to previous methods in treating sternal wound infections.

This report is based on a consecutive series of 47 sternal wound infections occurring in 2872 cardiac procedures (incidence 1.7%) over a four year period (1984-1988). The first 16 patients (group A) were treated with closed irrigation drainage (3), or debridement, dressing changes and closure either secondarily (4) or with a muscle flap (9). The subsequent 31 patients (group B) had a single stage procedure. Treatment grouping was not influenced by severity of infection. Immediately after the diagnosis of mediastinitis was made, the wounds were opened widely, necrotic tissue debrided, and sternum resected back to bleeding bone. Full thickness pectoralis major myocutan-eous flaps were created by dividing the muscle at the sternal insertion and elevating the flap off the anterior chest wall. The myocutaneous flaps were mobilized to close the sternal defect primarily. Closed-suction drains were placed under the flaps and were left in place for an average of 7 days. Other than suction drainage, no further attempt was made to obliterate dead space.

All patients had mediastinitis and sternal instability. 4 group A patients and 8 group B patients had osteomyelitis of the sternum. The causative organisms were similar in both groups: Staphylococcus epidermidis (Group A, n = 9, Group B, n = 14), Staphylococcus aureus (Group A, n = 3, Group B, n = 7), beta hemolytic Streptococcus (Group A, n=0, Group B, n = 3), gram negative organisms (Group A, n =4, Group B, n = 7). Antibiotics were directed by culture and sensitivity, and administered from 5 to 45 days (average 12 days). After operative treatment of mediastinitis, group B patients had a shorter mean hospital stay (20 vs 42 days, $p<0.05$) and fewer reoperations (4/31 vs. 13/16, $p<0.05$). Infection did not recur in any group B patient at a mean follow-up of 24.5 months (3 - 48 months). Mortality rates were not statistically different (Group A 6.2%, Group B 12.5%). Deaths were all due to cardiac or pulmonary dysfunction, not infection.

Single stage treatment consisting of debridement, immediate mobilization of bilateral pectoral myocutaneous flaps, and primary closure significantly reduces the morbidity caused by sternal wound infections, and should be adopted as the treatment of choice for this complication.

2:45 p.m. Intermission - Visit Exhibits

*By Invitation

3:30 p.m. SCIENTIFIC SESSION - HYNES BALLROOM

22. Surgical Management of Wolff-Parkinson-White Syndrome in Infants and Small Children

FREDA. CRAWFORD, JR., PAUL C. GILLETTE,*

MARTHA R. STROUD and VICKI ZIEGLER**

Charleston, South Carolina

Surgical ablation of accessory conduction pathways (ACP) has rarely been reported in infants and small children with Wolff-Parkinson-White Syndrome. In the interval January 1985 to September 1988, 19 infants and children age 5 or less have undergone surgical ablation of ACP because of recurrent supraventricular tachycardia (SVT). There were 12 (63%) males and 7 (37%) females. Age ranged from 4 to 66 months (mean 33.8 months). Nine infants were less than 24 months old. Weight ranged from 5.5 to 21.6 kg (mean 13.2 kg). All 19 patients were symptomatic with duration of symptoms ranging from 3 to 63 months (mean 21 months). ACP were classified preoperatively as left free wall in 4 (21%), right free wall in 9 (47%) and posterior septal in 6 (32%). No patient was recognized preoperatively to have multiple pathways. Left ventricular function was abnormal in 4 (21%) preoperatively. Free wall pathways (13) were surgically dissected and septal pathways (6) were cryoablated at -70°C. Mean cardiopulmonary bypass time was 60 ± 4 minutes. Mean cross-clamp time was 42 ± 2 minutes in those undergoing surgical dissection. Mean postoperative stay was 6.4 ± 0.2 days. There were no deaths, no significant postoperative complications, no incidence of complete heart block, and all patients were considered cured at the time of discharge. At a mean follow-up of 12.7 months, 18 (94.7%) remain cured. One patient with Ebstein's anomaly and a right freewall pathway developed a recurrent SVT 3 months postoperatively and repeat electro-physiologic study has shown a previously unsuspected anterior septal pathway. Ventricular function returned to normal in all 4 patients who had abnormal function preoperatively. Surgical ablation of accessory conduction pathways can be safely carried out in infants and small children with results equal to those obtained in adults.
*By Invitation

23. Peri-Nodal Cryosurgery for AV Node Reentry Tachycardia

JAMES L. COX, T. BRUCE FERGUSON, JR.,*

BRUCE D. LINDSAY, DENNIS M. CASSIDY* and*

*MICHAEL E. CAIN**

St. Louis, Missouri

AV node reentry tachycardia is the most common cause of paroxysmal atrial tachycardia (PAT). Available non-pharmacologic therapies include: 1) catheter ablation or cryosurgical ablation of the His bundle and insertion of a permanent pacemaker, 2) surgical dissection around the AV node or discrete cryosurgery of the peri-AV nodal tissues in an attempt to divide or ablate only one of the dual AV node conduction pathways responsible for the tachycardia while leaving the other intact. This report describes 20 consecutive patients with AV node reentry tachycardia who underwent the discrete cryosurgical procedure between August 13, 1982 and October 10, 1988.

The first patient in this series, a 38 year-old female, represents the first surgical cure of a patient with refractory AV node reentry tachycardia by a procedure designed to treat this arrhythmia. The 11 female and 9 male patients ranged in age from 12-56 years with an average age of 29 years. Eleven of the 20 patients (55%) had the WPW syndrome. Other associated arrhythmias included atrial flutter/fibrillation (1), right atrial reentrant tachycardia (1), junctional tachycardia (1), and a Mahaim fiber (1). Associated anatomic abnormalities included Ebstein's Anomaly in 2 patients and a large right atrial aneurysm in 1 patient. The discrete cryosurgical procedure was performed through a right atriotomy in the normothermic beating heart. Multiple 3mm diameter cryolesions were placed around the borders of the Triangle of Koch on the lower right atrial septum to alter the input pathways of the AV node.

There were no operative deaths in this series of patients. Postoperatively, all 20 patients have normal A-V conduction and no heart block has occurred in any patients during the follow-up period. All patients have remained free of AV node reentry tachycardia (and of the WPW syndrome) and none have required postoperative anti-arrhythmic drugs for either of these arrhythmias. We consider this simple, safe, easily performed and uniformly successful operation to be the procedure of choice for the treatment of medically refractory AV node reentry tachycardia.

*By Invitation

24. Transatrial Balloon Technique for Activation Mapping During Surgery for Recurrent Ventricular Tachycardia

LYNDA L. MICKLEBOROUGH, EUGENE DOWNAR*,*

AKIHIKO USUI, LOUISE HARRIS*, IAN PARSONS**

*and GORDON GRAY**

Toronto, Ontario, Canada

Sponsored by: Tirone E. David, Toronto, Ontario, Canada

Results of surgery for recurrent ventricular tachycardia have improved since methods of mapping have been developed which allow a directed approach to the problem. Using standard operative techniques (ventriculotomy and introduction of a hand-held probe or multiple electrode array), it has not always been possible to obtain satisfactory endocardial activation maps during the tachycardia. We have recently developed a new transatrial balloon approach which has greatly facilitated intraoperative mapping in these patients. A videotape will be presented which demonstrates this technique with particular attention given to the following:

1) description of the balloon array of 112 silver bead electrodes

2) technique for pressure-volume calibration of the balloon

3) technique of cardiopulmonary bypass and surgical approach for balloon insertion across the mitral valve

4) recording of local electrograms and on-line video display of the activation sequence used for intraoperative identification of the "target area"

5) correlation between position of target electrodes on the balloon and the internal geometry of the heart

6) choice and application of the appropriate ablation technique.

We have used this technique in 34 consecutive patients referred for surgical control of ventricular arrhythmias, 35% of whom had only nonsustained ventricular tachycardia at their preop electrophysiologic study. Thirty-eight percent of these patients had a grade IV ventricle, 32% had a previous posterior infarct, and 50% did not have a resectable aneurysm. All of these factors have been associated with poor operative results in other series. With the transatrial balloon technique we were able to induce and map ventricular tachycardia in 100% of patients (average 2.5 ± 1.3 morphologies per patient). Using a variety of ablation techniques (endocardial excision, cryoablation or balloon electric shock ablation) we have achieved surgical control of the arrhythmia in 80% of patients with an operative mortality of 15%. We recommend transatrial balloon mapping as the procedure of choice for intraoperative identification of arrhythmogenic foci in patients with recurrent ventricular tachycardia.
*By Invitation

25. Transannular Mapping and Cryoablation: A New Surgical Approach for Cure of Ventricular Tachycardia

GERALD M. LAWRIE, ANTONIO PACIFICO* and
*RAJ R. KAUSHIK**

Houston, Texas

Patients who require electrophysiological map-guided direct surgical procedure usually have depressed left ventricular function. In our own experience in 82 patients, the mean preoperative EF was 36%, range 11-61%. Operative mortality in most series has been in the range of 10-15% and in ours was 12.2%. Patients with no discrete left ventricular aneurysm who have diffuse ventricular impairment are at special risk.

In order to attempt to reduce this risk, we have begun to employ transan-nular cryoablation of arrhythmogenic areas without ventriculotomy or ventricular resection.

A balloon electrode array carrying 80 bipolar electrode pairs is employed for endocardial mapping through either the mitral or tricuspid annulus. Cryoablation has then been performed via the mitral annulus in two patients, the tricuspid annulus in two patients, and the aortic annulus in one patient.

There were three males and two females. Coronary disease was present in three patients and idiopathic cardiomyopathy in two patients. Mapping and cryoablation were uneventful. All patients survived operation. Ventricular tachycardia was non-inducible at post-operative electrophysiologic study in all patients and the ejection fractions were unchanged.

In conclusion, in patients who do not require resection of ventricular aneurysms for hemodynamic reasons, transannular mapping and cryoablation without ventriculotomy is our procedure of choice.

