1996 ANNUAL MEETING PROGRAM

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1996 Annual Meeting
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1996
AATS
Postgraduate
Course
Congenital Heart Disease

Sunday, April 28, 1996
8:00 a.m. - 4:30 p.m.
Room 14
San Diego
Convention Center
San Diego, California

Objectives

The 1996 Postgraduate Course in Congenital Heart Disease will address the following topics: Late Problems following the Fontan Procedure including arrhythmias, protein losing enteropathy, cirrhosis, stroke and developmental delay and subaortic stenosis. Diagnosis and management options will be discussed. In addition, various topics related to brain protection in pediatric cardiac surgery will be covered. Finally, several videos demonstrating surgical techniques for managing complex congenital anomalies will be shown.

This course will provide attendees the opportunity to interact with recognized experts involved in the research and development of new techniques and procedures in congenital heart disease. The format of the course will include lectures and videos of current issues within each of the topics areas, with ample time provided during each session for discussion of specific questions from the audience.

Registration

The registration fee is $75 per person and includes the course, coffee breaks and lunch.

Accreditation

The American Association for Thoracic Surgery is accredited by the Accreditation Council for Continuing Medical Education to sponsor continuing medical education for physicians. The American Association for Thoracic Surgery designates this continuing education activity for 6 credit hours in Category 1 of the Physicians Recognition Award of the American Medical Association.

Postgraduate Course on Congenital Heart Disease
San Diego Convention Center - Room 14

7:00 a.m. **REGISTRATION AND CONTINENTAL BREAKFAST**
8:00 a.m. **INTRODUCTION**
Richard A. Jonas, M.D., Course Chairman
Session I LATE PROBLEMS FOLLOWING THEFONTAN PROCEDURE
Moderator: John E. Mayer, M.D.
8:05 a.m. **Arrhythmia**
Charles B. Huddles ton, M.D., St. Louis, Missouri
8:25 a.m. **Protein Losing Enteropathy and Cirrhosis**
Gordon K. Danielson, M.D., Rochester, Minnesota
8:45 a.m. **Stroke and Developmental Delay**
John E. Mayer, M.D., Boston, Massachusetts
9:05 a.m. **Subaortic Stenosis and Restrictive Intraventricular Communication**
Marshall Jacobs, M.D., Browns Mills, New Jersey
9:25 a.m. **Conversion of the Traditional Fontan to Lateral Tunnel**
Hillel Laks, M.D., Los Angeles, California
9:45 a.m. **Panel Discussion**
10:00 a.m. **Coffee Break**
Session II BRAIN PROTECTION AND PEDIATRIC CARDIAC SURGERY
Moderator: Richard A. Jonas, M.D.
10:45 a.m. **Latest Results of the Boston Circulatory Arrest Study**
Jane Newburger, M.D., Boston, Massachusetts
11:05 a.m. **pH Stat Versus Alpha Stat**
Richard A. Jonas, M.D., Boston, Massachusetts
11:25 a.m. **Potential Benefits of Delayed Rewarming**
Erle H. Austin, III, M.D., Louisville, Kentucky
11:45 a.m. **Retrograde Cerebral Perfusion**
Randall B. Griepp, M.D., New York, New York
12:05 a.m. **Cerebroplegia**
Julie A. Swain, M.D., Kenosha, Wisconsin
12:25 a.m. **Modified Ultrafiltration and Cerebral Protection**
Ross M. Ungerleider, M.D., Durham, North Carolina
12:45 p.m. **Luncheon**
2:00 p.m. **VIDEO PRESENTATIONS OF SURGICAL TECHNIQUES OF CONGENITAL ANOMALIES**
5:00 p.m. **RECEPTION - EXHIBIT HALL**

1996
AATS
General
Thoracic
Surgery
Symposium

Progress, Technical Pitfalls And Management Of Complications

Sunday, April 28, 1996
8:00 a.m. - 5:00 p.m.
Room 15
San Diego
Convention Center
San Diego, California

Objectives

The 1996 General Thoracic Surgery Symposium entitled "Progress, Technical Pitfalls and Management of Complications" will provide an overview of the current "state of the art" of general thoracic surgery. Discussion topics will include the surgical treatment of myobacterial disease, malignant mesothelioma, gastroeso-phageal reflux, Barrett's esophagus and emphysema; technical challenges and complications of tracheal surgery, surgical treatment of stage IIIA and IIIB lung cancer, chest wall resection and reconstruction, and transhiatal esophagectomy; new directions in video assisted thoracic surgery; and new developments in molecular biology, which have exciting implications for gene therapy for malignant mesothelioma and diagnosis of adenocarcinoma of the cardia.

This symposium will provide attendees with the opportunity to interact with recognized experts in their respective fields. The course format will include lectures with ample time provided for discussion of specific questions from the audience.

Registration

The registration fee is $75 per person and includes the symposium, coffee breaks and lunch.

Accreditation

The American Association for Thoracic Surgery is accredited by the Accreditation Council for Continuing Medical Education to sponsor continuing medical education for physicians. The American Association for Thoracic Surgery designates this continuing education activity for 6.5 credit hours in Category 1 of the Physicians Recognition Award of the American Medical Association.

General Thoracic Surgery Symposium
San Diego Convention Center - Room 15

7:00 a.m. **REGISTRATION AND CONTINENTAL BREAKFAST**
8:00 a.m. **INTRODUCTION AND WELCOME**
Mark B. Orringer, M.D., Chairman
8:00 a.m. **Surgical Treatment of Mycobacterial Disease**
Marvin Pomerantz, M.D., Denver, Colorado
8:30 a.m. **Surgical Treatment of Malignant Mesothelioma**
David J. Sugarbaker, M.D., Boston, Massachusetts
9:00 a.m. **Gene Therapy for Malignant Mesothelioma**
Larry R. Kaiser, M.D., Philadelphia, Pennsylvania
9:30 a.m. **Trachea! Surgery - Pitfalls and Avoidance of Problems**
Douglas J. Mathisen, M.D., Boston, Massachusetts
10:00 a.m. **REFRESHMENT BREAK**
10:30 a.m. **Prosthetic Replacement of SVC**
Philippe G. Dartevelle, M.D., Le Plessis Robinson, France
11:00 a.m. **Technical Problems in the Management of Stage III A and IIIB Lung Cancer**
Robert J. Ginsberg, M.D., New York, New York
11:30a.m. **LUNCHEON**
1:00 p.m. **Treatment Strategy for Barren's Esophagus**
Victor F. Trastek, M.D., Rochester, Minnesota
1:30 p.m. **Adenocarcinoma of the Cardia - The Role of Molecular Biology in Diagnosis and Early Detection**
David G. Beer, Ph.D., Ann Arbor, Michigan
2:00 p.m. **REFRESHMENT BREAK**
2:30 p.m. **Pitfalls and Unusual Challenges of Chest Wall Resection and Reconstruction**
Peter C. Pairolero, M.D., Rochester, Minnesota
3:00 p.m. **Hiatal Hernia and Gastroesophageal Reflux Disease -Does Thoracic Surgery Still have a Role?**
F. Griffith Pearson, M.D., Toronto, Ontario, Canada
3:30 p.m. **Lung Volume Reduction for Generalized Emphysema**
Joseph I. Miller, M.D., Atlanta, Georgia
4:00 p.m. **Thoracoscopy - New Directions**
Rodney J. Landreneau, M.D., Pittsburgh, Pennsylvania
4:30 p.m. **Transhiatal Esophagectomy - Avoiding and Managing Complications**
Mark B. Orringer, M.D., Ann Arbor, Michigan
5:00 p.m. **RECEPTION - EXHIBIT HALL**

1996 AATS Adult Cardiac Surgery Symposium

Sunday, April 28, 1996
8:00 a.m. - 4:30 p.m.
Room 16
San Diego
Convention Center
San Diego, California

Objectives

The 1 996 Adult Cardiac Surgery Symposium is divided into four parts. The morning session provides a focused discussion of risk and cost prediction in coronary artery surgery with experts presenting data from databases accumulated over the last several years. The second half of the morning session will relay to the paricipant experience of experts in cost management focusing upon length of stay issues and hospital physician interaction for cost control.

The first part of the afternoon session will bring together four experts on aortic valve operations, presenting techniques and results with aortic valve repair, homograft aortic valve replacement, the Ross procedure, and prosthetic valve replacement of the aortic valve. A panel discussion will follow these four presentations. The final session will present two techniques for coronary revascularization which represent the frontier in revascularization: transmural laser channels for revascularization and direct revascularization with video assisted, limited access techniques.

Registration

The registration fee is $75 per person and includes the symposium, coffee breaks and lunch.

Accreditation

The American Association for Thoracic Surgery is accredited by the Accreditation Council for Continuing Medical Education to sponsor continuing medical education for physicians. The American Association for Thoracic Surgery designates this continuing education activity for 6.5 credit hours in Category 1 of the Physicians Recognition Award of the American Medical Association.

Adult Cardiac Surgery Symposium
San Diego Convention Center - Room 16

7:00 a.m. **REGISTRATION AND CONTINENTAL BREAKFAST**
8:00 a.m. **INTRODUCTION**
Robert A. Guyton, M.D., Chairman
Session I RISK AND COST PREDICTION IN CORONARY ARTERY SURGERY
8:05 a.m. **Prediction of Mortality, Length of Stay and Complications with Data from the STS Database**
Richard E. Clark, M.D., Pittsburgh, Pennsylvania
8:35 a.m. **Prediction of Mortality, Length of Stay and Complications with Data from the VA Database**
Frederick L. Graver, M.D., Denver, Colorado
9:05 a.m. **Clinical Application of Risk Assessment Techniques**
William Nugent, M.D., Lebanon, New Hampshire
9:35 a.m. **Prediction of Costs with Preoperative Variables**
Gregory L. Kay, M.D., Los Angeles, California
10:05 a.m. **COFFEE BREAK**
Session II COST MANAGEMENT IN CORONARY ARTERY SURGERY
10:30 a.m. **Aggressive Reduction in Hospital Stay - Discharge on the Third or Fourth Postoperative Day**
Joseph M. Graver, M.D., Atlanta, Georgia
11:00 a.m. **Physician-Hospital Collaboration for Cost Management**
Steven R. Gundry, M.D., Loma Linda, California
11:30a.m. **LUNCHEON**
Session III STATE OF THE ART IN AORTIC VALVE OPERATIONS
12:45 p.m. **Aortic Valve Repair: Indications, Techniques and Results**
Delos M. Cosgrove, M.D., Cleveland, Ohio
1:15 p.m. **Homograft Replacement of the Aortic Valve: Indications, Techniques and Results**
Robert B. Karp, M.D.. Chicago, Illinois
1:45 p.m. **Autograft Replacement of the Aortic Valve: Indications, Techniques and Results**
Nicholas T. Kouchoukos, M.D., St. Louis, Missouri
2:15 p.m. **New Options for Prosthetic Valve Replacement of the Aortic Valve**
Ellis L. Jones, M.D., Atlanta, Georgia
2:45 p.m. **PANEL DISCUSSION**
3:00 p.m. **COFFEE BREAK**
Session IV THE FRONTIER IN MYOCARDIAL REVASCULARIZATION
3:30 p.m. **Transmural Laser Channels for Revascularization in Advanced Coronary Artery Disease**
Lawrence H. Cohn, M.D., Boston, Massachusetts
4:00 p.m. **Direct Coronary Revascularization with Video-Assisted, Limited Access Techniques**
Thomas A. Burdon, M.D., Stanford, California
5:00 p.m. **RECEPTION - EXHIBIT HALL**

AMERICAN ASSOCIATION FOR THORACIC SURGERY
76TH ANNUAL MEETING
SAN DIEGO CONVENTION CENTER
APRIL 29-MAY 1, 1996

MONDAY MORNING, APRIL 29, 1996

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

8:45 a.m. SCIENTIFIC SESSION

Room 6, San Diego Convention Center

Moderators: Mortimer J. Buckley, M.D.

James L. Cox, M.D.

1. PAEDIATRIC TRACHEAL HOMOGRAFT TRANSPLANTATION.

Jeffrey P. Jacobs, M.D.*, Martin J. Elliott, M.D., FRCS*and Claus Herberhold, M.D.*

London, United Kingdom and Bonn, Germany

Sponsored by: †Marc R. de Leval, M.D., FRCS,

London, United Kingdom

Discussant: Thomas L. Spray, M.D.

Purpose: Tracheal stenosis is a life-threatening problem in children. Recurrent long segment tracheal stenosis is especially problematic. Tracheal homograft transplantation (THT) represents a new treatment option for this difficult group of patients.

Methods: Cadaveric trachea is harvested, fixed in formalin, washed in methiolate, and stored in acetone. The stenosed tracheal segment is opened to widely patent segments proximally and distally. The anterior cartilage is removed and the posterior trachealis muscle or tracheal wall remains. A temporary silastic intraluminal stent is placed and absorbable sutures secure the homograft. Regular postoperative bronchoscopy clears granulation tissue for several weeks. The stent is removed endoscopically after epithelialization over the homograft.

Twenty-four children (age 5 months to 18 years, mean ± standard error of the mean [SEM] = 8.18 ± 1.21 years) underwent THT. All had severe life-threatening tracheal stenosis and had undergone previous failed reconstructive attempts. Ten lesions were congenital, nine were post-traumatic, and five were secondary to prolonged intubation. Eighteen procedures used neck incisions only. Six children required sternotomy. Cardiopulmonary bypass (CPB) allowed THT for more distal lesions down to and beyond the carina and allowed THT in the small infant. CPB was necessary in five procedures. Three patients without functional airways required stabilization with preoperative extra-corporeal membrane oxygenation.

Results: Follow-up ranged from 5 months to 10 years (mean ± SEM = 3.73 ± 0.71 years). Twenty patients survived (20/24 = 83%), seventeen without any airway problems. Three patients are still undergoing treatment. One patient requiring emergent ECMO support preoperatively expired three weeks postoperatively. Another patient with severe preoperative mediastinal sepsis expired 20

weeks postoperatively. Two patients died with functional airways: one died from unrelated gastrointestinal problems eighteen months postoperatively and one died from cardiac failure.

Conclusions: THT represents a new treatment modality with encouraging short to medium-term results for children with severe recurrent long segment tracheal stenosis. Postoperative bronchoscopic and histologic studies provide evidence of epithelialization and support the expectation of good long-term results.

†1973-74 Graham Fellow

*By invitation

2. LOOKING FOR THE ARTERY OF ADAMKIEWICZ: A CLINICOPHYSIOLOGIC QUEST.

Randall B. Griepp, M.D., M. Arisen Ergin, M.D., Ph.D., Steven L. Lansman, M.D.*,

Jan D. Galla, M.D.*, Cid S. Quintana, M.D.* and Jock N. McCullough, M.D.*

New York, New York

Discussant: Joseph S. Coselli, M.D.

In a renewed effort to lower the incidence of postoperative paraplegia, all patients undergoing thoracic or thoracoabdominal aneurysm resection since October 1993 have had spinal cord function monitored with somatosensory evoked potentials (SSEP) intraoperatively, and postoperatively until awakening. In an attempt to identify segmental vessels critical to cord blood supply, each vessel in the segment to be resected was occluded temporarily: if no change in SSEP latency or amplitude occurred within 10-15 minutes, it was ligated and divided. Adjunctive measures to protect spinal cord function included mild generalized hypothermia (31-33°C), distal perfusion (in all but three cases), corticosteroid administration, maintenance of high normal blood pressure perioperatively, avoidance of nitroprusside, and cerebrospinal fluid drainage when multiple pairs of peridiaphragmatic intercostals were sacrificed.

