1991 ANNUAL MEETING PROGRAM

THE AMERICAN ASSOCIATION

FOR THORACIC SURGERY

1990 -1991

President Keith Reemtsma, New York, NY

Vice-President John A. Waldhausen, Hershey, PA

Secretary Martin F. McKneally, Toronto, Ontario, Canada

Treasurer William A. Gay, Jr., Salt Lake City, UT

Editor John W. Kirklin, Birmingham, AL

Councilors

Delos Cosgrove (1994), Cleveland, OH

Tom R. Demeester (1993), Los Angeles, CA

L. Henry Edmunds, Jr. (1992), Philadelphia, PA

Nicholas T. Kouchoukos (1991), St. Louis, MO

F. Griffith Pearson (1991), Toronto, Ontario, Canada

Membership Committee

Randall B. Griepp, Chairman, New York, NY

Delos M. Cosgrove, Cleveland, OH

James K. Kirklin, Birmingham, AL

Alex G. Little, Las Vegas, NV

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

James V. Maloney, Jr., Los Angeles, CA

John A. Waldhausen, Hershey, PA

Benson R. Wilcox, Chapel Hill, NC

Board of Governors, American College of Surgeons

Anthony R.C. Dobell (1990), Montreal, Quebec, Canada

Harold V. Liddle (1993), Salt Lake City, UT

COMMITTEES

LOCAL ARRANGEMENTS

Robert B. Wallace, Chairman

Benjamin L. Aaron

Bechara F. Akl

L. Thompson Bowles

Peter W. Conrad

Nevin M. Katz

John M. Keshishian

Edward A. LeFrak

Frank M. Midgley

Mitchell Mills

Judson G. Randolph

Robert I. Simmons

J. Rostik Zajtchuk

LADIES' HOSPITALITY COMMITTEE

Mrs. Robert B. Wallace, Committee Chairwoman

Mrs. Benjamin L. Aaron

Mrs. Bechara F. Akl

Mrs. L. Thompson Bowles

Mrs. Peter W. Conrad

Mrs. Nevin M. Katz

Mrs. John M. Keshishian

Mrs. Edward A. LeFrak

Mrs. Frank M. Midgley

Mrs. Mitchell Mills

Mrs. Judson G. Randolph

Mrs. Robert L. Simmons

Mrs. J. Rostik Zajtchuk

PROGRAM COMMITTEE

Chairman, Keith Reemtsma, M.D..... New York, New York

John W. Kirklin, M.D..... Birmingham, Alabama

Martin F. McKneally, M.D..... Toronto, Ontario

John A. Waldhausen, M.D..... Hershey, Pennsylvania

James L. Cox, M.D..... St. Louis, Missouri

Douglas J. Mathisen, M.D..... Boston, Massachusetts

Eric A. Rose, M.D..... New York, New York

EVARTS A. GRAHAM MEMORIAL TRAVELING

FELLOWSHIP COMMITTEE

Chairman, Bartley P. Griffith, M.D..... Pittsburgh, Pennsylvania

William A. Gay, Jr., M.D..... Salt Lake City, Utah

Robert A. Guyton, M.D..... Atlanta, Georgia

Martin F. McKneally, M.D..... Toronto, Ontario

G. Alexander Patterson, M.D..... Toronto, Ontario

Keith Reemtsma, M.D..... New York, New York

ETHICS COMMITTEE

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

Anthony R.C. Dobell, M.D..... Montreal, Quebec, Canada

Lucius D. Hill, III, M.D..... Seattle, Washington

Will C. Sealy, M.D..... Macon, Georgia

Larry W. Stephenson, M.D..... Detroit, Michigan

LIAISON COMMITTEE FOR THORACIC SURGERY

Chairman, Mark B. Orringer, M.D..... Ann Arbor, Michigan

Joel D. Cooper, M.D..... St. Louis, Missouri

Robert J. Ginsberg, M.D..... New York, New York

George J. Magovern, M.D..... Pittsburgh, Pennsylvania

Martin F. McKneally, M.D..... Toronto, Ontario

Andrew S. Wechsler, M.D..... Richmond, Virginia

GOVERNMENT RELATIONS COMMITTEE (AATS/STS)

Chairman, John E. Albers, M.D. (1991)..... Cincinnati, Ohio

Benjamin L. Aaron, M.D. (1992)..... Washington, DC

Arthur E. Baue, M.D. (1991)..... St. Louis, Missouri

Mortimer J. Buckley, M.D. (1992)..... Boston, Massachusetts

Willard A. Fry, M.D. (1991)..... Evanston, Illinois

Robert W. Jamplis, M.D. (1992)..... Palo Alto, California

George E. Miller, Jr., M.D. (1992)..... Sacramento, California

Hugh E. Scully, M.D. (1993)..... Toronto, Ontario

Robert M. Vanecko, M.D. (1993)..... Chicago, Illinois

Robert B. Wallace, M.D. (1991)..... Washington, DC

AMERICAN ASSOCIATION FOR THORACIC SURGERY

13 Elm Street

Manchester, Massachusetts 01944

(508) 526-8330/FAX (508) 526-4018

AMERICAN COLLEGE OF SURGEONS ADVISORY COUNCIL FOR CARDIOTHORACIC SURGERY

Floyd D. Loop, M.D., Cleveland, Ohio (1991)

John A. Waldhausen, M.D., Hershey, Pennsylvania (1991)

AMERICAN HEART ASSOCIATION COUNCIL OF CARDIOPULMONARY DISEASE

Mark K. Ferguson, M.D., Chicago, Illinois (1991)

AMERICAN MEDICAL ASSOCIATION HOUSE OF DELEGATES

Thomas D. Hartley, M.D., Pueblo West, Colorado (1991)

AMERICAN MEDICAL ASSOCIATION CPT-4 ADVISORY COMMITTEE

Milton Weinberg, Jr., M.D., Parkridge, Illinois (1992)

ASSOCIATION OF AMERICAN MEDICAL COLLEGES COUNCIL OF ACADEMIC SOCIETIES

Thomas C. King, M.D., New York, New York -Representative (1991)

Vincent L. Gott, M.D., Baltimore, Maryland - Alternate (1991)

ASSOCIATION OF PHYSICIANS' ASSISTANTS IN CARDIOVASCULAR SURGERY

Quentin R. Stiles, M.D., Los Angeles, California (1991)

COMMITTEE FOR COORDINATING CONTINUING EDUCATION IN THORACIC SURGERY

David B. Campbell, M.D., Hershey, Pennsylvania (1996)

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 (1991)

Richard G. Fosburg, M.D., La Jolla, California

Representative (1991)

Stanton P. Nolan, M.D., Charlottesville, Virginia

Representative (1991)

Hendrick B. Earner, M.D., St. Louis, Missouri - Alternate

(1991)

NATIONAL ASSOCIATION FOR BIOMEDICAL RESEARCH

Timothy J. Gardner, M.D., Baltimore, Maryland (1991)

TRANSFUSION MEDICINE SCIENTIFIC FORUM

Safuh Attar, M.D., Baltimore, Maryland, Representative (1991)

American Association for Thoracic Surgery
71ST ANNUAL MEETING
Washington Hilton Hotel, Washington, DC
MAY 6-8, 1991
Program Outline

MONDAY MORNING, May 6, 1991

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

8:45 a.m. SCIENTIFIC SESSION - International Ballroom

1. Pulmonary Transplantation: Early and Late Results

G. ALEXANDER PATTERSON, ALBERTO L. DeHOYAS*,

JUAN C. RAMIREZ*, JANET R. MAURER*

and TIMOTHY L. WINTON*

Toronto, Ontario, Canada

Isolated lung transplantation has been successfully applied in an increasing number of centres. A variety of end-stage lung diseases have been treated by isolated lung transplantation. Between November 1983 and October 1990, our centre performed 47 single (SLT) and 30 bilateral (BLT) lung transplants in 72 patients. Underlying diseases of patients receiving SLT included 22 idiopathic fibrosing alveolitis, 8 emphysema, 6 pulmonary vascular disease, 3 eosinophilic granuloma, 1 lymphangiomyomatosis, 1 extrinsic allergic alveolitis, 1 fibrosing mediastinitis. Five patients have undergone retransplan-tation; 3 of these were done early because of donor airway dehiscence and 2 performed late (one airway stricture and one bronchiolitis obliterans). Underlying diseases of BLT included 13 cystic fibrosis, 9 emphysema, 4 bronchiectasis, 1 each eosinophilic granuloma, bronchiolitis obliterans, primary pulmonary hypertension and 1 re-transplant for primary pulmonary hypertension. Eight SLT recipients (19%) died perioperatively and 8 (19%) died from 4 months to 6 years following transplantation. 28 of 44 SLT recipients (64%) are alive (2 weeks to 5 years). Eight BLT recipients (26%) died perioperatively and 1 (3%) died late. 21 of 30 BLT recipients (70%) are alive (4 weeks to 4 years). Airway dehiscence (2 SLT, 3 BLT) and infection (4 SLT, 5 BLT) have been the commonest cause of operative mortality. Late deaths have most commonly been due to chronic rejection complicated by infection (5 SLT, 1 BLT). One late death was due to renal failure. Of surviving SLT recipients, 2 of 28 (7%) are functionally limited by bronchiolitis obliterans. 2 of 21 BLT recipients (9%) have bronchiolitis obliterans but are not significantly impaired. 2 of 21 surviving BLT recipients (9%) have airway stenosis and infection ± rejection which provides significant functional impairment. Actuarial survival for SLT is 64% and 58%, and for BLT, 64% and 64% at 1 and 3 years respectively. Overall 47 of 49 surviving lung transplant recipients (67%) are surviving without functional disability.

*By Invitation

2. Cystic Fibrosis: Target Population for Lung Transplantation in North America in the 1990's

VAUGHN A. STARNES*, NORMANLEWISTON*,

JAMES THEODORE*, CHRISTOPHER STOEHR*,

EDWARD STINSON, NORMANE. SHUMWAY

and PHILIPS. OVER

Stanford, California

It is anticipated that about 3.5% of the 20,000 individuals with cystic fibrosis (CF) in North America will die each year of end-stage lung disease. Although this genetic disorder affects a number of organs, most of the mortality stems from a severe endobronchitis resulting from the basic molecular defect. Transplanted lungs do not have this defect and do not develop the pulmonary findings characteristic of this disease. Some 150 CF patients have received heart-lung or lung transplantation to date in North America and Europe, where it is now the single most common diagnosis for lung transplantation. We have evaluated 42 CF patients at our center, accepting 27 as transplant candidates. Thirty-three percent (9/27) have died while on the waiting list. Thirteen have received transplantion (12 HLT, one double lung) with a 77% survival. All survivors are NYHA Class I for activity. As we have found with other patients with pulmonary parenchymal disease, the incidence of obliterative bronchiolitis is much less than for patients with pulmonary vascular disease (none so far for CF, 30% for all PVD survivors).

CF patients present a number of challenges which are becoming less important with increased experience. The problem of pleural adhesions and/or previous limited surgical pleuradesis has largely been mastered with the use of the "clam shell" incision, a bilateral lateral thoracotomy with a bridging transverse sternal incision for better pleural exposure. Insulin-dependent diabetes mellitus and low dosage corticosteroid therapy are no longer considered absolute contraindications. Although both septic lungs must be removed, either an HLT with a "domino" donation of the heart or a double lung transplantation are appropriate for most candidates. This ability substantially expands the available donor pool. These data support the notion that lung transplantation has proven efficacy for selected CF patients.

*By invitation

3. Bilateral Lung Transplantation for Cystic Fibrosis

TIMOTHY L. WINTON*. JUAN C. RAMIREZ*,

ALBERTO L. DeHOYAS*, JANETR. MAURER*,

MASINA SCA VUZZO* and G. ALEXANDER PA TTERSON

Toronto, Ontario, Canada

Heart/lung transplant has been advocated as the preferred procedure for end-stage cystic fibrosis. Adequate cardiac function in these patients has permitted the use of the recipient's heart for a cardiac allograft (Domino procedure). Since 1988 our centre has performed 13 bilateral lung transplants in patients with cystic fibrosis including 8 male (mean age, 27.6 years) and 5 female (mean age, 26.6 years). Mean room air PaO2 was 53.6 ± 5.9 mmHg; FVC, 1.7 ± 0.64 L; FeV1, 0.85 ± 0.4 L and 6 minute walk, 511.7 ± 114.9 M. En bloc bilateral lung transplantation was performed in three patients. Ten patients underwent sequential bilateral single lung transplantation. Two patients underwent median sternotomy and the most recent eleven patients had transverse bilateral thoracotomy and sternotomy. Nine patients

required cardiopulmonary bypass with a mean time of 3.8 hours for the en bloc procedure and 2.4 hours for the sequential operation. Mean allograft ischemic times for en bloc procedures was 4.6 hours. For the sequential procedure, mean allograft ischemic time was 2.9 hours for the first implanted lung and 4.2hours for the second lung. Post operative cardiac function was adequate in all patients. There were 4 post operative deaths. One patient died of disseminated sepsis, 2 patients of bilateral gram negative pneumonia and 1 patient of cyclosporin neurotoxicity. Among 9 surviving patients, the most frequent complication was spesis; bilateral pneumonia 2, CMV pneumonia 1, empyema 2, lung abscess 1, mediastinal abscess 1, sepsis 1 and disseminated herpes 1. Airway stenosis requiring stent insertion occurred in 3 patients. Among operative survivors, 3 month follow up mean room air PaO2was 88.57 ± 5.94 mmHg ($P<0.0001$); FVC 2.85 ± 0.7 L ($P<0.0002$); FeV1, 2.69 ± 0.76 L ($P<0.0001$) and 6 minute walk, 656.6 ± 114.5 M ($P<0.02$). Despite significant morbidity and mortality in this challenging group of young patients, bilateral pulmonary transplantation is an effective surgical option associated with significant post operative functional improvement.

9:45 a.m. INTERMISSION - VISIT EXHIBITS

10:30 a.m. SCIENTIFIC SESSION - International Ballroom

4. Single Lung Transplantation for Primary Pulmonary Hypertension

MICHAEL K. PASQUE*, LARRYR. KAISER*,

CAROLYN DRESLER*, ELBERT TRULOCK*,

ANASTASIOS N. TRIANTAFILLOU* and

JOEL D. COOPER

St. Louis, Missouri

Shorter waiting times, relative technical simplicity, and satisfactory application to a broad spectrum of patients has made single lung transplantation (SLT) a viable option in the treatment of patients with end-stage primary pulmonary hypertension (PPH). Seven patients with PPH underwent SLT on cardiopulmonary bypass. Two associated atrial septal defects were closed at the time of SLT. Despite severely compromised pre-transplant right ventricular function in all patients, there has been no early or late mortality. Right ventricular functional recovery has been nearly uniform and is characterized by the following hemodynamic data (mean: 13 weeks post-transplant):

	PRE	POST	P
PA Sys (mmHg)	92 ± 7	29 ± 6	.001
PA Mean (mmHg)	64 ± 18	18 ± 5	.001
CVP (mmHg)	10 ± 6	1 ± 2	.02
PCWP (mmHg)	7 ± 2	8 ± 73	.54
Cardiac Index	2.54 ± 98	3.54 ± .7	.65

Pulm. Vase. Resistance Index	1924 ± 663	232 ± 73	.001
Syst. Vase. Resistance Index	2763 ± 766	2346 ± 759	.39

Echocardiography documented early recovery of right ventricular function and geometry. Radionuclide ventriculography (mean: 17 weeks post-transplant) documented a significant (p=.006) increase in right ventricular ejection fraction from 22% (± 15; S.D.) to 51% (± 11; S.D.). Left ventricular ejection fraction did not change significantly post-transplant. Pulmonary ventilation/perfusion scans (mean: 17 weeks post-transplant) documented a predominance of flow to the transplanted lung with a slight, but significant, decrease in ventilation of the transplanted side:

	PRE	POST	P
Ventilation (%± S.D.)	56 ± 6	49 ± 8	.004
Perfusion (% ± S.D.)	56 ± 6	89 ± 7	.001

Exercise tolerance has been excellent in all recipients. These early data cautiously support SLT as a transplant option in PPH although long-term follow-up of the functional and hemodynamic results is required before widespread application.

*By Invitation

5. Surgical Lessons Learned in Heart Transplantation for Complex Congenital Heart Disease

ROBERTE. MICHLER*. CRAIG R. SMITH*,

JAN M. QUAEGEBEUR * and ERIC A. ROSE

New York, New York

Cardiac transplantation provides an excellent therapeutic option to high-risk reparative surgery in the pediatric patient with complex congenital heart disease. Since many of these patients will have had prior corrective or palliative procedures, the surgeon must be prepared to manage the potentially distorting anatomical effects of these prior operations, as well as the underlying complex congenital anatomy. We have performed 54 cardiac transplants in 52 pediatric patients. Nineteen patients (37%) with congenital heart disease carried the following diagnoses: corrected transposition of the great arteries (4); single ventricle variants (6) including one with visceroartrial situs inversus; hypoplastic left heart syndrome (2); Ebstein's anomaly (1); VSD (2); Tetralogy of Fallot (1); A-V canal (1); and coarctation of the aorta with left ventricular outflow tract obstruction (2). There were 11 males and 8 females ranging in age from 5 days to 17.7 years (mean 9.4 ± 6.6 yrs) with a mean weight of 31.8 ± 25.3 (range 2.3-100 kg). The mean duration of illness pre-transplant was 17.8 ± 32.9 months. Fifteen patients (79%) had undergone 30 prior operations (14 palliative and 16 corrective). The mean interval between prior surgery and transplantation was 4.7 ± 5.9 yrs (range 2.6 mos-17.7 yrs). The mean ischemic time was 182 + 58 min (range 68-270 min).

No deaths were related to surgical technique. The most frequently encountered technical problem was distortion of the pulmonary artery (PA) secondary to bands, shunts, conduits, and Fontan connections. Stenoses were patched, dilated PA's were partially oversewn and the main orifice transferred leftward in patients with malposition of the great arteries. Discrepancies in size of the donor and recipient right atria were amenable to suture plication or cephalad extension of the recipient right atrium, and enlargement of the donor right atrium by incision through the SVC posterior to the SA node. Visceroatrial situs inversus

was successfully corrected by creating autologous tissue tunnels for SVC and IVC return. Heart transplantation for congenital heart disease can be performed with excellent results provided careful attention is paid to restoring functional anatomy.

11:15 a.m. PRESIDENTIAL ADDRESS

Discovery in Surgery: Reflections on a Golden Age

Keith Reemtsma, M.D., New York, New York

*By Invitation

MONDAY AFTERNOON, MAY 6, 1991

1:30 p.m. SCIENTIFIC SESSION - International Ballroom

6. Barrett's Ulcer: A Surgical Disease?

WARREN A. WILLIAMSON*, F. HENRY ELLIS, JR.,

PETER S. GIBB* and H. THOMAS ARETZ*

Burlington, Massachusetts

Published reports on the treatment of Barrett's ulcer are controversial and sparse. Our experience with the management of this disorder over the past 16 years suggests that surgical therapy is rarely indicated.

Two hundred eighty-five cases of Barrett's esophagus were treated from 1974 to 1990. Seventy-three of these patients either presented with adenocarcinoma in Barrett's or developed it while under surveillance. Of the remaining 212 patients with benign Barrett's esophagus, 30 were found to have Barrett's ulcer on endoscopy, for a prevalence of 14%. Ulcers varied in size from 0.5cm to 3cm, sixteen (53%) being 1 cm or larger. Heartburn (70%), dysphagia (60%), and bleeding or anemia (23%) were the most common presenting symptoms. No patient exhibited either free perforation or perforation into the mediastinal structures. Ten patients (30%) admitted to heavy alcohol consumption.

Initial treatment consisted of aggressive medical therapy including H2 antagonists and antacids as well as the usual dietary and anti-reflux measures. Three patients were lost to follow-up and twenty-seven were initially re-endoscoped in 2-4 months. Total endoscopic follow-up was 109 patient-years with a range of 2 months to 13 years and a median of 2.3 years. Complete healing occurred in 23 of 27 patients (85%) in two to 14 months (median 4 months). Seven of the 23 (30%) developed recurrent ulceration which healed in five with further medical therapy. A Nissen fundoplication or a Collis-Nissen was performed in four of the six patients with non-healing Barrett's ulcers, 1 to 1.5 cm in size, and they subsequently healed. Two patients refused surgery.

We conclude that the majority of Barrett's ulcers will heal on medical therapy, even large ulcers (2-3cm) and recurrent ulcers. We reserve surgical intervention for otherwise suitable operative candidates if there is no evidence of healing of the ulcer after a minimum of four months of medical therapy and prefer the

Nissen fundoplication or one of its modifications. Perforation, uncontrollable hemorrhage, and malignant degeneration, not encountered in this series, are of course, indications for urgent surgical intervention.

*By Invitation

7. Primary Repair of a Wide Spectrum of Esophageal Atresia

EDWARD M. BOYLE, JR*, ERICD. IRWIN*

and JOHN E. FOKER

Minneapolis, Minnesota

Surgical repair of esophageal atresia (EA) with or without tracheoesophageal fistula (TEF) in infants with a wide gap length has continued to be a difficult problem. This study demonstrates, however, that primary repair can be safely carried out in infants with a wide spectrum of esophageal gap lengths. From 1976 to 1989, 56 infants with EA, with (54) or without (2) associated TEF(s), underwent primary repair. A very wide gap length (‰¥ 2.5 cm) existed in 15/56 (27%). Three infants had gap lengths of 4-7 thoracic vertebrae, two with pure EA and one with a proximal TEF with a short blind distal segment. These three infants underwent gastrostomy placement but after six weeks had no significant decrease in gap length. The wide gap in five other infants was related to an associated vascular ring. Since 1983 no infant was staged for interposition, even with an ultrawide gap. All 56 patients had a primary anastomosis without lengthening procedures such as circular myotomy. Our results include no deaths related to the operation (0%), nor apparent leaks (0%) nor recurrent TEF's (0%). Eight (14%)late deaths occurred (16-300 days postop) secondary to prematurity or other anomalies, thus demonstrating that survival depends on the severity of associated problems. One infant required a second operation for an unsuspected cervical TEF. Significant gastroesophageal reflux (GER) was found in 32/56 (57%) and was most common in the wide gap group. Fun-doplication was required in 13/56 (23%) and severe tracheomalacia was treated by aortopexy in 5/56 (9%) infants. All patients underwent two prophylactic dilations with 19/56 (34%) requiring additional dilations of anastomotic narrowing. One patient (2%) required late resection of an anastomotic stricture. All 48 surviving patients are now eating a diet normal for age (follow-up 0.75-16 years). In summary: (1) Primary repair can be reliably performed for the full spectrum of EA defects. (2) With our technique, a gap which exceeds 2.5 cm does not preclude successful primary repair despite the resulting great anastomotic tension. (3) With wide gap EA later operations for GER and the need for additional dilations are more common. (4) We conclude that infants born with EA can expect an adequately functioning native esophagus after this type of repair.

*By Invitation

8. Surgery for Subclavian Vein Effort Thrombosis

J. ERNESTO MOLINA

Minneapolis, Minnesota

Effort subclavian vein thrombosis (ESCVTO) occurs suddenly in young active people without premonitory signs or symptoms. If not treated as an emergency, permanent disability is common. Our protocol entails direct lysis of the clot via a catheter in the subclavian vein (Urokinase at the dose of 3,000 u/kg/wt) for 12-24 hours followed by surgery. The operation is done via a subclavicular approach for 1st rib resection and subclavian vein patch angioplasty is done if necessary. Residual stricture is treated with in-traluminal balloon angioplasty. Out of 27 patients seen, 23 were treated with this regimen. 16 on the right and 7 on the left. Three groups were identified: a) acute, < 5 days (6 pts.). Tears in the veins but no

strictures were present, b) sub-acute, > 6 days (7 pts.), stricture present in 4 patients, c) chronic, > 2 weeks (10 pts.) comprising two categories: 1) short segment of subtotal obstruction (6 pts.), and 2) long segment of total obstruction (4 pts.). Vein patency with normal or near normal caliber and flow was achieved in all of groups a and b, and 7 patients in group c (87%). ESCVT must be treated as emergency for successful re-establishment of normal vein patency. The subclavicular approach to the first rib is simple direct and offers no risk of damaging vital structures. This method is less cumbersome than the transaxillary or posterior approaches and relieves any type of vascular thoracic outlet obstruction.