4:50 p.m. EXECUTIVE SESSION (Members Only)

7:00 p.m. PRESIDENT'S RECEPTION (Tickets Required) REPUBLIC BALLROOM, SHERATON

*By Invitation

WEDNESDAY MORNING, May 10, 1989

7:30 a.m. SYMPOSIUM I - AORTIC DISSECTION

MODERATOR: Randall B. Griepp, M.D., New York, NY

PANELISTS: E. Stanley Crawford, M.D., Houston, TX

M. Arisan Ergin, M.D., New York, NY

Nicholas Kouchoukos, M.D., St. Louis, MO

D. Craig Miller, M.D., Stanford, CA

7:30 a.m. SYMPOSIUM II - SURGERY OF EMPHYSEMA

MODERATOR: Jean DesLauriers, M.D., Sainte-Foy, Quebec, Canada

SURGICAL APPROACH TO BULLOUS EMPHYSEMA

Edward A. Gaensler, M.D., Boston, MA

OPERATIVE STRATEGIES AND RESULTS OF SURGERY

Jean-Paul Whitz, M.D., Strasbourg, France

ROLE OF SURGERY IN DYSPLASTIC TRACHEA

Mr. H. Hsrzog, Basel, Switzerland

NEW PERSPECTIVES IN SURGERY FOR DIFFUSE NON BULLOUS EMPHYSEMA

Marcel Dahan, M.D., Toulouse, France

9:00 a.m. SCIENTIFIC SESSION - HYNES BALLROOM

26. 30 Year Follow-Up of Superior Vena Cava-Pulmonary Artery Shunts

GARY S. KOPF. HILLEL LAKS,*

HORACE C. STANSEL, WILLIAM E. HELLENBRAND,*

CHARLES S. KLEINMANN and NORMAN S. TALNER**

New Haven, Connecticut

Superior vena cava-pulmonary artery shunt (SVC-PAS), (Glenn shunt) as first performed clinically at our institution in 1958. From then through September 1988, 91 patients have undergone SVC-PAS. We here report the follow up data available on all patients.

Patients' age ranged from 1 month to 46 years (mean 6.8 yrs). Diagnoses were: tricuspid atresia 27, single ventricle 22, tetralogy of Fallot 14, D-TGA +VSD + PS 9, TGA 5, Ebstein's anomaly 4, PA + intact septum 4, others 6. Hospital mortality was 7.7% (1 death in the last 53 patients 1.9%); 5 of the patients who died were 4 months of age or younger.

Arterio-venous fistula formation (AVF) occurred in 18 patients (19.7%), 6 of whom have undergone therapeutic embolization with improvement in saturation. The incidence of AVF

increased with time post shunt. Desatura-tion due to venous collaterals occurred in 2 patients, and a patent SVC-RA connection in 1. No shunt thrombosis or obstruction due to stricture formation occurred following discharge from the hospital. Improvement in saturation was obtained following SVC-PAS in 8 inoperable patients by creation of a right axillary arterio-venous fistula up to 12 years following shunt placement. Three patients had conversion of a right Blalock-Taussig shunt to a SVC-PAS to decrease the volume load on a failing ventricle and to improve saturation. Thirty patients underwent physiological repair (Fontan procedure or modification in 26 and biventricular repair in 4, with 4 deaths (13.3%). In 3 instances, the SVC-PAS was taken down to provide continuity between the left and right pulmonary arteries; otherwise the shunt was left intact. Compared to patients without prior SVC-PAS, hospital mortality was not significantly different. Thirty years following SVC-PAS, the first patient in this series is married and working full time, having undergone a modified Fontan procedure (RA-LPA connection) in 1981.

We conclude that SVC-PAS, usually with supplemental procedures to enhance oxygenation, has provided excellent physiologic palliation with low mortality up to thirty years with no late thrombosis or stricture formation. Pulmonary A-V fistula formation increases with embolization. Physiological repair following SVC-PAS carries a low mortality with good long term survival. Although currently used infrequently, SVC-PAS, particularly with end-to-side anastomosis, remains a useful method of palliation in selected patients.

*By Invitation

27. The Bi-Directional Cavopulmonary Shunts

JOHN J. LAMBERTI, ROBERT L. SPICER,*

TODD M. GREHL and J. DEANE WALDMAN**

San Diego, California

The bi-directional cavopulmonary shunt (CPS) improves systemic arterial oxygen saturation without increasing ventricular work or pulmonary vascular resistance. A CPS is ideal palliation for patients destined for a right atrium to pulmonary artery connection (RH-PAC). Since 1983, 15 patients have undergone CPS (5 primary operations, 10 secondary operations). Diagnoses were: asplenia; 2, single ventricle; 2, pulmonary atresia and intact ventricular septum; 6, tricuspid atresia; 3, hypoplastic left heart; 1, TGA with hypoplastic LV; 1. Age at primary operation ranged from 3.5 to 30 months (median = 6 mos.). Weight ranged from 3.5 kg. to 9.7 kg. Age at secondary operation ranged from 10 mos. to 14 yrs. (median = 15 mos.). Six CPS were performed through a right thoracotomy utilizing a temporary shunt. Nine CPS were performed on cardiopulmonary bypass (CPB). All CPB patients had additional procedures: Takedown modified Blalock-Taussig shunt, 7; revise right ventricular outflow tract, 4; reconstruct pulmonary arteries, 4; tricuspid valvuloplasty, 1. There was no operative mortality. One patient required early revision. Follow-up ranges from 1 to 53 months. Twelve of 15 had a good to excellent late result. Two patients died late (pulmonary vascular disease, 1 pt., pulmonary A-V malformations, 1 pt.). There was one late failure (converted to Glenn). The CPS is an excellent palliative procedure when RA-PAC must be deferred because of age, weight or anatomic considerations. In addition, at the time of RA-PAC (modified Fontan) the CPS approach may optimize the anatomic connection (8 additional patients).

*By Invitation

28. Unifocalization and Complete Repair of Patients with Pulmonary Atresia, Ventricular Septal Defect and Systemic Collaterals

FRANCISCO J. PUGA, FRANCO E. LEONI,*

PAUL R. JULSRUD and DOUGLAS D. MAIR**

Rochester, Minnesota and Edmonton, Alberta, Canada

Arborization abnormalities of the peripheral pulmonary arterial tree in patients with pulmonary atresia and ventricular septal defect have limited the success of attempts at complete surgical repair. Techniques at surgical Unifocalization have allowed disconnection of pulmonary arterial branches from systemic collateral arteries with concomitant anastomosis to other branches and creation of centrally accessible sources of pulmonary arterial blood flow. Thus, an anatomic situation is created which permits complete correction of the anomaly. From 1982-1988 we have operated 115 patients in whom 60 unifocalizations were performed. All patients had pulmonary atresia, ventricular septal defects, and systemic collateral arteries supplying the majority of pulmonary arterial segments. A total of 93 extrapericardial anastomoses were performed and 80 systemic collaterals interrupted. Procedures included: direct pulmonary arterial branch anastomosis (Type I), in-terpulmonary conduit interposition (Type II), patch angioplasty of communicating pulmonary arterial branches (Type III), and establishment of in-trapericardial pulmonary confluence (Type IV). There were 3 deaths resulting from the Unifocalization procedure (5%). Twenty-five patients have completed bilateral Unifocalization procedures. Two of these have been rejected for complete repair due to persistent restriction in pulmonary arterial runoff. One patient has been accepted for complete repair and is waiting for surgical correction. Twenty-two patients have undergone complete repair (elimination of extracardiac sources of pulmonary arterial blood flow, closure of septal defects, and insertion of valved conduit between the right ventricle and the pulmonary arteries). There were 21 survivors. Intraoperative, post-repair peak systolic pressure ratio between the right and left ventricles ranged from 0.4 to 0.85 (mean = 0.60, SD 0.25). We conclude that, in selected patients, staged reconstruction of peripheral arterial confluence may correct arborization problems sufficiently to allow complete surgical repair of patients formerly considered unrepairable.

10:00 a.m. Intermission - Visit Exhibits

*By Invitation

10:45 a.m. SCIENTIFIC SESSION - HYNES BALLROOM

29. The Size of the Pulmonary Arteries and the Results of the Fontan Type Repair

FRANCIS FONTAN, GUY FERNANDEZ,*

FRANCISCO COSTA, DAVID NAFTEL*,*

FRANCESCO TRITTO, EUGENE BLACKSTONE*

and JOHN W. KIRKLIN

Pessac-Bordeaux, France, Curitiba, Brazil

and Birmingham, Alabama

Among the 334 patients undergoing a Fontan type repair for congenital heart disease (between 1968 and August 1, 1988) in two widely separated institutions, cineangiograms were available in 234 patients (median age 7.0 years, range 8/12 to 38 years) for measurements of the McGoon ratio:

$$\frac{\text{diameter of right pulmonary artery + diameter of left pulmonary artery}}{}$$

diameter of descending aorta at diaphragm

Fifty-five patients (24%) died or had a takedown of the repair within two months of the operation *(the event).* 124 patients had tricuspid atresia (20 events) and 110 had other cardiac malformations, most commonly double inlet left ventricle (35 events) (P = .005). The new atrial connection was to the right ventricle in 73 patients (8 events) and to the pulmonary arteries in 161 (47 events) (P = .002). The median value of the McGoon ratio was 2.3. A smaller McGoon ratio was a risk factor (logistic multivariate analysis) for death or takedown within two months of the Fontan-type repair (P<.001) with increments being added by young age (P<.001), termination of the new right atrial connection on the pulmonary artery (rather than right ventricle) (P<.007) and the presence of mitral atresia (P = .03). The risk factors were identical when the event was analyzed in a time-related manner (hazard function). Solution of the multivariate equation, in a patient aged 5 years, without mitral atresia, and with a right atrial to right ventricular connection, predicted a 14%, 22%, and 41% prevalence of the event when the McGoon ratio was 2.0, 1.6 and 1.2 respectively; and when the connection was directly to the pulmonary arteries, the prevalence was 36%, 49% and 71% respectively.

To enhance usefulness in considering early takedown of the Fontan repair, an additional multivariate analysis was made incorporating the early postoperative right atrial pressure (P_{RA})- This considerably refined the risk factors, such that in the patient described, with a connection to the right ventricle and with a McGoon ratio of 2.0, the prevalence of the event varied from 5% (P_{RA} 13 mmHg) to 15% (P_{RA}17 mmHg) to 38% (P_{RA} 21mm Hg); with a McGoon ratio of 1.6 the prevalence of the event varied from 10% (P_{RA} 13 mmHg) to 27% (P_{RA} 17 mmHg) to 56% (P_{RA}21 mmHg); and with a McGoon ratio of 1.2 the prevalence of the event varied from 27% (P_{RA} 13 mmHg) *to* 55% (P_{RA} 17 mmHg) to 81% (P_{RA}21 mmHg). (These combinations were all represented in the experience.)