Neurological outcome in 73 consecutive patients so treated (Group II) was compared with a group of 138 consecutive patients operated on earlier who did not have SSEP monitoring (Group I). Preoperative clinical characteristics did not differ significantly between Group I and II patients: average age, 63 vs. 65 years; male, 64 vs. 56%; urgent or emergent operation 41 vs. 51%; dissection, 28 vs. 21%.
The incidence of permanent spinal cord injury was significantly lower in some categories of Group II patients:

Aneurysm Extent	**Group I** ***1/86-9/93***		**Group II** ***10/93-10/95***		**p value**
Thoracic	2/94	2%	0/42	0%	0.34
Thoracoabdominal (Crawford I & II)	8/24	33%	2/21	10%	0.05
All Thoracoabdominal	9/44	20%	2/31	6%	0.09
TOTAL	11/138	8%	2/73	3%	0.13

Transient evidence of spinal cord ischemia was observed in three patients postoperatively: all had return of function in response to supportive measures to increase spinal cord perfusion, including elevation of blood pressure and spinal fluid drainage.

The reversal of transient spinal cord ischemia as well as the lower incidence of permanent spinal cord injury in Group II patients suggest that careful monitoring of spinal cord function and a multimodal approach to maximizing spinal cord blood flow are effective in preventing paraplegia following thoracoabdominal aneurysm resection. Since intraoperative SSEP was not affected by gradual serial intercostal sacrifice in any patient, the low incidence of spinal cord injury in Group II patients was achieved without reimplantation of a single intercostal or lumbar artery. We conclude that spinal cord blood supply is unlikely to depend upon a single artery, or even a small number of critical segmental vessels, and that spinal cord perfusion can be effectively manipulated using generalized adjunctive measures. We question whether there is indeed an "Artery of Adamkiewicz" of physiologic significance.
*By invitation

3. RESULTS OF LUNG VOLUME REDUCTION SURGERY IN 120 CONSECUTIVE PATIENTS.

Joel D. Cooper, M.D., G. Alexander Patterson, M.D., R. Sudhir Sundaresan, M.D.*,

Elbert P. Trulock, M.D.*, Roger D. Yusen, M.D.* and Stephen S. Lefrak, M.D.*

St. Louis, Missouri
Discussant: John R. Benfield, M.D.

Between January 1993 and August 1995, 120 patients have undergone median sternotomy and bilateral lung volume reduction surgery for severe emphysema. Selection criteria include severe dyspnea, thoracic distention, and "target" areas in the lung which can be excised to reduce lung volume without significant sacrifice of functioning lung tissue. All candidates are enrolled in a structured exercise rehabilitation program for a minimum of six to eight weeks before final acceptance.
Early mortality (<90 days) has been 2.5%, all from respiratory complications. Late mortality (>90 days) has been an additional 2.5% (post-coronary bypass at 3 months; unknown causes at 5 months; stroke at 9 months). Mean hospital stay has been 14.5 days and median stay 11 days. Due to modifications in technique and post-operative management, the mean stay has been reduced to 10.5 days and the median stay to 8 days for the last 40 patients.

Follow-up is complete on 119 patients with a mean follow-up time of 358 days and median of 327 days. Results of on-site follow-up studies are as follows:

	Pre-op		Six Months		One Year	
	N=120		N=65		N=35	
FEV_1* „ “ (% pred)	.69	(24%)	1.08	(38%)	1.08	(37%)
FVC* „ “ (% pred)	2.5	(70%)	3.0	(88%)	2.9	(83%)
TLC^+ „ “ (%pred)	8.5	(148%)	7.2	(124%)	7.1	(124%)
RV^+ „ “ (%pred)	6.0	(291%)	4.1	(202%)	4.1	(205%)
FEV_1/FVC ratio*	28%		35%		37%	
% pts on oxygen (continuous)	55%		8%		9%	
% pts on oxygen (w/exercise)	91%		34%		29%	
MRC dyspnea scale	3.0		1.0		1.3	

* post-bronchodilator

[+] plethysmography

For selected patients with severe chronic obstructive pulmonary disease, lung volume reduction surgery improves respiratory mechanics, diminishes oxygen requirement, reduces dyspnea and improves the quality of life.

9:45 a.m. INTERMISSION - VISIT EXHIBITS

*By invitation

10:30 a.m. SCIENTIFIC SESSION

Room 6, San Diego Convention Center

Moderators: David B. Skinner, M.D.

James L. Cox, M.D.

4. REOPERATION FOR FAILED MITRAL VALVE REPAIR.

Marc Gillinov, M.D.*, Delos M. Cosgrove, M.D., Bruce W. Lytle, M.D., Paul C. Taylor, M.D.*, Robert W. Stewart, M.D.*, Patrick M. McCarthy, M.D., Nicholas G. Smedira, M.D.*, Derek Muehrcke, M.D* and Floyd D. Loop, M.D.

Cleveland, Ohio

Discussant: Tirone E. David, M.D.

Recurrent mitral regurgitation (MR) is a vexing complication of mitral valve (MV) repair. To determine the causes of failed MV repair, the surgical pathology of patients who underwent reoperation for a failed MV repair was examined. From 1986-1994, 2,548 patients underwent surgery for MR. Of these, 81 patients (3.1%) had 86 reoperations for recurrent MR after MV repair. Mean age was 59.2 ± 1.4 years (18-79 years); 55 were men. Primary valve pathology was degenerative in 47 patients (58%), rheumatic in 16 (20%), ischemic in 14 (17%), endocarditis in 3 (4%) and congenital in 1 (1%).

Time interval between initial MV repair and reoperation was 15.6 ±2.5 months. Findings at reoperation were:

	Degenerative N=48	**Rheumatic N=17**	**Ischemic N=16**	**Other N=5**
Progressive primary valve disease	58%	70%	38%	0%
Suture dehiscence	23%	12%	25%	80%
Inadequate initial repair	12%	6%	19%	0%
Other	6%	12%	19%	20%

Progression of primary valve disease was the most common cause of recurrent MR (46 patients [57%]). Rupture of previously shortened chordae was the mechanism of recurrent MR in 17 of 28 patients (61%) with progression of degenerative MV disease.

Operations included mitral valve replacement in 64 patients (79%) and repeat MV repair in 17 (21%). Five patients who had repeat MV repair required subsequent valve replacement. There were 6 hospital deaths (7.4%).

We conclude that: 1) progression of primary valve disease is the most common cause of late failure after MV repair; 2) chordal shortening is associated with late failure in patients with degenerative disease; and 3) repeat mitral valve repair results in successful treatment for a small minority of patients.

*By invitation

5. TRANSMYOCARDIAL LASER REVASCULARIZATION: RESULTS OF A MULTI-CENTER TRIAL USING TMLR AS SOLE THERAPY FOR END-STAGE CORONARY ARTERY DISEASE.

Keith A. Horvath, M.D.*, Lawrence H. Cohn, M.D., Denton A. Cooley, M.D., John R. Crew, M.D.*, O. Howard Frazier, M.D., Hartley P. Griffith, M.D., Kamuran Kadipasaoglu, Ph.D.*, Allan Lansing, M.D.*, Robert March, M.D.*, Mahmood R. Mirhoseini, M.D.* and Craig Smith, M.D.

Boston, Massachusetts; Houston, Texas; San Francisco, California; Pittsburgh, Pennsylvania, Louisville, Tennessee; Chicago, Illinois; Milwaukee, Wisconsin and New York, New York

Discussant: John L. Ochsner, M.D.

Transmyocardial laser revascularization was utilized as sole therapy for patients with ischemic heart disease not amenable to PTCA or CABG. This technique employs an 800W CO_2 laser to create transmyocardial channels for direct perfusion of the ischemic heart. Since 1992, 200 patients at eight U.S. hospitals, have undergone TMLR. One hundred seventy-five patients have 3-12 months follow-up for an accumulated 1190 patient/months of follow-up. Their age was 62 ± 11 years (mean ± SD) and their ejection fraction was 47 ± 12%. Eighty percent (140/175) had at least one previous CABG and 36% (63/175) had a prior PTCA. Preoperatively, the patients underwent nuclear SPECT perfusion scans to identify the extent and reversibility of their ischemia. These scans were repeated at 3, 6 and 12 months. Angina class (CCS) and admissions for angina were recorded. The peri-operative mortality was 8% (16/200). The data are expressed as mean ± SD with t-test for significance vs. preoperative values.

	Preop	3 months	6 months	12 months
Angina Class	3.8 ± 0.4	1.5 ± 1.2*	1.3 ± 1.2*	1.3 ± 1.2*
Perfusion Defects	5.1 ± 3.4	4.4 ± 3.5	2.9 ± 3.0#	2.8 ± 3.4#

*p = 0.000l, #p = 0.005

Improved perfusion was confirmed by PET scans at one center which demonstrated increased subendocardial vs. subepicardial resting perfusion at 12 months (0.96 ± 0.7 vs. 1.10 ± 0.02, $p < 0.001$, N = 11). In the year prior to their TMLR, the patients averaged 2.5 ± 1.7 admissions for angina; this decreased to an average of 0.3 ± 7 admissions in the year after the procedure ($p = 0.00002$).

These combined results indicate that TMLR may provide angina relief, decrease hospital admissions and improve perfusion in patients with severe coronary artery disease.

11:15 a.m. PRESIDENTIAL ADDRESS

I'd Like to be a Thoracic Surgeon

Mortimer J. Buckley, M.D., Boston Massachusetts

12:00 p.m. ADJOURN FOR LUNCH

*By invitation

MONDAY AFTERNOON, APRIL 29, 1996

1:30 p.m. SCIENTIFIC SESSION

Room 6, San Diego Convention Center

Moderators: D. Glenn Pennington, M.D.

Douglas J. Mathisen, M.D.

6. HETEROTOPIC CARDIAC TRANSPLANTATION IN INFANTS AND CHILDREN.

Francesco Santini, M.D.*, †Cornelius Dyke, M.D.*, Rosemary Radley-Smith, FRCP*, Asghar Khaghani, FRCS* and Magdi H. Yacoub, FRCS
Harefield, Middlesex, United Kingdom

Discussant: Charles B. Huddleston, M.D.

Children with advanced heart failure, particularly those with elevated pulmonary vascular resistance, pose a difficult management problem because of the fact that normal donor right ventricle cannot cope with the high pulmonary resistance, and the relative shortage of donor organs of an appropriate size for this age group. To address these issues, in the period between January 1, 1991 and October 31, 1995, 11 children, 6 boys and 5 girls, ranging in age between 11 months to 15 years (mean 7.2 years), and having a mean weight of 24.4 kg (range, 7.6 to 56.8 kg) underwent heterotopic heart transplantation (HHTx). Seven patients had dilated and 4 restrictive cardiomyopathy. Seven patients (63.6%) had significant elevation of pulmonary artery pressure (PAsys 68.7 ± 7.4, mean transpulmonary gradient = 20.3 ± 2.4). In all patients the donor pulmonary artery was anastomosed to the right atrium, avoiding the use of any prosthetic material. Ischemic time varied between 135 and 255 min (mean, 181.3 min). Immunosuppression regiment included cyclosporin and azathioprine. Steroids were not routinely used. One patient died in the hospital of acute rejection (p.o.d.=16). One patient developed right middle lobe collapse, treated successfully. Ten survivors (90.9%) are alive and active with a normal pattern of growth at a mean follow-up time of 20.8 months (range 2 to 52 months). Repeated cardiac catheterization performed in 4 patients showed slow progressive drop in pulmonary vascular resistance. Echocardiogram at 6 months postoperatively showed a mean donor heart shortening fraction of 38.7 ± 4.3%. There was no significant change in the function of the recipient hearts.

It is concluded that HHTx in children is feasible for a selected group of patients with good median term results, notably regression of pulmonary vascular disease, normal growth, and lack of chest complications.

†1995-96 Graham Fellow

*By invitation

7. NEUROPHYSIOLOGICAL MONITORING FOR OPTIMAL BRAIN PROTECTION DURING RETROGRADE CEREBRAL PERFUSION.

Brian L. Ganzel, M.D.*, Harvey L. Edmonds, Jr., Ph.D.* and John R. Pank, M.D.*

Louisville, Kentucky

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

Discussant: Randall B. Griepp, M.D.

Purpose: Selective RCP has been recently advocated as a means of protecting the brain during aortic surgery requiring hypothermic circulatory arrest. To aid in the establishment of optimal conditions for producing RCP, we here report on the electrical, hemodynamic and metabolic changes occurring before, during and after effective retroperfusion of the middle cerebral arteries. The results are compared with arrest using no RCP.

Methods: Monitoring was performed on 25 patients requiring aortic surgery (7 without RCP). Cerebral electrical activity was monitored by EEG and brainstem auditory evoked potentials, while transcranial Doppler ultrasound (TCD) measured the direction and velocity of cerebral blood flow bilaterally in the middle cerebral arteries. Metabolic activity was assessed continuously by regional cerebrovenous oxygen saturation (CVOS) using transcranial near-infrared spectroscopy. In all cases, patients were cooled to electrocerebral silence (ECS) prior to circulatory arrest.

Results: There was a wide variation in the nasopharyngeal temperature required to maintain complete ECS (21-8°C). Arrest times were similar in groups with (35 ± 17 min) and without (28 ± 16) RCP. Retrograde flow was verified by TCD in 13/18 patients during RCP. In the other 5, no middle cerebral arterial flow was evident at the onset of retrograde perfusion. TCD signals indicating reversed flow subsequently appeared in 2 of these patients after adjustment of the superior vena caval snare or increasing perfusion pressure. Corrective measures were ineffective in producing a TCD signal in the other 3 patients despite the effux of dark blood from the carotid arteries. In the RCP group with TCD-documented flow reversal, the CVOS decreased by 3 ±4% during retroperfusion. In contrast, CVOS decreased by 19 ± 14% in the arrest group (P = 0.03). The magnitude of cerebral oxygen desaturation in the 3 RCP patients without TCD-documented retrograde flow likewise decreased 19 ± 14%. Cerebral electrical activity returned in 17 ± 8 min in the RCP group with adequate flow compared with 60 ± 43 min in the arrest group (P = 0.007). The EEG in RCP patients without evidence of cortical flow returned in 36 ± 21 min (P - 0.03).

Conclusions: 1) The EEG defined the degree of hypothermia required for effective cerebral cooling prior to arrest. 2) TCD documented the establishment and maintenance of retrograde cerebral, as opposed to cranial, flow. 3) Optimally-adjusted RCP prevented further cerebral oxygen desaturation and facilitated the return of cerebral electrical activity with antegrade perfusion.

*By invitation

8. HEMODYNAMIC BENEFITS OF THE TORONTO STENTLESS PORCINE AORTIC VALVE PROSTHESIS.

Dario Del Rizzo, M.D.*, Bernard S. Goldman, M.D., George T. Christakis, M.D. and Tirone E. David, M.D.
Toronto, Ontario, Canada

Discussant: Delos M. Cosgrove, M.D.

The Toronto SPV™ is a stentless porcine heterograft used for replacement of the human aortic valve. Our institutions are participating in an ongoing multicentre international Phase II clinical trial to assess the efficacy of this valve. We report herein the hemodynamic benefits of this bioprosthesis. To assess valve function, echo Doppler studies were performed after operation, at 3-6 months, at 12 months, and annually thereafter. We previously demonstrated in 118 patients operated upon between March 1992 and December 1993, that effective orifice area (EOA) increased on average by 40% over a 1-year follow-up period while mean transvalvular gradient decreased by a similar 40%. With 3 year data now available, we report that these trends are persistent. Mean transvalvular gradient has continued to decrease such that at three years the average gradient is 2 mmHg for all valve sizes (23 mm to 29 mm) implanted. By repeated measures analysis with Wilks' Lambda (MANOVA), we showed that the regression of transvalvular gradient was significant in all valve sizes at all follow-up intervals, and that the rate of regression was independent of valve size.