2:30 p.m. BASIC SCIENCE LECTURE

Transplant Immunology: A Broadening of the Concept for the Future

Fritz H. Bach, M.D., Ph.D., Minneapolis, Minnesota

3:15 p.m. INTERMISSION - VISIT EXHIBITS

*By Invitation

4:00 p.m. SCIENTIFIC SESSION - International Ballroom

9. Transesophageal Echocardiography in the Emergency Surgical Management of Patients with Aortic Dissection

PAUL SIMON*, ALYSONNINA OWEN*,

MICHAEL HA VEL*, ERNST WOLNER,

MICHAEL HIESMAYR* and WERNER MOHL*

Vienna, Austria

To investigate the benefit of transesophageal echocardiography (TEE) in the diagnosis of aortic Type A dissection, 15 consecutive patients were studied.

Patients were evaluated immediately upon admission. The intimal flap was visualized in all 15 patients. The primary entry site was correctly identified in 14 patients (93%). The entry site which was misdiagnosed by TEE was also misdiagnosed by angiography and NMR. Significant aortic regurgitation was encountered in 2 patients and mild insufficiency in 3. Involvement of the coronary arteries was ruled out in all patients. Pericardial tamponade necessitating immediate surgery was detected in one case. Five patients of this series underwent surgery based on TEE alone. Intraoperative TEE monitoring was used to verify retrograde flow in the true lumen after femoral can-nulation. The surgical result was evaluated immediately after termination of cardio-pulmonary bypass. TEE documented no flow in the false lumen in 13 patients. In two patients small intimal tears were detected in the descending thoracic aorta. Aortic regurgitation had resolved in all patients.

We conclude that TEE allows expedient diagnosis of type A aortic dissections and recommend TEE as the primary bedside diagnostic modality. It can especially be used to identify patients requiring surgical intervention without further delay due to other diagnostic procedures.

*By Invitation

10. Atheroembolism from the Ascending Aorta: An Emerging Problem in Cardiac Surgery

CHRISTOPHER I. BLAUTH*, BRIAN WEBB*,

NORMAN B. RATLIFF*, BRUCE W. LYTLE,

FLOYD D. LOOP and DELOS M. COSGROVE

Cleveland, Ohio

As patients undergoing cardiac surgery increase in age, noncardiac causes of death have increased. To identify these causes, autopsy findings in 221 patients undergoing myocardial revascularization or valve surgery, between 1982 and 1989, were analyzed. Mean age was 65.6 ± 9.5 and ranged from 32 to 94 years; 130 (58.7%) were males. Autopsies were complete in 129 patients (58.4%) and limited to the chest and abdomen in the remainder.

Embolic pathology was identified in 69 patients (31.2%). Atheroemboli or pathology consistent with atheroemboli were identified in 48 (21.7%). Fourteen had thromboembolism; 7 had disseminated intravascular coagulation. The incidence of atheroembolic pathology increased dramatically from 4.5% in 1982 to 48.2% in 1989 ($p<0.0001$). Atheroembolic pathology was found in the brain in 16.3%, spleen in 10.9%, kidney in 10.4%, and pancreas in 6.8%. Thirty (62.5%) of the patients had multiple atheroembolic sites. Atheroemboli were more common in patients undergoing coronary artery procedures (43/165; 26.1%) than in those undergoing valve procedures (5/56; 8.9%) ($p = 0.008$).

There was a high correlation of atheroemboli with severe ascending aortic atherosclerosis. Atheroembolic events occurred in 46 of 123 patients (37.4%) with severe disease of the ascending aorta but only 2 in 98 patients (2%) without significant ascending aortic disease ($p<0.0001$). Forty-six of 48 patients (95.8%) who had evidence of atheroemboli had severe atherosclerosis of the ascending aorta.

There was a direct correlation between age, severe atherosclerosis of the ascending aorta, and atheroemboli.

Patient age	<60	61-70	‰¥70	p Value
	N = 46	N = 96	N = 79	
Aortic atherosclerosis	26.1%	61.5%	65.8%	<0.001
Atheroemboli	10.9%	20.8%	29.1%	0.05

We conclude that atheroembolism to the brain and other organs is emerging as a major problem in cardiac surgery, particularly in patients having coronary operations. The incidence correlates directly with age and severe atherosclerosis of the ascending aorta.

*By Invitation

11. Management of the Severely Atherosclerotic Aorta During Cardiac Operations: A Strategy for Detection and Treatment

THOMAS H. WAREING*, VICTOR G. DAVILA-ROMAN*,

BENICO BARZILAI", SUZAN F. MURPHY*

and NICHOLAS T. KOUCHOUKOS

St. Louis, Missouri

Embolization of atheroma from manipulation of the ascending aorta is one of the principal causes of stroke following cardiac surgical procedures. We have previously shown that intraoperative ultrasonographic scanning of the aorta using a high frequency (7.0 MHz) linear ultrasound transducer with longitudinal and transverse views rapidly, safely, and accurately identifies severe atheromatous disease in the ascending aorta and is far more effective for diagnosis than palpation of the aorta. Intraoperative ultrasonography of the ascending aorta was performed in 331 of a consecutive series of 362 patients (pts) 50 years of age or older (mean 69 years) who underwent a variety of cardiac surgical procedures. Forty-eight patients (14.5%) with a mean age of 72 years (range 55-85 years) were identified as having significant atheromatous disease in the ascending aorta at the usual sites for cannulation or clamping or diffusely throughout the ascending aorta, and were considered to be at increased risk for embolization. Palpation identified the disease in only 23 (48%) of these patients. A total of 107 modifications in the standard techniques for cannulation and clamping of the aorta were implemented. These included alterations in the sites of aortic cannulation (35 pts), aortic clamping (34 pts), attachment of the vein grafts (23 pts), and cannulation for infusion of cardioplegia (15 pts). Additionally, 8 patients with severe diffuse atheromatous disease underwent graft replacement of the ascending aorta using hypothermic circulatory arrest without aortic clamping, and one patient underwent coronary artery bypass grafting with femoral artery cannulation, hypothrmic fibrillatory arrest and internal mammary artery grafts. Thirty day mortality for the entire group was 2.4% (8 pts). Permanent neurological deficits occurred in 5 (1.5%) of the patients in the entire group but in none of the 48 patients with significant atheromatous disease in whom modifications in technique were employed.

We conclude that intraoperative ultrasonic imaging is currently the most reliable method of assessing the ascending aorta for the presence of severe atheromatous disease. Modification of standard cannulation and clamping techniques based on ultrasonography may reduce the frequency of stroke related to atheromatous embolization.

*By Invitation

TUESDAY MORNING, MAY 7, 1991

7:30 a.m. FORUM SESSION I - Cardiac Surgery

International Ballroom

F1. Age Dependent Sensitivity to Unprotected Cardiac Ischemia: The Senescent Myocardium

BRUCE D. MISARE*, IRVIN G. KRUKENKAMP*

and SIDNEY LEVITSKY

Boston, Massachusetts

Septuagenarians and octogenarians are presenting for complex cardiac surgical procedures in increasing numbers. To adjudicate whether in-traoperative myocardial management should be optimized for age-dependant ischemic tolerance, a new model of senile myocardium was developed in ovine hearts lacking

pathological cardiac hypertrophy, dilatation, or coronary stenosis. Six young, sexually mature and seven elderly (in the last decile of their captive lifespan) sheep of either sex were used (ages 0.75 +/- 0.11 versus 7.1 +/- 0.45 years, respectively). LV weight to body weight ratios were not significantly different between age groups (3.09 +/- 0.09 vs. 3.07 +/- 0.19 g/kg, old vs. young, NS). Global left ventricular mechanics were assessed using intracavitary micromanometery and ultrasonic dimension transducers while varying preload on right heart bypass both before and 30 min. following 15 min. of global normothermic ischemia induced by aortic cross-clamping. Contractility was quantitated by the slope of the linear preload recruitable stroke work relationship and diastolic mechanics by the slope of the linear end-diastolic pressure vs. volume relationship. The mechanics data are tabulated:

		OLD		YOUNG	
		Slope	r	Slope	r
Systolic	Pre	48.0 (5.9)	.979	55.0 (7.2)	.974
	Post	11.6 (4.8)*	.720	27.7 (5.1)*	.970
Diastolic	Pre	0.178 (0.029)	.859	0.228 (0.043)	.955
	Post	0.193 (0.041)	.960	0.471 (0.069)*	.963

Data: Mean (SEM); Sys:Joules/beat/100gmLV/ml; Dias:mmHg/ml; *p<0.05 ANOVA

Post-ischemic systolic functional injury was significantly greater in the older group (77.3% +/- 10.7% vs. 45.6% +/- 9.5% injury, old vs. young, p=0.05). In contrast, diastolic compliance was preserved in the older hearts (99.4% +/- 4.6% vs. 230% +/- 38% of control, old vs. young, p=0.05). This report is the first to identify a differential ischemic sensitivity for senescent myocardium. Future studies should emphasize specific myoprotective strategies to preserve both systolic and diastolic cardiac mechanics in the aged heart.

*By Invitation

F2. The Use of Combined Antegrade-Retrograde Blood Cardioplegia in Pediatric Open-Heart Surgery - The UCLA Experience

DAVIS C. DRINKWATER, JR. *, CHRIS K. CUSHEN*,

HILLEL LAKS AND GERALD B. BUCKBERG

Los Angeles, California

The benefits of combined antegrade-retrograde blood cardioplegia are becoming well known in adult coronary and valvular heart surgery. Many of these advantages relate directly to the pediatric patient. They include prompt arrest and even distribution, particularly with aortic insufficiency or open aortic root; avoiding or limiting ostial cannulation; allowing uninterrupted surgical procedures; and flushing air/debris from the coronary arteries. We therefore report on the first 86 pediatric patients at UCLA to receive myocar-dial protection using antegrade (aortic) infusion in conjunction with retrograde (coronary sinus) infusion of blood cardioplegia. We employed a retroplegia catheter with a self-inflating and deflating occlusion balloon on the tip of a pressure-monitored infusion cannula that remains in the coronary sinus during the operation. Induction blood cardioplegia, 30 ml/kg in equally divided doses is administered first antegrade at an aortic pressure < 80 mmHg, followed by retrograde infusion at < 40 mmHg in the coronary sinus. Maintenance cardioplegia (15 ml/kg) every 20 minutes is administered

through one or both of the infusion cannula depending on the procedure. Patients' ages ranged from 1 week to 16 years with a mean of 5.5 years. They included the following procedures in descending order: Fontan (14), VSD (and DORV) closure (13), Rastelli (10), AV valve repair or replacement (8), tetralogy of Fallot (8), aortic root/Konno (7), aortic valve repair/replacement (6), coronary reimplantation/fistula ligation (3), arterial switch (2), and AP window, Senning, Stansel (1 each). Aortic cross-clamp times ranged from 23 to 219 minutes with a mean of 98 minutes. One early death occurred in a patient with Truncus Arteriosus (1% 30-day mortality). There were no complications related to the catheter. From this initial positive experience, we conclude that: 1. Combined antegrade-retrograde blood cardioplegia can be safely used in an expanding number of pediatric heart operations in all age groups, and 2. Combined antegrade-retrograde blood cardioplegia may provide additional myocardial protection in complex congenital heart repairs with excellent patient outcome.

*By Invitation

F3. Electrode-Derived Myocardial pH Measurements Reflect Intracellular Myocardial Metabolism Assessed by 31p NMR Spectroscopy During Ischemia

TREVOR C. AXFORD*, JOSEPH A. DEARANI*,

IGOR KHAIT*, W. MICHAEL PARK*,

LEO NEURINGER*, C. ROBERT VALERI*,

MANISHA PATEL*, MHEIR DOURSOUNIAN*

and SHUKRIF. KHURI

Boston and West Roxbury, Massachusetts; Washington, D.C.

and New York, New York

To study the ability of extravascular myocardial tissue pH (MpHe) measured with an intramural electrode to reflect myocardial intracellular metabolic status during ischemia, 14 open-chest dogs had in vivo 31P NMR spectroscopy during left anterior descending coronary artery (LAD) occlusion (experimental group, n = 7) or following sham operation (control group, non-ischemic, n = 7). Spectra were acquired q5min at 4.7 Tesla (256 averages, TR = 1000 msec, pulse width = 30 μsec) with a 2 cm 2-turn RF surface coil. Intracellular myocardial adenosine triphosphate (ATP) peak area was normalized to an external phosphate standard (HCCTP). Change in ATP peak area was expressed as percent of baseline value. During three hours of nor-mothermic ischemia the observed MpHe correlated with NMR calculated myocardial pH in the ischemic dogs with an average r value of 0.94, $p<0.0001$. During this same interval, the fall in MpHe correlated with the loss of ATP in each dog with an average r value of 0.91, $p<0.0001$. Thus, myocardial pH, practically measured with an intramural electrode, correlates with NMR-derived myocardial pH and loss of myocyte ATP content and reflects the metabolic status of the myocyte during ischemia. These data validate the use of extravascular myocardial tissue pH to assess the adequacy of myocardial preservation during aortic cross-clamping in cardiac surgery.

*By Invitation

F4. Enhanced Myocardial Protection During Global Ischemia with 5'-Nucleotidase Inhibitors

STEVEN F. BOLLING*, DOUG C. OLSZANSKI*,

EDWARD L. BOVE, KEITH T. CHILDS*

and KIM P. GALLAGHER *

Ann Arbor, Michigan

Depletion of ATP precursors, such as myocardial adenosine, during global ischemia results in poor postischemic ATP repletion and functional recovery. Neonatal hearts are more resistant to this deleterious effect of ischemia hypothetically, because they are characterized by low 5'-nucleotidase activity, which may result in sustained endogenous myocardial adenosine levels during ischemia. Adult hearts, however, have high levels of 5'-nucleotidase activity leading to depleted myocardial adenosine levels during ischemia and poor post-ischemic functional recovery. Augmenting myocardial adenosine exogenously, during ischemia in adult hearts has a beneficial effect. The present study tests the hypothesis that preservation of adenosine, better ATP repletion and enhanced post-ischemic myocardial recovery in adult hearts could be achieved with a "neonatal" strategy. Therefore, 5'-nucleotidase inhibitors were administered to isolated perfused adult rabbit hearts subjected to 120 minutes of ischemia (at 34°C) to determine if this improved functional recovery or nucleotide precursor availability during ischemia. Hearts received St. Thomas cardioplegia, as controls (CTL); or cardioplegia containing pen-toxifylline (PENT, 500 mg/l pentoxifylline); 4-thioinosine (4TI, 2-(p-nitro-phenyl)-4-thioinosine, 20 μmol/1 in DMSO); or DMSO vehicle alone (0.5 ml DMSO/1 cardioplegia). Results are 45 min after reperfusion. Results are mean ± SD, * = p<.05 vs CTL.

	n	%DP	%dP/dt	dEDP(mmHg)	CF(cc/min)	%MVO2
CTL	10	37 + 8	43 + 10	19+11	38+12	51 + 19
DMSO	6	40 + 8	44 + 7	9+11*	38+13	74 + 6
4TI	8	60+10*	71 + 16*		8 + 5*	39 + 7 81 + 12*
PENT	8	59 + 8*	69+10*	7 + 5*	47 + 8	91 + 17*

Following ischemia and reperfusion, recovery of pre-ischemic systolic function (<%DP and %dP/dt), was significantly better with 5'-nucleotidase inhibition, as was diastolic function (dEDP) and myocardial oxygen consumption (%MVO2). No changes in coronary flow (CF) were noted. The data demonstrate that preventing the catabolism, transport and loss of endogenous adenosine from the heart during global ischemia, with 5'-nucleotidase inhibitors enhances post-ischemia functional recovery. We speculate and are persuing that the mechanism is due to maintenance of intracellular ATP precursor availability.

*By Invitation

F5. Leukocyte-Depleted Controlled Reperfusion of the Regionally Ischemic Myocardium Reduces Stunning, No-reflow and Infarct Size

JOHN G. BYRNE*, ROBERT F. APPLEYARD*,

C. CHIN LEE*, GREGORY S. COUPER*,

FRANK G. SCHOLL*, RITA G. LAURENCE*

and LAWRENCE H. COHN

Boston, Massachusetts

Prolonged ventricular dysfunction (stunning) following emergency coronary grafting for acute regional ischemia carries a high risk of early mortality. Controlling the initial events of reperfusion has been

advocated as essential for myocardial salvage. Since leukocytes and their products (oxygen free radicals) are the most important initiating mediators of reperfusion injury, we tested whether leukocyte-depleted controlled reperfusion would enhance myocardial salvage in a large animal model of acute coronary occlusion and surgical reperfusion.

Methods: After baseline measurements and 90 minutes LAD occlusion, sheep were placed on vented cardiopulmonary bypass (CPB). After 30 minutes cardioplegic arrest, simulating distal anastomoses, the LAD occlusion was released. Before removing the cross-clamp, controlled reperfusion (SOmmHg, 135ml/min) for the first 20 minutes was delivered at the aortic root with either unmodified whole blood (control, n = 7) or blood passed through leukocyte filters (filters, n = 7). The cross clamp was then removed and the animals weaned from CPB. Mean arterial pressure (MAP), the first derivative of LV pressure (dP/dt), LV stroke work index (SWI), regional area systolic shortening (%SS) and regional myocardial blood flow (RMBF) were determined after 3 hours reperfusion. Percent LV area at risk (Ar) and area of necrosis (area necrosis/area risk, An/Ar) were determined at the completion of the experiment.

Results: Filters removed 99% of leukocytes during controlled reperfusion ($p<0.001$ vs. control). There were no significant differences in baseline or end-ischemia (pre-CPB) values for any measurements between groups. Values after 3 hours reperfusion are expressed as mean ± SEM:

	MAP						
	mmHg	dP/dt					
	mmHg/sec	SWI					
	ergs/gm(x103)	%SS(x10-3)	RMBF				
	ml/min/gm	Ar					
	%LV	An/Ar					
Fillers	70 ± 7	1868 ± 105	35 ± 5*	12 ± 12	0.57 ± 0.11*	20 ± 2	40 + 6*
Control	56 ± 8	1592 ± 334	19 ± 4	3 ± 8	0.22 ± 0.05	19 ± 5	70 ± 5

*:$p<0.05$ vs. control.

Improved left ventricular SWI, increased RMBF and reduced An/Ar suggest amelioration of myocardial stunning and the no-reflow phenomenon, and decreased infarct size respectively.

Conclusion: Leukocyte-depleted controlled reperfusion is superior to whole-blood reperfusion for the surgical treatment of acute regional ischemia. Treating the initial events of reperfusion should include methods to prevent leukocyte-mediated reperfusion injury.

*By Invitation

F6. Donor Heart Valves: Electron Microscopic and Morphometric Assessment of Cellular Injury Induced by Warm Ischemia

DONALD G. CRESCENZO*, STEVEN L. HUBERT*,

MARY K. BARRICK*, PHILLIP C. CORCORAN*,

JAMES ST. LOUIS*, ROBERT H. MESSIER*,

ROBERT B. WALLACE and RICHARD A. HOPKINS*

Rockville, Maryland and Washington, D. C.

Cryopreserved allograft valves are being used more frequently as valvular replacements; use is limited by donor tissue availability. Leaflet fibroblast viability has been suggested to influence clinical durability; however, worldwide harvesting protocols currently allow widely variable warm ischemic times (WITs) ranging from zero to 72 hours. The WIT (i.e., time from cessation of donor heart beat to initial cooling in tissue storage solution) is thought to be a critical determinant of cell viability. Metabolic studies conducted in our laboratory have shown that 2 hours of WIT depleted ATP reserves and cells converted from aerobic to anaerobic metabolism to support cell viability, while lactate accumulation continues through 24-36 hours of WIT. The purpose of this study was to apply quantitative morphometric methods to characterize, by transmission electron microscopy (TEM), valvular cellular injury resulting from progressive WITs.

Porcine aortic valve (PAV) tissue was used due to the limited availability of human allograft valves and justified on the basis of comparable histology to human aortic valves. The PAVs were harvested and processed with methods currently used for the cyropreservation of allograft valves. PAVs were harvested with a spectrum of WITs (40 min., 2, 6, 12, 24 and 36 hours; five valves per WIT; N = 30). Following fixation, each leaflet was cut from the free edge to the base (N = 90), divided into five aliquots and processed using standard methods. To ensure randomized tissue selection within each WIT interval, three PAVs and three tissue blocks per valve were selected randomly and thin-sectioned (54 sections). The first ten cells in each thin-section were photographed and cellular injury assessed (cell disruption, dilation of en-doplasmic reticulum, cytoplasmic edema, nuclear and mitochondria! changes). 440 micrographs have been analyzed using Cochran-Mantel-Haenszel statistics to determine if there was a significant association between WIT and cellular injury.

The number of cells demonstrating morphologic evidence of injury at various WITs are as follows: 40 min, 23.2% (21/90); 2 hrs, 30.8% (12/39); 6 hrs, 66.7% (58/87); 12 hrs, 45.3% (43/75); 24 hrs, 79.7% (63/79); 36 hrs, 72.9% (51/70). The following effect of WIT on cell disruption was observed: 40 min, 3.3% (3/90); 2 hrs, 2.6% (1/39); 6 hrs, 10.3% (9/87); 12 hrs, 2.7% (2/75); 24 hrs, 10.1% (8/79); 36 hrs, 25.7% (18/70). Our Findings indicate that there is a significant association between WIT, the extent of cellular injury (WIT 24 hours; $p<0.001$) and cell disruption (WIT 36 hours; $p<0.0001$). These data represent the first ultrastructural (TEM) morphometric evaluation of the effects of WIT in a model analogous to human donor tissue harvesting. Our findings indicate that current allograft harvesting practices with very restrictive WIT allowances result in minimal fibroblast injury; however, these protocols may be overly restrictive leading to the loss of potentially usable tissue.

*By Invitation

F7. Growth of Composite Conduits Utilizing Longitudinal Arterial Autograft

KAZUO SAWATARI*, HIROAKI KAWATA*,

LOIS C. ARMIGER* and RICHARD A. JONAS

Boston, Massachusetts and Auckland, New Zealand

Many reconstructive procedures for congenital heart disease, particularly those requiring conduit insertion, would be more corrective than palliative if they were to incorporate growth potential. We have confirmed the growth potential of a longitudinal strip of autologous aortic wall incorporated in an

autologous pericardial conduit in 10 lambs (mean age 26 days). A 15 mm length of descending thoracic aorta (diameter 11.5 ± .7 mm) was excised and replaced with a composite autograft conduit of autologous pericardium with a longitudinally inserted aortic strip 5 mm in width taken from the excised aortic tissue. Radiopaque markers along all suture lines allowed determination of growth of the aortic autograft relative to growth of entire conduit in addition to growth assessment by pathological analysis. Plain x-rays and aor-tograms were performed at baseline and at 3, 6, 9 and 12 months. No graft became stenotic or aneursymal. Appropriate growth was demonstrated by minimal change in diameter ratio of conduit to distal aorta from 1.00 to 1.02 over 12 months. Aortic strips showed 172 ± 19%, 148 ± 15% and 256 ± 31% incrases in width, length and area, respectively. Histological study confirmed maintenance of normal architecture in the aortic strip. There was in-timal and medial proliferation colonizing the pericardial tissue. A clinical implant using an autologous aortic strip in an aortic homograft in a 4-year old with tetralogy and pulmonary atresia has also demonstrated growth from 15 mm to 21 mm diameter at one year follow-up angiography.

This study confirms that incorporation of autologous arterial wall into cardiac reconstructive procedure allows for subsequent growth. The experimental study also demonstrates the safety of autologous pericardium for aortic reconstruction in the neonatal lamb.

*By Invitation

F8. An Aortic Valve Sparing Operation for Patients with Aortic Incompetence and Aneurysm of the Ascending Aorta

TIRONE E. DAVID and CHRISTOPHER M. FEINDEL*

Toronto, Ontario, Canada

A number of patients (pts) with aortic incompetence (AI) and ascending aorta (AA) aneurysm have normal aortic valve (AV) leaflets. The AI is caused by annular distorsion and or dilatation. We have repaired the AV and replaced the AA in these pts employing the following technique. All three aortic sinuses were excised, leaving only 0.5 cm of aortic tissue attached to the annulus. Interrupted horizontal mattress sutures (4-0 polyester) were passed from inside to outside of the aortic root at a plane corresponding to the lowest level of the annulus. These sutures were then passed through one end of a collagen impregnated tubular Dacron graft in such a way to correct the annular dilatation. The diameter of the graft was 1.4 times greater than the distance from the base to the free margin of the smallest AV leaflet. The native AV was placed inside the graft and all sutures tied down. The AV commissures were resuspended and secured to the graft with a continuous suture (4-0 polypropylene) similarly to what is done when an AV homograft is implanted. Both coronary arteries were re-implanted and the graft was anastomosed to the distal AA.