Therefore, the size of the right and left pulmonary arteries, as determined by preoperative cineangiography, is an important risk factor for surgical failure, but its effect can be modified by other risk factors.

*By Invitation

30. Modified Fontan Procedure Reconstructive Surgery for Single or Dominant Right Ventricle

JOHN D. PIGOTT, ALVIN J. CHIN*,*

JOHN D. MURPHY and WILLIAM I. NORWOOD*

Philadelphia, Pennsylvania

Since January, 1984, 105 patients with a single or dominant right ventricle have undergone application of Fontan's procedure for definitive palliation. Diagnosis included hypoplastic left heart syndrome (60), heterotaxy syndrome (23), solitary right ventricle (4), other (18). Age at Fontan's procedure ranged from 4.5 months to 23 years (median 20 months). Surgery included baffling pulmonary venous return in 15 patients, closure of an ASD and atriopulmonary anastomosis in 6 patients, and baffling systemic venous return to the pulmonary arteries in 84 patients. Extensive pulmonary arterial augmentation using pulmonary artery homograft was performed in the last 85 patients. There were 26 early deaths. Suspected causes of early mortality include ventricular diastolic dysfunction (10), residual pulmonary arterial stenosis (4), pulmonary venous hypertension (3), elevated pulmonary vascular resistance (2), hemorrhage and infection (3 each) and hepatic

dysfunction (1). Thirty-five patients had 37 nonfatal complications, including prolonged pleural and pericardial effusions (30), complete heart block (3), prolonged ventilation and atrial arrhythmias (2 each). Early reoperation was undertaken in 13 patients: pulmonary artery angioplasty (4), change pulmonary venous to systemic venous baffle (3), pacemaker (2), others (6). Five of 26 early deaths occurred in patients undergoing early reoperation. There were 8 late deaths. Suspected causes of late mortality include persistent pleural and pericardial effusions (4), ventricular diastolic dysfunction (2), irreversible cerebral injury (1), elevated pulmonary vascular resistance (1). Patients with single or dominant right ventricles have structural issues not present in patients with single left ventricles. The thrust of this work has been to develop a systematic means to apply Fontan's principle to all patients with single ventricle, including those with unusual pulmonary and systemic venous return and right atrioventricular valve dysfunction. Important elements for improved survival in this series include: wide augmentation of branch pulmonary arteries and baffling of systemic venous rather than pulmonary venous return at initial operation as well as scrupulous avoidance of elevations in ventricular end diastolic pressure in the early postoperative period.

*By Invitation

31. Primary Repair of Tetralogy of Fallot in Infancy

GILLES D. TOUATI, PASCAL R. VOUHE*,*

PHILIPPE POUARD, FRANCINE LECA*,*

ANTONIO AMODEO* and JEAN-YVES NEVEUX*
Paris, France

Sponsored by: Aldo R. Castaneda, Boston, Massachusetts

From June 1983 to April 1988, 100 consecutive symptomatic infants with Tetralogy of Fallot (without pulmonary atresia) were operated upon. Age ranged from 0.5 to 12 months (mean = 7.3 ± 3.7). Twenty patients were 0.5 to 3 months, 21 were 3 to 6 months and 59 were 6 to 12 months of age. Mean weight was 6.5 Kg ± 1.7.

Seventy patients received a transannular patch. Only 16 patients had hypothermic circulatory arrest, all others had conventional cardiopulmonary bypass.

Hospital mortality was 3%; there were no late deaths; cummulative follow-up was 186 patients/years. Causes of deaths include: hypoplastic pulmonary arteries (4 and 5 months old) and right ventricular failure (4 months old).

The last 48 patients were operated on without mortality; during this period, operative management differed in: 1.) blood cardioplegia repeated every 20 minutes and 2.) ultrafiltration was added to bypass.

The predicted 30-day survivorship after repair was 96-98% (CL 70%) and was 90-99% (CL 95%). No ventricular arrythmias have so far been detected after repair (mean of follow-up = 21.6 months) and echocardiographic ventricular diameter ratios were: 0.60 ± 0.10.

These early results encourage continued primary repair of symptomatic infants with Tetralogy of Fallot thanks to improved surgical and anesthetic management.

*By Invitation

32. Neonatal Aortic Stenosis

KEVIN TURLEY, EDWARD L. BOVE,

*JOSEPH J. AMATO, MARK IANNETTONI**

*and JOHN YEH**

San Francisco, California, Ann Arbor, Michigan

and Newark, New Jersey

Aortic stenosis in the neonate has in the past been associated with a high surgical mortality. As a result, in the era of percutaneous balloon valvuloplasty, the optimal mode of therapy remains controversial. An approach of metabolic stabilization using cardiopulmonary bypass (CPB) followed by relief of left ventricular outflow tract obstruction (LVOTO) was employed by three institutions, and the results are presented.

During the period 1983-1988 (the valvuloplasty era), 33 neonates with isolated aortic stenosis, patent ductus arteriosus, and/or coarctation of the aortic underwent operative repair. Ages ranged from 1 to 30 days, median 11 days including 16 in the first week of life. There were 25 males and 8 females; and weights ranged from 2.5 to 5.5 kg., mean 3.7 kg. Preoperative conditions included: congestive heart failure in 32, mitral regurgitation in 14, and left ventricular/aortic gradients ranged from 15 to 130 mmHg. Operative therapy included the use of CPB to provide metabolic stabilization in all thirty-three prior to relief of LVOTO. There were 25 open valvulotomies and eight transventricular dilatations. Hospital survival was 85% (28/33) with n.s. difference between the methods (7/8, 21/25). There have been four reoperations with one late death (MVR-Apical-Aortic conduit) and one late sudden death.

The use of CPB for resuscitation of neonates with critical aortic stenosis combined with relief of LVOTO can result in a high operative and late survival. These results support the concept that this technique provides a milieu in which the nenonatal myocardium can optimally respond to relief of obstruction. The results of this technique are a standard against which closed methods such as percutaneous valvuloplasty should be compared.

12:00 p.m. Adjourn for Lunch

*By Invitation

1:30 p.m. SCIENTIFIC SESSION - HYNES BALLROOM

33. Aortic Valve Replacement with Stentless Porcine Aortic Bioprosthesis

*TIRONE E. DAVID, CHARLES POLLICK**

*and JOANNE BOS**

Toronto, Ontario, Canada

Fatigue tests indicate that the best stent for the aortic valve leaflets is the aortic root. That is probably why hand-sewn aortic valve homografts are more durable than stent-mounted homografts

used for aortic valve replacement. Artificial stents not only shorten the durability of aortic valve bioprostheses but also impair their hemodynamic performance.

We have initiated a clinical trial on aortic valve replacement using a Stentless porcine aortic valve which is processed in the same manner as the Hancock II bioprosthesis (fixed with glutaraldehyde under very low pressure and treated with sodium dodecyl sulphate to retard calcification). This bioprosthesis has been implanted in 20 patients. To evaluate its hemodynamic performance, these 20 patients were matched for age, valve lesion, body surface area and bioprosthetic size with 20 cohorts who underwent aortic valve replacement with Hancock II bioprosthesis. The hemodynamic performance of the bioprostheses was assessed by Doppler echocardiography. The results were the following:

Valve Size (mm)	Number of Patients	Peak Systolic Gradient (mmHg)		Aortic Valve Orifice (cm.sq.)	
		Stentless	Hancock II	Stentless	Hancock II
21	4	7.2 + /- 6.0	18 + /- 5.8	1.58 + /- 0.36	1.26 + /- 0.11
23	4	7.5 + /- 6.6	22 + /- 6.5	1.72 + /- 0.12	1.38 + /- 0.14
25	5	10.4 + /- 7.0	23 + /- 5.2	1.80 + /- 0.24	1.43 + /- 0.05
27	4	0.0 + /- 0.0	14 + /- 3.4	1.89 + /- 0.19	1.56 + /- 0.16
29	3	0.0 + /- 0.0	16 + /- 1.5	1.96 + /- 0.41	1.62 + /- 0.18

This Stentless porcine aortic valve has lower peak systolic gradient and greater effective orifice than its stented version in every bioprosthetic size. The steneless porcine aortic valves, like the hand-sewn aortic valve homografts, are expected to be more durable than the stended porcine aortic bioprostheses.

*By Invitation

34. Prosthetic Ring Mitral Valve Repair: The Second Decade

ALAIN DeLOCHE, VICTOR A. JEBARA*,*

PATRICK M. PERIER, JOHN Y. M. R ELL AND*,*

GILLES D. DREYFUS and ALAIN F. CARPENTIER*

Paris, France and Beirut, Lebanon

The 195 consecutive patients (pts) having undergone prosthetic ring mitral valve repair (PRMVR) between 1972 and 1979 in our institution were reviewed in order to assess long term function of this method of repair.

Patients age ranged from 18 years to 79 years (mean age 48.7 years). There were 107 (55%) males and 88 (45%) females. Mitral valve incompetence was secondary to degenerative valvular diseases in 113 cases (58%), rheumatic disease in 74 cases (38%), ischemia and various other causes in 8 cases (4%). 188 patients (97%) were in NYHA class III and class IV preoperatively and 94 (48%) had atrial fibrillation. The patients were divided into 3 functional groups:

- Type I (Normal leaflet motion) 35 patients (18%)

- Type II	(Leaflet prolapse)	147 patients (75%)
- Type III	(Restricted leaflet motion)	13 patients (7%)

The techniques used include prosthetic ring annuloplasty (187), partial leaflet resection (158), chordae shortening (89), leaflet mobilization (10) and papillary muscle reimplantation {1). Long term follow-up was available in 189 patients (96.8%) for a rate of 2316 patients/year. The actuarial survival at 15 years was 72.2 ± 4.4%. The occurrence of complications were analyzed in linear and actuarial terms:

Event	% Free	%/Pts/Year
- Valve related death	82.4 ± 3.9	1.2
- Reoperation	88.4 ± 5.3	1.0
- Thromboembolism	94 ± 6.8	0.4
- Endocarditis	96.6 ± 7. 8	0.2
- Anticoagulant hemorrhage	95.3 ± 7.5	0.3

Half of the reoperations were necessary in the first postoperative year and were proved to be due to technical errors. Among the 157 survivors, 117 (74%) were in NYHA class I and class II and 105 (66%) were in sinus rhythm. Risk factor analysis did not show any statistically significant difference in the long term results with regards to age, etiologies and functional groups and lesions.