Valve size (mm)	N	Time post operation (months)	Mean gradient (mmHg)	EOA (cm^2)
23	9	post operative	7.7 ± 2.9	1.4 ± 0.4
		36 months	2.2 ± 0.9	2.2 ± 0.8
25	26	post operative	7.0 ± 3.0	1.4 ± 0.6
		36 months	1.4 ± 1.2	3.5 ± 1.6
27	43	post operative	5.9 ± 2.4	1.6 ± 0.4
		36 months	2.5 ± 1.5	2.8 ± 1.3
29	37	post operative	4.4 ± 2.1	2.0 ± 0.5
		36 months	2.1 ± 0.4	3.1 ± 1.8

In the next phase of this study we investigated the relationship between changes in gradient and changes in left ventricular (LV) mass. We looked at 71 patients (50 males and 21 females; age 59.8 ± 11.9 years [range: 33-80]) between March 4, 1992 and June 30, 1995 (all operated upon at one centre). Valve pathology was stenosis in 44, insufficiency in 11, and mixed disease in 16 patients. In 90% of patients there was 0 or trace insufficiency, while there was minimal insufficiency (1+) in the remaining 10% of cases. No patient had moderate insufficiency (2+) and there was no deterioration in valve performance during the follow-up period. Again, we demonstrated an average 36% reduction in mean transvalvular gradient (10.8 ± 4.6 mmHg to 6.9 ± 3.6 mmHg) in the initial follow-up. Regression analysis revealed that Ag (change in gradient) was dependent on changes in transvalvular velocity (V_2) ($R = 0.93$, $p < 0.0001$). Mean LV mass was 363.8 ± 96.8 gm postoperative and decreased to 220.2 ± 76.8 gm at 3-6 months. Scatter plot analysis showed correlation between AV_2 and LV mass regression; we found congruency in 70% of patients ($p < 0.0001$) and LV mass regression in 80% of patients ($p < 0.0001$). The highest congruency was in aortic stenosis where LV mass regression occurred in 90% of cases. LV mass index (LV mass/body surface area) was found to decrease over time in all valve sizes. These results are consistent with the hypothesis that LV remodeling occurs following AVR with the SPV valve.

In conclusion, these observations suggest the Toronto SPV™ valve is an excellent substitute in patients deemed appropriate candidates for a tissue bioprosthesis, especially in those patients with marked LVH and in those in whom a mismatch between valve size and patient size might be anticipated. The reduction in LV mass and transvalvular gradient and the potential anticoagulation free durability may have beneficial prognostic implications.
*By invitation

9. ADENOID CYSTIC CARCINOMA OF THE AIRWAY: A THIRTY-TWO YEAR EXPERIENCE.

Donna E. Maziak, MDCM, FRCSC*, Thomas R.J. Todd, M.D., FRCSC, Shafique H. Keshavjee, M.D., FRCSC*, Timothy L. Winton, M.D., FRCSC*, Peter Van Nostrand, M.D.* and F. Griffith Pearson, M.D., FRCSC
Toronto, Ontario, Canada

Discussant: Douglas Wood, M.D.

We have reviewed our experience in 36 patients with adenoid cystic carcinoma of the upper airway seen during a 32 year period (1963-1995). The average age was 45 years (range: 15 - 80) with a male:female ratio of 1.1:1. Of these 36 patients, 30 were managed by resection and reconstruction (primary anastomosis 25; marlex prosthesis 5) of whom 25 received adjuvant radiotherapy. Six were managed with radiotherapy only.

Pathology revealed local invasion in all cases beyond the outer wall of the trachea. In most cases, microscopic extension was found in submucosal and perineural lymphatics, well beyond grossly viable or palpable tumour. Lymphatic metastases were rare: 4 of 30 (13.3%) resected cases. Subsequent haematogenous metastases occurred in 15 of 36 patients (41.6%). Twelve of 36 cases (35%) had pulmonary metastases.

Thirteen of 30 resections were complete and potentially curative. There were 2 operative deaths, and the mean survival in 11 surviving patients was 7.8 years (range: 6 months-18 years). Seventeen of 30 resections were incomplete (residual tumour at final pathology), with one operative death and a mean survival of 7.7 years (range: 9 months - 29 years). The 6 patients treated with radiation only had a mean survival of 3.3 years (range: 2 months -10 years). In the 12 patients with pulmonary metastases, mean survival was 3 years (range: 6 months - 6 years) following its identification.

Adenoid cystic carcinoma is a rare tumour, which is always locally invasive but frequently amenable to resection. Although late local recurrence following resection is common (up to 29 years) excellent long-term palliation is commonly achieved following both complete and incomplete resection. There was little difference in survival between complete and incompletely resected cases. Pulmonary metastases are common, usually slow growing, and are not necessarily a contraindication to palliative resection of the primary tumour. Long periods of control can be obtained with radiotherapy alone. The best results, however, are obtained by resection. Adjuvant radiotherapy is assumed to favourably influence survival.
*By invitation

10. CRICOPHARYNGEAL MYOTOMY FOR NEUROGENIC OROPHARYNGEAL DYSPHAGIA.

Nancy Claire Poirier, M.D.*, Luigi Bonavina, M.D.*, Raymond Taillefer, M.D.*, Attilio Nosadini, M.D.*, Alberto Peracchia, M.D. and Andre C.H. Duranceau, M.D

Montreal, Quebec, Canada and Milan, Italy

Discussant: Mark B. Orringer, M.D.

Between 1976 and 1994, 40 patients with neurological disorders associated with incapacitating upper esophageal sphincter (UES) dysfunction underwent a cricopharyngeal myotomy. Etiology for the dysphagia was a cerebrovascular accident (65%), amyotrophic lateral sclerosis (5%), trauma (5%), pseudobulbar palsy (7.5%), iatrogenic (2.5%), peripheral nerve disorders (IX-X-XII) (7.5%), Parkinson's disease (2.5%), Arnold Chiari (2.5%), and multiple sclerosis (2.5%). Patients were assessed pre- and post-operatively with a mean follow-up period of 48 months (1-255 months).

		Preop	Postop	p value
SYMPTOMS	Dysphagia	40/40	33/39	0.03
	Regurgitation			
	Pharyngooral	15	8	0.16
	Pharyngonasal	12	7	0.32
	Aspiration	33/40	13/39	<0.01
RADIOLOGY	Pharyngeal stasis	21/40	17/39	0.57
	UES incoordination	27/40	5/39	<0.01
	Aspiration	17/40	14/39	0.71
UES MANOMETRY	Resting pressure (mmHg)	64.9 ± 33.0	18.1 ± 14.5	<0.01
	Contraction pressure (mmHg)	69.1 ± 337.4	22.3 ± 26.8	<0.01
	Coordination	0.35 ± 0.40	0.48 ± 0.45	0.33
SCINTISCAN	Retention at 120 sec	8/8	8/8	

The mortality was 2.5% (1 patient) and resulted from aspiration pneumonia. Seven patients are asymptomatic, 23 patients are improved and 10 patients report no change in symptoms. Radiological evidence of functional obstruction at the pharyngoesophageal junction is reduced. UES resting and contraction pressures are significantly decreased in the UES but the coordination abnormalities are unchanged. Single liquid bolus radionuclide emptying studies of the pharynx show persistant stasis in all patients where this study was obtained.

3:15 p.m. INTERMISSION - VISIT EXHIBITS

*By invitation

4:00 p.m. SCIENTIFIC SESSION

Room 6, San Diego Convention Center

Moderators: Andrew S. Wechsler, M.D.

Bruce A. Reitz, M.D.

11. WHAT IS THE APPROPRIATE SIZE CRITERION FOR RESEARCH OF THORACIC AORTIC ANEURYSMS?

John A. Elefteriades, M.D., Michael A. Coady, M.D.*, John A. Rizzo, Ph.D.*, Kevin Johnson, M.D.*, Umer Darr, M.D.*, Ellen Frank, M.D., M.P.H.* and Gary S. Kopf, M.D.

New Haven, Connecticut

Discussant: O. Wayne Isom, M.D.

Although many papers have described techniques for resection of thoracic aortic aneurysms (TAA), limited information on the natural history is available to aid in defining criteria for surgical intervention.

Data on 225 patients with TAA at one center from 1985 to 1995 were analyzed. This computerized database included 638 imaging studies (MRI, CT, ECHO). Overall survival at 1 and 5 years was 88% and 65%, respectively. Patients having aortic dissection had markedly lower survival (83%, 1 year; 31%, 5 year) than the non-dissected cohort (90%, 1 year; 70%, 5 year).
Mean size of the thoracic aorta in these patients was 5.1 cm. The mean growth rate was 0.15 cm/year. Multivariate regression analysis to isolate risk factors for aortic expansion revealed a size of > 6.0 cm to be a significant predictor ($p<0.0001$).

Median size at time of rupture or dissection was 6.1 cm (5.9 cm for ascending; 7.0 cm for descending). The incidence of dissection or rupture increased with aneurysm size, as illustrated below.

Seventy-four patients underwent surgery for their TAA. For elective operations, the mortality was 9.7%; emergent operations had a mortality of 30.2%.

If the median value at the time of dissection or rupture were used as the intervention criterion, half of the patients would have suffered a devastating complication prior to surgery. Accordingly, a criterion lower than the median is appropriate. We recommend 5.5 cm as an acceptable size for elective resection of ascending aortic aneurysms, as these can be performed with relatively low mortality. For aneurysms of the descending aorta, where peri-operative complications are greater and the median size at time of complication is larger, we recommend intervention at 6.5 cm.

*By invitation

12. ONE STAGE COMPLETE UNIFOCALIZATION IN INFANTS: WHEN NOT TO CLOSE THE VENTRICULAR SEPTAL DEFECT.

Frank L. Hanley, M.D., V. Mohan Reddy, M.D.*, Edwin Petrossian, M.D.*, Phillip Moore, M.D.* and David F. Teitel, M.D.*
San Francisco, California

Discussant: Hillel Laks, M.D.

Background: The decision to close the ventricular septal defect at the time of unifocalization in patients with pulmonary atresia with ventricular septal defect and major aortopulmonary collaterals (PA.VSD.MAPCAS) may be difficult. Standard indices of true pulmonary arteries cannot be used since reconstruction using the collaterals makes the preoperative values meaningless. We have developed an index of total pulmonary vascular cross-sectional area which systematically evaluates true pulmonary arteries and all collateral vessels to be unifocalized. The purpose of this study is to determine if this new index is predictive of peak right ventricular systolic pressure following complete repair.

Patients and methods: Since July 1992, 17 infants (age: mean ± SD = 4.1 ± 3.3 months) with PA.VSD.MAPCAS have been managed at our institution. Complete unifocalization (n=16) from a midline approach and VSD closure (n=12) was the procedure of choice. In 4 patients the VSD was left open and only one patient with severe distal collateral artery stenosis was staged. The preoperative angiograms were evaluated to estimate the size of the true pulmonary arteries and size of the collaterals. The indexed cross-sectional areas of the true pulmonary arteries (PAI), of the MAPCAS (CAI), and the total bed (TI = PAI + CAI) were calculated. True pulmonary arteries were measured just proximal to the first branching point and the collaterals were measured at the narrowest point beyond the site of surgical unifocalization and proximal to any branches. The ratio of the peak systolic pressure in the right ventricle (pRV) to that in the left ventricle (pLV) was estimated from the absolute pressures obtained in the operating room.

Results: The median PAI was 40.5 mm^2/M^2 (range = 0 to 294.4 mm^2/M^2), the median CAI was 72.9 mm^2/M^2 (range = 22.5 to 422.1 mm^2/M^2), and the median TI was 174.7 mm^2/M^2(range = 50.5 to 422.1 mm^2/M^2). The median pRVipLV was 0.63 (range = 0.29 to 0.85). By unpaired /-Test there was a significant difference ($p = 0.011$) in the TI between the two groups. By linear regression there was a significant correlation ($p = 0.023$) between the TI and VSD closure. In addition, the pRV:pLV ratio appeared to be lower in patients with higher TI and approached significance ($p = 0.07$).

Conclusion: The total pulmonary vascular index appears to correlate with postrepair pRV:pLV. This index is useful in deciding when not to close the VSD.

*By invitation

13. DOES SUCCESSFUL BRIDGING WITH THE IMPLANTABLE LEFT VENTRICULAR ASSIST DEVICE AFFECT CARDIAC TRANSPLANTATION OUTCOME?

Malek G. Massad, M.D.*, Patrick M. McCarthy, M.D., Nicholas G. Smedira, M.D.*, Daniel J. Cook, Ph.D.*, Norman B. Ratliff, M.D.*, Marlene Goormastic, M.P.H.*, Jose Navia, M.D.*, James B. Young, M.D.* and Robert W. Stewart, M.D.*

Cleveland, Ohio

Discussant: Eric A. Rose, M.D.

Purpose: We sought to determine if cardiac transplant recipients who required a bridge-to-transplant with an implantable LVAD had a different outcome than patients (pts) who had primary cardiac transplants.

Methods: A retrospective study of 238 primary cardiac transplants at our institution between January 1992 and October 1995 included 44 pts who received the *HeartMate*® LVAD and 194 pts who had no LVAD support. LVAD transplant rate was 77%. Comparison of demographic and clinical factors was performed and associations were analyzed using Chi square test. Distribution of continuous factors was compared using Wilcoxon Rank Sum test. Kaplan-Meier survival estimates were compared using log-rank test.

Results: LVAD transplants constituted 7.5% of all primary transplants in 1992 (n=67), 17% in 1993 (n=58) and 1994 (n=63), and 36% in 1995 (n=50, YTD). LVAD and non-LVAD pts had similar age distribution (median 52 yrs) and female gender (16% vs 26%, NS). LVAD recipients had greater weight (81 kg vs 70 kg, $p = 0.003$) and body surface area (1.94 m^2 vs 1.81 m^2, $p = 0.05$). They were more likely to have type O blood group (52% vs 34%, $p = 0.07$), and ischemic cardiomyopathy (70% vs 42%, $p = 0.001$). All LVAD pts and 41% of non-LVAD pts had previous

cardiac operations (mean number per pt: 1.3 vs 0.3, $p < 0.001$). LVAD related sepsis occurred in 20/44 pts (45%) during support. More pts in the LVAD group had anti-HLA antibodies prior to transplant (T-cell PRA level >10% in 66% of LVAD pts vs 15% of non-LVAD pts, $p < 0.0001$). Blood utilization during LVAD support of more than 96 units of blood products (median) results in significantly higher anti-HLA antibody levels. Sixty-one percent of non-LVAD pts were UNOS status I. Time on the waiting list was longer for LVAD pts compared to non-LVAD status I pts (median 88 days vs 33 days, $p = 0.002$). There was no difference in the length of post-transplant hospital stay (median 15 days for each) and operative mortality (4.6% LVAD vs 5.2% non-LVAD). No significant difference was found in the Kaplan-Meier survival curves. One year survival was 93% in the LVAD group and 88% in the non-LVAD group (NS). Two-year survival was 82% vs 84% (NS). Comparison of post-transplant events showed no significant difference in CMV infection rates (23% vs 16%), or the incidence of vascular rejection (20% vs 15%) or moderate/severe cellular rejection (79% vs 71%) at one-year follow-up.

Conclusions: LVAD support intensified donor shortage by including recipients who otherwise would not have survived to transplant. Bridging affected transplant demographics favoring pts who are larger, have ischemic cardiomyopathy, have had previous cardiac surgery, are HLA sensitized, and have blood group O. Successfully bridged pts wait longer for transplant but have similar post-transplant hospital stay and operative mortality and achieve survival similar to that of pts not requiring LVAD support.

*By invitation

14. THE DISCHARGE OF PATIENTS WITH NEW-ONSET ATRIAL FIBRILLATION AFTER HEART SURGERY IN ATRIAL FIBRILLATION IS SAFE AND COST-EFFECTIVE.

Robert L. Hannan, M.D.*, Allen J. Solomon, M.D.*, Peter C. Kouretas, M.D.*, Richard A. Hopkins, M.D., Nevin M. Katz, M.D. and Robert B. Wallace, M.D.
Washington, DC

Discussant: Renee S. Hartz, M.D.

Atrial fibrillation is a frequent complication of open heart surgery and leads to prolonged hospitalization and increased cost. We retrospectively reviewed our experience over a one year period to determine if patients in new-onset atrial fibrillation (AF) could safely be discharged in atrial fibrillation after ventricular rate had been controlled and anticoagulation initiated. In the year ending in August 1995, 505 adult patients underwent heart surgery utilizing cardiopulmonary bypass. Of the 488 survivors (96.6%), 93 (19%) developed AF postoperatively. Of these 93 patients, 82 (88%) were discharged in sinus rhythm (NSR) and 11 (12%) were discharged in AF. Results are summarized in the table below: age is mean age in years, risk is by the modified Parsonnett score, postop LOS is length of stay postoperatively, and mean hospital cost and charge are normalized to 1.00 in patients without AF.