This operation has been successfully performed in 8 pts; 5 women and 3 men whose mean age was 43 years, range 21 to 63. Three pts had Marfan's syndrome and 2 had acute aortic dissection. The AI was severe in 6 pts and moderate in 2. There were no operative deaths nor serious postoperative complications. These pts have been followed from 2 to 21 months, mean of 7. Serial Doppler echocardiographic studies indicated that the AV reconstruction remained stable in all pts. Seven had no AI and one had mild AI. No anticoagulants were given and there has been no thromboembolic complication.

This operation provides excellent functional results in pts with AI and AA aneurysm with normal AV, and it may be preferable to composite replacement of the AV and AA with a prosthetic valve.

*By Invitation

F9. CABG Morbidity Decreased by EEC Monitoring

A. DAVID SLATER*, LYNN K. GRIFFITHS*,

JACOBA VAN DER LAKEN*, CHRISTOPHER B. SHIELDS*

and HARVEY L. EDMONDS, JR. *

Louisville, Kentucky

Sponsored by: Laman A. Gray, Jr., Louisville, Kentucky

We have previously demonstrated that persistent increases in slow wave EEC activity occurring during cardiopulmonary bypass predicted signs of postoperative neurologic dysfunction (PND). Without adjustment of surgical/anesthetic technique, PND was observed in 14/48 myocardial revascularization procedures. The current findings describe the effect of increasing cerebral perfusion in response to intraoperative EEG evidence of ischemia.

METHODS: In 30 consecutive patients with coronary artery bypass, the EEG was obtained from 8 bipolar electrode pairs fixed in standard positions. The EEG, ECG, and mean arterial pressure signals were amplified, digitalized, displayed, and stored using a signal analyzer (Cadwell Labs, Kennewick, WA). Ischemic events were identified by a 5 min 3 standard deviation increase in relative (% total) delta (1.5-3.5 Hz) EEG power referred to individualized reference norms established prior to insertion of the perfusion cannulae. Ischemic changes most often occurred during rewarming, and attempts were made to increase cerebral perfusion mechanically or pharmacologically.

RESULTS: Intraoperative ischemic events were detected in 9 of 30 surgeries. Increased perfusion promptly corrected the EEG abnormality in 6 cases. There was no evidence of PND in any of these 6 patients. Signs of PND appeared in only 3 patients, 2 of whom displayed ischemic EEG changes intraoperatively. PND in the third patient appeared to be unrelated to a specific intraoperative event.

DISCUSSION: Prior to interventional monitoring, our rate of PND following myocardial revascularization was 29%. By prompt identification of early cerebral ischemia and intervention, the PND rate has decreased significantly to 10% ($P<0.05$, Chi23.98).

Computerized EEC-based intervention decreased the incidence of PND.

*By Invitation

F10. Poloxamer 188 Improves Neurological Outcome Following Hypothermic Circulatory Arrest

CRAIG K. MEZROW*, MAURIZIO MAZZONI*,

DAVID WOLFE*, HOWARD H. SHIANG*,

ROBERT LITWAK and RANDALL B. GRIEPP

New York. New York

Poloxamer 188, a hydrophilic and hydrophobic copolymer possesses favorable cytoprotective and rheologic properties. We investigated the possibility of improving neurologic outcome with this agent following prolonged periods (150 min.) of hypothermic circulatory arrest (HCA).

PROTOCOL: Thirteen mongrel dogs (20-25 kg) anesthetized with Na pen-tobarbital were cooled to 10 C with combined surface/cardiopulmonary bypass (CPB), arrested for 150 minutes, rewarmed and weaned from CPB. Seven dogs were treated with poloxamer 188 before and following HCA. Six control dogs were saline treated. The Dogs were evaluated (blinded fashion) and observed daily (one week) following HCA for clinically overt neurologic deficits and/or behavioral changes. Neurologic outcome was graded with the following system: Gade I - death within the observation period; Grade II -comatose; Grade III - holds head up; Grade IV - sits up; Grade V - stands; Grade VI - normal in both behavior and gait.

RESULTS: There were no deaths in the Poloxamer 188 treated animals and they manifested significantly less neurologic dysfunction following HCA compared to the control group ($p<0.02$) (Fig. 1).

This study documents that Poloxamer 188 has a significant impact in improving neurological outcome in exceptionally long periods of HCA.

*By Invitation

TUESDAY MORNING, May 7, 1991

9:00 a.m. SCIENTIFIC SESSIONS - International Ballroom

12. Quality of Life After Myocardial Revascularization: Effect of Increasing Age

JOSEPH S. CAREY and RAMON A. CUKINGNAN

Torrance, California

Older patients are being referred for coronary artery bypass surgery (CAB). We studied the effect of increasing age on quality of life (QL), probability of survival (PS) and risk of reoperation (RR) in 2479 patients followed prospectively 1-20 years. QL was determined from annual questionnaires, calculating the health status index (HSI) from the patients' subjective response to surgery: asymptomatic, "greatly improved," HSI = 1.0; mild symptoms, "greatly improved," 0.8; moderate symptoms, "slightly improved," 0.6; "not improved" or "worse," 0.4. Four age groups were studied: = <49 (AG40), 50-59 (AG50), 60-69 (AG60), and = >70 (AG70). Associated problems (LV aneurysm, valve disease, acute myocardial infarction) requiring treatment were present in 17% (61/361) of AG40, 19% (164/858) of AG50, 23% (213/927) of AG60 and 31% (104/333) of AG70 patients. Hospital mortality (HM) for all patients was 6.9% (AG40), 3.7% (AG50), 6.3% (AG60) and 9.9% (AG70). HM in patients undergoing CAB grafts only with EF > .40 was 2.7% (AG40), 1.8% (AG50), 3.1% (AG60) and 4.2% (AG70).

PS and RR were calculated by the Kaplan-Meier method excluding valve and cardiogenic shock patients. PS at 5, 10 and 15 years was .85, .70, .55 (AG40); .87, .68, .54 (AG50); .81, .63, .43 (AG60) and .70, .50, .32 (AG70). RR at 15 years was 27% (AG40), 17% (AG50), 6% (AG60) and 8% (AG70). QL was determined by averaging the mean yearly HSI. For years 1-5, QL was .85 in AG40, .84 in AG50, .89 in AG60 and .90 in AG70. For years 6-10, QL was .81, .80, .86 and .89 respectively.

This study shows that in AG40 and AG50 patients, reoperation rate is high and QL is lower as compared to older patients, reflecting the influence of active coronary atherosclerosis. After age 60, the risk of reoperation drops off significantly and symptomatic relief is maintained, suggesting that the probability of

recurrent atherosclerosis is diminished. Therefore, despite a somewhat higher early risk of mortality, myocardial revascularization is likely to be of lasting benefit to older patients, supporting the rationale of coronary bypass grafting in this group.

*By Invitation

13. Operative Mortality is Less for Unstable Angina Patients Undergoing Coronary Bypass Surgery Early than Late

RAM SHARMA*, ROBERT H. DEUPREE*,

ELLIOTT SCHECTER*, SHUKRI F. KHURI,

ROBERT J. LUCHI* and STEWART M. SCOTT

West Roxbury, Massachusetts, West Haven,

Connecticut, Oklahoma City, Oklahoma, Houston, Texas and

Asheville, North Carolina

We conducted a VA multicenter prospective, randomized trial designed to compare medical (M) and early surgical therapy (S, within 7 days of randomization) in 468 patients (pts) with unstable angina (UA). Two hundred and thirty-seven pts were assigned to M and 231 to S. Of the 237 medical patients 79 patients had to be crossed over to S within the first two years because of failure of M. Of the 231 pts randomized to S, 207 received early surgery, 13 received surgery more than 30 days after randomization, and 11 were never operated upon; Operative mortality (i.e. mortality up to 30 days) in the 207 pts who received early surgery was compared to that in the 79 pts who failed M and crossed over to S (late).

OPERATIVE MORTALITY

	Early S	Late S	p Value
All UA PTS	9/207 (4.3%)	8/79 (10.1%)	0.06
c EF	1/69 (1.5%)	3/26 (11.5%)	0.02
c 3-Vessel Disease & EF }	0/35 (0%)	2/14 (14.3%)	0.03

Conclusion:

Coronary bypass surgery in unstable angina pts should be performed early; delaying surgery increases operative mortality, particularly in pts with reduced ejection fraction.

*By Invitation

14. Ventricular Assist Devices for Postcardiotomy Cardiogenic Shock: A Combined Registry Experience

WALTER E. PAE, JR., CYNTHIA A. MILLER*

and WILLIAM S. PIERCE

Hershey, Pennsylvania

Despite advances in myocardial preservation and cardiac surgical techniques, PCCS remains a significant cause of death. VADs have been employed failing conventional therapy to diminish myocardial work allowing time for metabolic recovery of the ‘stunned' myocardium. Since its inception in 1985, the Combined Registry for the Clinical Use of Mechanical Ventricular Assist Devices, investigators have voluntarily submitted data on patients (nts) receiving VADs for PCCS:

	Left VAD	Right VAD	Bi-VAD	TOTAL
Number Pts	454	107	283	844
Weaned (WN)	227 (50.0%)	43 (40.2%)	101 (35.7%)	371 (43.9%)
Discharged (DC)	122 (26.9%)	27 (25.2%)	48 (17.0%)	197 (23.3%)

In the rare instances of device dependency and no contraindications to transplant, 43 pts underwent "bridge" to cardiac transplant. Of these, 35 pts (81.4%) were transplanted and 20 (46.5%) survived. Coronary artery bypass grafting was the most prevalent original operation. Regardless of the surgical procedure or the type (centrifugal vs. pneumatic) of device used for support, the percent of pts WN and eventually DC was similar. Pts were supported with an mean duration of 3 to 5 days; those pts that were WN but not DC were supported for a significantly longer period of time. There is a linear relationship that as age increases, the probability of being WN and DC decreases, with the lowest rate of DC being 10.6% for pts over 70. Multivariate analysis of complications in pts supported with VAD's indicate: 1) inadequate cardiac output post VAD insertion, 2) pre VAD biventricular failure and 3) infection impact negatively on the ability to WN a pt. Similarly, if a pt is WN, renal failure impacted negatively on DC. Two year actuarial survival for DC pts was 92% for left VAD support, 88% for right VAD support and 94% for biventricular support. This multi-institional experience would continue to support the use of VAD in PCCS.

10:00 a.m. INTERMISSION - VISIT EXHIBITS

*By Invitation

10:45 a.m. SCIENTIFIC SESSIONS - International Ballroom

15. Applicability of Noncardioplegic Coronary Bypass to High Risk Patients

LAWRENCE I. BONCHEK, MARK W. BURLINGAME*,

BRAD E. VAZALES* and EDWARD F. LUNDY*

Lancaster, Pennsylvania

Although some surgeons still prefer noncardioplegic (NCP) coronary bypass (CAB), most surgeons are skeptical of its suitability for high risk patients. We used multivariate discriminant analysis to assess risk factors and results in the first 3000 pts. who had primary CAB without cardioplegia (CP) since our program's inception in 1983. Most grafts were done with intermittent aortic clamping, but local vessel

control was often used without clamping. Average number of grafts was 3.5/pt. Pts. with reoperations or valve operations were excluded. Multivariate predictors of operative death included age, sex, LV dysfunction, preop intraaortic IABP, and urgency of operation. Many pts. were in these high risk subgroups. 879 pts. (29%) were > 70 yrs. old; 346 (11.5%) had EF < .30 and another 77 (2.56%) had LV aneurysms; 196 (6.5%) had acute MI and another 397 (13%) had MI < 1 week preop; 917 (31%) had rest pain in hospital (preinfarction angina). Only 790 (26%) had elective operations.

Overall operative mortality was 1.3% (39/3000): elective 0.5% (4/790); urgent 1.4% (24/1687); emergency 2.1% (11/523). In pts. with EF < 30% mortality was 4.6% (16/346); with age > 70 it was 3.4% (30/879); with acute MI it was 3.1% (6/196); and with LV aneurysmectomy it was 0% (0/77). Only 6.6% (199 pts.) required inotropic support after leaving the operating room and only 1% (30 pts.) required a new IABP postop (only 2 of these 30 died).

Noncardioplegic technique has several advantages. Complexity of equipment, cost, and operating time are reduced. Familiarity with NCP techniques facilitates CAB in the growing number of pts. with aortic calcification that precludes crossclamping and CP. IMA use is simplified in acute coronary occlusion. These results provide reassurance that noncardioplegic CAB provides excellent myocardial protection and operating conditions for primary CAB, and is particularly suitable for high risk pts.

*By Invitation

16. Incremental Risk of Bypass Surgery for Patients with Left Ventricular Ejection Fractions Less than 20%

GEORGE T. CHRISTAKIS*, STEPHEN E. FREMES*,

RICHARD D. WEISEL, JOAN IVANOV*,

TIRONE E. DAVID and TOMAS A. SALERNO

Toronto, Ontario, Canada

Patients undergoing aortocoronary bypass surgery with severe ventricular impairment [left ventricular ejection fraction (LVEF) <20%] are at high risk of operative mortality. The incremental risk factors for mortality in patients with LVEF <20%, and the mechanisms by which they contribute to mortality are not well understood. Between January 1982 and June 1989, 11,177 patients underwent isolated aortocoronary bypass surgery. Forty perioperative variables were collected prospectively and analyzed by multivariate statistical techniques. 8,640 patients had preoperative LVEF >40% with an operative mortality of 2.3%. 2,286 patients had preoperative LVEF between 20 - 39% with an operative mortality of 5.1% and 431 patients had preoperative LVEF <20% with an operative mortality of 9.1%. Patients undergoing coronary bypass surgery with LVEF <20% had a higher incidence of left main stenosis (25%), preoperative intraaortic balloon pump (15%), and underwent urgent surgery for unstable angina more frequently. Stepwise logistic regression analysis revealed that urgent surgery for unstable angina was the only independent risk factor for mortality. Traditional risk factors including redo surgery, age, sex and left main stenosis did not contribute to the operative mortality in this highly selected group of patients with severe ventricular dysfunction. Patients undergoing elective surgery for stable angina with LVEF <20% had a 5% operative mortality. Patients undergoing semi-elective surgery had an 8% operative mortality while the operative mortality increased to 25% for patients undergoing urgent surgery for unstable angina.

CONCLUSION: The major independent predictor of operative mortality for patients with preoperative left ventricular ejection fractions <20% is urgent surgery for unstable angina. Traditional risk factors for

CABG contribute minimally to operative mortality in patients with poor ventricular function. Unstable patients with severe ventricular impairment face a high mortality and medical stabilization should be attempted prior to surgery. Perhaps, modified techniques of myocardial preservation designed to resuscitate the injured heart, should be assessed in this high risk sub-group.

11:30 p.m. ADDRESS BY HONORED SPEAKER

Long-Term Transplantation as a Model

Magdi Yacoub, M.D., London, England

12:15 p.m. CARDIOTHORACIC RESIDENTS' LUNCHEON

Jefferson Room (Tickets Required)

*By Invitation

TUESDAY AFTERNOON, MAY 7, 1991

1:45 p.m. SIMULTANEOUS SCIENTIFIC SESSION A - Congenital Heart Disease - Monroe Room

17. Hypoplastic Aortic Arch with Severe Coarctation in the Neonate: Repair Using Resection, Extended Aortotomy and Primary Anastomosis

GARY K. LOFLAND*, WILLIAM B. MOSKOWITZ*,

MONICA COBLE*, DANIEL PIERONI*,

ROBERT GINGELL * and ANDREW S. WECHSLER

Richmond, Virginia and Buffalo, New York

Although hospital mortality of isolated coarctation repair in infancy is less than 3%, coarctation presenting symptomatically in the neonatal period remains a surgical challenge, especially if there is associated aortic arch hypoplasia. Reported mortality rates for this lesion have ranged from 26 to 61%. Between July, 1987 and October, 1990, 50 consecutive infants (48 neonates) with significant arch hypoplasia and severe coarctation underwent surgical correction. Weights ranged from 1.6 to 3.5 kilograms. Cardiac anomalies included: VSD in 42 (84%), ASD in 37 (74%), TGA in 16 (32%), aortic valve anomalies in 10 (20%), single or common ventricle in 12 (24%), aberrant right subclavian artery in 4, mitral atresia and tricuspid atresia in 5 patients. Excluding PDA, 2.6 additional cardic anomalies were present per patient. Non cardiac anomalies included chromosomal abnormalities in 4 patients, broncho-pulmonary dysplasia in 2, renal failure in 2, cortical and brain stem dysfunction preoperatively in 4, and gastroschisis in 1 patient. Mean age of repair was 6.7 days. Mean weight at repair was 2.8 kilograms. All

patients underwent left thoracotomy, ligation, division, and oversewing of the ductus arteriosus, resection of all ductal and coarctation tissue, extended aortotomy along the aortic arch and proximal descending thoracic aorta and primary anastomosis using 7.0 polypropylene suture. Length of the anastomoses were 1.2-1.7 cm. Patients with significant VSD underwent PA banding with VSD closure at 4-8 months postoperatively. Hospital stay was 13.8 +/- 2.3 days for term infants. Postoperative stay was 9 +/- 2 days with duration of mechanical ventilation 36 +/- 4 hours for the entire series. 4 patients had gradients between 5 and 10 mm of mercury secondary to residual hypoplastic segments proximal to the extent of aortotomy. These gradients had reduced to 0 by 1 month follow-up. 2 patients developed gradients of 15 to 20 mm of mercury at 8 months follow-up, both of whom were treated with coarctation revision at the time of VSD closure. 4 patients had unacceptable gradients following completion of repair at the time of the original surgery, and underwent augmentation of the coarctation repair with either reverse subclavian flap or anastomosis between base of left carotid and base of left subclavian arteries. There were no major complications which lengthened hospital stay. 1 peri-operative death occurred (2%) in a patient with critical aortic stenosis, small (5mm) aortic annulus and LVOTO. Late deaths have occurred in 4 patients between 1 and 6 months post operatively, all from causes unrelated to coarctation or coarctation repair. There were no gradients between upper and lower extremities in any patients at time of death. All patients have been followed with serial upper and lower extremity blood pressure determinations and serial echocardiograms. Thus, short term operative results with this aggressive approach to the hypoplastic aortic arch utilizing totally autologous tissues and a long anastomosis have been very gratifying with reduction in operative mortality from as high as 59% to 2%. These patients will continue to be followed closely for growth of all areas of the aorta including the anastomosis.

*By Invitation

SIMULTANEOUS SCIENTIFIC SESSION A - Congenital Heart Disease - Monroe Room

18. Risk of Recoarctation in Neonates and Infants Following Repair with Patch Aortoplasty, Subclavian Flap and the Combined Resection-Flap Procedure

CHARLES A. DIETL*, ALBERTO R. TORRES*,

RENE G. FAVALORO, CINDY L. FESSLER* and

GARY L. GRUNKEMEIER*

Portland, Oregon and Buenos Aires, Argentina

Between September 1980 and August 1988, forty-seven patients younger than 12 months (27 neonates and 20 infants), underwent repair of aortic coarctation, utilizing three surgical techniques: patch aortoplasty (group A: 5 neonates and 3 infants, mean age 4.5 months), subclavian flap (group B: 11 neonates and 8 infants, mean age 3.1 months), and the combined resection-flap procedure (group C: 11 neonates and 9 infants, mean age 2.7 months). There were 8 early deaths (3 in group A, 3 in group B and 2 in group C), and 2 late deaths (both in group B, after subsequent intracardiac surgery), which occurred in patients with complex coexisting anomalies. 37 patients (5 in group A, 14 in group B, and 18 in group C) were followed between 24 and 108 months (mean 62.8, 56.4 and 45.5 months respectively). Residual gradients were detected in 4 out of 5 patients in group A (range 0 to 60 mmHg, mean 25 mmHg), in 11 out of 14 patients in group B (range 0 to 40 mmHg, mean 18.2 mmHg), and in 1 out of 18 patients in

group C (range 0 to 20 mmHg, mean 1.1 mmHg) ($p<0.01$). So far 4 patients operated during their neonatal period, with gradients greater than 30 mmHg, have required reoperation: 1/5 in group A (20% risk), 3/14 in group B (21% risk), and none in group C (0% risk) ($p<0.01$). In summary, residual gradients and risk of recoarctation are significantly higher when a patch or a subclavian flap were employed. The combined resection-flap procedure (an end-to-end anastomosis enlarged with a subclavian flap) is more effective in avoiding gradients and preventing recoarctation, specially in neonates.

*By Invitation

SIMULTANEOUS SCIENTIFIC SESSION A - Congenital Heart Disease - Monroe Room

19. Logical Approach for Surgical Management of Multiple VSD/118 Patients

CLAUDE PLANCHE*, ALAIN SERRAF*,

JACQUELINE BRUNIAUX*, FRANCOIS LACOUR-GAYET*,

RUTH OUAKNINE* and JEAN LOSAY*

Le Plessis Robinson, France

Sponsored by: John W. Kirklin, Birmingham, Alabama

From January 1980 through September 1990, 118 children underwent surgical closure of multiple VSD. Patients with associated major cardiac malformation were excluded. The mean age and the mean weight at operation were respectively 20 ± 22 months and 8 ± 4.4 kg. Fifty-one (43.2%) were below 1 year old.

Fifty-six children had pulmonary protection, either by previous pulmonary artery banding (n = 48) or by pulmonary valve stenosis, (n = 8). All other patients had severe pulmonary hypertension (mean systolic pressure 71.5 mmHg ± 22.3) and already disabling heart failure (NYHA: III - IV).

The surgical management was based on the localization of the defects and the ventricular dominance which were assessed pre and intra-operatively. Trabecular VSDs were always centered by the moderator band and were therefore divided into low trabecular, mid trabecular and high trabecular.

The perimembranous septum was involved in 89 cases, the trabecular in 113 and the infundibular in 6.

Fifty children had two large VSD, 22 had 3 VSD, 8 had 4 and thirty-eight patients presented the "swiss-cheese"form of the lesion.

Closure of the VSDs included Dacron patch and mattress sutures. They were always first approached through a right atriotomy, which was sufficient for complete repair in 74 cases. In mid trabecular VSDs, section of the moderator band (n = 20) allowed closure of all the defects with a single Dacron patch. In 44 patients the VSDs could not be closed only through a right atriotomy, and a right (n = 28) or a left (n = 14) particularly for low trabecular VSD or both right and left (n = 2) ventriculotomies were necessary to secure the repair.

In hospital mortality was 8.5% (70% CL: 11.9 - 6.5%, 10 patients). The causes of death were related to : low trabecular residual VSD in 5, pulmonary hypertension in 2, hypoplastic right (n = 1) or left (n = 1). ventricles and myocardial infarction in 1. Eighteen survivors had residual VSD. In 11 cases they were

located in the lower part of the trabecular septum and mandate reoperation in 4 with 2 deaths. Four patients required a permanent pacemaker for complete AV-block. Morbidity was significantly increased in low trabecular VSD ($n<0.001$). All but 8 survivors were reviewed. Five years, actuarial survival rate and freedom from reoperation were respectively 89.8% and 95.9%.

We conclude that a surgical management of multiple VSDs based on the anatomy of the defects and on the ventricular dominance constitutes a logical approach to these patients, however patients with low trabecular multiple VSDs represent a group with higher risk of morbidity.

2:45 p.m. INTERMISSION - VISIT EXHIBITS

*By Invitation

3:15 p.m. SIMULTANEOUS SCIENTIFIC SESSION A - Congenital Heart Disease - Monroe Room

20. Conal Enlargement for Diffuse Subaortic Stenosis

SERAFIN Y. DeLEON, MICHEL N. ILBAWI,

RENE A. ARCILLA*, OTTO G. THILENIUS*,

WILLIAM R. WILSON*, DAVID A. ROBERSON*,

ELISE C. DUFFY* and JOSE A. QUINONES*

Oak Lawn, Illinois

Conal enlargement (CE) to relieve diffuse subaortic stenosis (SAS) was done on 12 patients over a 3½year period. Ages ranged from 2 weeks to 12 years (mean 4.4 ± 4). The SAS was due to tunnel outflow in 11 and malattached mitral valve in 1. Subaortic gradient ranged from 10-70 mm Hg (mean 50 ± 21). Seven had ventricular septal defect (VSD) (1 with interrupted aortic arch (IAA), 1 with partial anomalous pulmonary venous drainage) and 2 had endocardial cushion defects (BCD). SAS was distal to VSD in 4 and proximal to VSD in 3. In 8 patients, significant obstruction occurred 2-7 years (mean 4 ± 2) following simple resection of SAS (2), VSD closure (2), VSD closure and SAS resection (2) and ECD repair (2). In 3 infants, the tunnel outflow distal to a large VSD was enlarged and closed with the VSD. One infant also had IAA repair. In 3 patients with SAS proximal to a previously repaired VSD, transatrial CE through the VSD was performed. Another patient had transatrial CE and the remaining 5 patients had modified Konno (aortic valve preserved). There was no early or late death. Two patients had complete heart block (1 had left bundle branch block [LBBB] following previous SAS resection, 1 had concurrent attempted resection and CE). Echographic outflow gradients detected 12 days to 3½ years (mean 1.2 ± 1) post-operatively, ranged from 0-25 mm Hg (mean 7 ± 11) and were mainly at the aortic valve level. All patients are clinically well up to 3½ years of follow-up (mean 1.5 ± 1). We conclude that transatrial or transventricular CE (modified Konno) can be safely performed for diffuse SAS in infants and children. Pre-operative LBBB and attempted resection with CE may predispose to complete heart block.