We conclude that PRMVR is associated with a low risk of complications and mortality when compared to other alternatives. The technique is reliable and provides stable long term results.

*By Invitation

35. Comparative Clinical Assessment of Mitral Valve Replacement With and Without Chordal Preservation

HANI A. HENNEIN, JULLE A. SWAIN*,*

CHARLES L. McINTOSH, CHRISTOPHER D. STONE*,*

ROBERT O. BONOW and RICHARD E. CLARK*

Bethesda, Maryland

Left ventricular (LV) function often deteriorates both acutely and chronically after mitral valve replacement (MVR) for pure mitral regurgitation (MR). Disruption of the mitral apparatus at operation has been proposed as a major cause of postoperative dysfunction. The hypothesis tested in this clinical study was that MVR with chordal preservation results in more favorable postoperative LV function. Forty-eight consecutive patients with pure MR were studied before and six months after operation by treadmill exercise testing, catheterizations, echocardiography, and radionuclide angiography. Thirteen patients underwent MVR with St. Jude and Biopros-thesis with preservation of chordae tendineae, and the remaining 35 had MVR without chordal preservation. Preoperatively, there were no differences in age, gender, New York Heart Association functional class, exercise capacity, cardiac index, rest EF, exercise EF, fractional shortening, mean arterial pressures, or cardiac index the two groups. There were three operative and six late deaths among the 35 patients with chordal resection, but no early or late deaths in patients who had MVR with chordal preservation (P = 0.05). In patients whose chordae were excised, exercise capacity was

unchanged after MVR and left ventricular function deteriorated with a reduction in resting EF (49 ± 11% to 34 ± 13%, PC 0.001), exercise EF (51 ± 14% to 38 ± 16%, PC 0.01), and fractional shortening (34 ± 10% to 23 ± 18%, p = 0.005). In contrast, exercise capacity increased after MVR in patients whose chordae were preserved (by 5.9 ± 2.2 minutes, PC 0.005) and function was maintained, with no charges in resting EF (45 ± 13% to 47 ± 11%,P = NS), exercise EF (47 ± 12% to 51 ± 14%, P = NS), and a decrease in LV end systolic dimension (from 40 ± 8% to 35 ± 6%, P < 0.05). Both group of patients demonstrated improvement in pulmonary artery pressure, mean left atrial pressure, LV end diastolic pressure, cardiac index, with no statistical differences between groups. These data demonstrate that patients who undergo MVR with chordal preservation have improved postoperative survival, exercise capacity, and LV function compared to patients in whom the chordae were excised.

*By Invitation

36. Mitral Valve Repair: Results and Decision Making Process of the Reconstruction

ARRIGO LESSANA, CARMINE CARBONE*.*

MAURO ROMANO, EVELYNE PALSKY*,*

YU HONG QUAN and GENEVIEVE LUTFALLA**

Aubervilliers and Paris, France and Beijin, China

From 1975 to 1988, 266 patients (pts) underwent mitral valve repair (MVR) procedures for pure or predominant mitral regurgitation (MR). The cause of MR was rheumatic in 176 pts (mean age: 28.4 ± 1.2, x ± SEM) and degenerative in 78 pts (mean age: *54.2*± 1.6). Fifty-five percent of the pts were in NYHA class III and IV prior to surgery. Intraoperative assessment of the MV led us to identify 3 major mechanisms of MR: 1) Restriction of leaflet motion by fibrosis (56 pts, Group I); 2) Enhancement of leaflet motion by leaflet and chordae extension and prolapse (137 pts, Group II) and 3) Combination of both (65 pts, Group III). Only 8 pts had isolated dilatation of the annulus. One hundred and forty-nine pts had isolated MR and 117 had associated aortic and/or tricuspid valve or coronary disease. To illustrate more comprehensively how surgical procedures were selected according to the type of valve disease, a 7-min motion pictures has been prepared and will illustrate the different steps of the decision-making process and the subsequent surgical maneuvers. Hospital mortality was 3.8% ± 2.2. Follow-up was 95.9% complete and totaled 1,182.86 patient-years. The postop assessment included clinical examination, and echodoppler study. At 13-year follow-up, survival was 91.9% in Group I, 89.2% in Group II and 96.5% in Group III. The percent freedom from reoperation was 69.8% in Group I, 81.6% in Group II and 64.2% in Group III; the percent freedom from emboli was 97.2%, 97.6%, and 92.5%, respectively. In the group of pts with isolated MR, the results were as follows:

LINEARIZED RATE (expressed as percent/pt-yr)

Event	Group 1 (n=25)		Group II (n=82)	Group III (n=34)
Emboli	0.0		0.3	0.0
Reoperation	0.9		0.0	1.5
Persistent MR	0.0		0.3	1.0
Recurrent MR	2.6	†□P<0.05†’	0.3	1.0

Recurrent rheumatic fever	2.6	†□p<0.05†'	0.3		1.0
Endocarditis	0.0		0.0		0.0
Late death	0.9		0.3		0.0
Cumulative morbidity	2.6	†□p<0.05†'	1.0	†□p<0.05†'	2.0

These results suggest that conservative surgery should be used with caution in Group I and III pts. In contrast MVR should be extended in Group II pts. This finding is clinically relevant, since, in Western countries, valve prolapse tends to be a major cause of MR.

*By Invitation

37. Five Hundred and Thirty Patients Undergoing Tricuspid Valve Surgery: 25 Year Assessment Early and Late Phase Hazards and Events

LYNN B. McGRATH, LORENZO GONZALEZ-LAVIN,*

BRIDGETM. BAILEY, GARY P. GRUNKEMEIR*,*

*JAVIER FERNANDEZ, FRANCIS P. SUTTER**

*and GLENN W. LAUB**

Browns Mills, New Jersey

From January 1, 1961 through December 31, 1987, 530 patients underwent an intracardiac repair which included tricuspid valve surgery. Three hundred fifty-one patients had tricuspid valve repair (66.2%) and 179 had tricuspid valve replacement (33.8%). Mean age at repair was 56.9 years, range 10 years to 79 years. Two hundred and fifty-six patients (48.3%) had undergone previous cardiac surgery. Preoperative variables predicting the requirement for tricuspid valve replacement included: ascites ($p = 0.02$), hepatomegaly ($p = 0.002$), pulsatile liver ($p = 0.01$), peripheral edema ($p = 0.01$), tricuspid stenosis ($p = 0.001$), previous cardiac operation ($p = 0.001$), increasing right atrial pressure ($p = 0.0001$), increasing preoperative angiographic severity of tricuspid valve incompetence ($p = 0.003$), concomitant mitral valve replacement ($p = 0.002$), and higher functional class ($p = 0.07$). There were 76 hospital deaths (14.3%). Incremental risk factors for the event hospital death included male gender ($p = 0.003$), higher functional class ($p = 0.07$), hepatomegaly ($p = 0.02$), ascites ($p = 0.04$), previous operation ($p = 0.009$), total cardiopulmonary bypass time ($p = 0.00001$), total aortic occlusion time ($p = 0.03$), and the use of an annuloplasty ring in the tricuspid valve repair group ($p = 0.05$). Ninety-seven percent of the patients were followed, at a mean of 50.6 months, range 0 to 315 months. There were 250 late deaths (55.1%). Actuarial survival was 33.5% at 120 months and 13.1% at 180 months. Seventy-eight patients (17.2%) had at least one reoperation. Actuarial freedom from reoperation was 83.1 % at 60 months and 29.3% at 180 months. There was no difference in reoperation rates ($p = 0.10$), nor actuarial survival ($p = 0.40$) according to the type of tricuspid valve procedure performed. Hazard function analysis for reoperation revealed early, intermediate, and late phase events.

We conclude that preoperative variables may predict the requirement for tricuspid valve replacement. As there is no difference in hospital nor late survival in patients undergoing tricuspid valve repair (versus replacement), and as there is an increased incidence of valve related events in the replacement group, we recommend tricuspid valve repair, without annuloplasty ring, whenever possible.

*By Invitation

38. The Operative Treatment of Patients with Hypertrophic Cardiomyopathy and Pulmonary Hypertension

CHRISTOPHER D. STONE, HANI A. HENNEIN*,*

CHARLES L. McINTOSH, ARSHED A. QUYYUMI**

and RICHARD E. CLARK Bethesda, Maryland

The clinical course and hemodynamic results in patients undergoing operation for obstructive hypertrophic cardiomyopathy (HCM) with preoperative pulmonary artery hypertension (PHT) was unknown. The hypothesis tested in this retrospective study was that operative relief of left ventricular outflow tract obstruction results in a substantial reduction in pulmonary artery pressures and mitral regurgitation (MR) without necessitating mitral valve replacement (MVR). Patients were included if their preoperative pulmonary systolic pressure was > 35 mmHg, and were, with the exception of MR, without concomitant cardiac pathology. Since 1962, 49 pts. who fit our criteria underwent left ventricular myotomy and myectomy (LVM&M) with 98% follow up. Mean follow up was 7.9 ± 4.5 (mean ± 1 S.D.) years with a range of .8 to 18.4 years. Early hospital mortality was 12% (N ± 6), 2 (4%) from low C.O. and 4 (8%) from arrhythmia. There were 43 (84%) hospital survivors and 19 late deaths; 4 (9%) from arrhythmia or sudden death, 5 (12%) from CHF, 3 (7%) died at repeat cardiac procedure, and 4 (9%) from other causes. Actuarial survival was 74 ± 6.5% (N = 31) at five years and 46 ± 8.4% (N = 8) at ten years. Three patients had late MVR. Of the 43 survivors 39 (91%) returned 7.7 ± 4.5 months later for follow up evaluation including cardiac catheterization. The majority (82%) experienced New York Heart Association functional class one or two status postoperatively. Cardiac catheterization indicated a fall in PA systolic pressure from 62 ± 17.4 (range = 36 to 105) to 38.5 ± 11.7 (range = 17 to 68) mmHg (p = .0001) with no difference in right atrial pressure or cardiac output. Pulmonary capillary wedge mean pressure decreased from 23.6 ±6.4 to 16.1 ± 5.3 mmHg (p = .0002) and, preoperative MR improved or was abolished in 85% of patients studied (N = 13). Rest and maximum provocable left ventricular outflow tract gradients decreased from 80.5 ± 45.4 and 103.4 ± 26.2 to 13.8 ± 19.2 and 46 ± 43 respectively (p = .0001). Analysis of the above pts. operated upon since 1982 and comparison to an age and functionally matched group with PHT and HCM treated with MVR showed no statistical difference in mortality, morbidity, hemodynamic or functional outcome with two years of mean follow up. We conclude that a consistent, significant reduction (mean =42% ± 18%) in preoperative pulmonary hypertension, clinical symptoms and mitral regurgitation occurs with relief of outflow tract obstruction by LVM&M and that MR and PHT are not indications for MVR in these patients.