	N	AGE	RISK	LOS	COST	CHARGE
No AF	385	59	12.5	6.2	1.00	1.00
AF, discharged NSR	82	65	15.7	11.6	1.75	1.71
AF, discharged AF	11	72	19.4	8.9	1.00	1.11

At mean follow-up period of 222 days, all 11 patients discharged in AF were alive and well: 9 patients were in sinus rhythm. Outpatient cardioversions were performed on 3 patients, one of whom remained in sinus rhythm. No systemic emboli or other complications were noted in any of

the patients discharged in AF. Patients discharged in AF had similar cost and charges to patients without AF: patients with AF discharged in NSR had increased cost and charges. We suggest that the discharge of patients in persistent AF after heart surgery is safe and cost-effective, and hypothesize that earlier discharge of patients in AF may be appropriate.
*By invitation

TUESDAY MORNING, APRIL 30, 1996

7:00 a.m. FORUM SESSION I - CARDIAC SURGERY

Room 6A/B, San Diego Convention Center

Moderators: Edward D. Verrier, M.D.

James K. Kirklin, M.D.

F1. EFFICIENT TRANSFER OF OLIGONUCLEOTIDE AND PLASMID DNA INTO WHOLE HEART THROUGH CORONARY ARTERY.

Yoshiki Sawa, M.D.*, Keishi Kadoba, M.D.*, Kazuhiro Taniguchi, M.D.*, Hong-zhi Bai, M.D.*, Ken Suzuki, M.D.*, Yasufumi Kaneda, M.D.* and Hikaru Matsuda, M.D.*

Osaka, Japan

Sponsored by: Yasunaru Kawashima, Osaka, Japan

At the time of harvesting donor heart, it may be possible to perform gene transfection to provide the myocardium protection from the ischemic injury and/or change the alloreactivity after transplantation. However, several of the current techniques for transfer of both oligonucleotide and plasmid DNA into the myocardium are impaired by low efficiency and toxicity. To improve gene transfer techniques especially into whole heart, a gene transfer method involving liposome with viral envelope (HVJ-liposome) was assayed as an alternative. In this study, in vivo gene transfection of FITC labeled oligonucleotide (F-ODN) and cDNA of p-galactosidase (P-gal) and Mn-SOD was examined using HVJ-liposome (H group) or cationic liposome (L group). In H group, F-ODN or cDNA of P-gal or MnSOD were complexed with liposomes, DNA binding nuclear protein (HMG-1) and the viral protein coat of HVJ. After the harvest of donor rat hearts arrested by cardioplegia, coronary artery was infused with liposome-gene complex through aortic cannula during cardioplegia arrest. Then the hearts were transplanted (anoxic time: 30 min.) into the abdomen of the recipient rat of the same strain and all hearts were sacrificed after 3 days of transplantation. All hearts showed no rejection at the time of sacrifice. FITC was detected in the nuclei of more than 80% of the myocytes (82 ± 17%) in H group than in L group (7 ± 5%). Intensity of FITC was significantly higher in H group (939 ± 112 F.I.) than L group (166 ± 78 F.I.). P-gal was expressed in the cytosol of more than 70% of the myocytes (71 ± 14%). After 3 days of gene transfection, the hearts transfected with Mn-SOD (S) showed significantly higher percentage of recoveries of LVDP (S vs C, 86 ± 3 vs 54 ± 12%) and coronary flow (98 ± 2 vs 66 ± 12%) than did the control hearts (C) when exposed to ischemia (30 min., 37°C) and reperfusion (30 min., 37°C) with Langendorff apparatus. These results clearly demonstrated that donor hearts were transfected with FITC-oligonucleotide and p-galactosidase gene in the whole layer of myocardium by coronary infusion of HVJ-liposome during cardioplegia arrest at the time of harvest. And the heart transfected with Mn-SOD showed significant improvement of tolerance against ischemia-reperfusion injury. Our method appears to be a novel in vivo gene transfer technique for the heart to be subjected to ischemia, and may provide a new tool for research and therapy of heart transplantation.

*By invitation

F2. TIROFIBAN PROVIDES "PLATELET ANESTHESIA" DURING CARDIOPULMONARY BYPASS IN BABOONS.

Yugi Hiramatsu, M.D.*, Nicolas Gikakis, B.S.E.*, Harry L. Anderson, M.D.*, Joseph H. Gorman, M.D.*, Robert J. Gould, M.D., Ph.D.*, Stefan Niewiarowski, M.D.* and L. Henry Edmunds, Jr., M.D.

Philadelphia, Pennsylvania

Tirofiban is a reversible, non-protein inhibitor of platelet GPIIb/IIIa receptors that is in Phase III clinical trials for some applications. We tested the efficacy of tirofiban to preserve platelet numbers and function and to shorten postoperative bleeding times in a baboon model of cardiopulmonary bypass (CPB).

Three groups of baboons were studied: control (n=9); low dose tirofiban (0.1 ug/kg/min, n=7); and high dose tirofiban (0.3 ug/kg/min, n=7). Tirofiban was infused 60 min before and during CPB. After heparin (300 n/kg), animals were perfused for 60 min at 50 ml/kg/min at 37°C using a bubble oxygenator (to increase platelet activation) and peripheral cannulation. Hemodynamics, platelet count, aggregation to ADP, release of PTG and template bleeding times (TBT) were measured before tirofiban infusion; before heparin; after heparin before CPB; after five and 55 min of CPB; after protamine (3 mg/kg); and 60 min after protamine. TBT were also measured 120 and 180 min after protamine. Platelet GPIIIa antigen was measured in Triton X-100 washes of the perfusion circuit after CPB.

High dose tirofiban completely prevented platelet loss and release of Î²TG. ADP aggregation was suppressed during CPB, but returned to control values 60 min after protamine. These measurements did not significantly differ from control with low dose tirofiban but did with the high dose. TBT in control animals was 11.6 and 10.5 min at 120 and 180 min after protamine. Both doses of tirofiban significantly ($p < 0.004 - 0.02$) shortened TBT at the same time points to 7 (120 min) and 5 min (180 min) after protamine. Surface GPIIIa antigen did not significantly differ between groups. Neither dose of tirofiban altered hemodynamics.

High dose tirofiban completely preserves platelet numbers and function during CPB in baboons and significantly accelerates restoration of normal template bleeding times.

*By invitation

F3. CORRELATION OF FUNCTIONAL PROGNOSIS WITH MYOCARDIAL MORPHOLOGY AND METABOLISM IN PATIENTS UNDERGOING CORONARY BYPASS SURGERY.

Christophe Depre, M.D.*, Jean-Louis Vanoverschelde, M.D., Ph.D.*, Bernard Gerber, M.D.*, Marcel Borgers, M.D., Ph.D.*, Jacques Melin, M.D., Ph.D.* and Robert Dion, M.D.*

Brussels, Belgium
Sponsored by: Bruce W. Lytle, M.D., Cleveland, Ohio

Little is known about the relation between postoperative functional recovery and the ultrastructural and metabolic alterations in myocardial ischemic dysfunction. In 40 patients with anterior wall dysfunction scheduled for bypass surgery, Positron Emission Tomography (PET) with ^{13}N-ammonia and ^{18}F-deoxy glucose was performed to assess myocardial blood flow and glucose uptake, respectively. Left ventricular function and regional wall motion score were assessed by contrast ventriculography and 2-dimensional (2-D) echo-cardiography before and 6 months after surgery. Transmural biopsies were obtained from the dysfunctional myocardial area during surgery and analyzed by light and electron microscopy to quantify fibrosis (an index of irreversible ischemic damage) and ischemia-altered but viable cardiomyocytes (characterized by a loss of myofilaments and cytosolic accumulation of glycogen).

A significant correlation was found between the reduction of regional blood flow and the surface of the biopsy covered by fibrosis ($r = 0.50$, $P<0.01$). Also, increased glucose uptake at PET significantly correlated with the density of altered cardiomyocytes ($r = 0.44$, $P<0.01$) and the amount of glycogen accumulation in these cells ($r = 0.42$, $P<0.01$). Six months after surgery, the changes in regional wall motion score were analyzed in 24 patients using 2D-echocardiography: 16 patients were considered to have viable myocardium and 8 patients to have non-viable myocardium. Retrospectively, at preoperative PET, viable myocardium had displayed higher blood flow (88 ± 23 vs 60 ± 12 ml.min^{-1} per 100 g, $P < 0.01$) and glucose uptake (50 ± 21 vs 30 ± 13 Hmol.min^{-1} per 100 g, $P<0.05$) than non-viable myocardium. Similarly, at morphological analysis, viable myocardium had shown less tissue fibrosis (24 ± 12 vs 45 ± 21% of surface, $P < 0.01$) and more altered cardiomyocytes (38 ± 15 vs 24 ± 15% of surface, $P < 0.05$) than non-viable myocardium. At follow-up, the ejection fraction measured by contrast ventriculography was increased by 20% in patients with viable myocardium and decreased by 25% in patients with non-viable myocardium ($P < 0.01$).

Therefore, in patients with left ventricular ischemic dysfunction, improvement of ventricular function after surgical revascularization is associated with a higher preoperative myocardial perfusion and glucose uptake, and with less tissue fibrosis and a higher amount of viable cardiomyocytes in the dysfunctional area. Such correlation provides a morphological basis for the prognostic value of non-invasive preoperative procedures in myocardial revascularization.

*By invitation

F4. ASPARTATE/GLUTAMATE ENRICHED BLOOD DOES NOT IMPROVE MYOCARDIAL ENERGY METABOLISM DURING ISCHEMIA-REPERFUSION: A ^{31}P MRS STUDY IN ISOLATED PIG HEARTS.

Hooman R. Ghomeshi, B.Sc.*, Ganghong Tian, M.D., Ph.D.*, Jian Ye, M.D.*, Jiankang Sun, M.Sc.*, Edward F. Hoffenberg, M.Sc.*, Tomas A. Salerno, M.D. and Roxanne Deslauriers, Ph.D.*

Winnipeg, Manitoba, Canada and Buffalo, New York

This study tests the effects of L-aspartate (asp) and L-glutamate (glu) with the hypothesis that depressed myocardial function and oxygen consumption (MVO_2) observed after an ischemic episode is not necessarily indicative of the lack of potential for efficient aerobic respiration, or of the need for manipulations of the perfusate as an attempt to improve such potential. ^{31}P Magnetic Resonance Spectroscopy (MRS) was used to observe cellular energetics in isolated pig hearts. The hearts were perfused with blood (group A) or blood enriched with 13 mM each of asp and glu (Group B), and then subjected to 30 min normothermic, zero-flow ischemia. The hearts were then reperfused with their respective perfusates for a period of 40 min (Stage I), after which they

received inotropic stimulation through a gradual increase in the perfusate (Ca^{++}) until the myocardial function reached a plateau and stabilized (Stage II). The data are summarized below, as percentage of pre-ischemic values. (Average pre-ischemic values for groups A and B, respectively: rate-pressure-product (RPP), 11100 and 11600 mmHg/min; -dP/dt, 1080 and 1020 mmHg/sec; MVO_2, 5.3 and 6.1 ml O_2/100g tissue/min; ATPandPCr, 100%).

		RPP	-dP/dt	MVO2	ATP	PCr
STAGE I	Group A	30	23	44	70	127
	Group B	27	17	47	68	134
STAGE II	Group A	89*	108*	76*	75	129
	Group B	83*	78*	74*	65	131

*$p < 0.05$ with respect to stage I

The MR spectra showed no decrease in the rate of energy decline during ischemia with asp/glu enriched perfusate. Furthermore, there were no significant differences between the two groups in terms of myocardial function, oxygen consumption, or the rate or extent of high-energy phosphate recovery, after either stage I or stage II of reperfusion. There was, however, a dramatic improvement in myocardial functional parameters and a significant improvement in MVO_2 with inotropic stimulation in stage II ($p < 0.05$). Furthermore, despite this near-complete recovery of function following inotropic stimulation, there were no changes in levels of ATP or PCr, demonstrating the cell's ability to recruit significant aerobic (data not shown) energy production when needed. The data suggest that in an isolated pig heart subjected to 30 minutes normothermic ischemia, it is possible to inotropically recruit significant functional recovery and sufficient aerobic respiration to support it, irrespective of asp/glu enrichment.

*By invitation

F5. NEAR-ZERO COOLING: A NEW STRATEGY FOR CEREBRAL AND MYOCARDIAL PROTECTION USING A BLOOD SUBSTITUTE.

George V. Letsou, M.D.*, John H. Braxton, M.D.*, Spyros Condos, Ph.D.*, Hal Sternberg, Ph.D.*, Paul Segall, Ph.D.*, William B. Shaffer, CPP*, Hazim J. Safi, M.D. and John C. Baldwin, M.D.

Houston, Texas; New Haven, Connecticut and Berkeley, California

Profound hypothermia is being used with increasing frequency in cardiothoracic surgery. However, current techniques are limited in cooling to only 15°C for 1 hour. We examined the use of a colloid-based blood substitute to facilitate cooling animals to 1-4°C for 4 hours.

Six dogs were placed on cardiopulmonary bypass via the femoral vessels. A centrifugal pump and heat exchanger were used for systemic cooling supplemented by a cooling blanket. At 21-25°C each animal's blood was completely replaced with a colloid-based blood substitute (BioTime, Inc., Berkeley, CA). Cooling then continued to a rectal temperature of 1-4°C. Ventricular fibrillation occurred spontaneously at 17-25°C. Below 15°C, all animals were asystolic. Animals were maintained below a rectal temperature of 4°C for 4 hours. Systemic rewarming was then accomplished via the bypass circuit and a warming blanket. At 10-18°C, the colloid-based blood substitute was replaced with the animals' stored blood. All animals were weaned from cardiopulmonary bypass at 35-37°C; no animal required pressor support. All (6 of 6) were

extubated and recovered normal cardiopulmonary function and behavior. Five of six animals survived 6 months to 1 year (1 died at 1 week from aspiration) with normal exercise capacity and behavior.

This blood replacement strategy allows for cooling below 4°C and results in extension of the safe period of neurologic and cardiopulmonary protection in the dog.

*By invitation

F6. ENDOTHELIAL STUNNING AND MYOCYTE RECOVERY AFTER REPERFUSION OF JEOPARDIZED MUSCLE: A ROLE OF L-ARGININE BLOOD CARDIOPLEGIA.

Asatoshi Mizuno, M.D.*, Rufus Baretti, M.D.*, Gerald D. Buckberg, M.D., Jakob

Vinten-Johansen, Ph.D.*, Helen H. Young, Ph.D.* and Louis J. Ignarro, Ph.D.*

Los Angeles, California

Early sudden death sometimes follows elective or emergency operations in patients who are apparently well. Emergent placement on bypass (CPB) shows open grafts. This study tests the hypothesis that this is caused by myocyte recovery in hearts undergoing endothelial stunning (reduced nitric oxide, NO synthase activity) causing vascular hypercontraction, platelet adherence and leukocyte aggregation and subsequent muscle damage.
Thirty pigs (25 kg) underwent cardiopulmonary bypass. Six received standard glutamate/aspartate blood cardioplegia (BCP) *without* intermittent ischemia. Twenty-four others underwent 20 minutes of 37°C ischemia. In 6, the clamp was removed after twenty minutes, *without* cardioplegia infusion (injury). In 18, the clamp was left on for 30 more minutes and all received the same blood cardioplegia solution. Of these, in 6 the BCP was normal, in 6 contained L-arginine (a NO precursor) at 2 mmol/L, and in 6 contained L-NAME (an NO synthetase inhibitor), at 1 mmol/L. Measurements included contractility by LV pressure volume loops and Starling curves, NO production, endothelial dependent (acetylcholine) and independent (nitroprusside) function, and myeloperoxidase (MPO) determination for endothelial leukocyte adherence.