*By Invitation

21. The Relation of Right Ventricle and Pulmonary Artery Morphology and Treatment Protocols to Survival after Repair of Tetralogy of Fallot: A Two-Institution Study

JOHN W. KIRKLIN, RICHARD A. JONAS,

YASUHISA SHIMAZAKI* and

EUGENE H. BLACKSTONE

Birmingham, Alabama, Boston, Massachusetts and

Osaka, Japan

The cineangiograms were reviewed of 100 consecutive patients (1985-1990) with tetralogy and pulmonary stenosis and no prior procedure, in each of two institutions, Institution A (whose protocols were generally for routine primary repair) and B (whose protocols were generally for preliminary shunting in patients < 9-12 months old). The dimensions of the right ventricular outflow tract, pulmonary trunk (PT), and the entirety of the right (RPA) and left pulmonary arteries and their branches were measured, and related to early and intermediate term outcomes. The youngest patient was 1 day old at catheterization, the median age was 5.7 months, 18 (9%) had important coexisting cardiac anomalies, including complete AV canal, and 7 (3.5%) had large AP collateral arteries.

Survival for 1 /12, 1, and 3 years after study was 97 %, 91 %, and 90%, and was not different both overall and risk-adjusted (P=0.5) between Institutions. Median age at repair (n = 172) was 10.3 months at A, 18.7 at B. Survival overall for 1/12, 1 and 3 years after repair was 94%, 91%, and 91%, not different overall and risk-adjusted in the two institutions. The diameter (as Z-value) of the "annulus," and of the RPA just beyond its origin, and the presence of large AP collateral arteries (APCs) were risk factors for death, as were multiple VSDs, young age at repair, and Down syndrome. Institution, transannular patching, and shunting prior to repair were not. The decreased survival in patients receiving transannular patching (at 1/12, 1, and 3 years, was 90%, 85%, and 85% vs. 98%, 97%, and 97% without) was explained by the higher prevalence in them of severe pulmonary trunk and artery hypoplasia. The strength and shape of the risk factors indicated that survival for at least 12 months (age at repair 6 months, single VSD, no large APCs, no Down) was:

Anulus

Z Prox

RPA

Z Survival Anulus

Z Prox

RPA

Z Survival Anulus

Z Prox

RPA								
Z	Survival							
-3	>-3	98%	-6	>-3	97%	-8	>-3	94%
-3	‰¤-3	94%	-6	‰¤-3	89%	-8	‰¤-3	82%

The protocol of essentially routine primary repair provides as good survival as one of 2-stage repair for infants. In both protocols the probability of survival is related primarily to the degree of RV, PT, and PA hypoplasia, and the incremental risk of young age is now small (predicted 12 month survival after repair, anulus Z -3, RPA Z >-3, age 6 months 98%, age 1 month 94%).

*By Invitation

SIMULTANEOUS SCIENTIFIC SESSION A - Congenital Heart Disease - Monroe Room

22. Transposition of Great Arteries, Ventricular Septal Defect and Pulmonary Outflow Tract Obstruction: Rastelli or Lecompte Procedure?

PASCAL R. VOUHÃ‰, DANIEL TAMISIER*,

FRANCINE LECA * RUTH OUAKNINE*,

YVES LECOMPTE* and JEAN-YVES NEVEUX*

Paris, France and Milano, Italy

Sponsored by: Eduardo Arciniegas, Detroit, Michigan

Between Jan. 1, 1980 and Jan. 1, 1990, 62 pts underwent complete repair for TGA, VSD, POTO. Twenty-two pts (35%) (mean age = 97 ± 87 mths) underwent operation of the Rastelli type: the VSD was enlarged anteriorly in 8 cases, the right ventricle (RV) - pulmonary artery (PA) continuity was established using an extracardiac valved (9/22) or non-valved (13/22) conduit. There were 2 early deaths (9% - 70% CL = 39/0-20%) and one late death (5% - CL = 1%-16%). Late survival rate was 83% ± 9% at 5 years. Six pts (32% - CL = 20%-46%) underwent reoperation after a mean follow-up of 5.5 yrs (residual VSD in one, obstructed conduit in 5). At last follow-up (mean follow-up=4.3 yrs), all pts but one were in functional class I; residual RV-PA gradient (>25 mmHg) was present in 7 cases (37% - CL = 24%-51%). Forty pts (65%) (mean age = 40 ± 39 mths) underwent the Lecompte modification: the infundibular septum was completely resected when present (30/40), anterior translocation of the pulmonary bifurcation was performed in 32 cases, the RV-PA continuity was established by direct anastomosis without prosthetic conduit. There were 5 early deaths (12.5% -CL = 7%-20%) and one late death (3% - CL = 0%-9%). Late survival rate was 84% ± 6% at 5 years. Four pts (12% - CL = 6%-20%) underwent reoperation after a mean follow-up of 3.8 yrs (residual VSD in 2, residual POTO in 2). At last follow-up (mean follow-up=3.7 yrs), all pts were in functional class I; residual RV-PA gradient was present in 6 cases (18% - CL = 11%-27%).

Conclusions: 1) Both procedures provide satisfactory early and late results in patients with TGA, VSD, POTO. 2) The Lecompte procedure allows complete repair at a younger age (40 mths vs 97 mths -

p=0.0007) and may reduce the need for reoperation (12% vs 32% - p=0.08) and the incidence of residual POTO (18% vs 37% - p=0.11).

*By Invitation

SIMULTANEOUS SCIENTIFIC SESSION A - Congenital Heart Disease - Monroe Room

23. Fenestrated Fontan with Delayed Catheter ASD Closure: Improved Results in High Risk Patients

GARY S. KOPF, CHARLES S. KLEINMAN*,

ZIYAD M. HIJAZI*, JOHN T. FAHEY*, MICHAEL L. DEWAR*

and WILLIAME. HELLENBRAND*

New Haven, Connecticut

In the past 12 months, all patients with univentricular hearts and two or more risk factors for a Fontan operation have undergone a fenestrated Fontan procedure with delayed catheter-device closure of the fenestration before discharge three patients had total cavo-pulmonary artery connections (TCPC) and three had direct right atrial-pulmonary artery connections. Ages ranged from 9 months to 2.5 years. Fenestration was carried out with a 4 or 5 mm aortic punch in the ASD patch or intraatrial baffle, depending on size of patient. Preoperative risk factors included increased pulmonary vascular resistance in 4, need for pulmonary artery reconstruction in 3, arrythmias in 2, and decreased ventricular function in 2.

Despite moderate postoperative desaturation, there were no operative deaths. Analysis of the length of ventilatory support, need for inotropic agents, amount of pleural drainage and length of hospital stay showed no significant difference with a matched control group of low risk patients. The fenestration was successfully closed in the cath lab with a catheter tipped umbrella device in 5 patients prior to discharge with marked improvement in arterial saturation, but a 2 to 3 mmHG rise in right atrial pressure and decrease in cardiac output in two patients. In addition one patient had con-committant pulmonary artery dilation and one had closure of a second small atrial septal defect undiscovered at the time of surgery. One patient was discharged with a small ASD due to high right atrial pressures.

In conclusion, the fenestrated Fontan procedure, with a carefully sized fenestration, can be accomplished in high risk patients with low mortality and low post-operative morbidity. Fenestrations can be closed in most cases with catheter tipped devices prior to discharge with maintenance of good hemodynamics. The technique may help lower morbidity and mortality for patients otherwise considered high risk or inoperable for the Fontan procedure.

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

*By Invitation

TUESDAY AFTERNOON, May 7, 1991

1:45 p.m. SIMULTANEOUS SCIENTIFIC SESSION B - General Thoracic Surgery - Lincoln Room

24. Solitary Brain Metastases from Non-Small Cell Lung Cancer: Results of Therapy

MICHAEL BURT*, MAREK WRONSKI*,

JOSEPH GALICICH*, NAEL MARTINI

and ROBERT GINSBERG

New York, New York

The treatment of patients with a solitary brain metastasis (SBM) from NSCLC has been evolving, with most centers recommending resection in patients with good performance status. In order to evaluate the long term results of therapy, we reviewed our 15 yr experience. Methods: Records of 185 consecutive patients who underwent resection of a SBM from NSCLC at our institution from 1974 to 1989 were reviewed. Survival was calculated by Kaplan-Meier method; comparisons by log rank analysis; multivariate analysis by Cox proportional hazards model; significance defined as $p<0.05$. Results: Age: 34-75 yr (median 54); M:F 1:1. Sixty-five (35%) were synchronous (SYN) and 120 (65%) metachronous SBM (META). Discounting the brain metastasis, 68 (37%) were stage 1,13 (7%) stage 2, 62 (33%) stage 3A, 30 (16%) stage 3B, and 12 (6%) stage 4. Histology included 130 (70%) with adeno, 39 (21%) epidermoid, and 16 (9%) large cell carcinoma. There was no significant difference in age, stage, or histology between SYN and META. Complete resection (CR) of the primary NSCLC was accomplished in 115 (63%) overall; 32 (49%) with SYN and 83 (69%) META. Of those having no or incomplete resection, most received external radiation therapy with or without chemotherapy. The overall survival was 1 yr: 55%, 2 yr: 27%, 3 yr: 18%, 5 yr: 13%, and 10 yr: 7% (median 14 mos). There was no significant difference in survival from time of diagnosis of SBM in SYN vs META. The median time to development of META was 12 mos. There was a significant increase in survival for patients with SYN undergoing CR of the lung primary (median 20.8 mos; $n = 32$) versus those not having CR (median 9.9 mos; $n = 33$). When stage (discounting the SBM) and resectability were analyzed by the Cox model, stage had no significant ($p=0.97$) effect, but resectability was still highly significant ($p=0.002$). The ten year survival of SYN with CR of the primary NSCLC was 16 percent. Conclusion: Patients who present with SYN, or develop a META SBM benefit from resection of the SBM. These data also suggest that complete resection of the primary NSCLC in patients undergoing resection of the SBM is important and translates into long term survival. In addition, the local stage of NSCLC, even if extensive, should not preclude resection of primary and SBM for prolonged survival.

*By Invitation

25. Wedge and Segmental Resection for Primary Lung Carcinomas

JOHN C. WAIN*, DOUGLAS J. MATHISEN,

ALAN D. HILGENBERG, ASHBY C. MONCURE

and HERMES C. GRILLO

Boston, Massachusetts

Primary lung cancer excision by parenchyma sparing (PS) methods, either wedge or segmental resection, may be used as a method of choice or to minimize the loss of non-neoplastic lung in patients with compromised pulmonary function. To ascertain the efficacy of PS for these indications, the results of 196 resections from 1978 to 1989 were reviewed. Patients included 94 males, 102 females, mean age 66 ± 10 years (range 29 - 91 years). PS was performed by choice in 128/196 (65%, Group I) or because of compromised pulmonary function (FEV1 ‰¤ 1.0 or <40% predicted, or prior pneumonectomy) in 68/196 (35%, Group II).

Eighty-six wedge and 110 segmental resections were performed. 96 patients underwent mediastinoscopy; all cases had intrathoracic node sampling. Operative mortality was 5/196 (3%). Complications included pneumonia (10), empyema (2), pulmonary embolism (3), myocardial infarction (3) and stroke (2).

Histologically, adenocarcinomas (98/196), squamous cell (54/196) and bronchoalveolar (28/196) carcinomas predominated. Pathologic staging revealed 164 Stage I (113 T1NO, 51 T2NO), 5 Stage II, 19 Stage III-a, 5 Stage III-b and 3 Stage IV lesions. For Stage I lesions, median survival was 44.7 months in Group I, 43.8 months in Group II (p=0.65). Local recurrence was seen in 6/128 (5%) in Group I, in 2/68 (3%) in Group II (p=0.69).

Parenchyma sparing wedge or segmental resections are effective techniques for lung cancer therapy with a low operative mortality and a low incidence of recurrence. No significant difference in survival or in the incidence of local recurrence was found when these methods were used electively for small tumors or to minimize loss of pulmonary tissue in patients with compromised pulmonary function.

*By Invitation

SIMULTANEOUS SCIENTIFIC SESSION B - General Thoracic Surgery - Lincoln Room

26. Anterior Pericardial Tracheoplasty for Congenital Tracheal Stenosis

DAVID A. HEIMANSOHN*, KENNETH K. KESLER*,

MARK TURRENTINE*, YOUSEF MAHOMED*,

LYNN MEANS*, BRUCE MATT* and JOHN W. BROWN

Indianapolis, Indiana

Congenital tracheal stenosis may present as a life threatening anomaly not relieved by endotracheal or tracheostomy intubation. Anterior pericardia! tracheoplasty (APT) has been utilized at our institution for

congenital long segment tracheal stenosis (CLSTS) in infants with impending airway obstruction. These patients have been reviewed to assess the utility, limits and long term results with this technique.

Between 1984-1990, eight infants (age 1-24 mo.) (wt. 1-14 Kg.) with CLSTS have been evaluated with conventional radiographs, CT scans and bronchoscopy. All have required preoperative tracheal intubation to maintain ventilation. Seven have undergone APT through a median sternotomy approach on partial normothermic cardiopulmonary bypass. An average of 10 tracheal rings (range 5-22) were divided anteriorly and a patch of fresh autologous pericardium utilized to enlarge the trachea by 1.5 times the predicted diameter. Fine absorbable monofilament sutures using a running technique were used to attach the pericardium to all tracheal layers except the mucosa. After insertion, the patch and hypoplastic tracheal cartilages were suspended by interrupted sutures to sorrounding vascular and pericardial structures. These infants were sedated and paralyzed for 10 postoperative days while maintaining positive end-expiratory airway pressure to assure patch adherence to the mediastinal tissues. Repeat bronchoscopy was performed prior to extubation to assess stability of the repair.

All 7 infants survived with no patch dehiscence or wound infections. Five were extubated within 14 days and are asymptomatic. They remain free of bronchoscopic narrowing at 1 to 60 months follow-up. The remaining 2 patients were markedly improved but have residual stenosis above or below the operative site secondary to complications of prior tracheostomy. They however, are well palliated after 1 and 30 months. Our review of APT has demonstrated the safety, utility and at least medium-term benefit of this procedure in infants of any age and weight.

2:45 p.m. INTERMISSION - VISIT EXHIBITS

*By Invitation

3:15 p.m. SIMULTANEOUS SCIENTIFIC SESSION B - General Thoracic Surgery - Lincoln Room

27. A New Operation for the Treatment of Innominate Artery Compression of the Trachea

STEVEN M. CLARK*, WARREN W. BAILEY*

and JOHN A. HAWKINS*

Cincinnati, Ohio

Sponsored by: Tom D. Ivey, M.D., Cincinnati, Ohio

Innominate artery compression of the trachea (IACOT) is a well recognized cause of airway obstruction in infants and children. The innominate artery normally originates partially or completely to the left of the trachea in young children and can cause anterior compression of the trachea. Innominate artery suspension has been the standard technique for treating IACOT, but there have been problems with recurrence using this technique. The purpose of this paper is to describe a new technique, innominate artery reimplantation (IAR), and to report our results.

From June 1982 to June 1990, 22 children underwent IAR to correct their IACOT. Ages ranged from 6 weeks to 15 years with a median age of 8 months. All children were evaluated with microlaryngoscopy and bronchoscopy preoperatively to document anterior vascular compression of the trachea. Ten of these children had the diagnosis confirmed with MRI and another child with CT. Indications for operations were reflex apnea in 59.1% (13/22), recurrent bronchopulmonary infection in 18.2% (4/22), severe stridor in 18.2% (4/22), and exercise induced asthma and stridor in 4.5% (1/22).

IAR is performed via a median sternotomy without the use of car-diopulmonary bypass or a shunt. The innominate artery is mobilized and transected at its origin from the aortic arch. The artery is then reimplanted more proximally on the aortic arch using interrupted polypropylene suture. This places the origin of the innominate artery to the right of the trachea thereby alleviating anterior compression of the trachea.

There were no early or late deaths during the study. Follow up was complete in all patients and ranged from 3 weeks to 4 years (mean = 18 months). One patient had a postoperative wound infection. There were no neurologic deficits and all children had a normal brachial pulse at follow up. Twenty of the 22 patients (91%) were completely asymptomatic at follow up. Two patients (9%) had partial resolution of their symptoms at follow up. One of these patients had residual stridor and another patient had resolution of reflex apnea but continued bronchopulmonary infections. Both of these patients had complete resolution of tracheal compression on postoperative micro laryngoscopy and bronchoscopy.

In conclusion, IAR is a viable alternative to innominate artery suspension in the treatment of IACOT. IAR completely removes the anatomic substrate for compression of the trachea and can be performed with minimal morbidity and good long term results.

*By Invitation

SIMULTANEOUS SCIENTIFIC SESSION B - General Thoracic Surgery - Lincoln Room

28. Pulmonary Arterial Reactivity Following Transplantation: Differential Effects of Denervation and Rejection

VIRGINIA M. MILLER*, FOLKE NILSSON* and

CHRISTOPHER G.A. McGREGOR

Rochester, Minnesota and Gothenburg, Sweden

Little is known of blood vessel function in transplanted solid organs during rejection. The separate effects of denervation and rejection have not been elucidated. To study how these two components may affect pulmonary arterial function following lung transplantation, pulmonary arteries were studied from rejecting allotransplanted lungs, autotransplanted lungs and control nonoperated lungs. Pulmonary arteries were dissected, cut into rings and suspended in organ chambers for the measurements of isometric force. In selected rings the endothelium was removed deliberately so that endothelial and smooth muscle function could be studied separately. There was no significant difference between groups in contractions to potassium chloride suggesting no important functional smooth muscle atrophy. Smooth muscle contractions to norepinephrine and the endothelium-derived contractile protein endothelin were reduced in both transplanted groups compared to controls. In rings with endothelium contractions to angiotensin I

were similar in control and autotransplanted groups but reduced significantly in the rejecting group. Serum levels of angiotensin converting enzyme were also reduced significantly during acute rejection: 9.9 ± 1.5 U/L prerejection and 4.2 ± 0.5 U/L postrejection, (n = 12). Relaxations to adenosine diphosphate which are dependent on the presence of intact endothelial cells were reduced significantly only in the rejecting group. Relaxations of the smooth muscle to histamine were greater in the autotransplanted arteries compared to control and were further increased significantly with rejection. These results suggest that pulmonary arterial function changes in transplanted lungs and that such changes are distinct following denervation alone compared with denervation accompanied by rejection. Altered function of the pulmonary arteries may affect vascular resistance in the transplanted rejecting lung. Further, circulating levels of angiotensin coverting enzyme may prove to be a useful indicator of acute pulmonary rejection.

*By Invitation

SIMULTANEOUS SCIENTIFIC SESSION B - General Thoracic Surgery - Lincoln Room

29. Single Lung Transplantation - Factors in Postoperative Infections

J. KENT TRINKLE, JOHN H. CALHOON*.

LARHEA NICHOLS*, J. ROGER DAVIS*,

CALIANN T. LUM* and FREDERICK L. GROVER

San Antonio, Texas

Single lung transplantation (SLTX) is a new option for patients with end-stage lung disease. SLTX evolved slowly due to problems with infection, rejection, preservation, and bronchial healing. Infection remains the most perplexing problem. Chi-Square Analysis was performed on risk factors for cytomegalovirus (CMV) and all postoperative infections (PI) in SLTX operations in 31 patients. Eighteen patients were on preoperative Prednisone. Each patient received postoperative cyclosporine, Imuran, and high-dose methylprednisolone followed by Prednisone. Nonrandomized cytolytic therapy varied between OKT3 (n = 10), ALG (n = 12), or none (n = 9).

PI occurred in 21 patients, 11 of which had CMV. There were no bronchial complications. Preoperative Prednisone did not correlate with PI (p=.641), CMV (p=.739), or death (p=.5347). Postoperative CMV resulted in death in 5 of 11 patients (p=.0766), but when two highly probable, but not culture-proven, deaths are included (p=.0097). With cytolytic therapy 17 of 22 patients had PI, compared to 3 of 9 with no cytolytic therapy (p=.0354). All PI with no cytolytic therapy were minor. However, CMV was present in 13 of 22 with cytolytic therapy, and 0 of 9 with no cytolytic therapy (p=.0003). CMV occurred in 9 of 10 patients with OKT3 compared to 4/12 with ALG (p=.0071). CMV occurred in 0/9 with no cytolytic (p=.0542) compared to ALG. Prophylactic Ganciclovir and immune globulin were used in 17 patients with only 2 CMV in 6 patients also receiving ALG. There was no difference in clinical incidence of rejection, an imprecise diagnosis in SLTX, in the three groups.

CONCLUSIONS: Preoperative Prednisone in SLTX does not increase bronchial complications, PI, CMV, or mortality. CMV is a serious complication in SLTX. Ganciclovir and immune globulin may be effective

prophylaxis for CMV. OKT3 causes a significant increase in CMV. Cytolytic therapy with OKT3 or ALG increases the incidence of PI and CMV in SLTX patients and should not be used!

*By Invitation

SIMULTANEOUS SCIENTIFIC SESSION B - General Thoracic Surgery - Lincoln Room

30. Long Segment Colon Substitution for the Esophagus

DOUGLAS J. MATHISEN, JOHN C. WAIN*,

EARLE W. WILKINS, JR., HERMES C. GRILLO and

ASHBY C. MONCURE Boston, Massachusetts

Long segments of colon have been used for total esophageal substitution for neoplastic or non-neoplastic disorders. To determine the utility of this method of complete esophageal substitution, the use of long segment colon in a single institution from 1955 to 1989 was reviewed. 136 patients were identified - 100 males, 36 females, mean age 51 years (range 3 months - 83 years).

Indications for use were neoplasm in 88 (including 8 tracheoesophageal fistulas) and non-neoplastic disorders in 48 (intractable stricture-35, congenital atresia-10, motility disturbances-3). Colon was used for bypass in 80 or replacement in 56. Left colon was used in 100/136 (74%) and right colon in 36/136 (26%).

Major acute complications included ischemic necrosis (4/100 left colon, 6/36 right colon), cervical anastomotic leak (8) and acute non-vascular perforation (1). Thirty day operative mortality: neoplastic group 16% [14/88 -colon necrosis (7), respiratory failure (5), metastatic disease (1), sudden cardiac death (1)], non-neoplastic group 0%).

Among operative survivors, excellent function (no dysphagia, stable weight) was obtained in 88% (107/122) and good function (mild dysphagia, stable weight) in 10% (12/122). Late complications included proximal anastomotic stenosis (3), graft redundancy (3), bile reflux (2) and esophageal mucocoele (1). Median survival: neoplastic group 9 months (range 2 months -10 years), non-neoplastic group 7.75 years (range 2-25 years).

For selected neoplasms, when conduit choice is limited, or for non-neoplastic disorders, where operative mortality is low and long term function is adequate, long segments of colon are useful conduits for total esophageal substitution. Left colon is significantly less likely to develop ischemic necrosis ($p<0.003$).

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

International Ballroom

*By Invitation

TUESDAY AFTERNOON, May 7, 1991

1:45 p.m. SIMULTANEOUS SCIENTIFIC SESSION C - Cardiac Surgery - International Ballroom

31. Tricuspid Valvulectomy Without Replacement: 20 Years Experience

AGUSTINARBULU, INGIDA ASFAW*

and ROBERT J. HOLMES*

Detroit, Michigan

Since September, 1970, we have operated upon 55 patients with intractable right-sided endocarditis. All patients were Heroin addicts. Fifty-three underwent Tricuspid Valve Excision without replacement (TVE) and two had TVE and Pulmonic Valve Excision without replacement (TVE + PE). Twenty-four (44%) patients returned to their addiction. Six patients (11%) required insertion of a prosthesis: two days to ten years after the TVE in five, and 13 years after TVE + PE in one. In all these six, the indications for surgery was medically refractory right heart failure. Sixteen patients died (29%). Six were EARLY, (11%) within 45 days after the TVE. Only one (2%) of these was related to the TVE. The other five were due to uncontrollable infection. Ten (18%) deaths occurred LATE: nine months to 13 years after the TVE or TVE + PE. Nine of these were related to the drug addiction and one was due to progressive right ventricular failure two months after insertion of a bioprosthesis, 10 years after the TVE. Four of the deaths occurred in patients that required insertion of a tricuspid prosthesis: one on the 5th P.O. day due to low cardiac output (the only EARLY death related to TVE), another died two months later, (the LATE death due to progressive right ventricular failure), and two were due to drug addiction.