*By Invitation

SCIENTIFIC SESSION - ALTERNATE

Hypoplastic Left Heart Syndrome: Palliation Without Cardiopulmonary Bypass

WILLIAM Y. TUCKER, ROBERT C. McKONE*,*

KENNETH M. WEESNER and NEAL D. KON**

Winston-Salem, North Carolina

Sponsored by: A. Robert Cordell, Winston-Salem,

North Carolina

Hypoplastic left heart syndrome (HLH) continues to be a surgical challenge. Mortality with a first stage palliative repair utilizing car-diopulmonary bypass (CPB) is high. Cardiac transplantation in neonates with HLH has severe limits of time, donor avilability and patient management.

Our 100% mortality with first stage HLH repair using CPB led us to a procedure not requiring CPB. In 9 consecutive patients, after medical stabilization, a left thoracotomy was performed and an 8 mm woven Dacron graft placed from the main pulmonary artery to the descending thoracic aorta. The patent ductus arteriosus was ligated and the main pulmonary artery banded distal to the graft and proximal to the bifurcation.

Age at surgery ranged from 2-6 days. Weights were 2.3-3.7 kg (mean 3.2 kg). Pre-op diagnosis was made by ECHO in 4 patients and by cath in 5. Caths were accompanied by balloon atrial septostomy. Two patients diagnosed by ECHO underwent post-op cath and balloon septostomy prior to discharge. Five patients were extubated within 4 days (range 1-4). Only low dose Dopamine was needed for inotropic support. There were no bleeding problems. Five patients were discharged 11-80 days postop (mean 38 days). There were 4 hospital deaths; 1 from Candida sepsis at 81 days, 1 from low cardiac output at 2 days, and 2 from a restrictive ASD at 3 and 5 days. Both of the latter had pre-op ECHO with cath and balloon septostomy planned prior to discharge. Patients followed up to 3 years have had normal growth and development.

Palliation of HLH without CPB has allowed hospital discharge in 5 of 9 patients and has decreased post-op hemodynamic, respiratory and bleeding problems. We now do pre-op balloon septostomies on all patients and feel that this will improve survival rate. This simpler initial approach may allow for a staged Fontan or cardiac transplant at a later date for more definitive treatment.

3:30 p.m. ADJOURN

*By Invitation

NECROLOGY

Lyman A. Brewer, III, M.D. South Pasadena, CA
Jacques Bruneau, M.D. Outremont, PQ
Max G. Carter, M.D. New Haven, CT
George H. Clowes, Jr., M.D. Boston, MA
Robert E. Gross, M.D. Kingston, MA
Peter Hairston, M.D. Charleston, SC
Elgie K. Johnson, M.D. New Paltz, NY
Julian Johnson, M.D. Gladwyne, PA
Ormand C. Julian, M.D. San Rafael, CA
Earlc B. Mahoney, M.D. Rochester, NY
Clarence A. McIntosh, M.D. Toronto, ON
Allen I. Midell, M.D. Chicago, IL
William T. Mustard, M.D. Dorset, ON
W. L. Rogers, M.D. San Francisco, CA
Sir Thomas Holmes Sellers, M.D. Buckinghamshire, England
Arthur M. Vineberg, M.D. Westmount, PQ
Julius L. Wilson, M.D. Tyron, NC

American Association for Thoracic Surgery, 1988-1989

(Listed by Countries, States, Provinces and Cities)