Complete myocyte and endothelial recovery occurred in all non-jeopardized muscle. Complete myocyte systolic and diastolic recovery occurred also in BCP and BCP + L-arginine studies (approximately 90%). Conversely, recovery was <40% in the injury group (20 minutes ischemia alone), and BCP + L-NAME group. Substantial changes occurred with NO production, as only the BCP + L-arginine group produced 85 ± 10% of normal NO. In contrast, <25% recovery was with BCP alone, and no recovery with BCP + L-NAME. Coronary endothelial dependent vasodilatation (with acetylcholine) recovered 75 ±10% with BCP + L-arginine, but <20% in all others. Conversely, independent (nitroprusside) vasodilatation was unaltered in all groups. Myeloperoxidase activity (relative to control), rose 2.3 ± 0.2 after 20 minutes of ischemia only, 1.6 ± 0.4 with BCP, and rose only 0.3 ±0.2 with BCP + L-arginine.

This discrepancy between myocyte recovery and endothelial stunning in jeopardized muscle can be offset by adding L-arginine to the blood cardio-plegic solution. This may (a) suppress production of endothelial derived contracting factor (EDCF), and (b) counteract EDCF type responses, thereby avoiding need for leukocyte filters and anti-adhesion molecules. L-arginine is a cheap, naturally occurring substance in everyone that normalizes endothelial function. This limits endothelial vasospasm and neutrophil and platelet activity, and may have major importance in subsequent cardioplegic formulations.

*By invitation

F7. ANGIOGENESIS AND COLLATERAL BLOOD VESSEL GROWTH IN CORONARY ARTERY BYPASS PATIENTS.

Roland Fasol, M.D.*, Teddy Fischlein, M.D.* and Christian Holubarsch, M.D., Ph.D.*

Bad Neustadt, Munich and Freiburg, Germany
Sponsored by: Tirone E. David, M.D., Toronto, Ontario, Canada

Background. Controlled blood vessel growth and site-directed neovessel formation in vivo by the application of angiogenic growth factors have been investigated over the past few years in experimental models. Although the application of growth factors as a possible therapeutic intervention in the management of advanced coronary artery obstructive disease may have some potential value, no clinical data are available.

Methods and Results. To assess the angiogenic potency of purified basic fibroblast growth factor (bFGF), a modified chorioallantoic membrane assay was performed, which showed significant new blood vessel growth. The growth promoting activity of purified bFGF was studied by comparing adult human endothelial cell (AHEC) control cultures, without adding growth factor to the culture medium, with AHEC cultures with 0.5, 1.0 and 5.0 ng/ml bFGF added to the culture medium. Tritiated thymidine counts proved the significant growth promoting activity of bFGF.

In a subsequent clinical study with five selected coronary artery bypass patients, 3 ml of modified fibrin glue containing 20 ng/ml bFGF was implanted between the distal internal mammary artery (IMA) pedicle and the myocardium of the anterior wall, in the area between the left anterior descending artery (LAD) and the diagonal branch. In addition, 4 ml of purified bFGF with a concentration of 20 ng/ml was applied subepicardial into the myocardium of the left ventricle in this area.

Arterial digitized computed angiography of the IMA transplanted to the LAD was performed postoperatively at the time of discharge to confirm the patency. Coronary angiography was performed after a follow-up period of one year which showed significant growth of new blood vessels in every investigated patient, indicative of coronary angiogenesis and collateral growth at the site of bFGF implantation. To quantitate the results, digital grey scale analysis of the corresponding myocardial areas in pre- and postoperative angiograms of every patient was performed. This analysis showed a significantly higher grey scale, indicative of a significantly higher density of blood vessels, at the site of bFGF implantation in the postoperative angiogram, as compared to preoperative as well as to other myocardial segments postoperatively.

Conclusions. Our data suggest that significant coronary angiogenesis and new coronary collateral blood vessel growth is possible in patients receiving angiogenic growth factor implants to the heart.

*By invitation

F8. L-ARGININE REDUCES HETEROGENEITY OF CORONARY BLOOD FLOW, REDUCES NEUTROPHIL ADHESION, AND IMPROVES RECOVERY OF VENTRICULAR FUNCTION AFTER HYPOTHERMIC ISCHEMIA

Takeshi Hiramatsu, M.D.*, Toshiharu Shin'oka, M.D.*, Marc L. Schermerhorn, M.D.*, Gregor Zund, M.D.*, Takuya Miura, M.D.*, David P. Nelson, M.D.* and John E. Mayer, Jr., M.D.

Boston, Massachusetts

Prior experiments suggest that vascular events such as the "no reflow phenomenon" are involved in ischemia (1) and reperfusion (R) injury, and histologically the injury following global ischemia is typically focal. Regional disparities (heterogeneity-Het) in post-ischemic blood flow have been demonstrated. But the relationship between vascular events and I/R myocardial injury remain unclear. We investigated the relationships between regional variations in blood flow (each LV divided into 48 pieces) using microspheres, regional neutrophil accumulation (myeloperoxidase [MPO] assayed in each LV piece), and the recovery of global ventricular function (LV max developed pressure-DP and dP/dt) in 12 isolated blood-perfused neonatal lamb hearts subjected to 2 hours of hypothermic (15°C) cardioplegic ischemia followed by reperfusion. One group received 3 mM L-arginine during R to augment endothelium mediated vasodilation and inhibit neutrophil-endothelial interactions. Controls received only cardioplegia. Endothelial function was assessed by coronary resistance response (CRR) to 10^{-7} M acetylcholine (an endothelium dependent vasodilator), and regional coronary blood flow (CBF) was measured pre-ischemia and at 5, 15, and 30 min of R. A heterogeneity index for each heart at each time point was calculated as the coefficient of variation of CBF among 48 segments (100 x SD/mean). Correlation coefficients (CC) relating CBF at 5 min of R and MPO for each of the 288 heart segments in each group were calculated (# = $p<0.01$; * = $p<0.05$ vs controls; % = % recovery of pre-ischemic values).

Group	n	Mean Het index	MPO (U/g)	CC	CBF (%)	max DP (%)	+maxdP/dt (%)	CRR (%)
controls	6	72.4	5.14	-0.44#	86.8	68.2	61.7	49.5
L-arginine	6	39.9*	0.85*	-0.52#	121.1*	90.2*	85.4*	70.8*

The inverse correlations (CC) between regional neutrophil accumulation (MPO activity) and CBF during early R suggest that microvascular occlusion by neutrophils causes regional ischemia and reduced recovery of global ventricular function. L-arginine reduced the heterogeneities in CBF between regions of the heart during early R, reduced neutrophil accumulation, and was associated with improved recovery of global LV function and endothelial function. Strategies such as L-arginine infusion which reduced neutrophil adhesion and enhance CBF during early R seem likely to be useful in limiting I/R injury.
*By invitation

9:00 a.m. SCIENTIFIC SESSIONS

Room 6, San Diego Convention Center

Moderators: G. Alexander Patterson,. M.D.

Joseph I. Miller, M.D.

15. LIVING DONOR LOBAR LUNG TRANSPLANTATION EXPERIENCE: INTERMEDIATE RESULTS.

Vaughn A. Starnes, M.D., James E. Davis, M.D.*, Mark L. Barr, M.D.*, Felicia A. Schenkel, R.N.*, Monica V. Horn, R.N.*, Winfield J. Wells, M.D.*, Jeffrey A. Hagen, M.D.*, Robbin G. Cohen, M.D.* and The University of Southern California Lung Transplant Group

Los Angeles, California

Discussant: Charles B. Huddleston, M.D.

Living donor lobar lung transplantation offers an alternative form of treatment for patients with end-stage lung disease who might otherwise die while waiting for traditional cadaveric lung transplantation. Thirty-four adult (25) and pediatric (9) patients (14 females, 20 males) have undergone living lobar transplantation since January 1993. The mean age was 22.5 ± 1.1 yrs (range 9.3 to 36 yrs). Thirty-one patients had cystic fibrosis. Other indications were pulmonary hypertension, pulmonary fibrosis and obliterative bronchiolitis. Patients were selected for transplantation based on increasing frequency or severity of infections, as well as developing antibiotic resistance or deteriorating pulmonary function tests. The mean weight was 43.7 ± 1.7 kg. The mean pCO_2 and pO_2 were 65 ± 5 and 70 ± 5 mmHg, respectively. Preoperatively, 23 patients required supplemental oxygen or mechanical ventilation. Twenty-five procedures were done emergently or urgently. The mean lobe ischemic time was 65 ± 6 minutes. Immunosuppression consisted of cyclosporine, azathioprine, and corticosteroids. Gancyclovir was used in 28 cases where either the recipient or donor was cytomegalovirus (CMV) positive. The mean survival time was 20.2 ± 2.1 months with a 1-year survival of 66.1 ± 9.1%. There were 7 in hospital deaths and 4 late deaths. Rejection occurred in 16 patients (0.07 rejections/patient month) and were treated with steroid pulse therapy. Twenty-three patients developed post-operative infections which were most often Pseudomonal pneumonias but included 7 patients with fungal infections and 7 with CMV infections. Postoperatively, pulmonary function tests improved significantly ($p<0.001$). FEV_1 and FVC increased from 23 ± 2% to 68 ± 4% and 37 ± 2% to 67 ± 4% of predicted, respectively. The most significant improvement occurred in the FEF25.75 measurements (8.5 ± 2.2% to 68 ± 6%). Normal cardiac and pulmonary hemodynamics were present postoperatively. Fourteen patients were catheterized. Mean right atrial pressure, pulmonary wedge pressure, pulmonary artery systolic and diastolic pressures were 4 ± 1 mmHg, 10.4 ± 1.6 mmHg, 32.1 ± 2.7 mmHg, and 13.4 ± 1.7 mmHg, respectively. The mean NYHA class improved from 3.1 ±0.1 to 1.1 ±0.1 ($p<0.001$). These intermediate results demonstrate that living donor lobar lung transplantation is in effective therapy for critically ill patients in need of urgent transplantation.

*By invitation

16. SINGLE OR BILATERAL LUNG TRANSPLANTATION FOR EMPHYSEMA?

Sudhir Sundaresan, M.D.*, Yuji Shiraishi, M.D.*, Jenny Manley, R.N.*, Dottie Bigger, R.N.*, Mary Pohl, R.N.*, Elbert P. Trulock, M.D.*, Joel D. Cooper, M.D. and G. Alexander Patterson, M.D.

St. Louis, Missouri

Discussant: J. Kent Trinkle, M.D.

The optimal lung transplant procedure for Chronic Obstructive Pulmonary Disease (emphysema and Alpha-1 Antitrypsin Deficiency) continues to be a matter of controversy. To date, no long-term follow-up study with comparison of late results is available. Although most programs favor single lung transplantation (SLT), we have limited SLT mainly to older patients and those with small body habitus. We analyzed our experience with 118 consecutive lung transplants for emphysema between 1989-1994 (50 SLT and 68 bilateral lung transplants [BLT]) to try to determine which might be the superior option. The SLT group was older and had a higher proportion of females (68% vs. 43% in BLT group, $p < 0.01$). However, pulmonary function (FEV_1), arterial oxygen tension (PaO2), and exercise tolerance (distance covered in a 6-minute walk test) were similar. Post-transplantation, 90-day mortality (SLT 10% vs BLT 8%; $p = 0.74$), and duration of mechanical ventilation, intensive care unit stay, and hospitalization were similar. A significant improvement in FEV_1; PaCO2, PaO2, and exercise tolerance was noted within 3 months post-transplantation in both groups, and was sustained. However, the improvements in FEV_1, PaO2, and exercise tolerance were consistently and significantly better in the BLT group at and beyond 6 months (data shown in table).

Parameter		Time Point				
		Pre-op	3 Mos	6 Mos	1 Yr	3 Yrs
FEV_1	SLT	18 ± 1	56 ± 2*«	57 ± 2	54 ± 2	45 ± 3
(% predicted)	BLT	17 ± 1	84 ± 3*†	91 ± 2†	94 ± 3†	80 ± 7†
PaO2	SLT	54 ± *2*	80 ± 2*	81 ± 1	81 ± 2	80 ± 2
(mmHg)	BLT	57 ± 1	87 ± 2†	89 ± 2†	92 ± 2†	89 ± 4†
6 minute walk	SLT	980 ± 46	1509 ± 46*	1529 ± 46	1494 ± 53	1553 ± 52
(feet)	BLT	991 ± 50	1624 ± 46*	1704 ± 48†	1713 ± 52t	1817 ± 109†

All data mean ± S.E.M.

*Significant vs. pre-op value

†Significant vs. corresponding SLT value

Obliterative bronchiolitis (OB) was equally prevalent in both groups (BLT 37% vs SLT 38%; $p = 0.99$). Survival was similar in both groups until 3 years (BLT 83% vs SLT 81%), but subsequently showed a tendency to better survival in the BLT group (5 year actuarial survival: BLT 65% vs SLT 42%). These data demonstrate that: (1) both SLT and BLT are satisfactory options in emphysema and produce durable results; (2) BLT is not associated with increased operative mortality or morbidity, and produces significantly better improvement in spirometry, oxygenation, exercise tolerance, and possibly late survival than SLT; and (3) the observed benefit following BLT on late survival and function may be attributed to the presence of more pulmonary reserve after the onset of OB.

*By invitation

17. LONG-TERM RESULTS OF LUNG METASTASECTOMY: REPORT ON 5,206 CASES FROM THE INTERNATIONAL REGISTRY OF LUNG METASTASES.

Ugo Pastorino, M.D.*, Robert Ginsburg, M.D., Patricia McCormack, M.D., Joe Putnam, M.D., Harvey Pass, M.D., Michael Johnston, M.D.* and Peter Goldstraw, M.D.*

London, United Kingdom; New York, New York; Houston, Texas; Bethesda, Maryland and Toronto, Ontario, Canada

Discussant: Valerie W. Rusch, M.D.

An International Registry of Lung Metastases was established in 1991 to assess the long-term results of pulmonary metastasectomy. Data has been collected from 18 major departments of thoracic surgery in Europe (13), United States (4) and Canada.

The Registry has accrued a total of 5207 cases of lung metastasectomy, of which 4572 (88%) underwent complete surgical resection. Main patient features were: 2932 males and mean age 44 (range 2-93). In 2260 cases the pulmonary metastases were from an epithelial tumour, 2203 from sarcomas, 363 had germ cell deposits and 328 melanomas. 1603 cases experienced a disease-free interval (DPI) of 0-11 months (mos), 1857 cases had DPI 12-35 mos and 1620 cases had DPI 36+ mos. There were 3687 thoracotomies and 1415 sternotomies. Single proven metastases accounted for 2383 cases, and 2726 had multiple lesions. Mean follow-up of the whole series was 46 mos. Analysis of the data, including Kaplan-Meier estimates of the survival curves, related risks of death (RR), proportional hazard and multivariate Cox model was performed by an independent centre in Brussels.

The survival after complete metastasectomy was 36% at 5 years, 26% at 10 years and 22% at 15 years, with a median survival of 35 mos; the corresponding values for incomplete resection were 13% at 5 years and 7% at 10 years, with a median of 15 mos. Among complete resections, the 5-year and median survival was 33% and 29 mos for patients with DPI of 0-11 mos, 31% and 30 mos for DPI of 12-35 mos, 45% and 49 mos for DPI of 36+ mos. The 5-year and median survival for complete resection of single lesion cases was 43% and 43 and mos, 34% and 31 mos for 2-3 lesions, 27% and 27 mos for 4+ lesions.

At multivariate analysis, the primary tumor type, DPI and number of metastases emerged as significant prognostic factors. In particular, germ cell and Wilms' tumors showed the best prognosis (RR=0.4) and melanoma the worst prognosis (RR=2.1); DPI of 36+ mos had a better prognosis (RR=0.6), as well as single metastases (RR=0.7).
On this basis, further analyses are in progress with the aim of defining a system of classification in three to four prognostic groups, valid for the various primary tumor types.

10:00 a.m. INTERMISSION - VISIT EXHIBITS

10:45 a.m. SCIENTIFIC SESSIONS

Room 6, San Diego Convention Center

Moderators: Mortimer J. Buckley. M.D.

Fred A. Crawford, Jr., M.D.