Of the 39 patients that are alive since the TVE or TVE + PE, 37 (67%) had not required insertion of a prosthesis. Only two (4%) had mild right ventricular failure. Thirty-five (63%) are hemodynamically stable and none had liver impairment. CONCLUSIONS: 1) Drug addiction is a serious recurrent and lethal disease. It must be carefully considered in the choice of operation for this type of patient; 2) Only 11 % (6/55) of patients that had a TVE or TVE + PE required the insertion of a prosthesis to control medically refractory right heart failure; 3) TVE and TVE + PE remain the operation(s) of choice in the treatment of intractable right-sided endocarditis among drug addicts; and 4) In a small percentage of patients TVE may lead to severe distention and permanent impairment of the right ventricular function.

*By Invitation

SIMULTANEOUS SCIENTIFIC SESSION C - Cardiac Surgery - International Ballroom

32. Reduction and Elimination of Systemic Heparinization During Cardiopulmonary Bypass: Experimental Basis and Clinical Application

LUDWIG K. VON SEGESSER*, BRANKO M. WEISS*,

ELIGIO GARCIA*, ARTHUR VONFELTEN*

and MARCO I. TURINA

Zurich, Switzerland

Heparin-coated perfusion equipment was compared to uncoated equipment during left-heart bypass (LHBP) with roller pump (canine experiments: n = 20; bovine experiments: n = 20) or centrifugal pump (bovine experiments: n= 10), and during closed chest cardiopulmonary bypass (CPB) (bovine experiments: n = 30), or open chest CPB (canine experiments: n =40). Improved biocompatibility of heparin-coated equipment was demonstrated by scanning electron microscopy, hematology, biochemistry, coagulation and hemodynamics. Absence of systemic heparinization resulted in improved hemostasis, superior hemodynamics, preserved renal function, and attenuated hormonal stimulation.

Heparin-coated equipment was clinically evaluated in three different groups of patients (n =46):

1) Resection of descending thoracic aortic aneurysms (n = 23) was performed with heparin-coated LHBP (n = 12) or partial CPB (n = 11) for distal protection and proximal unloading. A single dose of heparin (5000 I.U.) was given to facilitate the use of autotransfusion. All devices remained functional throughout the procedures and no systemic emboli were detected. The sole death in this series (1/24: 4%) occurred (unrelated to the technique) in a patient with ruptured thoraco-abdominal aneurysm requiring surgery in extremis. Paraparesis with spontaneous recovery occurred in 1 patient (1/24: 4%).

2)Coronary artery revascularization randomized for low (ACTMSOs) versus full (ACTMSOs) systemic heparinization was prospectively analyzed in 22 patients perfused with heparin-coated equipment. All patients survived the procedure and no myocardial infaction was diagnosed. Low dose of heparin (8041 ± 1270 I.U. versus 52500 ± 17100 I.U.: $p<0.0005$) resulted in reduced protamin requirements (7875 ± 1918 I.U. versus 31400 ± 14000 I.U.: $p<0.0005$), reduced blood loss (831 ± 373 ml versus 2345 ± 1815 ml: $p<0.01$), reduced blood transfusion requirements (281 ±415 ml versus 2731 ± 2258 ml: $p<0.001$) and less patients receiving blood products (5/12 versus 10/10: $p<0.05$).

3) Rewarming in accidental hypothermia by CPB was successfully performed without systemic heparinization in a patient with hypothermic cardiac arrest (23.3°C) and intracranial trauma.

Conclusion: CPB can be performed without systemic anticoagulation. Our preliminary clinical experience using equipment with improved biocom-patibility demonstrates reduced bypass morbidity.

*By Invitation

SIMULTANEOUS SCIENTIFIC SESSION C - Cardiac Surgery - International Ballroom

33. Surgery or Atrioventricular Node Reentry Tachycardia: Skeletonization of the Atrioventricular Node and Discrete Perinodal Cryosurgery

YOUSUFMOHAMED*, ROBERTD. KING,

DOUGLAS P. ZIPES*, WILLIAM M. MILES*,

LA WRENCE S. KLEIN* and JOHN W. BROWN

Indianapolis, Indiana

Surgical treatment options for interruption of atrioventricular node reentrant tachycardia (AVNRT) include: (1) skeletonization of the atrioventricular node (AVN) by dissecting it from most of its atrial inputs, (2) discrete cryosurgery of the perinodal tissues by applying a series of sequential cryole-sions to the atrial tissues immediately adjacent to the AVN. Both these techniques attempt to interrupt one of the dual AVN conduction pathways while preserving the other.

This report describes 17 consecutive patients (pts) who underwent surgical treatment, 10 pts with skeletonization of the AVN and 7 pts with discrete perinodal cryosurgery. There were 10 females and 7 males and their ages ranged from 28 to 56 years (mean 38). Two of the 17 pts had Wolff-Parkinson-White Syndrome and their accessory pathways were interrupted prior to ablating the AVNRT. All the procedures were performed in a normothermic beating heart while monitoring AV conduction closely. In the skeletonization technique, the right atrial septum was mobilized and the AVN exposed anterior to the tendon of the Todaro. The perinodal cryosurgery procedure was also performed through a right atriotomy and a series of sequential 3 mm cryolesions were placed around the borders of the triangle of Koch on the inferior right atrial septum. There were no operative deaths. Two pts who underwent the skeletonization operation, developed heart block requiring pacemaker therapy. At postoperative electrophysiology study, there were no echoes or AVNRT inducible in any of the 17 pts. All patients have remained free of arrhythmia recurrence and have required no anti-arrhythmic therapy after a follow-up of 5 to 28 months (mean 14).

In conclusion, both AVN skeletonization and perinodal cryosurgery successfully ablate AVNRT however, perinodal cryosurgery appears to be safer in avoiding heart block, is more easily performed and is our procedure of choice for the treatment of medically refractory AVNRT.

2:45 p.m. INTERMISSION - VISIT EXHIBITS

*By Invitation

3:15 p.m. SIMULTANEOUS SCIENTIFIC SESSION C - Cardiac Surgery - International Ballroom

34. Vein Graft Disease: The Clinical Impact of Stenoses in Saphenous Vein to Coronary Artery Bypass Grafts

BRUCE W. LYTLE, FLOYD D. LOOP,

MARLENE GOORMASTIC*, PAUL C. TAYLOR*

and DELOS M. COSGROVE

Cleveland, Ohio

The influence of coronary artery stenoses on patient survival and event-free survival is known but no studies have documented the long-term outcome of patients with stenoses in saphenous vein bypass grafts (SVG). We retrospectively studied 723 patients who underwent a postoperative angiographic study that documented a stenosis of 20-99% in at least one SVG and for whom the initial treatment plan was non-

operative. Patients with only totally occluded SVGs or who underwent reoperation or PTCA within one year after the catheterization that documented the SVG stenosis were excluded. Six hundred nineteen patients had one, 98 had 2 and 6 had 3 stenotic SVGs. Follow-up (mean post-cath interval 83 months, range 1-237 months) documented survival of 83% and 68% and event-free survival of 66% and 43% at five and ten postcatheterization years, respectively. Cox regression analyses were used to identify predictors of late survival and event-free survival. For the entire group, moderate or severe impairment of left ventricular function ($p<0.0001$), increasing interval between operation and catheterization ($p<0.0001$), older age ($p=0.0004$), triple-vessel or left main coronary artery disease ($p=0.0034$), and left anterior descending (LAD) SVG stenosis ($p=0.09$) were associated with decreased late survival. Patients with an operation to catheterization interval ‰¥5 years were at particularly high risk and for that subgroup a stenotic SVG to LAD was a strong predictor of decreased survival ($p=0.0001$) and event-free survival ($p=0.0001$). Symptom status at the time of the catheterization and the degree of SVG stenosis did not significantly influence outcome. Patients ‰¥5 years postoperatively with a SVG to LAD stenosis ‰¥50% has survival of 67% and 45% at 2 and 5 postcatheterization years compared to 97% and 80% for those with a native coronary LAD stenosis ‰¥50% ($p=0.003$). Compared with native coronary stenoses, late vein graft stenoses are more dangerous. Late stenoses in saphenous vein grafts to the LAD coronary artery predict a high rate of death and cardiac events and are an indication for reoperation.

*By Invitation

SIMULTANEOUS SCIENTIFIC SESSION C - Cardiac Surgery - International Ballroom

35. Use of Inferior Epigastric Artery for Coronary By-Pass

JEAN-CLAUDE SCHOEVAERDTS*, MICHEL BUCHE*,

YVES LOUAGIE*, ROBERT DION*,

ER WIN SCHROEDER *, BAUDOIN MARCHANDISE*

and CHARLES CHALANT*

Yvoir and Brussels, Belgium

Sponsored by: Albert Starr, Portland, Oregon

During a learning period (Dec. 88-Dec. 89) 9 CABG operations using a free inferior Epigastric artery (IEA) and both in situ internal Mammary arteries (IMA) were performed in patients with three-vessel disease requiring complete revascularization without saphenous vein available. Favourable early and late angiographic controls and histological studies (Histol. similarity between IMA and IEA) encouraged us to start a prospective study.

MATERIAL: Since January 1990, 33 IEA were used in 32 patients (13 to distal right cor. artery, 11 to post-descending branch of RCA, 2 to posterolateral branch of RCA, 4 to Marginal Circumflex, 1 to Diagonal branch and 2 sequential IEA on 2 Marginal Circumflex branches). In addition, left IMA was anastomosed on LAD in all patients (as single or sequential graft) and right IMA in 28 patients on marginal Cx or distal RCA. The use of IEA was decided because of: no sitable saphenous vein in 12

patients, desire of complete arterial revascularisation in 12 young patients and 4 redo operations, no suitable RIMA in 4 patients (preop. angiographic evidence of unsuitability and/or severe emphysema).

Extraperitoneal approach of IEA is simple but requires standardised technique.

RESULTS: 28/32 patients underwent postoperative coronary angiographic control (on day 10 after operation) and all have accepted a 6 months postoperative angiographic control.

1. One death on postop day 2 from acute tamponnade

2. No postoperative myocardial infarction

3. 28 angiographic postop controls (day 10):

all 28 IEA grafts are patent (30/30) anastomoses are patent) similarly 53/53 IMA grafts (76/77 anastomoses) are patent

4. All patients belong to NYHA functional class I

CONCLUSIONS: If long term patency of IEA and IMA grafts remains comparable, the IEA could be a complementary material for coronary bypass (especially on distal RCA and distal Cx branches).

*By Invitation

SIMULTANEOUS SCIENTIFIC SESSION C - Cardiac Surgery - International Ballroom

36. Reactivity of Gastroepiploic and Internal Mammary Arteries: Relevance to Coronary Artery Bypass Grafting

REBECCA J. DIGNAN*, THOMAS YEH, JR. *

CORNELIUS DYKE*, K. FRANCIS LEE*,

GAR Y BENTON*, MAI DING* and

ANDREWS. WECHSLER

Richmond, Virginia

The GEA is an alternate conduit for CABG. This study tests the hypothesis that its vasoreactive properties are different from the IMA. Human GEA (n = 7) and IMA (n = 44) ring segments were mounted on a strain gauge in oxygenated, normothermic, physiologic saline. Segments were normalized for size according to compliance curves, and stretched to optimal resting length (90% of the internal circumference at 100 mmHg). Potassium chloride (KCL), serotonin (STN), and noradrenaline (NA) were chosen to simulate physiologic vasospasm. Contractions to KCL and a dose response curve to STN or NA were obtained. Sodium nitroprusside (SNP) was used to assess relaxation.

Artery	KCL	NA	EC50**	STN	EC50	SNP		
GEA	17.6±1.5*		15.8±2.1	1.8E-7M		14.0±4*	0.9E-7M	-94±7%
(N), SEM		(23)	(10)	±.34E-7M	(7)	±0.25E-7M	(5)	

IMA (N), SEM	12.1±0.64 (117)	16.1±1.3 (19)	2.8E-7M ±1.4E-7M	4.2±0.6 (7)	1.6E-7M ±1.1E-7M	-89±19% (21)

*p<0.05 by ANOVA; values are pressure expressed in mN/mm2 ± SEM

%Percent relaxation of contraction

**EC50: concentration which produces 50% maximum contraction

Thus, the GEA has increased reactivity (stronger contractions) to KCL (a depolarizing agent) and STN (a product of platelet aggregation) but equal reactivity compared to the IMA to NA (adrenergic) stimulation. The GEA is equally as sensitive (EC50) to NA and STN as the IMA. There is no difference in relaxation to SNP. These data suggest that prevention of platelet or KCL-induced vasospasm may be more important when the GEA is used as an alternate conduit for CABG, reinforcing consideration of platelet inhibitors in the peri-operative interval.

*By Invitation

SIMULTANEOUS SCIENTIFIC SESSION C - Cardiac Surgery - International Ballroom

37. Imparted Relaxation of the Human Mammary Artery After Temporary Clamping

JAMES D. FONGER*, XIMING YANG*,

RICHARD A. COHEN*,

CHRISTIAN C. HAUDENSCHILD*

and RICHARD J. SHEMIN

Boston, Massachusetts

The internal mammary artery (IMA) is now used routinely for coronary artery bypass grafting because of its long-term freedom from atherosclerosis. Preservation of the IMA endothelial cell lining may play an important role in both IMA vasoreactivity and resistance to atherosclerosis. Temporary occlusion of the vascular pedicle with external clamps has the potential for endothelial cell injury. We studied the degree of clamp injury and altered vasoreactivity caused by currently available hard and soft jaw clamps.

IMA specimens from 17 patients were harvested. Each specimen was divided into three vascular rings. A control ring was stored in saline while the other two were clamped for 30 minutes with either a hard or soft jaw clamp. Isometric tensions were measured in all three rings in an organ chamber and the rings were contracted with a thromboxane A2 mimetic, U46619. Vascular relaxation was induced with the endoihelium-dependent drug acetycholine and the endothelium-indepenent drug sodium nitroprusside. Papaverine which is a direct smooth muscle relaxant was given after each trial. The rings were silver stained and graded for percentage of intact endothelial cells by an independent pathologist.

Endothelium-dependent maximal relaxation of the rings was significantly impaired from control after soft (20% vs 91 %; p<0.01) and hard (1% vs 91%; p<0.01) jaw clamping. Relaxation after hard jaw clamping was also significantly less than soft jaw clamping (1%vs 20%; p<0.05). Endothelium-independent

maximal relaxation was not significantly impaired from control after soft jaw clamping (89% vs 97%) but was significantly impaired after hard jaw clamping compaired to control (73% vs 97%; $p<0.01$) and compared to soft jaw clamping (73% vs 89%; $p<0.05$). Papaverine produced complete relaxation of ail rings. Histologic grading of endothelial cell integrity demonstrated a significant reduction after soft jaw clamping versus control (39% vs 79%; $p<0.01$) and a further reduction with hard versus soft jaw clamping (15% vs 39%; $p<0.02$).

In conclusion, external occlusive pressure on the mammary artery pedicle significantly impairs mammary artery endolhelium-dependent relaxation after both soft and hard jaw clamping. Endothelium-independent relaxation was not effected by soft jaw clamping but was significantly impaired by hard jaw clamping. Soft jaw clamping caused significantly less impairment of both endothelium-dependent and independent relaxation. Papaverine will produce maximal relaxation in spite of these impairments. Histology confirmed that less endothelial cell damage occurs with soft jaw clamping. Since temporary occlusive clamping is necessary during the IMA anastomosis, soft jaw clamps significantly reduce the morphologic and physiologic dysfunction of the human internal mammary.

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

International Ballroom

7:00 p.m. MEMBERS' RECEPTION (Tickets Required)

International Ballroom West

*By Invitation

WEDNESDAY MORNING, MAY 8, 1991

7:30 a.m. FORUM SESSION II - General Thoracic Surgery

International Ballroom

F11. Platelet Activating Factor Antagonist Enhances Lung Preservation in a Canine Model of Single Lung Allotransplantation

PHILIP C. CORCORAN*, YINING WANG*,

NEVIN M. KATZ, MARIE L. FOEGH*,

ALI R. ANALOUEI* and ROBERT B. WALLACE

Silver Spring, Maryland and Washington, D. C.

Optimal techniques for lung preservation have yet to be defined. Platelet Activating Factor is a phospholipid released by a variety of cells which promotes inflammation and produces pulmonary abnormalities similar to post-transplantation pulmonary dysfunction. We investigated the effect of the Platelet Activating Factor Antagonist BN 52021 (P) as compared to that of Desferroxamine (D), a commonly available iron chelator known to improve lung preservation. Differential lung function and pulmonary hemodynamics were used to assess preservation after a six hour period of cold ischemic storage in a canine model of left lung allotransplantation. Thirty size- and weight-matched mongrel male canines were used for 15 transplant procedures randomized to one of three preservation techniques. The University of Wisconsin (UW) Solution was used as the basic flush solution. P was added to the flush solution in one group (10 mg/kg-N = 5), D was added to the flush solution in a second group (10 mg/kg-N = 5). No additives were used for the control animals (N = 5). P and D were administered to respective donor animals 30 min prior to organ harvest (10 mg/kg) and recipient animals 30 min prior to reperfusion (10 mg/kg). The pulmonary artery flush solution was administered at 40 ml/kg over 4 min. Recipient animals were monitored with balloon-tipped, flow-directed catheters in both pulmonary arteries and ultrasonic flow probes around each pulmonary artery. Solid state micromanometers measured pressures in the pulmonary artery, the left atrium and the left ventricle. Systemic arterial (SA), right and left pulmonary venous (RPV and LPV) and mixed venous blood samples were analyzed at 1, 2, 4 and 6 hours post-transplantation. Transplanted lung pulmonary venous oxygen tension (PVO2, alveolar-arterial gradient (A-aGRAD), in-trapulmonary shunt fraction (Qs/Qt). pulmonary vascular resistance (PVR), dynamic pulmonary compliance (DPC) and systemic vascular resistance (SVR) at six hours post-transplantation are reported below as mean ± standard error.

	PVO2 (mmHg)				A-aGRAD (mmHg)			
	UW	UW+P	UW+D		UW	UW+P	UW+D	
LPV	202±81	282±53*	307±96*	LPV	468±80	271±69*	252±88†	
	QSQT RATIO (%)				PVR (Dyneseccm-5)			
	UW	UW+P	UW+D		UW	UW+P	UW+D	
LPV	0.54±0.19	0.21±0.04*	0.16±0.05*	LL	319±54	149±71*	115±62*	
	DPC (ml/cm H2O				SVR(Dyneseccm-5)			
	UW	UW+P	UW+D		UW	UW+P	UW+D	
LL	32±6	52±6†	50±3†	SC	1146±171	1090±95*	1063±440*	

(*P<0.05,†P<0.01,*P = No Significance as compared to control, LL = left lung, SC = systemic circulation)

Proton Magnetic Resonance bpectroscopy was performed on specimens from recipient animals to determine total extravascular lung water content (TEVLW). PhadaTEVLWof 57.3 ± 6.4% as compared to 51.9 ± 7.7% for D (P = NS) and 88.6 ± 9.2% for controls (P<0.05). We conclude from these data that the Platelet Activating Factor Antagonist BN 52021 enhances lung preservation to a similar degree as Desferroxamine in a model of canine single lung allotransplantation.

*By Invitation

F12. Future Horizons of Lung Preservation by Application of PAF-Antagonists Compared to Current Clinical Standard (Euro-Collins Flush-Perfusion Versus Donor Core Cooling by Extracorporeal Circulation)

THORSTEN WAHLERS*, STEFAN HIRT*,

HANS-GERD FIEGUTH*, MICHAEL JURMANN*,

AXEL HAVERICH* and HANS GEORG BORST

Hannover, Germany

With the introduction of platelet activating factor antagonists (PAFa) a direct inhibition of ischemia induced reperfusion injury can be achieved by prevention of platelet activation, reduction of microvascular leakage and PAF-induced bronchoconstriction.

Meanwhile two preservation methods are established for clinical lung preservation: donor core cooling by extracorporeal circulation (DCC) and pulmonary artery flush using prostracyclin and Euro-Collins solution (P/ECS). In order to improve results obtained with both methods, we compared the application of a PAF-antagonist (WEB 2170 BS) (0.3 mg/kg bodyweight [bw]/h) for the donor, perfusion solution and throughout the first 6 hours of reperfusion in combination with prostacyclin (20 ng/kg bw/min) and Euro-Collins solution (60cc/kg bw) (P/ECS/PAFa).

Eighteen canine heterotopic heart, orthotopic left lung transplants (tx) were performed in 3 groups of six dogs each after 6 hours of cold ischemia (group A: DCC, group B: P/ECS, group C: P/ECS/PAFa). Myocardial preservation was achieved using St. Thomas Hospital solution (20cc/kg bw) in all groups. After tx cardiorespiratory function was assessed at FiO2 of 0.4.

Results: Post tx superior results were observed with P/ECS/PAFa as expressed by significantly improved oxygenation (PO2) (tab), while cardiac output and pulmonary artery pressure only showed insignificant changes.

tab.: arterial PO2 (mmHg) (mean values + standard deviation)

hours post tx:	3	6	9	12
Group A (DCC):	82.6 + -41.7	76.7 + -41.1	73.6 + -40.4	50.8 + -27.1
Group B (P/ECS):	179 + -70.2	162.3 + -61.4	118.6 + -38.8	122.8 + -34.4
Group C (P/ECS/PAFa):	273.0 + -26.4**/*	238.8 + -63.1**/*	236.0 + -27.0**/*	22.5 + -41.7**/*

p-values (C vs A/C vs B): *p<0.05, **p<0.01

It is concluded, that using the PAF-antagonistic activity of WEB 2170 BS in lung preservation compared to current clinical standards, superior results can be obtained as demonstrated by significantly improved oxygenation following 6 hours of cold ischemia in a canine transplant model.

*By Invitation

F13. Experimental Study on Ischemia-reperfusion Lung Injury in Cardiopulmonary Bypass Using a Rabbit Model

HIKARU MATSUDA *, TOHUR KURATANI*,

YOSHIKI SA WA *, MITSUNORIKANEKO*, SUSUMU

NAKANO* and YASUNARUKAWASHIMA

Osaka, Japan

Pulmonary dysfunction after cardiopulmonary bypass (CPB) may relate to possible ischemia-reperfusion lung injury from secession of pulmonary artery (PA) blood flow. Adults white rabbits (n = 33) were subjected to partial CPB (80 ml/min/kg) under median sternotomy at 32 C with homologous blood prime. Unilateral PA was occluded (PAO+) simulating total CPB using the other as control (PAO-). After 2 hours, CPB was terminated with separate perfusion for PAO+ lung (reperfusion:REP, 60 min) by either of whole blood, leukocyte-depletion (LD by filter), or inhibition of complement activation by FUT (nafamstat mesilate). Lungs were deflated in these groups, and inflated by oxygen in the 4th group (OX). Pulmonary tissue ATP, tissue blood flow (TBF), trans-pulmonary gradients of leukocytes count and C5a level(d-LK, d-C5a), and AaDo2 were measured.

In the control group, PAO + lung showed significant decreases in ATP and TBF at the end of CPB with subsequent significant changes in all indices at REP compared to pre-value. The LD and FUT groups showed significant preventions in these changes and OX-group in part.

	Control	LD	FUT	OX			
	PAO -	PAO +					
	CPB	REP	CPB	REP	REP	REP	REP
ATP (% to pre)	99	98#	*52	*80	920	97#	98#
TBF (% to pre)	*48	96#	*16	*58	92#	94#	64
d-LK (/mm!)	-	74#	-	*611	51#	52#	*432
d-C5a (ng/ml)	-	4#	-	*25	9#	2#	*23
AaDo2 (mmHg)		-	154#	-	*406	154#	162#	159#

(CPB & REP: at each end, C5a:at 15 min. *: significant to pre, #: significant to PAO +, mean value are presented)

These results indicate that complete secession of PA flow during CPB may have a risk to cause ischemia-reperfusion lung injuries with involvements of leukocyte and complement activations.