Geographical - UNITED STATES

ALABAMA

Birmingham

Blackstone, Eugene H

Blakemore, William S

Kahn, Donald R

Kessler, Charles R

Kirklin, James K

Kirklin, John W

McElvein, Richard B

Pacifico, Albert D

Montgomery

Simmons, Earl M

ALASKA

Anchorage

Phillips, Francis J

ARIZONA

Green Valley

McClenathan, James E

Paradise Valley

Nelson, Arthur R

Phoenix

Brown, Lee B

Cornell, William P

Kerth, William J

Scottsdale

Fisk, R Leighton

Pluth, James R

Sun City

Read, C Thomas

Tucson

Burbank, Benjamin

Copeland, Jack G, III

Sanderson, Richard G

Sethi, Gulshan K

ARKANSAS

CALIFORNIA

Anaheim

Main, F Beachley

Chico

Becker, Ronald M

Covina

Carter, P Richard

Del Mar

Angell, William W

El Macero

Andrews, Neil C

Escondido

Mannix, Edgar P, Jr

Fresno

Evans, Byron H

Indian Wells

Salyer, John M

Irvine

Connolly, John E

Miller, Don R

La Jolla

Fosburg, Richard G

Hutchin, Peter

Oury, James H

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

Jasper

Hudson, W A

Little Rock

Campbell, Gilbert S

Read, Raymond C

Williams, G Doyne

Buckberg, Gerald D

Davis, Lowell L

Fonkalsrud, Eric W

Holmes, E Carmack

Hughes, Richard K

Kay, Jerome Harold

Khonsari, Siavosh

Laks, Hillel

Lee, Myles E

Lindesmith, George G

Longmire, William, Jr

Maloney, James V, Jr

Mandal, Ashis K

Matloff, Jack M

Meyer, Bert W

Morton, Donald L

Mulder, Donald G

Stiles, Quentin R

Los Osos

Aronstam, Elmore M

Martinez

Guernsey, James M

Montebello

Lui, Alfred H F

Oakland

Ecker, Roger R

May, Ivan A

Orange

Gazzaniga, Alan B

Mason, G Robert

Wakabayashi, Akio

Oxnard

Dart, Charles H, Jr

Pacific Palisades

Ramsay, Beatty H

Daily, Pat O

Lamberti, John J, Jr

Moreno-Cabral, Ricardo

Peters, Richard M

Trummer, Max J

San Francisco

Culiner, Morris M

Ellis, Robert J

Gardner, Richard E

Grimes, Orville F

Hill, J Donald

Leeds, San ford E

Magilligan, D J, Jr

McEnany, M Terry

Richards, Victor

Roe, Benson B

Thomas, Arthur N

Turley, Kevin

Ullyot, Daniel J

San Jose

Oakes, David D

San Marino

Tsuji, Harold K

Santa Ana

Pratt, Lawrence A

Santa Barbara

Higginson, John F

Palm Springs

Goldman, Alfred

Palo Alto

Cohn, Roy B

Fogarty, Thomas J

Jamplis, Robert W

McFadden, Paul M

Wilson, John L

Pasadena

Ingram, Ivan N

Newman, Melvin M

Penido, John R F

Rancho Mirage

Bjork, Viking O

Sacramento

Benfield, John R

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

Jahnke, Edward J, Jr

Lewis, F John

Love, Jack W

Santa Cruz

Fishman, Noel H

Seal Beach

Silver, Arthur W

South Laguna

Oatway, William H, Jr

St Helena

Dugan, David J

Stanford

Mark, James B D

Miller, D Craig

Oyer, Philip E

Shochat, Stephen J

Shumway, Norman E

Stinson, Edward B

Tiburon

Heydorn, William H

Torrance

Carey, Joseph S

Cukingnan, Ramon A

Moore, Thomas C

Nelson, Ronald J

State, David

COLORADO

Aspen

Zaroff, Lawrence I

Colorado Springs

Yee, Edward S

Denver

Brown, Robert K

Burrington, John D

Wilmington

Pecora, David V

DISTRICT OF COLUMBIA

Washington

Aaron, Benjamin L

Bowles, L Thompson

Graeber, Geoffrey M

Hufnagel, Charles A

Clarke, David R

Condon, William B

Eiseman, Ben

Grow, John B

Harken, Alden H

Harper, Frederick R

Hopeman, Alan R

Pappas, George

Paton, Bruce C

Pomerantz, Marvin

Rainer, W Gerald

Waddell, William R

Wright, George W

Englewood

Kovarik, Joseph L

Lakewood

Swan, Henry

Pueblo

Bartley, Thomas D

Vail

Fuller, Josiah

CONNECTICUT

Bridgeport

Rose, Daniel M

Hartford

Kemler, R Leonard

New Haven

Baldwin, John C

Carter, Max G

Glenn, William W L

Hammond, Graeme L

Lindskog, Gustaf E

Stansel, Horace C, Jr

Stern, Harold

Norwich

Kelley, Winfield O

Keshishian, John M

Midgley, Frank M

Mills, Mitchell

Randolph, Judson G

Simmons, Robert L

Smyth, Nicholas P D

Wallace, Robert B

FLORIDA

Atlantic Beach

Stranahan, Allan

Belleair

Lasley, Charles H

Boca Raton

Seley, Gabriel P

Clearwater

Wheat, Myron W, Jr

Coconut Grove

Center, Sol

Coral Gables

Cooke, Francis N

Gainesville

Alexander, James A

Jacksonville

Barnhorst, Donald A

Koster, J Kenneth, Jr

Stephenson, Sam, Jr

Lakeland

Brown, Ivan W, Jr

Marathon

Mangiardi, Joseph L

Miami

Bolooki, Hooshang

Chesney, John G

Daughtry, Dewitt C

Gentsch, Thomas O

Jude, James R

Sharon

Wylie, Robert H

Wilton

Pool, John L

DELAWARE

Newark

Lemole, Gerald M

Kaiser, Gerard A

MacGregor, David C

Papper, Emanuel M

Reis, Robert L

Subramanian, S

Thurer, Richard J

Miami Beach
Greenberg, Jack J

Grondin, Pierre

Ripstein, Charles B

Naples

Linberg, Eugene J

North Miami Beach

Spear, Harold C

North Palm Beach
Dorsey, John M

Orlando

Scott, Meredith L

Sherman, Paul H

Ponte Vedra Beach

Gilbert, Joseph, Jr

St Petersburg

Clerf, Louis H

Daicoff, George R

Dematteis, Albert

Tallahassee

Kraeft, Nelson H

Tampa

Connar, Richard G

Seiler, Hawley H

Winter Haven

Maurer, Elmer P R

Winter Park

Bloodwell, Robert D

HAWAII
Honolulu

Ching, Nathaniel P

Gebauer, Paul W

McNamara, Joseph J

IDAHO
Boise

Herr, Rodney H

ILLINOIS
Chicago

Barker, Walter L

Campbell, Charles D

Ebert, Paul A

Faber, L Penfield

Goldin, Marshall D

Hanlon, C Rollins

Head, Louis R

Hunter, James A

Idriss, Farouk S

Ilbawi, Michel N

Jensik, Robert J

Karp, Robert B

Kittle, C Frederick

Leininger, Bernard J

Levitsky, Sidney

Michaelis, Lawrence

Najafi, Hassan

Raffensperger, John

GEORGIA

Atlanta

Graver, Joseph M

Hatcher, Charles, Jr

Hopkins, William A

Jones, Ellis L

King, Richard

Lee, Arthur B, Jr

Logan, William D, Jr

Mansour, Kamal A

Miller, Joseph I

Rivkin, Laurence M

Symbas, Panagiotis

Williams, Willis H

Augusta

Ellison, Robert G

Rubin, Joseph W

Macon

Sealy, Will C

Savannah

Langston, Hiram T

Yeh, Thomas J

Replogle, Robert L

Shields, Thomas W

Silverman, Norman A

Thomas, Paul A, Jr

Vanecko, Robert M

Evanston

Anderson, Robert W

Fry, Willard A

Tatooles, Constantine

Glencoe

Rubenstein, L H

Maywood

Keeley, John L

Pifarre, Roque

Oak Brook

Hudson, Theodore R

Javid, Hushang

Nigro, Salvatore L

Park Ridge

Weinberg, Milton, Jr

Peoria

DeBord, Robert A

Skokie

Baffes, Thomas G

Springfield

Wellons, Harry A, Jr

Winnetka

Mackler, S Allen

INDIANA

Indianapolis

Battersby, James S

Brown, John W

King, Harold

King, Robert D

Mandelbaum, Isidore

Siderys, Harry

Marrero

O'Neill, Martin J, Jr

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

IOWA

Cedar Rapids

Lawrence, Montague S

Council Bluffs

Sellers, Robert D

Des Moines

Dorner, Ralph A

Phillips, Steven J

Watkins, David H

Zeff, Robert H

Iowa City

Behrendt, Douglas M

Ehrenhaft, Johann L

Rossi, Nicholas P

Stanford, William

KANSAS

Cunningham

Allbritten, F F, Jr

Wichita

Tocker, Alfred M

KENTUCKY

Lexington

Crutcher, Richard R

Dillon, Marcus L, Jr

Todd, Edward P J

Louisville

Gray, Laman A, Jr

Mahaffey, Daniel E

Mavroudis, Constantine

Ransdell, Herbert, Jr

LOUISIANA

Alexandria

Knoepp, Louis F

Baton Rouge

Berry, B Eugene

Beskin, Charles A

Schramel, Robert J

Webb, Watts R

MAINE

Liberty

Hurwitz, Alfred

Portland

Drake, Emerson H

Hiebert, Clement

Rockport

Swenson, Orvar

MARYLAND

Annapolis

Blair, Emil

Baltimore

Attar, Safuh

Baker, R Robinson

Baumgartner, William A

Cowley, R Adams

Dodrill, Forest D

Gardner, Timothy J

Gott, Vincent L

Haller, J Alex, Jr

Hankins, John R

McLaughlin, Joseph S

Michelson, Elliott

Reitz, Bruce A

Turney, Stephen Z

Watkins, Levi, Jr

Belhesda

Clark, Richard E

Jones, Michael

Shumacker, Harris B, Jr

Chevy Chase

Iovine, Vincent M

Potomac

Zajtchuk, Rostik

Towson

Brawley, Robert K

Worton
Walkup, Harry E

MASSACHUSETTS
Acton

Boyd, Thomas F

Boston

Akins, Gary W

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

Cohn, Lawrence H

Collins, John J

Daggett, Willard M

Daly, Benedict D T

Deterling, Ralph, Jr

Frank, Howard A

Gaensler, Edward A

Grillo, Hermes C

Hilgenberg, Alan D

Moncure, Ashby C

Neptune, Wilford B

Overholt, Richard H

Rheinlander, Harold F

Russell, Paul S

Scannell, J Gordon

Schuster, Samuel R

Shemin, Richard J

Dover
Black, Harrison

Lynnfield
Wesolowski, Sigmund A

Medford

Desforges, Gerard

Methuen

Wilson, Norman J

North Andover

Cook, William A

So Weymouth

Malcolm, John A

Springfield

Breyer, Robert H

Engelman, Richard M

Rousou, John A

West Roxbury

Khuri, Shukri F

Westport Harbor

Findlay, Charles W, Jr

Winchester

Taylor, Warren J

Worcester

Vander Salm, Thomas J

MICHIGAN

Ann Arbor

Bartlett, Robert H

Bove, Edward L

Gago, Otto

Greenfield, Lazar J

Kirsh, Marvin M

Morris, Joe D

Neerken, A John

Orringer, Mark B

Starkey, George W B

Thurer, Robert L

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

Chestnut Hill

Laforet, Eugene G

Strieder, John W

Concord

Soutter, Lamar

Prager, Richard L

Sloan, Herbert

Birmingham

Timmis, Hilary H

Detroit

Arbulu, Agustin

Arciniegas, Eduardo

Day, J Claude

Levine, Frederick H

Steiger, Zwi

Wilson, Robert F

Farmington Hills

Lam, Conrad R

Grand Rapids

Harrison, Robert W

Meade, Richard H

Rasmussen, Richard A

Tomatis, Luis A

Grosse Pointe

Benson, Clifford D

Taber, Rodman E

Leonard

Gerbasi, Francis S

MINNESOTA
Minneapolis

Arom, Kit V

Foker, John E

Gannon, Paul G

Garamella, Joseph J

Helseth, Hovald K

Humphrey, Edward W

Jamieson, Stuart W

Johnson, Frank E

Kaye, Michael P

Kiser, Joseph C

Mayer, John H, Jr

Padula, Richard T

Piehler, Jeffrey M

Reed, William A

VanWay, Charles W, III

Mount Vernon

Campbell, Daniel C, Jr

St Louis

Earner, Hendrick B

Baue, Arthur E

Bergmann, Martin

Bolman, R Morton, III

Connors, John P

Cox, James L

Ferguson, Thomas B

Flye, M Wayne

Kaiser, George C

Molina, J Ernesto

Nicoloff, Demetre M

Rochester

Bernatz, Philip E

Clagett, O Theron

Danielson, G K

McGoon, Dwight C

Olsen, Arthur M

Pairolero, Peter C

Payne, W Spencer

Puga, Francisco J

Schaff, Hartzell V

St Paul

Lillehei, C Walton

Miller, Fletcher A

Perry, John F, Jr

MISSISSIPPI

Jackson

Dalton, Martin L, Jr

Hardy, James D

Johnston, J H, Jr

Netterville, Rush E

MISSOURI

Bridgeton

Codd, John E

Columbia

Bryant, Lester R

Silver, Donald

Kansas City

Adelman, Arthur

Ashcraft, Keith W

Benoit, Hector W, Jr

Holder, Thomas M

Killen, Duncan A

Kouchoukos, Nicholas T

Lewis, J Eugene, Jr