18. SUPERIOR VENA CAVA TO PULMONARY ARTERY ANASTOMOSIS: AN ADJUNCT TO BIVENTRICULAR REPAIR.

Glen S. VanArsdell, M.D.*, William G. Williams, M.D., Catherine M. Maser, R.N.*,

Kim Streitenberger, R.N.*, Ivan M. Rebeyka, M.D.*, John G. Coles, M.D. and Robert

M. Freedom, M.D.*

Toronto, Ontario, Canada

Discussant: Frank L. Hanley, M.D.

A morphologically small or poorly functioning right ventricle (RV) can increase the risk of biventricular repair. It has been proposed that a superior vena cava to pulmonary artery (SVC-PA) anastomosis can unload an inadequate RV thereby enhancing the success of biventricular repair. From May 1981 to September 1995, 41 patients received a SVC-PA anastomosis in association with biventricular repair. Indications were: group A (19 pts), small physiologic right ventricle defined as tricuspid annulus z value of < -2 ([mean -3.7 ±1.4] [14/19]) or predicted RV volume of 69-87% ([mean 77.8 ± 8] [5/19]); group B ([11 pts] [9/11 Ebsteinis]), chronic RV dysfunction; group C (4 pts), facilitation of repair (e.g. LSVC and AVSD); group D (4 pts), postoperative RV dysfunction. Three patients were excluded from analysis because the SVC-PA anastomosis was unrelated to biventricular repair. Age ranged from 5 months to 51 years (median 3.5 years). Diagnostic subsets were heterogenous: Ebsteinis anomaly (9), d-TGA(4), L-TGA(4), VSD (3), TOP (3), PS (3), pulmonary atresia with IVS (3), ASD (2), DORV (2), AVSD (2), DILV with TGA (1), AVSD with TOP (1), and left atrial isomerism complex (!)* Twenty-five patients received a right bidirectional cavopulmonary anastomosis (BCPA), 6 a side-to-side cavopulmonary anastomosis, 4 a left BCPA, 2 a classic Glenn shunt, and 1 a bilateral BCPA anastomosis. Operative mortality was: group A 2/19 (10.5%), group B 1/11 (9%), group C 0/4 (0%) and group D 3/4 (75%). Follow-up is complete in 37/38 patients (97%), ranging from 1 to 174 months (mean 45.6 ± 37.3) with one late mortality (1/31, 3%). NYHA class is I (22) or II (8). There is no clinical evidence of pulmonary AV fistulae or protein losing enteropathy. In the setting of an inadequate RV, this series demonstrates an acceptable intermediate term outcome for SVC-PA anastomosis and biventricular repair. In a subset, SVC-PA anastomosis safely facilitates repair. Results for this procedure when used as a salvage operation for severe postoperative RV dysfunction have not been satisfactory.
*By invitation

19. IS A HIGH RISK BIVENTRICULAR REPAIR ALWAYS PREFERABLE TO CONVERSION TO A SINGLE VENTRICLE REPAIR?

Ralph E. Delius, M.D.*, Marc A. Radermecker, M.D.*, tMarc R. de Leval, M.D.,

Martin J. Elliott, M.D.* and Jaroslav Stark M.D.

London, United Kingdom

Discussant: Vaughn A. Starnes, M.D.

AIM: A commonly held concept is that in the presence of two ventricles, a two ventricle repair is always preferable over a single ventricle repair. Whether this policy should be adhered to for high-risk biventricular repair remains controversial. The aim of this report is to examine the short and intermediate term outcome of a complex two ventricle repair with a single ventricle repair in patients with two functional ventricles.

PATIENT POPULATION: Since 1986, 34 patients with atrioventricular (AV) concordance or discordance, ventriculoarterial (VA) discordance, ventricular septal defect (VSD), and pulmonary stenosis or atresia (PS/PA) have undergone VSD closure and placement of a valved conduit between the pulmonary ventricle and pulmonary artery (Group I). Another group of 16 patients (Group II) with the same diagnoses have undergone a single ventricle repair consisting of a total cavopulmonary connection (TCPC) because of either a straddling AV valve (11 patients) or an uncommitted VSD (5 patients). These patients would have otherwise been candidates for a two ventricle repair.

RESULTS: The mean length of follow-up in the Group I was 3.9 years (range 1-10 years) and 3.0 years in Group II (range 0.5-9 years). There were 12 late reoperations in 11 patients in Group I and no late reoperations in Group II. Freedom from reoperation at 5 years was 60.5% in Group I and 100% in Group II ($p < 0.03$). There were 5 early and 3 late deaths in Group I. There was one early death in Group II and no late deaths. The actuarial estimate of survival at 5 years was 68.0% in Group I and 93.8% in Group II ($p < 0.05$). In Group I there were 2 patients (6.5%) that were Class III or Class IV; the remainder were Class I or II. All patients in Group II were NYHA Class I or II.

CONCLUSION: There appeared to be greater short and intermediate term morbidity and mortality in patients who underwent a conventional two ventricle repair compared to a similar group of patients who underwent a TCPC, even though the patients managed with a two ventricle repair were ideal candidates for this approach in contrast to the patients managed with a TCPC, who would have been at high risk for a two ventricle repair because of a straddling AV valve or an uncommitted VSD. It is unknown if the funtional long-term results of a TCPC in patients with 2 ventricles are as good as those obtained with a two ventricle approach. However, these findings suggest that there may be situations in which the short and intermediate term risks of a complex two ventricle repair may outweigh the long-term disadvantages of a single ventricle approach.

11:25 a.m. HONORED GUEST SPEAKER

Tolerance to Allogeneic and Xenogeneic Transplants
David H. Sachs, M.D., Boston, Massachusetts

12:10 p.m. ADJOURN FOR LUNCH - IN EXHIBIT HALL

12:10 p.m. CARDIOTHORACIC RESIDENTS' LUNCHEON

†1973-74 Graham Fellow

*By invitation

WEDNESDAY MORNING, MAY 1, 1996

7:00 a.m. FORUM SESSION II - GENERAL THORACIC SURGERY

Room 6C/F, San Diego Convention Center

Moderators: Keith S. Naunheim, M.D.

Larry R. Kaiser, M.D.

F9. REGULATORY EFFECTS OF INTERLEUKIN-10 ON LUNG ISCHEMIA-REPERFUSION INJURY.

Michael J. Eppinger, M.D.*, Peter A. Ward, M.D.*, Steven F. Bolling, M.D. and G. Michael Deeb, M.D.

Ann Arbor, Michigan

Lung reperfusion injury may predispose the transplanted lung to poor function and early rejection. Interleukin-10 (IL-10), a cytokine with primarily anti-inflammatory effects, was studied to determine its effects on the development of early lung reperfusion injury.
Adult male rats underwent clamping of the left bronchus, pulmonary artery, and pulmonary vein for 90 minutes of ischemia, followed by 4 hours of reperfusion (controls, n=6). Time-matched shams underwent hilar dissection but not lung ischemia (n=4). Lung injury was measured by vascular permeability to ^{125}I-BSA (cpm/g lung tissue/ml blood). To evaluate the effect of exogenous IL-10, additional animals (n=4) received 10 Î¼g IL-10 intravenously prior to ischemia. To assess the role of endogenous IL-10, animals received 200 Î¼g of either rabbit anti-mouse IL-10 IgG or pre-immune IgG (n=5 each) prior to ischemia.

Compared to shams, controls demonstrated significantly more lung injury (permeability index 0.358±0.035 vs. 0.102±0.009, $p<0.01$). Animals receiving IL-10 had significantly less lung injury compared to controls (0.167±0.028, $p<0.01$). Animals receiving IgG against IL-10 had significantly more lung injury than animals receiving pre-immune IgG (0.411±0.056 vs 0.237±0.044, $p<0.05$). Alveolar macrophages from animals after 90 minutes of lung ischemia produced more TNF-a in culture than unstimulated macrophages; this production was reduced significantly by the addition of IL-10 to the culture medium. Northern blot analysis of whole lung RNA demonstrated that the reduction in TNF-a occurred at the mRNA level.

We conclude that endogenous IL-10 has a protective effect against lung reperfusion injury during this early phase; and that IL-10 administration can reduce lung reperfusion injury, at least in part through its ability to reduce production of TNF-a by alveolar macrophages.

*By invitation

F10. CHANGES IN LUNG COMPLIANCE AFTER VOLUME REDUCTION SURGERY IN A RABBIT MODEL OF BULLOUS EMPHYSEMA.

Fernando E. Kafie, M.D.*, Matthew Brenner, M.D.*, John C. Chen, M.D.*, Edward A. Stemmer, M.D., Michael Budd, B.S.* and Michael W. Berns, M.D.*

Orange, California

Purpose: Staple lung volume reduction surgery has recently been described for treatment of emphysema resulting in improvement in Forced Expiratory Volume (FEV_1). Little is known regarding physiologic mechanisms of response in surgically treated emphysema patients. We hypothesized that volume reduction surgery in animals with pulmonary bullous emphysema would result in decreased lung compliance. Reduction in compliance may decrease airway resistance and improve Forced Expiratory Volume.

Methods: Seventeen New Zealand (NZ) white rabbits were induced with emphysema according to our previously published model with sephadex beads and carrageenan. This animal model has been previously used to study surgical treatments for emphysema. Pressure-volume relationships were measured at 60, 40 and 20 cc inflation pre- and post-operatively in anesthetized animals. Thoracoscopy was performed prior to thoracotomy to document bullae formation. A mini-

thoracotomy was performed on the side of bullae formation. Resection of areas with emphysema was accomplished with a standard pediatric multirow surgical stapler.

Results: Comparison of pressure-volume curves pre- and post-op demonstrate significant decrease in static compliance.

Pressure-Volume Data

Vol (cc H_20)	PRE-OP (mmHg)*	POST-OP (mmHg)*	p-VALUE (chi-test)
20	6.8	6.0	<0.01
40	18.1	15.7	<0.01
60	21.9	19.4	<0.01

* average of 17 experiments

Conclusion: Lung compliance is decreased following lung volume reduction surgery in New Zealand White rabbits. This finding suggests that increased elastic recoil and airway support may contribute to the mechanism of improved function following lung volume reduction surgery.

*By invitation

F11. GENETICALLY ENGINEERED PORCINE LUNGS IN A HUMAN XENOTRANSPLANTATION MODEL.

Casey W. Daggett, M.D.*, Mark Yeatman, FRCS*, Andrew J. Lodge, M.D.*, JeffH. Lawson, M.D.*, Edward P. Chen, M.D.*, Meera Srinivasan, B.A.*, Peter Van Trigt, M.D., Gerry Byrne, Ph.D.*, John Logan, Ph.D.*, Jeff L. Platt, M.D.* and Robert D. Davis, M.D.*

Durham, North Carolina

Pulmonary xenotransplantation is currently limited by an abrupt rise in pulmonary vascular resistance, capillary leak, loss of compliance, and poor gas exchange. Complement activation is believed to be a central event in this process. The human complement regulatory proteins, decay accelerating factor (hDAF) and CD59 (hCD59), inhibit both the classical and the alternative pathways. Using an *ex vivo* model has made it possible to study specific aspects of acute pulmonary dysfunction in an heterologous combination. The pulmonary function of swine expressing hDAF/hCD59 (n=7) was compared to that of the lungs from farm bred animals (n=6) while the lungs were perfused with human fresh frozen plasma (FFP). Lungs from adult swine were isolated and preserved with Euro-Collins solution. The perfusate consisted of freshly thawed, heparinized, pooled O+FFP reconstituted in 40% Lactated Ringer's solution. Perfusion fluid was delivered to the pulmonary artery at 37°C via a gravity reservoir and recirculated by a roller pump. The lungs were ventilated with 60% oxygen and the tidal volume was controlled to keep the peak airway pressure between 35-40 cm H_2O. After two hours of perfusion the control lungs had lost an average of 74 ± 16% of their static pulmonary compliance versus a 6 ± 17% loss by the transgenic lungs

($p<0.001$). The controls had an average transalveolar capillary leak of 561.7 ml compared to 5.9 ml by transgenic lungs ($p<0.05$). The control lungs achieved an oxygen concentration in the perfusate of 259 ± 42 mmHg compared to 383 ± 42 mmHg in transgenic lungs ($p<0.001$). The pulmonary vascular resistance was 20.3 ± 12.6 mmHg/L/min in controls and 10.9 ±2.0 mmHg/L/min in transgenic lungs ($p = 0.17$).

In conclusion, the lungs from swine expressing hDAF/hCD59 demonstrated superior pulmonary function compared to lungs from farm bred swine when perfused with human plasma. The compliance, capillary leak, oxygenation, and pulmonary vascular resistance were all significantly improved in the transgenic lungs as compared to controls. These data indicate that complement activation is in part responsible for acute pulmonary dysfunction in xenotransplantation and that inhibiting complement function with hDAF and hCD59 can improve several aspects of pulmonary function in porcine-to-human pulmonary transplantation.

*By invitation

F12. ISOLATED LUNG PERFUSION WITH MELPHALAN FOR TREATMENT OF METASTATIC PULMONARY SARCOMA.

Sumihiko Nawata, M.D.*, Howard M. Ross, M.D.*, Nuno Abecasis, M.D.*, Komal S. Sachar, B.S.*, Huiming Cheng, M.A.* and Michael E. Hurt, M.D., Ph.D.

New York, New York

Introduction: Metastatic pulmonary sarcoma remains a significant clinical problem with systemic chemotherapy offering little hope for cure. Isolated lung perfusion (ILP) avoids systemic chemotherapeutic toxicity, but the most efficacious agent remains unknown. Melphalan (MN) is active in the treatment of extremity sarcoma via isolated limb perfusion and therefore MN activity in a pulmonary sarcoma metastases model was investigated.

Methods: Toxicity Study: Nineteen F344 rats underwent left ILP with MN at total doses of 20 mg (n=2), 5 mg (n=6), 2 mg (n=6), or buffered hespan (BH) (n=5). Rats underwent contralateral pneumonectomy on day 21 post-perfusion to evaluate left lung toxicity. Efficacy Study: On day 0, 41 F344 rats were injected with $5x10^6$ MCA sarcoma cells via the external jugular vein. On day 7, rats received either 2 mg MN i.v. (n=10), 1 mg MN i.v. (n=8), or underwent ILP with MN (2 mg) (n=12) or BH (n=11). On day 14, rats were sacrificed and lung sarcoma nodules were counted. Statistical analysis was performed with ANOVA and Student T test.

Results: Toxicity: All rats perfused with 20 mg or 5 mg of MN died perioperatively. Rats perfused with 2 mg of MN or BH survived contralateral pneumonectomy at rates of 67% and 80%, respectively. Efficacy: The number of left lung lesions decreased significantly in the animals receiving MN via ILP as compared to all the other groups ($p<0.05$). In addition, MN ILP resulted in significant reduction of tumor nodules in treated lung as compared to right lung ($p<0.02$). All rats that received MN 2 mg i.v. died within 5 days of injection.

Group		number of lesions in left lung	right lung
MN 1 mg i.v.	(n=8)	60 ± 21	66 ± 23
MNILP	(n=12)	7 ± 10	185 ± 70
BHILP	(n=ll)	84 ± 52	201 ± 51

Conclusion: Melphalan ILP is well-tolerated at a dose that leads to 100% mortality intravenously. Melphalan ILP significantly decreased the number of metastatic pulmonary nodules compared to i.v. treatment. Melphalan can be considered an effective agent for ILP of metastatic sarcoma and studies evaluating Melphalan by ILP in man are warranted.

*By invitation

F13. GENE THERAPY FOR LUNG CANCER: ENHANCEMENT OF TUMOR SUPPRESSION BY A COMBINATION OF SYSTEMIC CISPLATINUM AND ADENOVIRUS-MEDIATED P53 GENE TRANSFER.

Dao M. Nguyen, M.D., FRCSC*, Sandra A. Weihle, B.Sc.*, Patricia E. Koch, M.Sc.*,

Richard J. Cristiano, Ph.D.* and Jack A. Roth, M.D.