*By Invitation

F14. Evaluation of Lung Metabolism During Successful 24-hour Canine Lung Preservation

HIROSHI DATE*, AKIHIDE MA TSUMURA*,

JILL K. MANCHESTER*, HIDEFUMI OBO*,

ORIANE LIMA*, JOSHUA M. COOPER*, SUDHIR SUNDARESAN*

and OLIVER H. LOWRY*

St. Louis, Missouri

Using a canine left lung allotransplantation model, we evaluated 24-hour lung preservation using two electrolyte preservation solutions, LPD (low potassium dextran) and LPDG (LPD solution plus 1% glucose). Both donor lungs were flushed with either LPD (group I, n = 6) or LPDG (group II, n = 6), inflated with 100% oxygen and preserved for 24 hours at 10 degrees C. The left lungs were implanted using a pulmonary cooling jacket to prevent re-warming of the lung graft and omentopexy was performed around the bronchial anastomosis. An inflatable cuff with a subcutaneously placed injection port, was placed around the right pulmonary artery at the time of the transplant. Biopsies of the right lung were performed at intervals during preservation for metabolic studies, which included ATP, PCr, glucose, G-6-P, lactate, citrate and malate measured via enzyme assay. Function of the transplanted lung was assessed by measurement of arterial blood gases during temporary occlusion of the contralateral pulmonary artery. Immediately after transplant the PaO2 (FiO2 = 1.0) during right pulmonary artery occlusion was significantly greater in group II than group I (518 ± 50 versus 376 ± 56 mmHg; $p<0.05$). Surviving animals were restudied at intervals up to 22 days at which time they were sacrificed.

In group I, 3 animals survived for 22 days with good lung function while the other 3 were sacrificed after 1, 2 and 10 days because of lung edema, pneumonia and rejection respectively. In group II, 4 animals survived until elective sacrifice while 2 others were sacrificed on day 8 and day 22 because of LA thrombus and rejection respectively. PaO2 during right pulmonary artery occlusion in group II was 546 ± 20 mmHg at 3 days (n = 6), 468 ± 45 mmHg at 8 days (n = 5), and 426 ± 30 mmHg at 22 days (n = 4) which were not significantly different from results in group I. Metabolic studies of the right lung at the end of 24 hours preservation revealed the following results:

	ATP	PCr	Glucose	G-6-P	lactate	citrate	malate
Group I	1.15±0.10	0.28±0.03	02±0.0	11.7±3.6	0.19±0.03	33.1±9.0	15.7±4.5
Group II	1.23±0.09	0.34±0.04	23.5±2.7	82.4±17.2	1.58±0.35	170.8±23.9	58.2±10.0
p Value	NS	NS	<.001	<.01	<.01	<.001	<.01

(Concentrations are u mole/g wet weight for ATP, PCr, glucose and lactate, and n mole/g wet weight for G-6-P citrate and malate).

We conclude that lung preservation at 10 degrees C is associated with maintenance of aerobic cell metabolism and that the addition of glucose to the preservation solution improves lung preservation. Both glycolysis and the citric acid cycle are maintained during such preservation as an energy source, thus protecting lung cells.

*By Invitation

F15. Unilateral Donor Lung Dysfunction Does Not Preclude Successful Contralateral Single Lung Transplantation

JOHN D. PUSKAS*, TIMOTHY L. WINTON*,

JOHN MILLER*, MASINA SCAVUZZO* and

G. ALEXANDER PATTERSON

Toronto, Ontario, Canada

Application of single lung transplantation remains limited by a severe shortage of suitable donor lungs. Potential lung donors are often deemed unsuitable because accepted criteria for lung donors (clear CXR bilaterally; PaO2>300 mmHg with FiO2 = 1.0, PEEP = 5 cm H2O; absence of purulent secretions) may not distinguish between unilateral and bilateral pulmonary pathology. Many adequate single lung grafts may be discarded as a result of contralateral aspiration or pulmonary trauma. We have recently employed intraoperative unilateral ventilation and perfusion to assess single lung function in potential donors with contralateral lung pathology. In the 11-month period ending October 1, 1990, we performed 18 single lung transplants. In 4 of these cases (22%), donor CXR and/or bronchoscopy demonstrated significant unilateral lung injury. Donor PaO2 (FiO2 1.0; PEEP 5 cm H2O) was below the accepted level in each case (246 ± 47 mmHg, mean ± STD). Through the sternotomy employed for multiple organ harvest, the pulmonary artery to the injured lung was clamped. A double-lumen endotracheal tube or endobronchial balloon occlusion catheter was used to permit ventilation of the uninjured lung alone. Repeat PaO2 (FiO2 1.0; PEEP 5 cm H2O) revealed excellent unilateral lung function in all 4 cases (499.5 ± 43 mmHg; $p<0.0004$). These single lung grafts (3 right, 1 left) were transplanted uneventfully into 4 recipients (3 pulmonary fibrosis, 1 primary pulmonary hypertension). Early post-transplant lung function was adequate in all patients. Two patients were extubated within 24 hours. There was 1 late death due to rejection and aspergillus infection; the other 3 patients are alive and doing well. We conclude that assessment of unilateral lung function in potential lung donors is indicated in selected cases, may be quickly and easily performed and may significantly increase the availability of single lung grafts.

*By Invitation

F16. Short and Long Term Results of Experimental Wrapping Techniques for Bronchial Anastomosis

JOSEPH LOCICERO, III*, MALEK MASSAD*,

JUNICHI OBA*, MICHAEL BRESTICKER*

and RODNEY GREENE*

Chicago, Illinois

Sponsored by: Robert W. Anderson, Chicago, Illinois

Major complications of bronchial anastomoses for either transplantation or sleeve resection include early leak, fistula formation, granulation tissue and stenosis. To evaluate the impact of technique on these complications we designed a non-immunocompromised canine model with a totally ischemic bronchial segment. We wished to discover the incidence of early and late complications of a telescoping anastomosis and if wrapping techniques modify them. We autotransplanted 2.5 cm of left mainstem bronchus by telescoping 1mm of proximal into distal bronchus sutured with interrupted 4-0 polyglactin.

The animals were divided into four groups: no wrap (I); omental pedicle wrap (II); detached free omental wrap (III); and gelfoam soaked porcine omental extract (Angiomedical Corp., New York) (IV). Weekly bronchoscopy assessed airway stenosis. Following euthansia at 70 days, the luminal areas of the proximal and distal anastomoses were compared to the origin of the main bronchus.

RESULTS

	Control (I)	Pedicle (II)	Free (III)	Extract (IV)	
Visible Stenosis	3/10	2/10	3/10	1/10	
Proximal Ratio (%)	70 +/-13		82 +/-16	68 +/-14	88 +/-10
Distal Ratio (%)	73 +/-13		80 +/-12	64 +/-13	88 +/-10

No airleak or infection occurred in any group at any time. We conclude that wrapping of a telescoped anastomosis is not necessary to prevent early complications. However, no method completely eliminates stenosis development. Further experiments are required to determine the effects of immunomodulation on this model.

*By Invitation

F17. Oncogene Activation in Esophageal Cancer

ALAN G. CASSON*. TAPAS MUKHOPADHYAY*,

KAREN R. CLEARY*, JAE Y. RO*, SUSAN R. CAFFERTY*

and JACK A. ROTH

Houston, Texas

The molecular genetic events that contribute to the development of esophageal cancer are unknown. The aim of these studies was to screen esophageal tumors for mutations in selected oncogenes. DNA was recovered from 24 archival pathology specimens (10 squamous and 14 adenocarcinoma, with normal esophagus from the resection margin) and target oncogene sequences of interest were amplified in vitro using the polymerase chain reaction (PCR). To screen for mutations in the p53 oncogene, the technique of single stranded conformational polymorphism analysis (SSCP) was developed where PCR-amplified DNA was labelled with radioisotope. Paired samples (tumor and corresponding normal tissue) were then elec-trophoresed across non-denaturing polyacrylamide gels. Relative differences in electrophoretic mobility between radiolabelled samples were found to occur with mutations in the DNA sequence studied, and have now been detected in 5 of the 24 tumor samples (20%). One adenocarcinoma has been sequenced, and a mutation (CAT, histidine) confirmed at codon 273 (normal: CGT, arginine). No mutation was detected in Barrett's epithelium (with low grade-dysplasia) adjacent to this tumor.

In summary, this is the first report of a mutated oncogene in esophageal cancer, implicating p53 in tumorigenesis. Such molecular events may well have clinical prognostic significance for patients with Barrett's epithelium and high-grade dysplasias, as an early marker of tumor development.

*By Invitation

F18. Laser Sealing of hand Sewn Esophageal Anastomoses

JOSEPH S. A UTERI*, MEHMET C. OZ*,

JUAN A. SANCHEZ*, VALLUVAN JEEVANANDAM*,

MICHAEL R. TREAT* and CRAIG R. SMITH*

New York, New York

Sponsored by: Keith Reemtsma, New York, New York

Dehiscence rates of 5-20% have been reported for esophageal anastomoses. Causative factors include ischemia, tension, foreign body reaction, microabscesses, and negative pressure within the thoracic cavity. Because laser assisted tissue sealing (LATS) has been shown to improve anastomotic strength in other tissues, the efficacy of LATS was assessed in a canine model of intrathoracic single-layer hand-sewn esophageal anastomosis. Paired 2 cm transverse incisions (one laser sealed, one control) were made in the proximal and distal esophagus in eight dogs. Both were closed with interrupted single layer 4-0 polyglycolic acid sutures. One closure in each pair was selected at random for LATS, beginning with application of sealant solution (SS), followed by 3 minutes of exposure to diode laser energy (wavelength 808 nm, power density 9.6 W/cm2). SS combines albumin (0.2 cc) and sodium hyaluronate (0.4 cc), used to provide a protein matrix across the anastomosis for ingrowth of fibroblasts, with indocyanine green (1 gtt), which increases uptake of laser energy by the targeted tissues because of selective absorption at a wavelength (805 nm) matching that of the diode laser. Each esophagus was removed and infused with saline under pressure, either at the time of sealing or 7 days postoperatively. Bursting pressures, defined as the intraluminal pressures at which saline leakage appears, are summarized below:

Time	Control	Laser Sealed	
0 days	79 ± 58 mmHg	204 ± 98 mmHg	$p<.05$
7 days	166 ± 87 mmHg	259 ± 38 mmHg	p = ns

HPS staining of laser sealed anastomoses revealed minimal thermal injury to the mucosal surface initially, with regeneration of intact mucosal lining by 7 days postoperatively. Foreign body reaction to SS was not seen. LATS is a rapid, simple technique which increases the strength of single layer hand sewn esophageal anastomoses, and may decrease the incidence of anastomotic leakage in clinical practice.

*By Invitation

F19. Immunotherapy Alters Lung Cancer Response to Oxidative Stress

HELEN W. POGREBNIAK*, WILBERT D. MATTHEWS*

and HARVEY I. PASS*

Bethesda, Maryland

Sponsored by: Robert B. Wallace, Washington, D.C.

Selected immunotherapy (tumor necrosis factor [TNF]) and chemotherapies generate reactive oxygen species (ROS). It is unknown whether lung cancer (A549) sensitivity to ROS therapy is altered by TNF, i.e. are cells made "resistant" via increased oxidative buffering through TNF-induction of manganese superoxide dismutase (MnSOD). To answer this question, we examined A549 cytotoxicity after exposure

to hypoxanthine (H)/xanthine oxidase (XO) with/without TNF pretreatment, and documented changes in MnSOD due to the TNF pretreatment. METHODS: A549 cells, treated with 0, 0.1, 1.0, or 10 ug/ml TNF for 24 hours (n = 13 experiments), were then exposed to 1 mM H/0.1 u/ml XO for 7.5-60 minutes. Controls received H, XO, or media alone. All cells were then washed, and incubated for 5 days, at which time viability was quantitated as the surviving fraction (SF^o) of cells compared to controls using the tetrazolium reduction assay. TNF exposed/unexposed cells were examined for MnSOD and actin using p32 labelled cDNA probes, with calculationn of the expression index (E.I.)- RESULTS: HXO caused a time-dependent decrease in survival; however, pretreatment with TNF at any dose increased cell survival significantly. TNF-exposed cells also increased their expression of MnSOD.

MnSOD	E.I.	[TNF]	7.5 min	15 min	30 min	45 min	60 min
1.00	0	SF:74±3	53 ± 3°	31 ± 2°	23 ± 1°	15 ± 1°	
4.49	0.1	SF:81±3	65 ± 3*°	39 ± 2*°	27 ± 1*°	19 ± 2*°	
4.81	1.0	SF:83±2*	66 ± 3*°	39 ± 2*°	27 ± 1*°	19 ± 2*°	
5.54	10.0	SF:84±3*	69 ± 3*°	38 ± 2*°	28 ± 1*°	18 ± 1*°	

*p2<0.05 from 0 ug/ml TNF; op2<0.05 from 7.5 minutes

CONCLUSIONS: TNF pretreatment, even at small doses, may confer resistance of lung cancer cells to subsequent ROS-based therapies. The resistance of these clones may be due to increased expression of MnSOD. It is possible that alterations in lung cancer cell oxidatitive buffering capacity by immunotherapy based regimens could lead to subsequent treatment failure, especially if the TNF is given concurrently with the other therapies.

*By Invitation

F20. Expanded Applications of Diagnostic and Operative Thoracoscopies

AKIO WAKABAYASHI Orange, California

Thoracoscopy was originally developed 80 years ago but has been underutilized in recent years. Over the past 20 years, the author performed thoracoscopy with increased frequency; 150 cases from 1971 to 1986 (9.4/year) vs. 146 cases from 1987 to 1990 (36.5/year). This increase was due to its expanded applications using the improved optic/video systems and carbon dioxide (CO2) or neodymium yttrium aluminum garnet (YAG) lasers. Diagnostic thoracoscopies included; 59 cases without biopsy and 144 cases with biopsies (pleura 99, mediastinal and aortopulmonary window lymph nodes 25, lungs 15, or others 5). Operative thoracoscopies that were rarely performed before have been carried out more frequently than diagnostic thoracoscopies since 1987 (88 vs. 58 cases). The applications of the operative thoracoscopy included: (1) debridgement of empyema 19, (2) YAG laser vaporization of malignant pleural implants in the treatment of recurrent massive pleural effusion in 3, (3) electrocautery or CO2 laser treatment of spontaneous pneumothorax in 39, (4) CO2 laser contraction of bullae in the treatment of diffuse bullous emphysema in 28; and (5) others 4. The operative mortality rate was low, 0.7% (2/296), the morbidity minimal, and postoperative recovery periods were markedly shorter than those after thoracotomy. The diagnostic thoracoscopy prevented many unnecessary exploratory thoracotomies and achieved very high diagnostic accuracy of 99% (201/203). The operative thoracoscopy replaced thoracotomy in many areas and has opened a new horizon in the management of previously untouched areas.

*By Invitation

WEDNESDAY MORNING, May 8, 1991

9:00 a.m. SCIENTIFIC SESSIONS - International Ballroom

38. Operative Treatment of Ebstein's Anomaly

GORDONK. DANIELSON, DAVID J. DRISCOLL*,

DOUGLAS D. MAIR*, CAROLE A. WARNES* and

WILLIAM C. OLIVER, JR. *

Rochester, Minnesota

Between 1972 and June 1990, 179 patients with Ebstein's anomaly have undergone repair. Ages ranged from 11 months to 64 years. In 72%, tricuspid valve reconstruction was possible and in 26% a prosthetic valve, usually a bioprosthesis, was inserted. Two percent underwent plication and a modified Fontan procedure. There were 12 hospital deaths (6.7%). All 21 patients who had an accessory conduction pathway (Wolff-Parkinson-White syndrome) underwent successful division of the pathway as part of their operative treatment. Follow-up of patients for more than two postoperative years shows 92% to be in New York Heart Association Class I or II. There were 6 late deaths, most sudden and presumably secondary to cardiac arrhythmia. Only 2 patients (1.7%) required reoperation following valve reconstruction (1 and 7 years later, respectively). Exercise testing in patients preoperatively and again late postoperatively snowed a significant improvement in performance; maximal oxygen consumption increased from a mean of 50% of predicted value before operation to a mean of 80% of predicted value after surgery. Postoperative echo-Doppler assessment in patients having valve reconstruction has shown excellent tricuspid valve function in most patients.

*By Invitation

39. Ventricular Septal Defect with Tricuspid Pouch with and without Transposition: Anatomic and Surgical Considerations

FAROUK S. IDRISS, ALEXANDER J. MUSTER*,

MILTON H. PAUL*, CARL L. BACKER* and

CONSTANTINE MAVROUDIS

Chicago, Illinois

Tricuspid valve pouch (TVP) covering a ventricular septal defect (VSD), which decreases its effective orifice, and the left to right shunt, may be misleading the surgeon. Conversely, in transposition of the great arteries (TGA), the bulging TVP may result in left ventricular outflow tract (LVOT) obstruction. In a 10 year review, operated patients were divided into two groups since the effect of the TVP is influenced by which ventricle has the higher pressure. Group I: VSD without TGA and Group II: VSD with TGA. Group I: In 72 of 392 patients, tricuspid valve leaflet (TV) was incised to expose the edges of the true VSD in order to accomplish proper repair. Of those, there were 48 with TVP. The diagnosis of TVP was established by angiography, echocardiography or at surgery. Ages at operation ranged from 5 months to 22 years and the Qp/Qs from 1 to 2.8. 16 had shunts less than 1.5. In one, the TVP produced 40 mm/Hg

gradient in the right ventricular outflow tract. At surgery, through a transatrial approach the pouch was opened radially and the actual VSD patched and the TV repaired. There was no mortality, no significant complications, or TV dysfunction. The average postoperative hospital stay was 4 days. Group II: 6 patients out of 83 operated for TGA with VSD had significant LVOT obstruction from TVP. 5 of those had a Mustard procedure, 2 requiring a LV to PA conduit and in 2 of the 5 the VSD was closed through the PA. One patient had heart transplant following atrial repair and TV replacement. The sixth patient in Group II had a successful arterial switch at 9 years of age and after the presence of LVOT obstruction was proven to be due to TVP.

We conclude that presence of TVP in Group I may mislead to closing false small openings produced by TVP rather than the actual VSD. Incising the TVP is safe and essential for proper exposure and closure of the defect. We recommend surgical repair of anatomically large VSD occluded by TVP even though the left to right shunt may be small. TVP should not be confused with the rare aneurysm of the membranous septum. In Group II, the systemic RV pressure can push the TVP into the LVOT causing significant obstruction and may also be responsible for TV insufficiency following atrial baffle repair. Arterial switch is preferred since it places the obstructive tricuspid tissue in the lower pressure ventricle.

*By Invitation

40. Intermediate-term Survival and Functional Results After Arterial Repair for Transposition of the Great Arteries

FLAVIAN M. LUPINETTI*, EDWARD L. BOVE,

A. REBECCA SINDER*, LOUISE B. CALLOW*,

JOH N. MELIONES*, DENNIS C. CROWLEY*.

ROBERTH. BEEKMAN*, L. LUANN MINICK*

and AMNON ROSENTHAL*

Ann Arbor, Michigan

An assessment of late morbidity and mortality is essential before arterial repair can be considered truly corrective for patients with transposition of the great arteries (TGA). We report our experience with 112 patients who underwent arterial repair in order to evaluate early and intermediate-term results. Operation was performed at a median age of 6 days with 73 patients operated on within the first 14 days of life. Coronary artery anatomy was abnormal in 27 patients. Simultaneous procedures were ventricular septal defect closure (n = 33) and repair of interrupted aortic arch (n =2) or coarctation (n = 5). Hospital mortality was 7/112 (6%), with 3 deaths in the most recent 90 patients (3%). There was one late, noncardiac death. Reoperation for pulmonary artery (PA) stenosis was required in 9 of the first 63 patients (14%), all of whom underwent PA reconstruction with separate patches for closure of the coronary excision sites. Of the last 49 patients, all of whom had PA reconstruction with a single pantaloon pericardial patch, 1 (2%) required reoperation for PA stenosis. No other patients have undergone reoperation. Doppler/echocardiograms performed in 102 of 105 surviving patients at a mean of 13 months after repair have demonstrated normal left ventricular function, an absence of left ventricular outflow gradients, and no more than trivial aortic regurgitation. Gradients across the PA were 19 ± 3 mm Hg in patients with separate PA patches and 7 ± 3 mm Hg in those with a single pantaloon patch ($p = 0.011$). Follow-up is 96%) complete from 1 month to 8 years after operation (mean 2.5 years). Actuarial survival at 5 years including operative mortality was 93%. All patients are in sinus rhythm and none requires

antiarrhythmic medications. These data suggest that PA reconstruction with a single pantaloon patch may be associated with a decreased requirement for reoperation. Intermediate-term survival and functional results are excellent following aterial repair for TGA.

*By Invitation

41. Factors Associated with Marked Reduction in Mortality for Fontan Operations in Patients with Single Ventricle

JOHN E. MAYER, JR., NANCYD. BRIDGES*,

RICHARD A. JONAS, FRANK L. HANLEY*,

JAMES E. LOCK* and ALDO R. CASTANEDA

Boston, Massachusetts

Fontan operations (FO) have historically carried higher operative mortality for patients with single ventricle (SV) than for those with tricuspid atresia. Between 7/88 and 6/90, 87 of 90 patients (96.7%) with SV survived FO (Group A) in one institution, while in the immediately preceeding two years (Group B), only 58 of 72 (80.6%) survived FO for SV (X2= 9.5, p<.01). Comparisons of preoperative factors in groups A and B showed no differences (all with p>. 10) in mean age (A = 6.1 +/- 5.2, B = 7.3 +/- 6.1 yrs), mean pulmonary artery (PA) pressure (A = 12.7 +/- 3.4, B = 12.3 +/- 3.8 mmHg), indexed pulmonary arteriolar resistance (A = 1.6 +/- .8, B = 1.14 +/- .6U), or fractions of patients with systemic or pulmonary venous anomalies (A =17/90, B = 19/72), with PA distortion (A = 13/90, B= 16/72), with age <4 yrs (A = 38/90, B = 26/72), with PA pressure >15 mmHg (A =15/90, B = 10/72, or with pulmonary arteriolar resistance >2 U (A =12/90, B = 4/72). Striking survival differences existed for subgroups with left AV valve atresia/stenosis (A =18/18, B = 14/21, p<.01) and PA distortion (A= 12/13, B = 8/16, p<.01). Differences in surgical management between the two groups included 1) use of cavo-pulmonary connections (A = 84/90, B = 45/72, p< .01), 2) minimizing the size of the intra-atrial baffle (A = 72/90, B = 16/72, p<.01). 3) prior bidirbidirec-tional Glenn for interim palliation (A = 10/90, B = 0/72, p<.01), and 4) fenestration of the intra-atrial baffle (A = 39/90, B =0/72, p<.01). We conclude that with these management modifications FO can now be carried out for patients with complex forms of SV with low mortality risk.

10:20 a.m. INTERMISSION - VISIT EXHIBITS

*By Invitation

11:05 SCIENTIFIC SESSIONS

42. Pulmonary Artery Sling: Results of Surgical Repair in Infancy

CARL L. BACKER*, FAROUK S. IDRISS, LAUREN D. HOLINGER*

and CONSTANTINE MAVROUDIS

Chicago, Illinois

Pulmonary artery (PA) sling is a rare congenital vascular anomaly in which the left PA originates from the right PA and encircles the right bronchus and trachea before entering the left hilum, passing between the trachea and esophagus. This causes severe compression of the trachea and right mainstem bronchus and most infants with this anomaly present with severe respiratory distress within the first year of life. Previous reviews of infants treated surgically for PA sling have reported high mortality with poor patency of the left PA in survivors.

Between 1953 and 1990, eleven children underwent surgical intervention for PA sling, nine males and two females. Age ranged from 8 days to 9 months (mean age 5 months). Bronchoscopy was performed in all patients and complete tracheal rings were the most common associated lesion (5 patients). Six patients had cardiac catheterization. Most recently, computed tomography and magnetic resonance imaging have been used to diagnose PA sling and associated complete tracheal rings when present. Surgical repair consisted of transection of the left PA at its origin from the right PA and reimplantation of the left PA into the main PA anterior to the trachea; via right thoracotomy (1), left thoracotomy (6), or median sternotomy (4). The last three patients in the series have had simultaneous pericardial patch tracheoplasty utilizing extracorporeal circulation at the time of pulmonary artery reimplantation.

There were no operative deaths. Two late deaths occurred, both from airway complications in infants who also had complete tracheal rings and tracheoplasty, at 7 months and 2.5 years postoperatively. Nine patients have had postoperative studies to determine the patency of the left pulmonary artery. Seven anastomoses are patent (78%).