Pennington, D Glenn

Roper, Charles L

Willman, Vallee L

NEBRASKA

Omaha

Demeester, Tom R

Fleming, William H

Moulton, Anthony L

Schultz, Richard D

NEVADA

Las Vegas

Little, Alex G

NEW HAMPSHIRE

Hanover

Crandell, Walter B

Peterborough

Woods, Francis M

NEW JERSEY

Alpine

Holswade, George R

Belleville

Jurado, Roy A

Bellville

Gerard, Franklyn P

Belmar

Bailey, Charles P

Browns Mills

Fernandez, Javier

Gonzalez-Lavin, Lorenzo

Camden

Camishion, Rudolph C

Cherry Hill

Brooklyn

Pierucci, Louis, Jr

East Orange

Auerbach, Oscar

Hackensack

Hutchinson, John E, III

Jersey City

Demos, Nicholas J

Millburn

Personnel, Victor

Moorestown

Morse, Dryden P

Morristown

Parr, Grant V S

New Brunswick

Kunderman, Philip J

Lewis, Ralph J

MacKenzie, James W

Newark

Abel, Ronald M

Amato, Joseph J

Gielchinsky, Isaac

Hochberg, Mark S

Neville, William E

No Caldwell

Wychulus, Adam R

Paterson

Bregman, David

Short Hills

Timmes, Joseph L

Tenafly

Gerst, Paul H

NEW MEXICO

Albuquerque

Edwards, W Sterling

Las Vegas

Thai, Alan P

Santa Fe

Davila, Julio C

Cunningham, J N, Jr

Levowitz, Bernard S

Sawyer, Philip N

Buffalo

Adler, Richard H

Andersen, Murray N

Bhayana, Joginder N

Lajos, Thomas Z

MacManus, Joseph E

Mentzer, Robert M, Jr

Cooperstown

Blumenstock, David A

East Meadow

Strevey, Tracy E, Jr

Fayetteville

Bugden, Walter F

Effler, Donald B

Floral Park

Crastnopol, Philip

Irvington

Altai, Lari A

Lido Beach

Hines, George L

Loudonville

Alley, Ralph D

New Hyde Park

Tyras, Denis H

New York

Acinapura, Anthony J

Adams, Peter X

Bains, Manjit S

Beallie, Edward, Jr

Bloomberg, Allan E

Bowman, Frederick, Jr

Boyd, Arthur D

Cahan, William G

Clauss, Roy H

Conklin, Edward F

NEW YORK

Albany

Foster, Eric D

Kausel, Harvey W

McKneally, Martin F

Bay Shore

Ryan, Bernard J

Bronx

Brodman, Richard F

Fell, Stanley C

Ford, Joseph M

Frater, Robert W M

Hirose, Teruo

Rubin, Morris

Cournand, Andre

Cracovaner, Arthur J

Culliford, Alfred T

Friedlander, Ralph

Green, George E

Griepp, Randall B

Holman, Cranston W

Isom, O Wayne

Jaretzki, Alfred, III

King, Thomas C

Kirschner, Paul A

Lambert, Adrian

Litwak, Roberl S

Maier, Herbert C

Malm, James R

Martini, Nael

McCord, Colin W

McCormack, Patricia M

Nealon, Thomas F, Jr

Okinaka, Arthur J

Redo, S Frank

Reemtsma, Keith

Rose, Eric A

Skinner, David B

Spencer, Frank C

Spotnitz, Henry M

Steichen, Felicien M

Subramanian, V A

Tice, David A

Veith, Frank J

Wallsh, Eugene

Weldon, Clarence S

Wichern, Walter, Jr

Wolff, William I

Valhalla

Moggio, Richard A

Reed, George E

Westhampton Beach

Sarot, Irving A

NORTH CAROLINA

Asheville

Betts, Reeve H

Scott, Stewart M

Takaro, Timothy

Chapel Hill

Keagy, Blair A

Starek, Peter J K

Wilcox, Benson R

Charlotte

Robicsek, Francis

Taylor, Frederick H

Durham

Jones, Robert H

Lowe, James E

Patchogue

Finnerty, James

Plattsburg

Potter, Robert T

Rochester

Craver, William L

DeWeese, James A

Hicks, George L

Schwartz, Seymour I

Stewart, Scott

Roslyn

Thomson, Norman B, Jr

Wisoff, B George

Saranac Lake

Decker, Alfred M, Jr

Scarsdale

Robinson, George

Scottsville

Emerson, George L

Southampton

Heroy, William W

Staten Island

Garzon, Antonio A

Stony Brook

Anagnostopoulos, C

Dennis, Clarence

Soroff, Harry S

Syracuse

Brandt, Berkeley, III

Bredenberg, Carl E

Meyer, John A

Parker, Frederick, Jr

Ivey, Tom D

Wilson, James M

Oldham, H N, Jr

Rankin, J Scott

Sabiston, David C

Wolfe, Walter G

Young, W Glenn, Jr

Oriental

Deaton, W Ralph, Jr

Pinehurst

Fischer, Walter W

Winston-Salem

Cordell, A Robert

Crosby, Ivan K

Hudspeth, Allen S

Johnston, Frank R

Meredith, Jesse H

Mills, Stephen A

NORTH DAKOTA

Grand Forks

James, Edwin C

OHIO

Akron

Falor, William H

Chagrin Falls

Cross, Frederick S

Cincinnati

Albers, John E

Callard, George M

Flege, John B, Jr

Gonzalez, Luis L

Helmsworth, James A

Hiratzka, Loren F

PENNSYLVANIA

Abington

Wright, Creighton B

Cleveland

Ankeney, Jay L

Cosgrove, Delos M

Geha, Alexander S

Grondin, Claude M

Groves, Laurence K

Kay, Earle B

Loop, Floyd D

Lytle, Bruce W

Van Heeckeren, Daniel W

Columbus

Clatworthy, H W, Jr

Kakos, Gerard S

Kilman, James W

Meckstroth, Charles

Myerowitz, P David

Vasko, John S

Williams, Thomas E, Jr

Dayton

Dewall, Richard A

Pepper Pike

Mendelsohn, Harvey J

Toledo

Davis, John T

Youngstown

McPhail, Jasper L

OKLAHOMA

Jenks

LeBeck, Martin B

Oklahoma City

Elkins, Ronald C

Felton, Warren L, II

Fisher, R Darryl

Greer, Allen E

Munnell, Edward R

Williams, G Rainey

Frobese, Alfred S

Bethlehem

Snyder, John M

Bryn Mawr

Haupt, George J

Mundth, Eldred D

Camp Hill

Pennock, John L

Carlisle

Demuth, William, Jr

Darby

McKeown, John J, Jr

Hamburg

Judd, Archibald R

Hershey

Campbell, David B

Pae, Walter E, Jr

Pierce, William S

Waldhausen, John A

Lancaster

Bonchek, Lawrence I

Witmer, Robert H

Philadelphia

Brockman, Stanley K

Donahoo, James

Dunn, Jeffrey M

Edie, Richard N

Edmunds, L Henry, Jr

Fineberg, Charles

MacVaugh, Horace, III

Mendelssohn, Edwin

Nemir, Paul, Jr

Norwood, William I

Rosemond, George P

Stephenson, Larry W

Van De Water, Joseph M

Wallace, Herbert W

Zuhdi, M Nazih

OREGON

Days Creek

Miller, Arthur C

Portland

Cobanoglu, Adnan

Krause, Albert H

Okies, J Edward

Poppe, J Karl

Starr, Albert

Pittsburgh

Bahnson, Henry T

Ford, William B

Griffith, Bartley P

Hardesty, Robert L

Magovern, George J

Pontius, Robert G

Rams, James J

Ravitch, Mark M

Rosemont

Templeton, John, III

Sayre

Sewell, William H

Wayne

Lemmon, William M

Wilkes-Barre

Roberts, Arthur J

Yardley

Sommer, George N, Jr

RHODE ISLAND

Providence

Karlson, Karl E

Moran, John M

Simeone, Fiorindo A

Singh, Arun K

SOUTH CAROLINA

Charleston

Bradham, R Randolph

Parker, Edward F

Sade, Robert M

Columbia

Almond, Carl H

Lundrum

Stayman, Joseph W

Mount Pleasant

Merrill, Walter H

Sawyers, John L

Scott, Henry W, Jr

Stoney, William S

Thomas, Clarence, Jr

Sparta

Labrosse, Claude C

TEXAS

Amarillo

Sutherland, R Duncan

Austin

Hood, R Maurice

Burnet

Ross, Raleigh R

Dallas

Adam, Maurice

Holland, Robert H

Lambert, Gary J

Mills, Lawrence J

Paulson, Donald L

Platt, Melvin R

Razzuk, Maruf A

Crawford, Fred A, Jr

Spartanburg

Utley, Joe R

TENNESSEE

Chattanooga

Hall, David P

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

Watson, Donald C

Nashville

Alford, William, Jr

Bender, Harvey W, Jr

Dale, W Andrew

Gobbel, Walter G, Jr

Hammon, John W, Jr

Johnson, Hollis E

Ring, W Steves

Seybold, William D

Shaw, Robert R

Sugg, Winfred L

Urschel, Harold, Jr

Dilley

Hood, Richard H, Jr

El Paso

Glass, Bertram A

Galveston

Conti, Vincent R

Derrick, John R

Houston

Beall, Arthur C, Jr

Burdette, Walter J

Cooley, Denton A

Crawford, E Stanley

DeBakey, Michael E

Frazier, Oscar H

Hallman, Grady L

Henly, Walter S

Lawrie, Gerald M

Mattox, Kenneth L

Morris, George C, Jr

Mountain, Clifton F

Ott, David A

Overstreet, John W

Reul, George J, Jr

Walker, William E

Wukasch, Don C

Kaufman

Davis, Milton V

Lubbock

Bricker, Donald L

Feola, Mario

Falls Church

Wolcott, Mark W

Lynchburg

Moore, Richmond L

Richmond

San Antonio

Dooley, Byron N

French, Sanford, III

Grover, Frederick L

Heaney, John P

Treasure, Robert L

Trinkle, J Kent

Temple

Brindley, G V, Jr

Woodville

Harrison, Albert W

UTAH

Salt Lake City

Cutler, Preston R

Doty, Donald B

Gay, William A, Jr

Liddle, Harold V

Mortensen, J D

Nelson, Russell M

VERMONT

Burlington

Coffin, Laurence H

Miller, Donald B

Chester Depot

Adams, Herbert D

West Dover

Humphreys, G H, II

White River Junction

Tyson, M Dawson

VIRGIN ISLANDS

St Thomas

Wilder, Robert J

VIRGINIA

Annandale

Akl, Bechara F

Lefrak, Edward A

Bosher, Lewis H, Jr

Brooks, James W

Cole, Dean B

Gwathmey, Owen

Lower, Richard R

Wechsler, Andrew S

WASHINGTON

Bellevue

Li, Wei-I

Manhas, Dev R

Bellingham

Varco, Richard L

Friday Harbor

Fox, Robert T

Lawrence, G Hugh

Kirkland

Mills, Waldo O

Lacey

Fell, Egbert H

Poulsbo

Malette, William G

Seattle

Anderson, Richard P

Ashbaugh, David G

Dillard, David H

Hill, Lucius D, III

Jarvis, Fred J

Jones, Thomas W

Mansfield, Peter B

Miller, Donald W, Jr

Rittenhouse, Edward A

Sauvage, Lester

Thomas, George I

Spokane

Berg, Ralph, Jr

WEST VIRGINIA

Arlington

Conrad, Peter W

Klepser, Roy G

Charlottesville

Dammann, John F

Kron, Irving L

Minor, George R

Muller, William, Jr

Nolan, Stanton P

Charlestown

Walker, James H

Daniels

Littlefield, James B

Huntington

Norman, John C

Morgantown

Murray, Gordon F

Warden, Herbert E

Parkersburg

Tarnay, Thomas J

WISCONSIN

La Crosse

Gundersen, Erik A

Madison

Berkoff, Herbert A

Chopra, Paramjeet S

Kroncke, George M

Young, William P

Marshfield

Myers, William O

Ray, Jefferson F, III

Sautter, Richard D

Milwaukee

Flemma, Robert J

Hausmann, Paul F

Johnson, W Dudley

Litwin, S Bert

Mullen, Donald C

Narodick, Benjamin

Olinger, Gordon N

Tector, Alfred J

West Bend

Gardner, Robert J

WYOMING

Cheyenne

Lefemine, Armand A

Teton Village

Kaunitz, Victor H

CANADA

ALBERTA

Calgary

Miller, George E

Edmonton

Callaghan, John C

Gelfand, Elliot T

Sterns, Laurence P

BRITISH COLUMBIA

Kelowna

NEWFOUNDLAND

St John's

Brownrigg, Garrett M

NOVA SCOTIA

Halifax

Landymore, Roderick W

Murphy, David A

Kentville

Quinlan, John J

Couves, Cecil M

Vancouver

Allen, Peter

Ashmore, Phillip G

Jamieson, W R Eric

Tyers, G Frank O

Victoria

Stenstrom, John D

West Vancouver

Robertson, Ross

MANITOBA

Winnipeg

Barwinsky, Jaroslaw

Cohen, Morley

Mabou

Thomas, Gordon W

ONTARIO

Hamilton

Sullivan, Herbert J

Kingston

Charrette, Edward J P

London

Heimbecker, Raymond

Nottawa

Key, James A

Ottawa

Keon, Wilbert J

Sudbury

Field, Paul

Walker, George R

Toronto

Baird, Ronald J

Bigelow, Wilfred G

Cooper, Joel D

David, Tirone E

Ginsberg, Robert J

Goldberg, Melvyn

Goldman, Bernard S

Joynt, George H C

Pearson, F Griffith

Salerno, Tomas A

Scully, Hugh E

Todd, Thomas R J

Trimble, Alan S

Trusler, George A

Weisel, Richard D

Williams, William G

Westbrook

Lynn, R Beverley

PROVINCE OF QUEBEC

Montreal

Blundell, Peter E

Chiu, Chu-Jeng (Ray)