Houston, Texas

Mutations of the p53 tumor suppressor gene occur in up to 70% of human non-small cell lung cancers. Restoration of the normal p53 function by gene replacement therapy in cancer cells with an abnormal p53 gene leads to G_1cell cycle arrest or apoptosis (programmed cell death). We observed that brief exposure of H1299 lung cancer cells (deleted p53) to low doses of cisplatinum (CDDP) prior to gene transfer resulted in a 2-fold elevation of reporter gene expression. To determine if such treatment would potentiate the tumor suppression effect of the AdV-CMV-p53 (recombinant adenovirus carrying the p53 gene driven by the cytomegalovirus [CMV] enhancer/promoter), H1299 cells were treated with CDDP (0.062 Î¼g/ml x 24 hrs) 2 days prior to transfection with AdV-CMV-p53 at multiplicities of infection (MOI) of 1 and 5 viral particles per cell (n=6 per group). Prior exposure to CDDP resulted in a 35% (MOI = 1) to 61% (MOI = 5) enhanced inhibition of tumor cell proliferation 3 and 5 days after AdV-CMV-p53 transfection as compared to that of similarly treated cells without prior CDDP exposure. In vitro transfection of CDDP-treated cells with AdV-CMV-p53 led to earlier, higher levels of p53 gene expression as well as increased apoptosis. Subcutaneous H1299 tumors were created in irradiated nude mice. A combination of **sequential** intraperitoneal CDDP (5 Î¼g/g of body weight) and injections of AdV-CMV-p53 ($5x10^9$ viral particles/injection) into H1299 tumors (200 mm^3) 2, 4, 6 days following CDDP administration resulted in a profound and prolonged inhibition of tumor growth of H1299 tumors in nude mice (n=5 per group). While systemic administration of CDDP had a small effect on H1299 tumor growth (3000±218 mm^3) compared to saline-injected tumors (3550±240 mm^3, 20 days after injection), tumors treated by a combination of CDDP and AdV-CMV-p53 were significantly smaller (1570±140 mm^3) than those treated with AdV-CMV-p53 alone (3100±260 mm^3, 32 days after treatment, $p<0.0001$). The timing of systemic CDDP administration relative to gene transfer was identified to be critical as simultaneous intraperitoneal CDDP and intratumoral AdV-CMV-p53 injections were less effective than sequential treatment (2300±196 mm^3 vs 1570±140 mm^3, $p<0.001$). A second cycle of combined CDDP and gene therapy given 10 days after completion of the first one led to further suppression of tumor growth (679±89 mm^3 vs 1570±140 mm^3, $p<0.001$). In conclusion, the combination of sequential systemic CDDP and intratumoral injection of AdV-CMV-p53 results in a superior tumor suppression effect. Inhibition of tumor growth can be maintained by repeated cycles of gene therapy. This gene therapy strategy has been incorporated into a phase I clinical trial for the treatment of lung cancer and provides the basis for the development of improved therapeutic protocols.

*By invitation

F14. THE DUAL FACES OF INHALED NITRIC OXIDE: IMPROVED LUNG PRESERVATION WITH EXOGENOUS NITRIC OXIDE GIVEN AT THE TIME OF HARVEST BUT NOT WHEN GIVEN DURING REPERFUSION.

Yoshifumi Naka, M.D., Ph.D.*, Dilip K. Roy, M.D.*, Hui Liao, M.D.*, David M. Stern, M.D.*, Arthur J. Smerling, M.D.*, Robert E. Michler, M.D., David J. Pinsky, M.D.* and †Mehmet C. Oz, M.D.*

New York, New York

Although inhaled nitric oxide (NO) lowers pulmonary vascular resistance in ARDS, its usefulness in the setting of lung transplantation remains controversial. We hypothesized that NO may have either beneficial or harmful effects depending upon the circumstances in which it is given. If NO given to the pulmonary donor raises endogenous (tissue) cGMP levels, this should benefit lung preservation by promoting vascular function, as cGMP analog supplementation is known to do. NO administered during reperfusion may rapidly combined with superoxide to become either ineffective or toxic (forming peroxynitrite and hydroxyl radical). Using an orthotopic rat left lung transplant model in which hemodynamics and functional parameters can be measured independent of the native lung [following ligation-of the right pulmonary artery (PA)], 4 experimental groups were established using male Lewis rats: (1) no supplemental gas given (No NO); (2) NO given at the time of harvest (65 ppm measured by chemiluminescence, Harvest NO); (3) supplementation of the preservation solution with a membrane permeable cGMP analog 8-Bromo-cGMP under No NO conditions (500 nM, cGMP); and (4) NO given during reperfusion (65 ppm, Reperfusion NO). For all groups, lungs were preserved for 6 hours at 4°C in Euro-Collins solution. Thirty minutes following ligation of the native PA, PA flow (ml/min), arterial oxygenation (pO_2, mmHg), graft neutrophil infiltration (myeloperoxidase activity, MPO, Î”absorbance/min at 460 nm), and recipient survival were determined.

Condition	PA flow	pO_2	MPO	Survival
No NO (n=25)	3.9 ± 1.5	94 ± 13	2.8 ± 0.1	20%
Harvest NO (n=9)	14.1 ± 3.6*†	165 ± 33*†	2.3 ± 0.3*†	67%*
CGMP (n=11)	17.9 ± 4.0*†	165 ± 31*†	1.9 ± 0.1*†	73%**†
Reperfusion NO (n=11)	4.9 ± 2.2	94 ± 20	2.8 ± 0.2	27%

(Means±SEMS are shown: *=p<0.05, and **=p<0.01 vs No NO, and †=P<0.05 vs Reperfusion NO)

To explore potential mechanisms underlying these beneficial effects of Harvest NO, and knowing that NO stimulates the soluble guanylate cyclase to produce endogenous cGMP, we determined (by ELISA) that Harvest NO increases endogenous pulmonary cGMP (by 38% vs No NO, p<0.05). These data suggest that stimulating the NO/cGMP pathway (such as by Harvest NO or by supplementing the preservation solution with a cGMP analog) is beneficial. We conclude that inhaled NO can be either beneficial or neutral, depending upon the circumstances in which it is given.

†Robert E. Gross Research Scholar

*By invitation

F15. MITIGATION OF INJURY IN CANINE LUNG GRAFTS BY EXOGENOUS SURFACTANT THERAPY.

Ken E. Gehman, M.D.*, Richard J. Novick, M.D., Andrea A. Gilpin, HBSc.*, Imtiaz S. AH, M.D.*, Ruud A.W. Veldhuizen, Ph.D.*, Jenifer Duplan, AHT*, Lynn Denning, AHT*, Fred Possmayer, Ph.D.* and James F. Lewis, M.D.*

London, Ontario and Edmonton, Alberta, Canada

We have previously demonstrated alterations in endogenous surfactant after lung transplantation and improved pulmonary function after 36 hour preservation of normalcanine lung grafts using donor bovine lipid extract surfactant (bLES) therapy. The objective of the current study was to determine whether exogenous bLES can mitigate the damage in lung grafts induced by high volume ventilation before procurement. Five control donor dogs were subjected to 8 hours of mechanical ventilation using a tidal volume of 45 ml/kg. This produced a significant decrease in PO_2 values ($p<0.01$) and significant increases in bronchoalveolar lavage (BAL) neutrophil count ($p = 0.05$), BAL protein concentration ($p<0.01$) and the ratio of poorly-functioning small surfactant aggregates (SA) to superior-functioning large aggregates (LA, $p = 0.02$) [see Table 1]. Animals (n=5) given instilled bLES (100 mg/kg) and subsequently ventilated with a tidal volume of 45 ml/kg demonstrated no significant change in PO_2 values over 8 hours and a decrease in BAL protein concentration ($p = 0.04$ versus control) and SA/LA ratio ($p = 0.01$ versus control).

Experimental Group	Time (hours)	PO_2/FiO_2 (mmHg)	BAL Neutrophil Count ($x10^6$/L)	BAL Protein (mg/kg)	BAL SA/LA
Control	0	476 ± 26	43 ± 39	0.42 ± 0.13	0.86 ± 0.1 5
	8	337 ± 27	1387 ± 502	2.54 ± 0.31	2.36 ± 0.41
Instilled bLES	0	512 ± 28	11± 6	0.31 ± 0.08	0.66 ± 0.36
	8	518 ± 23	665 ± 554	1.06 ± 0.53	0.91 ± 0.22

All 10 lung grafts were then flushed with 60 ml/kg modified Euro-Collins solution and stored for 18 hours at 4°C. Left lungs were transplanted into recipient dogs and reperfused for 6 hours. No additional bLES therapy was used. Results after 6 hours of reperfusion, including SA/LA in whole lung lavages from transplanted grafts, are shown in Table 2 (Recipient Animal data):

Experimental Group	PO_2/FiO_2 (mmHg)	PCO_2 (mmHg)	Peak Inspired Pressure (cm H_2O)	Transplanted Lung SA/LA
Control	73 ± 14	47.8 ± 1.4	34 ± 3.3	0.77 ± 0.17
Instilled bLES	307 ± 63	38.2 ± 4.1	24 ± 2.1	0.17 ± 0.04
p value	0.007	0.058	0.03	0.009

We conclude that instillation of exogenous bLES prior to 8 hours of high volume ventilation decreased protein leak, decreased surfactant SA/LA ratio and prevented the subsequent deterioration of PO_2 values in donor animals. Moreover, when these lungs were transplanted into recipients, bLES-treated grafts had superior PO_2 values, improved ventilation efficiency and a higher proportion of superior-functioning surfactant aggregate forms in the alveolar space than control grafts. bLES therapy can thus protect lung grafts from ventilation-induced injury and may offer a promising means to expand the donor pool.

*By invitation

F16. EXOGENOUS SURFACTANT TREATMENT BEFORE AND AFTER 16-HOUR ISCHEMIA IN EXPERIMENTAL LUNG TRANSPLANTATION.

Bernard Hausen, M.D.*, Wolfgang Bernhard*, Charles Hewitt, M.D., Ph.D.*, Frank Schroder*, Maike Beuke* and Hans-Joachim Schafers, M.D.*

Hannover, Germany and Camden, New Jersey

Sponsored by: Hans-George Borst, M.D., Hannover, Germany

Severe alterations in surfactant content of lung grafts occur following extended ischemia. A syngeneic, acute, in situ transplant model in the rat was used to determine the impact of exogenous surfactant treatment. Double lung blocs were flush perfused and preserved for 16 hours and then reperfused for 120 minutes. Group I received intratracheal surfactant (200 mg/kg; Curosurf) before perfusion and donor harvesting (n=6), group II after ischemia and before reperfusion (n=6). Untreated lungs served as controls (group III). Serial measurements of graft pulmonary vascular resistance (PVR), alveolar arterial oxygen difference ($AADO_2$), compliance and resistance were obtained. Final graft assessment included weight gain and histological analysis. Data is listed as mean ± standard error (*$p<0.05$ by ANOVA).
The mean survival after reperfusion in group I was 120 min. versus 113 ± 3 in group II and 117 ± 3 in group III. The weight increase was 12 ± 4% in group I, 105 ± 15% in group II and 87 ± 17% in group III.

	20 min. of reperfusion			120 min. of reperfusion			
Group	I	II	III Controls	I	II	III Controls	
$AAD0_2$	85 ± 16*	108 ± 18	147 ± 36	244 ± 60'	403 ± 99	487 ± 56	mmHg
PVR	104 ± 18*	411 ± 102	161 ± 46	72 ± 11*	411 ± 130	349 ± 59	mmHg/ml/min
Compliance	56 ± 5*	32 ± 4	42 ± 4	52 ± 3'	28 ± 6	28 ± 4	ml/cmH_2O
Resistance	307 ± 17	695 ± 95	435 ± 45	287 ± 10*	720 ± 130	672 ± 120	cmH_2O/1/sec

There was no significant difference in the histological analysis regarding interstitial and intra-alveolar edema or pulmonary hemorrhage.
Graft pretreatment before perfusion resulted in significantly improved oxygenation and compliance as well as decreased vascular resistance when compared to controls or treatment before reperfusion. It is therefore concluded that donor surfactant pretreatment is advantageous for preservation of overall graft function after 16 hours of ischemia.
*By invitation

F17. BOTH BLOOD AND CRYSTALLOID BASED EXTRACELLULAR SOLUTIONS ARE SUPERIOR TO INTRACELLULAR SOLUTIONS IN LUNG PRESERVATION.

Oliver A.R. Binns, M.D.*, Nuno F. DeLima, M.D.*, Scott A. Buchanan, M.D.*, Jeff T. Cope, M.D.*, Robert C. King, M.D.*, Chris A. Marek, B.S.*, Curtis G. Tribble, M.D. and Irving L. Kron, M.D.

Charlottesville, Virginia

Lung transplantation remains limited by donor ischemic time, inadequate graft preservation, and reperfusion injury. We evaluated the effects of an extracellular preservation solution, with or without the addition of blood, as compared to the standard intracellular solution Euro-Collins. Using an isolated, whole blood perfused/ventilated rabbit lung model, we studied three groups of animals. Lungs were flushed with either Euro-Collins (EC), low-potassium-dextran (LPD), or a 20% blood/low-potassium-dextran solution (BLPD). All lungs were harvested en bloc, stored inflated at 4°C for 18 hrs, and then reperfused at 60 ml/min with whole blood. Continuous measurements of pulmonary artery pressure (PAP), pulmonary vascular resistance (PVR), left atrial pressure, dynamic airway compliance (CPL), and weight gain were obtained. Fresh, non-recirculated venous blood was used to determine the single pass pulmonary venous-arterial O_2gradient (V-A O_2). All data are reported as means ± SEM after 30 minutes of reperfusion and analyzed by ANOVA.

Group	**PAP** (mmHg)	**PVR** (Dynes.sec.cm-5)	**% change CPL**	**Wet/Dry Ratio**	**V-A O_2** (mmHg)
EC (n=8)	40.8 ± 2.2*	46.0 ± 3.1*	-21.9 ± 4.7*	7.4 ± 0.3*	37.2 ± 4.6*
LPD (n=8)	28.9 ± 2.4	29.0 ± 4.2	1.8 ± 3.3	5.6 ± 0.1	296.3 ± 54.6
BLPD(n=7)	28.3 ± 1.5	28.8± 2.3	1.4 ± 6.2	5.7 ± 0.3	290.2 ± 66.4
ANOVA results:	*p=0.0003 vs. LPD & BLPD	*p=0.0005 vs. LPD & BLPD	*p=0.002 vs. LPD & BLPD	*p=0.0001 vs. LPD & BLPD	*p=0.001 vs. LPD & BLPD

We conclude that extracellular solutions provide superior preservation of pulmonary function as demonstrated by increased oxygenation, decreased pulmonary artery pressure, decreased pulmonary vascular resistance, improved airway compliance, and decreased edema formation as measured by wet-to-dry ratios. However, the addition of blood does not confer any demonstrable advantage over LPD alone in this model of 18 hour cold ischemia. A potential mechanism of injury by intracellular solutions may involve endothelial damage of the pulmonary vasculature.
*By invitation

F18. NICORANDIL, K^+ CHANNEL OPENER, AMELIORATES LUNG REPERFUSION INJURY.

Motohiro Yamashita, M.D.*, Ralph A. Schmid, M.D.*, Shozo Fujino, M.D.*, Koei Ando, M.D.*, Joel D. Cooper, M.D. and G. Alexander Patterson, M.D.