PA sling can be repaired in infancy with low operative mortality and excellent long-term patency utilizing division and reimplantation of the left PA anterior to the trachea. We currently recommend repair at the time of diagnosis with median sternotomy and extracorporeal circulation with simultaneous pericardial patch tracheoplasty if complete tracheal rings are associated.

*By Invitation

43. Cardiac Preservation in Patients Undergoing Transplantation: A Clinical Trial Comparing University of Wisconsin Solution and Stanford Solution

DARRYL G. STEIN*, DAVIS C. DRINKWATER*,

HILLEL LAKS, LESTER C. PERMUT*,

SUSHEELA SANG WAN*, HOWARD I. CHAIT*,

JOHN S. CHILD* and SUNITA BHUTA *

Los Angeles, California

Recent laboratory investigations have shown significantly improved donor heart preservation and function when the University of Wisconsin solution (UW) is used for arrest and storage. These findings prompted us to compare UW to Stanford solution in a clinical trial. With the approval of our Investigational Review Board and notification of the FDA, patient enrollment began in February 1990. After obtaining informed consent, patients were blindly randomized to receive a heart arrested and stored in UW or a heart arrested in Stanford solution and stored in normal saline. Orthotopic transplants were performed in a routine manner. All hearts were initially reperfused with aspartate/glutamate enriched warm blood cardioplegia for 3 minutes followed by whole blood reperfusion. All patients received

dopamine (5 ug/kg/min) and dobutamine (5 ug/kg/min) at the conclusion of cardiopulmonary bypass. Fourteen patients with a mean age of 50.9 were randomized to UW (Group 1), while fifteen patients with a mean age of 53.7 were randomized to Stanford (Group 2). Donor ages were similar with a mean age of 26.9 in group 1 and a mean age of 23.3 in group 2. Mean ischemic time in group 1 (150.7 minutes) was somewhat longer than in group 2 (131.3 minutes). Several differences were observed intraoperatively. The number of patients requiring defibrillation intraoperatively differed between groups with 14%(2/14) of patients in group 1 needing defibrillation compared to 53% (8/15) in group 2 ($p<0.05$, Fischer's Exact Test). The number of patients requiring temporary intraoperative pacing for junctional rhythm or heart block also showed a significant difference with 7% (1/14) of group 1 patients versus 47% (7/15) of group 2 patients requiring pacing ($p<0.05$). Intraoperative inotropic support in addition to our standardized protocol was required in 14% (2/14) of group 1 patients versus 40% (6/15) of group 2 patients. This reached near significance with a p value of 0.13. Ejection fractions as determined by transthoracic echocardiography on the first postoperative day were similar in both groups, 64 ± 3% (mean ± SEM) in group 1 and 65 ± 1% in group 2. With a follow-up of 1 week to 8.5 months, there were no early deaths (30 day) in either group and one late death in group 1. The death occurred in a patient who developed sepsis and multiorgan system failure 72 days after transplantation. We conclude: 1. UW is a safe and effective preservation solution for human cardiac transplantation. 2. Considering the decreased defibrillations, decreased intraoperative pacing, and decreased requirement for inotropic support in the UW group, UW appears to be superior to Stanford for donor heart preservation.

*By Invitation

44. University of Wisconsin Solution Versus Cystalloid Cardioplegia for Donor Heart Perservation: A Randomized Blinded Prospective Human Trial

VALLUVAN JEEVANANDAM*, MARK A. BARR*,

JUAN A. SANCHEZ*, JOSEPHS. AUTERI*,

CHARLES M. MARBOE*, FELICIA A. SCHANKEL*,

CRAIG R. SMITH* and ERIC A. ROSE

New York, New York

University of Wisconsin solution (UWS) improves and prolongs preservation of kidney, liver and pancreas grafts, but concerns about its viscosity and high potassium concentration have hindered its use in heart transplantation. After demonstrating the ability of UWS to prolong preservation times up to 18 hours with orthotopic baboon allografts and showing safety and efficacy in a pilot human trial, we started a randomized prospective trial to compare preservation, within a four hour donor ischemia limit, using UWS to crystalloid cardioplegia and saline storage (CCS).

Forty-two adult heart transplants performed between May and September 1990 were randomized into two groups: 1) UWS (n = 22) - donor hearts arrested (15 ml/kg, 80 mm/hg, 4C) and stored (4C) in UWS (n = 22), and 2) CCS (n = 20) - donor hearts arrested with crystalloid cardioplegia (15 ml/kg, 80 mm/hg, 4C) and stored in saline (4C). The recipient surgeon, data analyst, and pathologist were blinded to the randomization. Recipient age, gender, heart disease and preoperative inotropic support and donor age, gender, inotropic support, and mean ischemic time in hours (UWS 2'36" [1'36" to 2'53"], CCS 2'20" [2'20" to 2'44"] p = ns) were similar. Differences observed between the two groups included: 1) Mean time (minutes) to achieve stable sinus rhythm from reperfusion - UWS 5" (1' to 15'), CCS 19" (3" to 47") $p<.02$; 2) Intraoperative defibrillations - UWS 4/22, CCS 12/20 $p<.02$; 3) Transient cardiac pacing - UWS

2/22, CCS 10/20 p<.02; 4) Integrated postoperative enzyme (I/U) release over 48 hours - CPK: UWS 801.2 ± 468.7, CCS 1187 ± 717.7 p<.01, AST: UWS 207.6 ± 83.7, CCS 282.9 ± 69.0 p<.001. There was no difference in postoperative EKG, endomyocar-dial biopsy or hemodynamics (measured by mixed venous 02 sat and CO) at one week. One UWS patient died from sepsis.

UWS is safe for donor organ arrest and preservation despite high viscosity and potassium concentration. When compared to CCS, hearts preserved in UWS regained electrical activity quicker and had better myocardial protection as demonstrated by enzymatic analysis. Further investigation is required to determine the effects of UWS preservation on long term survival, incidence of rejection and graft atheroscerlosis, and to test the ability of UWS to extend donor ischemic time in human cardiac transplantation.

12:10 p.m. ADJOURN FOR LUNCH

*By Invitation

WEDNESDAY AFTERNOON, MAY 8, 1991

1:30 p.m. SCIENTIFIC SESSION - International Ballroom

45. Rescue Warm Substrate Blood Enriched Cardioplegia for Perioperative Sudden Death

FRIEDHELM BEYERSDORF*, MARVIN M. KIRSH,

GERALD D. BUCKBERG and BRADLEY S. ALLEN*

Frankfurt, Germany, Ann Arbor Michigan and Los Angeles, California

We reported previously experimental and clinical evidence that warm substrate enriched (glutamate/aspartate) blood cardioplegia (rescue car-dioplegia) can resuscitate energy depleted hearts and showed experimentally that myocardial salvage is possible in intractably fibrillating hearts supported by CPR for two hours.

This multi-center report describes an initial experience applying this approach to 11 patients with witnessed perioperative arrest; five elective CABG and 6 with extending infarction. All were stable hemodynamically in the cath lab, operating room or ICU when sudden irreversible ventricular fibrillation developed and progressed to electromechanical arrest in 6 patients. Conventional CPR and defibrillation were unsuccessful and extracorporeal circulation was started from 22 to 112 minutes (mean 62 minutes) after arrest. The LV was vented, the aorta clamped, and 20 minutes of warm 37°C aspartate/glutamate blood cardioplegia was given at 150 cc per minute. All bypass grafts were open with good flows in patients post-CABG, and CABG was done in the two who arrested pre-operatively.

All hearts resumed normal sinus rhythm after aortic unclamping. Ten patients had complete hemodynamic recovery, did not evolve ECG infarction, and improved postoperative ejection fraction measured at one week (42-77%; mean 57%) compared to preoperative levels (17-60%; mean 48%).* One patient was revascularized incompletely and died after 3 days of progressive cardiogenic shock. Nine patients were discharged an average of 10.5 days postoperatively. One died three months later from

mediastinitis. One patient died one month postoperative from a CVA, and another sustained a small CVA (mild left hand weakness).

We conclude that witnessed peri-operative arrest with intractable ventricular fibrillation should be treated aggressively by administering CPR during prompt transfer to the operating room for institution of total vented bypass and delivery of a warm substrate enriched blood cardioplegic solution. This treatment may rescue hearts thought to be damaged irreversibly and may represent a new approach to intractable witnessed cardiac arrest, provided CPR maintains satisfactory cerebral perfusion pressure.

*$p<0.05$

*By Invitation

46. Experience with 28 Cases of Systolic Anterior Motion after Carpentier Mitral Valve Reconstruction

EUGENE A. GROSSI*, AUBREY C. GALLOWAY*,

STEPHENS. COLVIN*, MICHAEL A. PARISH*,

TOHRU ASAI*,. AARON J. GINDEA*,

ITZHAK KRONZON* and FRANK C. SPENCER

New York, New York

Systolic anterior motion (SAM) of the mitral valve with left ventricular outflow obstruction (LVOT) following Carpentier ring annuloplasty has led some surgeons to abandon an otherwise successful mitral reconstruction or to avoid use of a rigid annuloplasty ring. In order to determine the long-term significance of SAM, a detailed study was done on a large group of patients who had Carpentier reconstruction with ring annuloplasty (CR).

Between 3/1/81 and 6/1/90, 439 consecutive patients had CR with or without a concomitant procedure at our institution. Hospital mortality was 4.8% (21/439); LVOT obstruction may have contributed to death in one of these patients with a congenitally small aortic annulus (16 mm). Postoperative echocardiography was performed on all patients except this one, and SAM was found in 28 patients (6.4%, 28/438). Two of the 28 (7.1%) had preexisting IHSS, and 17 (60.7%) had a greater than 3 cm. resection of the posterior mitral leaflet. Only eight of the 28 patients (28.6%) had a resting LVOT gradient (mean = 53 mm Hg). These eight were treated with beta-blockers and remained asymptomatic; none required reoperation. Subsequent echocardiographic studies revealed the disappearance of SAM in 13 of the 28 patients (46.4%) and absence of any LVOT gradient in seven of eight patients (87.5%).

These data demonstrate that SAM after CR with the rigid ring: 1) occurs with a low frequency, 2) is self limiting, and 3) should be managed medically.

*By Invitation

47. Ischemic Mitral Valve Disease: A Classification and Systematic Approach to Management

JAMES H. OURY, JOSEPH C. CLEVELAND*,

WILLIAM W. ANGELL and CARLOS G. DURAN

Missoula, Montana, Tampa, Florida and Riyadh, Saudi Arabia

One-hundred-sixty-one consecutive patients with coronary artery disease (CAD) and mitral valve pathology operated during the past five years were reviewed (98% follow-up). Eighty patients underwent repair and eighty-one replacement with concomitant coronary artery bypass grafting (CABG), Av # grafts = 3. An analysis of this patient group and a classification based on anatomic pathology of the mitral apparatus is shown below.

Classification	# of Pts.	Repair	Replace	Mortality Operative <30 day	Late 5 Year acturial Survival
I. Associated:					
CAD with leaflet or chordal pathology (including redo)	105	48	57	5/105=5%	65%
II. Functional:					
CAD with annular dilatation	32	29	3	4/32=15.5%	45%
III. Organic					
CAD with ischemic Dysfunctional or Infracted papillary muscle	24	3	21	7/24=29%	50%
Totals	161	80	81	16=10%	

The average age of patients in this series was 69 (range 48 to 92). Tissue valves were utilized in the majority of patients when necessary because of patient age and associated medical conditions precluding anticoagulation. Operative technique consisted of meticulous myocardial protection (antegrade and/or retrograde, i.e., coronary sinus cardioplegia), in-traoperative volume loading with transesophageal echo to monitor mitral valve hemodynamics pre and post repair, and a systematic search for the presence of pathology at each level of the mitral apparatus. Flexible ring an-nuloplasty was utilized in all repairs and chordal sparing techniques in all valve replacements. This may well account for the finding of equal mortality for replacement versus repair in this subset of high risk patients. Dysfunction of repaired valves was more common (5/74, 7.0%) than primary tissue valve failure (0 patients).

This classification by specific pathologic anatomy is recommended because it provides a basis for the choice of the appropriate valve procedure, the expected operative mortality, and long-term prognosis. Choice of surgical procedure may be selected according to this classification and the patient's age, with valve repair appropriate for younger (<60 year-old) patients in classification I and II with well-defined valve pathology.

*By Invitation

48. Universal Method for Insertion of Unstented Autografts, Homografts and Xenografts

WILLIAM W. ANGELL, DENNIS PUPELLO*

and STEPHEN HIRO*

Tampa, Florida

In order of decreasing popularity 4 methods have been described for the insertion of unstented valves in the aortic position: (1) double suture line, fully scalloped, (2) intact noncoronary sinus, (3) miniroot inclusion implant with coronary osteal approximation, (4) free aortic root insertion with pedicle coronary osteal implant.

In our series of the first 150 unstented homografts implanted using the fully scalloped double suture line method, 9 patients required reoperation for aortic regurgitation unrelated to valve deterioration within the first 36 postoperative months (2.1%/patient-year). Another 12% of patients developed early diastolic murmur. These early failures are believed to have been technical in origin due to inexact orientation of the free valve resulting in leaflet malalignment and inadequate coaptation inherent in most methods of implantation of the unstented aortic valve. This problem is not observed with the use of the free aortic root as a replacement.

A method of implanting unstented valves in the aortic position has been developed which decreases the probability of early failure resulting from technical malalignment of the valve at the time of implantation. Additionally, this technique permits the surgeon to plan and execute a standard procedure for all cases regardless of the anatomy and pathology of the recipient aortic root or the type of unstented valve selected (homograft, autograft, or xenograft). The salient features of this method are: (1) longitudinal aortotomy to the aortic annulus in the midportion of the noncoronary sinus, (2) proximal interrupted suture line with the valve oriented in the anatomic position, (3) circumferential running prolene side to side approximation of the donor coronary ostea to the recipient coronary ostea, (4) untethered com-misures to all three leaflets, (5) a distal running aortic suture line, (6) nonclosure of the aortotomy.

As with insertion of the free aortic root (method #4), this technique allows the tissue valve to assume its own natural configuration with the only dimensional requirement being an appropriate fit at the proximal suture line. It also has the advantage of leaving the recipient aortic root intact.

This method was used in a consecutive series of 21 patients including 12 homografts, 7 autografts, and 2 xenografts. Intraoperative transvalvular gradients and echocardiograms were followed by repeated echocardiogram at three month intervals. Aortic insufficiency was 0 to trivial in all cases. After one year of follow-up in 12 patients, there is no more than trace aortic insufficiency by echo, no diastolic murmur, and no instance of progressive aortic insufficiency.

In conclusion, we are proposing a method of unstented aortic valve implantation which potentially eliminates early aortic insufficiency secondary to valve malalignment and additionally leaves the recipient aortic root intact.

*By Invitation

49. Durability of Porcine Valves at 15 Years in a Representative North American Population

TOM A. BURDON*, D. CRAIG MILLER,

EDWARD S. STINSON, R. SCOTT MITCHELL*,

PHILIP E. OYER, VAUGHN A. STARNES*

and NORMAN E. SHUMWAY

Stanford, California

Broad use of bioprosthetic tissue valves has been increasingly questioned as more of these valves fail over time due to leaflet fibrocalcification. One thousand six hundred and fifty adult patients (862 AYR and 788 MVR) received a first-generation porcine xenograft bioprosthesis between 1971 and 1980. Follow-up extended to over fifteen years (total cumulative follow-up = 12,012 patient years) and was 95% complete. The average ages for AYR and MYR patients were 59 and 56 years, respectively (range = 16-87 years). Operative mortality rate was 4.5 ± 0.7% (± 70% CL) for AYR and 8.0 ± 0.9% for MVR. Two hundred and fifty-two patients are currently alive with their original bioprostheses (78 ± 1%, 55 ± 2%, and 30 ± 4% of the AYR cohort and 70 ± 2%, 50 ± 2%, and 31 ± 3% of the MVR cohort after 5, 10, and 15 years, respectively). Three hundred and seventy-two patients were withdrawn alive from the analysis after successful reoperation (either replacement of the bioprosthesis or insertion of another valve in a different site). Three hundred and ninety-eight patients underwent reoperation (REDO) to replace the original bioprosthesis; REDO AYR carried a 6.5 ± 1.9% mortality risk; this figure was 6.6 ± 1.7% for REDO MVR. A valve-related morbid or fatal event occurred in 480 patients (224 AYR and 256 MVR). Currently, 13% of AYR and 50% of MVR patients are on long-term anticoagulant therapy. Actuarial rates of freedom (± 1 SEM) from valve-related morbid events were as follows:

TIME	Structural Deterioration	Nonstnictural Dysfunction	Thrombo-embollsni	Prosthetic ValveEndocarditis	Anticoagulant-Related Hemorrhage
AYR					
5 years	99 ± .3%	98 ± .4%	95 ± .8%	96 ± .8%	96 ± .6%
10 years	80 ± 2%	96 ± .9%	90 ± 1%	93 ± 1%	96 ± .7%
15 years	58 ± 4%	84 ± 4%	84 ± 3%	88 ± 2%	96 ± .7%
MVR					
5 years	98 ± 6%	98 ± 5%	90 ± 1%	97 ±.6%	94 ± 1%
10 years	74 ± 2%	95 ± 1%	84 ± 2%	94 ± 1%	91 ± 1%
15 years	36 ± 4%	87 ± 6%	72 ± 6%	87 ± 3%	89 ± 2%

CONCLUSIONS: The durability of these first-generation porcine valves at 15 years was satisfactory; moreover, valve-related morbidity and mortality rates were remarkably low. Most AYR patients and at least one-half of MVR patients can safely be spared from indefinite anticoagulation without excessive risk of thromboembolism. Although one-third of the patients required reoperation, the mortality associated with first REOP was less than 7%. These observations constitute a strong argument favoring the use of porcine valves in most adult patients.