Dobell, Anthony R

Duranceau, Andre C H

Lepage, Gilles

MacLean, Lloyd D

Morin, Jean E

Mulder, David S

Pelletier, Conrad L

Scott, Henry J

Quebec

Gravel, Joffre-Andre

Sainte-Foy

Deslauriers, Jean

OTHER COUNTRIES

ARGENTINA
Buenos Aires

Favaloro, Rene G

AUSTRALIA

South Australia

Piccadilly

Sutherland, H D'Arcy

Victoria

Melbourne

Nossal, Gustav J V

AUSTRIA

Salzburg

Unger, Felix H

BRAZIL

Sao Paulo

Jatene, Adib D

Zerbini, E J

ENGLAND

Bath, Avon

Belsey, Ronald

Hereford

Thompson, Vernon C

Herefordshire

Smith, Roger A

ITALY
Bergamo

Parenzan, Lucio

Padova
Peracchia, Alberto

JAPAN

Kitakyushu

Miyamoto, Alfonso T

Osaka

Kawashima, Yasunaru

London
Braimbridge, Mark V

Kennedy, John H

Lennox, Stuart C

Lincoln, Christopher R

Ross, Donald N

FRANCE

Bordeaux-Pessac

Fontan, Francis M

Paris

Binet, Jean-Paul

Blondeau, Philip

Cabrol, Christian E A

Carpentier, Alain F

Dubost, Charles

GUATEMALA

Guatemala City

Herrera, Rodolfo

INDIA

Raiputana

Van Allen, Chester M

IRELAND

Dublin

O'Malley, Eoin

SCOTLAND
Edinburgh

Logan, Andrew

SPAIN
Madrid

Rivera, Ramiro

SWITZERLAND

Arzier

Hahn, Charles J

Zurich

Sendai

Mohri, Hitoshi

Tokyo

Wada, Juro J

NEW ZEALAND

Auckland

Barratt-Boyes, Sir Brian

P.R. OF CHINA

Beijing

Wu, Ying-Kai

PORTUGAL

Lisbon

Macedo, Manuel E M

SAUDI ARABIA

Riyadh

Deniord, Richard N

Duran, Carlos Gomez

Merendino, K Alvin

Senning, Ake

Turina, Marko I

UNITED ARAB EMERIT

Abu Dhabi

Brom, A Gerard

USSR

Moscow

Burakovsky, Vladimir I

VENEZUELA

Caracas

Tricerri, Fernando E

WEST GERMANY

Aschen

Messmer, Bruno J

Hamburg

Rodewald, Georg

Hannover

Borst, Hans G

THE AMERICAN ASSOCIATION

FOR THORACIC SURGERY

Charter Members
June 7, 1917

E. Wyllis Andrews

John Auer

Edward R. Baldwin

Walter M. Booihby

William Branower

Harlow Brooks

Lawrason Brown

Kenneth Bulkley

Alexis Carrel

Norman B. Carson

J. Frank Corbe:t

Armistead C. Crump

Charles N. Dovd

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

Kennon Dunham	Leo S. Peterson
Edmond Melchior Eberts	Eugene H. Pool
Max Einhorn	Walther I. Rathbun
Herman Fischer	Martin Rehling
Albert H. Garvin	B. Merrill Ricketts
Nathan W. Green	Samuel Robinson
John R. Hartwdl	Charles I. Scudder
George J. Heuer	William H. Stewart
Chevalier Jackson	Franz Torek
H. H. Janeway	Martin W. Ware
James H. Kenyon	Abraham O. Wilensky
Adrian V. S. Lambert	Sidney Yankauer

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. Purpose

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-five 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 to 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 *m* 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 65 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, arid 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 re-elected 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 re-elected 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 re-elected 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 01 desirable. All such committees shall render their reports at an executive session of ths 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 pr spare 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.

Section 10. The Committee on Manpower shall be a Joint Committee of this Association and The Society of Thoracic Surgeons. The Committee shall consist of two members of this Association, two members of The Society of Thoracic Surgeons, and a Chairman who shall be a member of this Association and The Society of Thoracic Surgeons. The duties of this Committee, and the manner of appointment and term of its members and chairman, shall be determined jointly by the Council of this Association and the Council of The Society of Thoracic Surgeons.

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 a id 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 may be 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 ‘:he 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 and 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 $150.00 and shall include a year's subscription to THE JOURNAL OF THORACIC AND CARDIOVASCULAR SURGERY.

Section 3. Annual dues for Associate Members shall be $150.00 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.

As amended, Tuesday, April 19, 1988.

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

1983-Atlanta..... President, Frank C. Spencer

1984-New York..... President, Dwight C. McGoon

1985-New Orleans..... President, David C. Sabiston

1986-New York..... President, James, R. Malm

1987-Chicago..... President, Norman E. Shumway

1988-Los Angeles..... President, Paul A. Ebert

GRAHAM EDUCATION AND RESEARCH FOUNDATION

13 Elm Street, Manchester, Massachusetts 01944, (508) 526-8330

President Martin F. McKneally, M.D., Albany, New York

Vice President Floyd D. Loop, M.D., Cleveland, Ohio

Secretary-Treasurer William T. Maloney, Manchester, Massachusetts

Director Bruce A. Reitz, M.D., Baltimore, Maryland

EVARTS A. GRAHAM MEMORIAL TRAVELING FELLOWSHIP

The Evarts A. Graham Memorial Traveling Fellowship was established in 1958 by The American Association for Thoracic Surgery. Administered through the Graham Education and Research Foundation, it provides grants to young surgeons from outside North America who have completed their formal training in general, thoracic, and cardiovascular surge -y. The award allows the recipient to study a year in North America to intensify his training in a program of special interest and to travel to several sites to broaden his averall training and increase his contacts with North American thoracic surgeons. Awards are made to surgeons of unique promise who have been regarded as having trie potential for later international thoracic surgical leadership.

Since the inception cif the Graham Fellowship, 39 young surgeons from 20 foreign countries have trained at thoracic surgical centers throughout North America.

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	Fri z Helmer, M.D. Second Surgical Clinic, University of Vienna, Vienna, AUSTRIA
10th	1962-63	Thuodor M. Scheinin, M.D. Tammisalonitie 20, Helsinki, 00830, 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.
		18 Alexander Sq., 5W3 2AX, London, ENGLAND
14th	1964-65	Elias Carapistolis, M.D., F.A.C.S.
		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-4040, AUSTRIA
21st	1970-71	Michel S. Slim, M.D.
		New York Medical College, Division of Pediatric Surgery
		New York, New York 10595 USA
22nd	1971-72	Severi Pellervo Manila, 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.

		1406 City Park Medical Center, 181 Longmarket St., Cape Town 8001, REPUBLIC OF SOUTH AFRICA
26th	1975-76	Mieczyslaw Trenkner, M.D. Institute of Surgery, 80-211 Ul, Deinsky 7, Gdansk, POLAND
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 Sydney, AUSTRALIA
29th	1978-79	Eduardo Otero Coto, M.D. Servicio de Cirugia Cardiovascular, Ciudad Sanitaria "Le Fe" Valencia, SPAIN
30th	1980-81	Richard K. Firmin, M.D. "Moss Grove", 5 Knighton Grange Road, Stoneygate, Leicester LE2 2LF, ENGLAND
31st	1981-82	Claidio A. Salles, M.D. Av Gelso Porfirio Machado, 370, Bairro Belvedere Belo Horizonte MG, BRAZIL
32nd	1982-83	Yasuhisa Shimazaki, M.D. Firsi Dept. of Surgery, Osaka Univ. Medical School Fukushima-ku, Osaka 553, JAPAN
33rd	1983-84	Georg S. Kobinia, M.D. LKH Klagenfurt, Dept. of Cardiac Surgery, Klagenfurt, 902d, AUSTRIA
34th	1984-85	Aram Smolinsky, M.D. Department of Cardiac Surgery, The Sheba Medical Center Tel Hashomer, 52621, ISRAEL
35th	1985-86	Florentine J. Vargas, M.D. San Martin 1353, Buenos Aires, ARGENTINA
36th	1986-87	Ari L. J. Harjula, M.D.

		Mitiditte 2 A, 4 02680 Espoo68, SF, Finland
37th	1987-88	Byung-Chul Chang, M.D. Dept. of Thoracic and Cardiovascular Surgery, Yonsei University College of Medicine, CPO Box 8044, Seoul, Korea
38th	1988-89	Wang Cheng, M.D. 30P Weymouth St., #104, Durham, North Carolina 27707 USA
39th	1989-90	Christopher John Knott-Craig, M.D. University of Stellenbosch, Department of Surgery, P.O. Box 63, Tygerberg 7505, Cape Town, SOUTH AFRICA

THE AMERICAN ASSOCIATION FOR THORACIC SURGERY RESEARCH SCHOLARSHIP

The American Association for Thoracic Surgery Research Scholarship was established by the Association in 1985. Funded by the Association and individual contributions, the Research Scholarship provides an opportunity for research, training and experience for North American surgeons committed to pursuing an academic career in cardiothoracic surgery. Administered by the Graham Education and Research Foundation, the program is undertaken within the first three years after completion of an approved cardiothoracic residency and is about two years in duration.

EDWARD D. CHUUCHILL RESEARCH SCHOLARSHIP

"Pharmacology of the Pulmonary Lymphatics"

1986-1988 Mark K. Ferguson, M.D.

University of Chicago, Department of Surgery, Box 255 5841 South Maryland Avenue, Chicago, Illinois 60637

ALFRED BLALOCK RESEARCH SCHOLARSHIP

"Efficacy and Toxicity of a New Blood Substitute: Polymerized, Ultra-Pure, Stroma-Free Bovine Hemoglobin"

1988-1990 Gus J. Vlahakes, M.D.

Massachusetts General Hospital and Harvard Medical School Department of Surgery, Boston, Massachusetts 02114