ADJOURN

*By Invitation

GEOGRAPHICAL ROSTER

NECROLOGY

O. Theron Clagett, M.D. Rochester, MN

Louis H. Clerf, M.D. St. Petersburg, FL

Richard G. Connar, M.D. Tampa, FL

W. Andrew Dale, M.D. Nashville, TN

J. Claude Day, M.D. Detroit, MI

Charles Dubost, M.D. Paris, France

William H. Falor, M.D. Akron, OH

Robert J. Flemma, M.D. Milwaukee, WI

Conrad R. Lam, M.D. Farmington Hills, MI

Richard H. Overholt, M.D. Boston, MA

John F. Perry, Jr., M.D. St. Paul, MN

John J. Quinlan, M.D. Kentville, NS

Fiorindo A. Simeone, M.D. Providence, RI

Geographical - UNITED STATES

USA

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

CALIFORNIA

Anaheim

Main, F Beachley

Capistrano Beach

Flynn, Pierce J

Chico

Becker, Ronald M

Coronado

Silver, Arthur W

Covina

Carter, P Richard

Del Mar

Angell, William W

El Cajon

Long, David M, Jr

Fisk, R Leighton

Pappas, George

ARIZONA

Green Valley

McClenathan, James E

Paradise Valley

Nelson, Arthur R

Phoenix

Brown, Lee B

Cornell, William P

Scottsdale

Pluth, James R

Sun City

Read, C Thomas

Tucson

Burbank, Benjamin

Copeland, Jack G, III

Sanderson, Richard G

Sethi, Gulshan K

ARKANSAS

Jasper

Hudson, W A

Little Rock

Campbell, Gilbert S

Read, Raymond C

Williams, G Doyne

El Macero

Andrews, Neil C

Escondido

Mannix, Edgar P, Jr

Flintridge

Penido, John R F

Fresno

Evans, Byron H

Indian Wells

Salyer, John M

Inglewood

Lee, Myles E

Irvine

Connolly, John E

Mason, G Robert

Miller, Don R

La Canada

Meyer, Bert W

La Jolla

Fosburg, Richard G

Hutchin, Peter

Laguna Hills

Oatway, William H, Jr

Loma Linda

Bailey, Leonard L

Wareham, Ellsworth E

Sacramento

Long Beach

Bloomer, William E

Carlson, Herbert A

Stemmer, Edward A

Los Angeles

Buckberg, Gerald D

Davis, Lowell L

DeMeester, Tom R

Benfield, John R

Berkoff, Herbert A

Follette, David M

Harlan, Bradley J

Hurley, Edward J

Miller, George E, Jr

Smeloff, Edward A

Fonkalsrud, Eric W

Holmes, E Carmack

Hughes, Richard K

Kay, Jerome Harold

Khonsari, Siavosh

Laks, Hillel

Lindesmith, George G

Longmire, William, Jr

Maloney, James V, Jr

Mandal, Ashis K

Matloff, Jack M

Morton, Donald L

Mulder, Donald G

Stiles, Quentin R

Waters, Paul F

Los Osos

Aronstam, Elmore M

Martinez

Guernsey, James M

Montebello

Lui, Alfred H F

Oakland

Ecker, Roger R

Iverson, Leigh I G

May, Ivan A

Orange

Gazzaniga, Alan B

Wakabayashi, Akio

Oxnard

Dart, Charles H, Jr

Palm Springs

Goldman, Alfred

Palo Alto

Conn, Roy B

Fogarty, Thomas J

Jamplis, Robert W

McFadden, Paul M

Tyson, Kenneth R T

San Bernardino

Misbach, Gregory A

San Diego

Baronofsky, Ivan D

Chambers, John S, Jr

Daily, Pat O

Jamieson, Stuart W

Kaye, Michael P

Lamberti, John J, Jr

Moreno-Cabral, Ricardo

Peters, Richard M

Trummer, Max J

San Francisco

Ellis, Robert J

Gardner, Richard E

Grimes, Orville F

Hill, J Donald

Leeds, Sanford E

McEnany, M Terry

Rankin, J Scott

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

Jahnke, Edward J, Jr

Lewis, F John

Love, Jack W

Wilson, John L

Pasadena

Ingram, Ivan N

Newman, Melvin M

Pebble Beach
Ramsay, Beatty H

Santa Cruz

Fishman, Noel H

Santa Monica

Nelson, Ronald J

Santa Rosa
Neville, William E

Seal Beach
Baisch, Bruce F

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

State, David

Victorville

Jurado, Roy A

COLORADO

Aspen

Zaroff, Lawrence I

Beulah

Bartley, Thomas D

Colorado Springs

Yee, Edward S

Denver

Brown, Robert K

Clarke, David R

New Haven
Baldwin, John C

Glenn, William W L

Hammond, Graeme L

Kopf, Gary S

Lindskog, Gustaf E

Stern, Harold

New Milford

Okinaka, Arthur J

Norwich
Kelley, Winfield O

Sharon
Wylie, Robert H

Wilton

Pool, John L

Woodbridge

Stansel, Horace

DELAWARE

Newark

Lemole, Gerald M

Wilmington

Pecora, David V

DISTRICT OF COLUMBIA

Washington

Aaron, Benjamin L

Bowles, L Thompson

Katz, Nevin M

Keshishian, John M

Condon, William B

Eiseman, Ben

Grow, John B

Harken, Alden H

Hopeman, Alan R

Paton, Bruce C

Pomerantz, Marvin

Rainer, W Gerald

Wright, George W

Englewood
Kovarik, Joseph L

Lakewood
Swan, Henry

Steamboat Springs
Burrington, John D

Vail
Fuller, Josiah

CONNECTICUT
Avon

Maier, Herbert C

Bridgeport

Rose, Daniel M

Hartford
Kemler, R Leonard

Midgley, Frank M

Randolph, Judson G

Simmons, Robert L

Wallace, Robert B

FLORIDA

Atlantic Beach

Stranahan, Allan

Belleair

Lasley, Charles H

Boca Raton
Seley, Gabriel P

Bonita Springs
Schuster, Samuel R

Clearwater
Wheat, Myron W, Jr

Coconut Grove
Center, Sol

Coral Gables
Cooke, Francis N

Gainesville

Alexander, James A

Jacksonville

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

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

Chickamanga

Kaiser, Gerard A

MacGregor, David C

Papper, Emanuel M

Reis, Robert L

Subramanian, S

Thurer, Richard J

Miami Beach

Greenberg, Jack J

Ripstein, Charles B

Spear, Harold C

Naples

Battersby, James S

Linberg, Eugene J

Smyth, Nicholas P D

North Palm Beach

Dorsey, John M

Orlando

Scott, Meredith L

Sherman, Paul H

Ponte Vedra Beach

Gilbert, Joseph, Jr

Punta Gorda

Taber, Rodman E

St Petersburg

Daicoff, George R

DeMatteis, Albert

Tallahassee

Kraeft, Nelson H

Tampa

Seller, Hawley H

Winter Haven

Maurer, Elmer P R

Winter Park

Bloodwell, Robert D

GEORGIA

Atlanta

Hall, David P

Macon

Dalton, Martin L, Jr

Sealy, Will C

Savannah

Langston, Hiram T

Yeh, Thomas J

HAWAII

Honolulu

Ching, Nathaniel P

Gebauer, Paul W

McNamara, Joseph J

IDAHO

Boise

Herr, Rodney H

ILLINOIS

Chicago

Barker, Walter L

Breyer, Robert H

Campbell, Charles D

Ebert, Paul A

Faber, L Penfield

Ferguson, Mark K

Goldin, Marshall D

Hanlon, C Rollins

Head, Louis R

Hunter, James A

Idriss, Farouk S

Karp, Robert B

Kittle, C Frederick

Leininger, Bernard J

Mavroudis, Constantino

Michaelis, Lawrence

Najafi, Hassan

Raffensperger, John

Replogle, Robert L

Graver, Joseph M

Guyton, Robert A

Hatcher, Charles, Jr

Hopkins, William A

Shields, Thomas W

Thomas, Paul A, Jr

Vanecko, Robert M

Evanston

Anderson, Robert W

Fry, Willard A

Tatooles, Constantine

Glencoe

Rubenstein, L H

Maywood

Keeley, John L

Montoya, Alvaro

Pifarre, Roque

Sullivan, Henry J

Oak Brook

Hudson, Theodore R

Ilbawi, Michel N

Javid, Hushang

Jensik, Robert J

Nigro, Salvatore L

Oak Lawn

DeLeon, Serafin Y

Park Ridge

Baffes, Thomas G

Weinberg, Milton, Jr

Peoria

DeBord, Robert A

Springfield

Wellons, Harry A, Jr

Winnetka

Mackler, S Allen

INDIANA

Indianapolis

Brown, John W

KANSAS

Cunningham

Allbritten, Frank F, Jr

Shawnee Mission

Adelman, Arthur

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

Norman, John C

Ransdell, Herbert, Jr

LOUISIANA

Alexandria

Knoepp, Louis F

Baton Rouge

Berry, B Eugene

Beskin, Charles A

Marrero

O'Neill, Martin J, Jr

Metairie

Ochsner, Alton, Jr

New Orleans

Blalock, John B

DeCamp, Paul T

Hewitt, Robert L

King, Harold

King, Robert D

Mandelbaum, Isidore

Shumacker, Harris B, Jr

Siderys, Harry

IOWA

Cedar Rapids

Lawrence, Montague S

Council Bluffs

Sellers, Robert D

Des Moines

Dorner, Ralph A

Phillips, Steven J

Zeff, Robert H

Iowa City

Behrendt, Douglas M

Ehrenhaft, Johann L

Rossi, Nicholas P

Stanford, William

Lindsey, Edward S

Mills, Noel L

Moulder, Peter V

Ochsner, John L

Pearce, Charles W

Rosenberg, Dennis M

Schramel, Robert J

Webb, Watts R

MAINE

Liberty

Hurwitz, Alfred

Portland

Bredenberg, Carl E

Drake, Emerson H

Rockport

Swenson, Orvar

MARYLAND

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

McLaughlin, Joseph S

Michelson, Elliott

Reitz, Bruce A

Turney, Stephen Z

Watkins, Levi, Jr

Jonas, Richard A

Lazar, Harold L

Levitsky, Sidney

Mathisen, Douglas J

Mayer, John E

Moncure, Ashby C

Neptune, Wilford B

Rheinlander, Harold F

Russell, Paul S

Scannell, J Gordon

Shemin, Richard J

Starkey, George W B

Thurer, Robert L

Weintraub, Ronald

Boylston

Bethesda

Jones, Michael

Chevy Chase

Iovine, Vincent M

Mills, Mitchell

Ellicott City

MacManus, Joseph E

Potomac

Zajtchuk, Rostik

Rockville

Blair, Emil

Towson

Brawley, Robert K

Worton

Walkup, Harry E

MASSACHUSETTS

Boston

Akins, Gary W

Austen, W Gerald

Barsamian, Ernest M

Berger, Robert L

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

Grille, Hermes C

Hilgenberg, Alan D

Okike, Okike N

Brookline

Madoff, Irving M

Burlington

Ellis, F Henry, Jr

Shahian, David M

Watkins, Elton, Jr

Cambridge

Harken, Dwight E

Malcolm, John A

Chestnut Hill

Laforet, Eugene G

Strieder, John W

Concord

Boyd, Thomas F

Soutter, Lamar

Dover

Black, Harrison

Lynnfleld

Wesolowski, Sigmund A

Medford

Desforges, Gerard

Methuen

Wilson, Norman J

North Andover

Cook, William A

Shrewsbury

Moran, John M

Springfield

Engelman, Richard M

Rousou, John A

West Roxbury

Bernhard, William F

Khuri, Shukri F

Westport Harbor

Findlay, Charles W, Jr

Williamstown
Wilkins, Earle 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

Prager, Richard L

Sloan, Herbert

Birmingham

Timmis, Hilary H

Detroit

Arbulu, Agustin

Arciniegas, Eduardo

Levine, Frederick H

Silverman, Norman A

Steiger, Zwi

Stephenson, Larry W

Wilson, Robert F

Grand Rapids
Harrison, Robert W

Meade, Richard H

Rasmussen, Richard A

Tomatis, Luis A

Grosse Pointe

Rochester
Bernatz, Philip E

Danielson, G K

McGoon, Dwight C

McGregor, Christopher G A

Olsen, Arthur M

Orszulak, Thomas A

Pairolero, Peter C

Payne, W Spencer

Puga, Francisco J

Schaff, Hartzell V

St Paul

Lfflehei, C Walton

Miller, Fletcher A

Waubun

DeNiord, Richard N

MISSISSIPPI

Jackson

Hardy, James D

Johnston, J Harvey, Jr

Netterville, Rush E

MISSOURI

Bridgeton

Codd, John E

Chesterfield

Bergmann, Martin

Columbia

Bryant, Lester R

Curtis, Jack J

Silver, Donald

Kansas City
Ashcraft, Keith W

Benoit, Hector W, Jr

Borkon, A Michael

Benson, Clifford D

Leonard
Gerbasi, Francis S

MINNESOTA

Minneapolis

Arom, Kit V

Bolman, R Morton, III

Emery, Robert W

Foker, John E

Gannon, Paul G

Garamella, Joseph J

Helseth, Hovald K

Humphrey, Edward W

Kiser, Joseph C

Molina, J Ernesto

Nicoloff, Demetre M

Johnson, Frank E

Kaiser, George C

Kouchoukos, Nicholas T

Lewis, J Eugene, Jr

Pennington, D Glenn

Roper, Charles L

Spray, Thomas L

Weldon, Clarence S

Willman, Vallee L

MONTANA

Missoula

Oury, James H

NEBRASKA

Omaha

Fleming, William H

Moulton, Anthony L

Schultz, Richard D

NEVADA

Holder, Thomas M

Killen, Duncan A

Mayer, John H, Jr

Padula, Richard T

Piehler, Jeffrey M

Reed, William A

Van Way, Charles W, III

Mount Vernon

Campbell, Daniel C, Jr

St Louis

Earner, Hendrick B

Baue, Arthur E

Connors, John P

Cooper, Joel D

Cox, James L

Ferguson, Thomas B

Flye, M Wayne

New Brunswick

Kunderman, Philip J

Lewis, Ralph J

MacKenzie, James W

Scholz, Peter M

Newark

Abel, Ronald M

Donahoo, James

Gielchinsky, Isaac

Hochberg, Mark S

Swan, Kenneth G

Paterson

Bregman, David

Princeton

Mullen, Donald C

Short Hills

Timmes, Joseph L

Tenafly

Las Vegas

Little, Alex G

NEW HAMPSHIRE

Jaffrey Center

Woods, Francis M

NEW JERSEY

Alpine

Holswade, George R

Bellville

Gerard, Franklyn P

Browns Mills

Fernandez, Javier

McGrath, Lynn B

Camden

Camishion, Rudolph C

East Orange

Auerbach, Oscar

Hackensack

Hutchinson, John E, III

Jersey City

Demos, Nicholas J

Millburn

Parsonnet, Victor

Moorestown

Morse, Dryden P

Morristown

Parr, Grant V S

Mount Laurel

Pierucci, Louis, Jr

Neptune

Bailey, Charles P

Roberts, Arthur J

Gerst, Paul H

NEW MEXICO

Albuquerque

Edwards, W Sterling

Las Vegas

Thai, Alan P

Santa Fe

Davila, Julio C

Silver City

Waddell, William R

NEW YORK

Albany

Foster, Eric D

Bay Shore

Ryan, Bernard J

Bronx

Attai, Lari A

Brodman, Richard F

Fell, Stanley C

Ford, Joseph M

Prater, Robert W M

Hirose, Teruo

Brooklyn

Cunningham, J N, Jr

Levowitz, Bernard S

Sawyer, Philip N

Buffalo

Adler, Richard H

Andersen, Murray N

Bhayana, Joginder N

Hoover, Eddie L

Lajos, Thomas Z

Cooperstown

Blumenstock, David A

East Meadow

Strevey, Tracy E, Jr

Spotnitz, Henry M

Steichen, Felicien M

Fayetteville

Bugden, Walter F

Effler, Donald B

Floral Park

Crastnopol, Philip

Lido Beach

Hines, George L

Loudonville

Alley, Ralph D

New Hyde Park

Amato, Joseph J

Tyras, Denis H

Van De Water, Joseph M

New Rochelle

Rubin, Morris

New York

Acinapura, Anthony J

Adams, Peter X

Bains, Manjit S

Beattie, Edward, Jr

Bloomberg, Allan E

Bowman, Frederick, Jr

Boyd, Arthur D

Cahan, William G

Clauss, Roy H

Conklin, Edward F

Cracovaner, Arthur J

Culliford, Alfred T

Ergin, M Arisan

Friedlander, Ralph

Ginsberg, Robert J

Green, George E

Griepp, Randall B

Holman, Cranston W

Isom, O Wayne

Jaretzki, Alfred, III

King, Thomas C

Subramanian, Valavanur A

Tice, David A

Veith, Frank J

Wichern, Walter, Jr

Wolff, William I

Patchogue

Finnerty, James

Plattsburg

Potter, Robert T

Rochester

Graver, 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

Slingerlands

Kausel, Harvey W

Staten Island

Garzon, Antonio A

Stony Brook

Anagnostopoulos, C

Dennis, Clarence

Soroff, Harry S

Syracuse

Brandt, Berkeley, III

Meyer, John A

Parker, Frederick, Jr

Valhalla

Kirschner, Paul A

Krieger, Karl H

Lambert, Adrian

Litwak, Robert S

Malm, James R

Martini, Nael

McCord, Colin W

McCormack, Patricia M

Nealon, Thomas F, Jr

Redo, S Frank

Reemtsma, Keith

Rose, Eric A

Skinner, David B

Spencer, Frank C

Charlotte

Robicsek, Francis

Selle, Jay G

Taylor, Frederick H

Durham

Jones, Robert H

Lowe, James E

Oldham, H N, Jr

Sabiston, David C

Wolfe, Walter G

Young, W Glenn, Jr

Greenville

Chitwood, Walter R

Oriental

Deaton, W Ralph, Jr

Pinehurst

Fischer, Walter W

Winston-Salem

Cordell, A Robert

Crosby, Ivan K

Hudspeth, Allen S

Moggio, Richard A

Reed, George E

Westhampton Beach

Sarot, Irving A

NORTH CAROLINA

Asheville

Belts, Reeve H

Scott, Stewart M

Takaro, Timothy

Chapel Hill

Keagy, Blair A

Starek, Peter J K

Wilcox, Benson R

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

OKLAHOMA

Jenks

LeBeck, Martin B

Lawton

Barnhorst, Donald A

Oklahoma City

Elkins, Ronald C

Johnston, Frank R

Meredith, Jesse H

Mills, Stephen A

NORTH DAKOTA

Grand Forks

James, Edwin C

OHIO

Canton

Wallsh, Eugene

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

Ivey, Tom D

Wilson, James M

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

Snow, Norman J

Van Heeckeren, Daniel W

Darby

McKeown, John J, Jr

Erie

Kerth, William J

Felton, Warren L, II

Fisher, R Darryl

Greer, Allen E

Munnell, Edward R

Williams, G Rainey

Zuhdi, M Nazih

OREGON

Days Creek

Miller, Arthur C

Portland

Cobanoglu, Adnan

Krause, Albert H

Okies, J Edward

Poppe, J Karl

Salomon, Neal W

Starr, Albert

PENNSYLVANIA

Abington

Frobese, Alfred S

Ardmore

Hargrove, W Clark, III

Bethlehem

Snyder, John M

Bryn Mawr

Haupt, George J

Mundth, Eldred D

Camp Hill

Pennock, John L

Carlisle

DeMuth, William, Jr

RHODE ISLAND

Providence

Karlson, Karl E

Singh, Arun K

Hamburg
Judd, Archibald R

Hershey

Campbell, David B

Pae, Walter E, Jr

Pierce, William S

Waldhausen, John A

Johnstown

Kolff, Jacob

Lancaster

Bonchek, Lawrence I

Rosemond, George P

Witmer, Robert H

Philadelphia

Addonizio, Paul V

Brockman, Stanley K

DiSesa, Verdi J

Dunn, Jeffrey M

Edie, Richard N

Edmunds, L Henry, Jr

Fineberg, Charles

MacVaugh, Horace, III

Nemir, Paul, Jr

Norwood, William I

Wallace, Herbert W

Pittsburgh

Bahnson, Henry T

Clark, Richard E

Ford, William B

Griffith, Bartley P

Hardesty, Robert L

Magovern, George J

Pontius, Robert G

Rams, James J

Rosemont

Sink, James D

SOUTH CAROLINA

Charleston

Bradham, R Randolph

Crawford, Fred A, Jr

Parker, Edward F

Sade, Robert M

Columbia

Almond, Carl H

Landrum

Stayman, Joseph W

Spartanburg

Utley, Joe R

TENNESSEE

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

Gobbel, Walter G, Jr

Hammon, John W, Jr

Merrill, Walter H

Sawyers, John L

Templeton, John, III

Sayre

Sewell, William H

Wayne

Lemmon, William M

Wyncote

Mendelssohn, Edwin

Yardley

Sommer, George N, Jr

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

Coppell

McPhail, Jasper L

Dallas

Adam, Maurice

Estrera, Aaron S

Holland, Robert H

Lambert, Cary J

Mills, Lawrence J

Paulson, Donald L

Platt, Melvin R

Razzuk, Maruf A

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

San Antonio

Cohen, David J

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

Wolcott, Mark W

VERMONT

Burlington

Coffin, Laurence H

Miller, Donald B

Chester Depot

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

Roth, Jack A

Walker, William E

Wukasch, Don C

Kaufman

Davis, Milton V

Lubbock

Bricker, Donald L

Feola, Mario

Richmond

Bosher, Lewis H, Jr

Brooks, James W

Cole, Dean B

Gwathmey, Owen

Lower, Richard R

Wechsler, Andrew S

WASHINGTON

Bellevue

Manhas, Dev R

Bellingham

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

Arlington

Conrad, Peter W

Klepser, Roy G

Charlottesville

Dammann, John F

Kron, Irving L

Minor, George R

Muller, William, Jr

Nolan, Stanton P

Lynchburg

Moore, Richmond L

Huntington

Gonzalez-La vin, Lorenzo

Morgantown

Graeber, Geoffrey M

Murray, Gordon F

Warden, Herbert E

Parkersburg

Tarnay, Thomas J

WISCONSIN

Delafield

Hausmann, Paul F

Varco, Richard L

Friday Harbor

Fox, Robert T

Lawrence, G Hugh

Issaquah

Jarvis, Fred J

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

Jones, Thomas W

Li, Wei-I

Mansfield, Peter B

Miller, Donald W, Jr

Rittenhouse, Edward A

Sauvage, Lester

Thomas, George I

Spokane

Berg, Ralph, Jr

WEST VIRGINIA

Charlestown

Walker, James H

La Crosse

Gundersen, Erik A

Madison

Chopra, Paramjeet S

Kroncke, George M

Mentzer, Robert M, Jr

Young, William P

Marshfield

Myers, William O

Ray, Jefferson F, III

Sautter, Richard D

Mequon

Narodick, Benjamin

Milwaukee

Johnson, W Dudley

Li twin, S Bert

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

Ottawa

Keon, Wilbert J

Todd, Thomas R J

Sudbury

Field, Paul

Gelfand, Elliot T

Sterns, Laurence P

BRITISH COLUMBIA

Kelowna

Couves, Cecil M

Vancouve r

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

NEWFOUNDLAND

St John's

Brownrigg, Garrett M

NOVA SCOTIA

Halifax

Landymore, Roderick W

Murphy, David A

Mabou

Thomas, Gordon W

ONTARIO

Collingwood

Heimbecker, Raymond

Hamilton

Sullivan, Herbert J

North York

Goldman, Bernard S

Nottawa

Key, James A

Walker, George R

Toronto

Delarue, Norman C

Goldberg, Melvyn

Hiebert, Clement

Joynt, George H C

McKneally, Martin F

Mickleborough, Lynda L

Patterson, G Alec

Pearson, F Griffith

Salerno, Tomas A

Scully, Hugh E

Trimble, Alan S

Trusler, George A

Weisel, Richard D

Williams, William G

Westbrook

Lynn, R Beverley

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

Sillery

Grondin, Pierre

OTHER COUNTRIES

AFGHANISTAN
Kabul

Hankins, John R

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

Vienna

Wolner, Ernst

BRAZIL

Sao Paulo

Jatene, Adib D

Zerbini, E J

ENGLAND
Bath, Avon

Belsey, Ronald

Cambridge

Kennedy, John H

Hereford

Thompson, Vernon C

Herefordshire
Smith, Roger A

London

Braimbridge, Mark V

Lennox, Stuart C

Lincoln, Christopher R

Ross, Donald N

Paris
Binet, Jean-Paul

Blondeau, Philip

Cabrol, Christian E A

Carpentier, Alain F

Piwnica, Armand H

Pessac-Bordeaux
Fontan, Francis M

GERMANY

Aachen

Messmer, Bruno J

Hamburg

Rodewald, Georg

Hannover
Borst, Hans G

Neuss

Bircks, Wolfgang H

GUATEMALA

Guatemala City

Herrera, Rodolfo

INDIA

Raiputana

Van Allen, Chester M

IRELAND

Dublin

O'Malley, Eoin

ITALY

Bergamo

Parenzan, Lucio

Padova
Gallucci, Vincenzo

Peracchia, Alberto

JAPAN

Kanazawa

Iwa, Takashi

Kitakyushu

Stark, Jaroslav F

Taylor, Kenneth M

FINLAND

Helsinki

Mattila, Severi P

FRANCE
Meudon

Cachera, Jean Paul

MONACO
Monte Carlo

Dor, Vincent

NEW ZEALAND
Auckland

Barratt-Boyes, Sir Brian

P.R. OF CHINA
Beijing

Ying-Kai, Wu

PORTUGAL

Lisbon

Macedo, Manuel E M

SAUDI ARABIA

Riyadh

Duran, Carlos Gomez

Merendino, K Alvin

SCOTLAND

Edinburgh

Logan, Andrew

Glasgow

Wheatley, David J

SPAIN
Madrid

Rivera, Ramiro

Miyamoto, Alfonso T

Osaka

Kawashima, Yasunaru

Sendai

Mohri, Hitoshi

Tokyo
Wada, Juro J

SWEDEN
Sollentuna

Bjork, Viking O

SWITZERLAND
Arzier

Hahn, Charles J

Pully
Naef, Andreas P

Zurich

Senning, Prof Ake

Turina, Marko I

UNITED ARAB EMERIT

Abu Dhabi

Brom, A Gerard

USSR

Moscow

Burakovsky, Vladimir I

VENEZUELA

Caracas

Tricerri, Fernando E

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

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

BY-LAWS OF

THE AMERICAN ASSOCIATION

FOR THORACIC SURGERY

ARTICLE I. Name

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

ARTICLE II. 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 an additional period of Associate Membership or dropped from the rolls of the Association.

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

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

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

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

Section 11. Notwithstanding Section 10, any member of the Association over 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, and shall have full power to manage and act on all affairs of the Association, except as follows:

a. It may not alter the initiation fees or annual dues, or levy any general assessments against the membership, except that it may, in individual cases, waive annual dues or assessments.

b. It may not change the Articles of Incorporation or By-Laws.

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

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

Section 3. At the conclusion of the annual meeting, the retiring President shall automatically become a Councilor for a one-year term of office. One of the other four Councilors shall be elected at each annual meeting of the Association to serve for a four-year term of office in the place of the elected Councilor whose term expires at such meeting, but no Councilor may be 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 or desirable. All such committees shall render their reports at an executive session of the Association, except that no ad hoc committee need report unless so directed by the Council.

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

Section 3. The Program Committee shall consist of at least six members: the President, the Vice President, the Secretary and the Editor of the Association, and at least two members-at-large appointed by the President. The duties of this Committee shall be to arrange, in conformity with instructions from the Council, the scientific program for the annual meeting.

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

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

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

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

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

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

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 January and end on the last day of December each year.

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

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

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

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

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

1. Appointment of necessary committees.

2. Miscellaneous business of an urgent nature

Section 7. The second executive session of the Association shall be held during the afternoon of the second day of the meeting. The business at this session shall include, but is not limited to:

1. Reading or waiver of reading of the minutes of the preceding meetings of the Association and the Council.

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

3. Audit Report.

4. Report of the Necrology Committee.

5. Report of the Program Committee.

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

7. Action on recommendations emanating from the Council.

8. Unfinished Business.

9. New Business.

10. Report of the Membership Committee.

11. Election of new members.

12. Report of the Nominating Committee.

13. Election of officers.

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

Section 9. Fifty voting members present in person shall constitute a quorum at a meeting of members.

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

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

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

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

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

ARTICLE IX. Indemnification 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, May 8, 1990

ANNUAL MEETING DATES

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

1989-Boston..... President, W. Gerald Austen

1990-Toronto..... President, F. Griffith Pearson

AWARDS

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 William A. Gay, Jr., M.D., Salt Lake City, Utah

Secretary-Treasurer William T. Maloney, Manchester, Massachusetts

Director Hartley P. Griffith, M.D., Pittsburgh, Pennsylvania

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 surgery. 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 overall training and increase his contacts with North American thoracic surgeons. Awards are made to surgeons of unique promise who have been regarded as having the potential for later international thoracic surgical leadership. Since the inception of the Graham Fellowship, 41 young surgeons from 21 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 Fritz Helmer, M.D.

Second Surgical Clinic, University of Vienna, Vienna, AUSTRIA

10th 1962-63 Theodor 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 Mattila, M.D.

Department of Thoracic Surgery, Helsinki University Central Hospital, Helsinki 29, FINLAND

23rd 1972-73 Yasuyuki Fujiwara, M.D.

Department of Cardiovascular Surgery, Tokyo Medical College Hospital, Shinjuku, Tokyo, JAPAN

24th 1973-74 Marc Roger deLeval, M.D.

8 Thornton Way, Hampstead Garden Suburb, London NW 11, ENGLAND

25th 1974-75 J. J. DeWet Lubbe, M.D.

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 Claudio A. Salles, M.D.

Av Celso Porfirio Machado, 370, Bairro Belvedere

Belo Horizonte MG, BRAZIL

32nd 1982-83 Yasuhisa Shimazaki, M.D.

First 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, 9020, 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.

Mitalitte 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.

Department of Cardiac Surgry, Beijing Heart, Lung, Blood Vessel Medical Center & Anzhen Hospital, Andingmenwai, Beijing, PEOPLE'S REPUBLIC OF CHINA

39th 1989-90 Christopher John Knott-Craig, M.D.

Univ. of Stellenbosch, P.O. Box 65, Tygerberg, South Africa 7505

40th 1990-91 Bojidar G. Bakalov, M.D.

kvartal "Tchaika", bl. 20, ent. A, fl. 12, app. 68, 9005 Varna, PEOPLE'S REPUBLIC OF BULGARIA

41st 1991-92 Ko Bando, M.D., Ph.D.

Division of Cardiac Surgery, Okayama University Medical School, 5-1 Shikatcho 2 Chome, Okayama City 700 Japan

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. CHURCHILL 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 Todcity 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

JOHN F. GIBBON, JR., RESEARCH SCHOLARSHIP

"Load-Independent Assessment of Cardiac Performance by Noninvasive Means"

1990-1992 Donald D. Glower, M.D.

Duke University Medical Center, Box 31064

Durham, North Carolina 27710