St. Louis, Missouri

Adenosine triphosphate-sensitive K^+ (K_{ATP}) channels are a class of ionic channels recently found important in ischemic injury. Nicorandil (Nic) acts as a K_{ATP}channel opener. Nic also acts as a nitric oxide donor and through that mechanism may reduce lung allograft reperfusion injury. In this study, we examined the effect of Nic on post-transplant function of preserved lung allografts. Donor lungs were flushed with modified Euro-Collins solution and stored for 21 hours at 1°C. Immediately following transplantation, the contralateral right main pulmonary artery and bronchus were ligated to assess isolated allograft function. Hemodynamics and arterial blood gas analysis (FiO_2 1.0) were assessed for 6 hours prior to sacrifice. Allograft myeloperoxidase (MPO) activity was assessed as an index of leukocyte sequestration. Group I (n=5) animals received no Nic. In group II (n=5), Nic (24 mg/L) was added to the flush solution, recipient animals received Nic (0.5 mg/kg, IV) just prior

to reperfusion and a continous infusion of Nic (0.74 ± 0.03 mg/kg/hr) during the assessment period. In group III (n=4), Nic was administered as in group II. In addition, group III animals received glibenclamide, a potent K_{ATP} channel antagonist (3 mg/kg) 15 minutes before Nic administration. Superior gas exchange (Fig. 1), hemodynamics and MPO data (Table 1) were noted in group II. The improvement of gas exchange and hemodynamics was suppressed by glibenclamide. These findings suggest Nic administration in the flush solution and during the reperfusion period ameliorates allograft function, improves cardiac output, and reduces pulmonary vascular resistance (PVR) and MPO activity in the transplanted lung. Lung allograft reperfusion injury is reduced by Nic likely as a result of its effect on K_{ATP} channels.

Table 1

	C.O. l/min	PAP mmHg	PVR dynes.sec.m2/cm5	MPO Î”OD/mg/min
Group I	1.44 ± 0.17*	27 ± 2	1000 ± 80*	0.40 ± 0.01*
Group II	2.51 ± 0.17	28 ± 4	620 ± 120	0.30 ± 0.03
Group III	1.34 ± 0.17*	26 ± 5	1200 ± 130*	0.38 ± 0.05

C.O.: Cardiac output; PAP: Pulmonary artery pressure; (mean ± SE)

*p<0.05 (ANOVA) vs Group II

*By invitation

7:00 a.m. FORUM SESSION III - CARDIAC SURGERY

Room 6A/B, San Diego Convention Center

Moderators: D. Craig Miller, M.D.

Randall B. Griepp, M.D.

F19. MECHANISMS UNDERLYING DEGENERATION OF CRYOPRESERVED HOMOGRAFTS.

JosÃ© P. Neves, M.D.*, SÃ©rgio Gulbenkian, MSc., Ph.D.*, Ana P. Martins, M.D.*, AntÃ³nio M. Ferreira, Pharm.D., Ph.D.,* Ramiro Mascarenhas, Vet.D., Ph.D.*, Ricardo N. Santos, MSc.* and JoÃ£o Q. Melo, M.D, Ph.D.*

Lisbon and Santarem, Portugal

Sponsored by: Manuel E.M. Macedo, Lisbon, Portugal

Recent studies comparing heart valve (HV) homografts and valves of transplanted hearts showed that while the latter contained fibroblasts of both donor and recipient origin, the former were mostly acellular. These differences could be either due to the occurrence of an immune response in HV recipients that was prevented or abrogated by the immunosuppressive therapy administered to transplanted patients but not to the HV recipients, or to the cryopreservation process to which the HV homografts were subjected. To distinguish between these two alternatives, an experimental model was designed in which the behavior of cryopreserved autografts (CA) and homo-grafts (CH), implanted in the same animal, were compared. Fresh autografts (FA) were used to analyze the role of denervation and devascularization.

Cryopreserved aortic conduit homografts were implanted in the descending thoracic aorta of 15 sheep (6 males), aged 2 to 18 months. The excised aortic segment was then subjected to the same cryopreservation process used for the treatment of the homograft. One to eight weeks later, the CA was implanted, 1 to 2 cm below the CH. The intermediate segment of the native aorta was, at this point, dissected to be used as an FA control. Animals were sacrificed at different intervals (2 weeks, 1, 3, 6, 12, and 24 months) and the implanted segments harvested together with a portion of native aorta. Histological and immunohistochemical analysis as well as cell viability assessment were then performed on each of the explanted segments. Similar studies were also conducted on fragments of CA and CH collected before implantation.

With the exception of a partial loss of the endothelial cells, cryopreserved specimens had preserved cell viability and histology prior to implantation. Explanted CH, however, showed profound histological changes that affected all strata, as well as a decline in cell viability. Thus, after an initial period of non-specific inflammatory reaction which in most cases subsided after one month, progressive neuronal and smooth muscle degeneration was observed, which led, in later stages, to the disappearance of axons and Schwann cells, fibrosis, hyalinization and calcification. Most likely due to this process, one CH ruptured after 17 months. Lymphocyte infiltrates were found up to 12 months after implantation. Endothelial cells were absent in all cases. In contrast, re-endothelization occurred in CA. After an initial inflammatory reaction as in all other segments, CA showed immunohistochemical signs of nerve degeneration with loss of Schwann cells and axons. After 1 month, however, progressive re-innervation occurred with re-establishment of the normal nervous tissue pattern being achieved 6 months after surgery. Histologically, a single alteration was present in these explants, consisting of an intimal thickening. Cell viability was similar to that of native aorta. Histological and immunohistochemical findings with regard to the FA were similar to those of the cryopreserved autografts, with the exception of the thickening of the intima, which did not occur.

In conclusion, it appears that the immunological reaction rather than the cryopreservation process is responsible for the degeneration that occurs in CH. Of particular interest were the findings that re-innervation, re-endothelization and regeneration of vasa vasorum occurred both on CA and FA. These conclusions are also important to the knowledge of the long-term behavior of aortic root replacement with CH or FA (Ross operation) in patients.

*By invitation

F20. BOTH PAPILLARY TIPS KEEP CONSTANT DISTANCE FROM THE MITRAL ANNULAR PLANE UNDER VARIOUS CONDITIONS.

Masashi Komeda, M.D., Ph.D.*, Julie R. Glasson, M.D.*, Ann F. Bolger, M.D.*, George T. Daughters, M.S.*, Neil B. Ingels, Ph.D.* and D. Craig Miller, M.D.

Stanford and Palo Alto, California

Mitral valve homografts are drawing more attention because they may preserve normal mechanics of the mitral subvalvular apparatus and improve postoperative LV performance, similar to reparative valve surgery. The dynamic nature of the LV, however, complicates precise preoperative and intraoperative assessment of LV geometry essential for homograft placement or complex valve repairs. To study various effects on 3-D mitral geometry, we investigated eight closed-chest dogs using implanted radiopaque markers under 4 conditions: **1) Baseline:** automatic blockade (esmolol at 50-100 ug/kg/min), **2) Caval Occlusion:** reduced preload (EDV fell from 143 ± 16 to 104 ± 13 ml [$p<0.001$], **3)Tachycardia:** atrial pacing (heart rate increased 108 ± 11 to 131 ± 5 min^{-1} [$p<0.001$], and **4)Nitroprusside:** decreased afterload (2-5 Î¼g/kg/min) (maximum LV pressure decreased from 132 ± 23 to 108 ± 29 mmHg [$p<0.001$]. Using cylindrical coordinates with the origin at the midpoint of the line connecting the anterior and posterior commissures and the LV long axis (z-axis) defined by the origin and the LV apex, D_{TIP-MA}(the distance along the z-axis

between the papillary muscle [PM] tip and mitral annular plane) was measured at end-diastole and end-systole (mm, mean ± 1SD; n=8 for posterior PM, and n=7 for anterior PM):

(D_{TIP-MA})		Baseline	Caval Occlusion	Nitroprusside	Tachycardia
Posterior PM:	End-Diastole	25.8 ± 4.8	25.1 ± 5.2	25.6 ± 4.8	25.1 ± 4.7
	End-Systole	25.5 ± 4.5	25.5 ± 4.5	25.5 ± 4.4	25.0 ± 4.5
Anterior PM:	End-Diastole	20.7 ± 2.7	21.1 ± 2.6	20.9 ± 2.6	20.5 ± 2.5
	End-Systole	20.8 ± 2.8	20.7 ± 2.8	20.8 ± 2.8	20.4 ± 2.7

There were no significant differences in any dimensions by ANOVA. The distance between each PM tip and mitral annular plane was constant regardless of time during the cardiac cycle, or changes in preload, afterload, and heart rate. The mechanisms of maintaining this fixed tip-annulus distance are not known; however, these findings raise the possibility that the PM tip-annular distance might be a useful parameter to determine mitral homograft chordal length or help create more precise intraoperative strategies for complex valve repairs. Further investigations in dilated LV models with MR and the clinical setting are obviously necessary to define mechanisms and confirm these observations.
*By invitation

F21. PORT-ACCESS MITRAL VALVE REPLACEMENT IN DOGS.

Mario F. Pompili, M.D.*, John H. Stevens, M.D.*, Thomas A. Burdon, M.D.*,

William S. Peters, M.B., Ch.B.*, Lawrence C. Siegel, M.D.*, Greg H. Ribakove,

M.D.* and Bruce A. Reitz, M.D.

Palo Alto and Stanford, California; New York, New York

Introduction: Minimally invasive techniques have been elusive in cardiac surgery. We describe a method of MVR using an endovascular CPB system and one 35 mm by 17 mm oval port and two 10 mm lateral thoracic ports in dogs.

Methods: Fifteen dogs, 28 ± 3 kg (mean ± SD), were studied using the port-access MVR system (Heartport, Redwood City). Eleven dogs underwent acute studies and were euthanized immediately following the procedure. Four dogs were recovered and euthanized 4 weeks after surgery. CPB was conducted via femoral cannulae using an endovascular balloon catheter for aortic occlusion, root venting and delivery of antegrade cardioplegia. Catheters were inserted in the jugular veins for pulmonary artery venting and retrograde cardioplegia delivery. Through the oval port, a prosthesis (St. Jude or Carbomedics) was inserted via the left atrial appendage and secured to the annulus with 8 to 12 sutures. De-airing was performed.

Results: All animals were weaned from CPB in sinus rhythm. There was no MR by left ventriculography or PAOP v-wave in all but 2 dogs. In these 2 dogs, there was interference with prosthetic valve closure by residual native anterior leaflet tissue. Pathologic examination otherwise showed normal healing without peri-valvular discontinuity. Microscopic and SEM studies showed no damage to the valve surfaces. Cardiac output and PAOP were unchanged (2.8 ± 0.7 1/min and 7 ± 3 mmHg preop vs. 2.6±0.6 and 9 ± 4 postop). CPB duration was 113 ± 23 minutes and aortic clamp duration was 71 ± 15 minutes. Transthoracic echo of the 4 chronic dogs showed normal ventricular function and prosthetic valve function four weeks postoperatively.

Discussion: Mitral valve replacement with a minimally invasive method has been demonstrated in dogs. A clinical trial is appropriate.

*By invitation

F22. THE INDUCTION OF TOLERANCE TO AN EXPERIMENTAL CARDIAC ALLOGRAFT REQUIRES INTRATHYMIC INOCULATION OF CLASS IIMHC DISPARATE ANTIGENS.

Zhenya Shen, M.D.*, Muhammad Mohiuddin, M.D.*, Hitoshi Yokoyama, M.D., Ph.D.*, G. Russell Reiss, M.D.* and Verdi J. DiSesa, M.D.

Philadelphia, Pennsylvania

Indefinite donor-specific tolerance to a cardiac allograft disparate in both Class I and Class II major histocompatibility (MHC) antigens has been achieved in our laboratory and others by the pre-transplant intrathymic (IT) injection of donor spleen cells and a single intraperitoneal (IP) injection of anti-lymphocyte serum (ALS). This study was designed to determine whether this phenomenon was reproducible with either Class I MHC only or Class II MHC only disparate grafts. Three strains of inbred rats were studied in these experiments. Donors of cells and hearts in all experiments were RP rats which are rat MHC RT1 (AuBlDl). Class I MHC disparate grafts were performed by placing an RP heart into a Lewis recipient (RT1 A1B1D1C1) and Class II disparate grafts were performed with RP donors and Wistar Furth (WF) recipients (RT1 AuBuDuCu). Lewis (n=10) and WF (n=10) recipients underwent intra-peritoneal injection of 1 ml ALS and intrathymic injection of $5x10^7$ RP spleen cells. Three weeks later heterotopic cardiac transplantation was done using a heart from an RP rat. Control rats had no pretreatment or ALS alone. Without any pretreatment, RP hearts survive 7-9 (mean 8) days in Lewis recipients (n=5) and 9-14 (mean 12) days in WF recipients (n=5). ALS alone produces slight prolongation of graft survival (12 days in Lewis recipients [n=5] and 14 days in WF recipients [n=5]). Lewis rats pretreated with Class I disparate RP splenocytes and ALS had graft survivals of 8 - 27 (mean 14) days not significantly different from the effect of ALS alone. Class II disparate RP grafts placed in pretreated WF rats had significant prolongation of graft survival with 4 out of 5 grafts surviving more than 60 days ($p<0.01$ vs ALS alone). These results suggest that a disparity at the Class II locus of the MHC is critical for the induction of cardiac allograft tolerance after by intrathymic inoculation of allogeneic cells. This implies that the specific requirements for antigen presentation in the thymus are quite stringent even in this rodent model.

*By invitation

F23. INCREASED GRAFT AND SYSTEMIC VASCULAR PERMEABILITY DURING EARLY CARDIAC ALLOGRAFT REJECTION IS MEDIATED BY GRAFT AND SYSTEMIC EXPRESSION OF INDUCIBLE NITRIC OXIDE SYNTHASE.

Neil K. Worrall, M.D.*, Kathy Chang, Ph.D.*, Patrick M. Sullivan, B.A.*, Thomas P. Misko, Ph.D.*, Jia-Ji Hui, M.D.*, Joseph R. Williamson, M.D.* and T. Bruce Ferguson, Jr., M.D.

St. Louis, Missouri

We recently demonstrated that inducible nitric oxide synthase (iNOS) expression results in increased nitric oxide (NO) production during cardiac allograft rejection. In contrast to the

physiologically protective role of NO in decreasing leukocyte adherence to endothelium, NO has also been implicated in mediating increased vascular permeability caused by various pathophysiological mediators, including LPS, TNF, and histamine. The present study examined whether NO contributes to increased vascular permeability to macromolecules during the early stages of graft rejection. Given the clinical systemic sequelae of rejection, we also examined whether early allograft rejection was associated with increased systemic vascular permeability. A double tracer permeation and microsphere method was used to examine vascular permeation (VP) during early graft rejection (POD 4) in a rat heterotopic cardiac transplant model: at time 0, ^{125}I-albumin was injected iv to measure VP; 8 min. later ^{131}I-albumin was injected iv (intravascular space marker); and 1 min. later ^{46}Sc-labeled microspheres were injected iv (blood flow). One min. later the tissues were excised for Î³-spectrometry, weighed, and 1) ^{125}I-albumin VP (intravascular tracer corrected), 2) blood flow, and 3) water content (wet/dry weight ratio) determined for each tissue. Allografts (Lewis to ACI) had increased VP and wet/dry weights in the grafted heart, lung, and brain compared to isografts (ACI to ACI) and controls (SEE TABLE). Increased allograft VP was associated with increased NO production (serum nitrite/nitrate levels) and with iNOS mRNA expression (determined by ribonuclease protection assay) in the grafted heart and the lung (not examined in brain; not detectable in the control or isograft tissues). iNOS inhibition with aminoguanidine (AG; 375 mg/kg/d iv) prevented the increased graft and systemic VP and water content, and normalized the serum nitrite/nitrate levels. Blood flow, cardiac output, and BP were not different between groups and were not affected by AG (not shown). AG had no effect on: 1) mild histological rejection score in allografts (1.7 ± 0.4 vs 1.6 ± 0.3; 0-5 scale); and 2) VP in isografts and controls (data not shown). These data demonstrate the novel observations that: 1) early allograft rejection increases vascular permeability and tissue water content in the systemic vasculature; 2) increased allograft heart and systemic vascular permeability is associated with increased NO production and iNOS mRNA expression in the allograft heart and lung; and 3) inhibition of NO production by iNOS prevents allograft heart and systemic vascular barrier dysfunction during early rejection.

Group	Grafted Heart		Lung		Brain		Nitrite/Nitrate
	VP	W/D Wt	VP	W/D Wt	VP	W/D Wt	(Î¼M)
Con (n=15)	NA	NA	1324 ± 443	4.5 ± 0.2	52 ± 15	5.0 ± 0.1	14.6 ± 2.4
Iso (n=10)	1317 ± 451	4.6 ± 0.1	1524 ± 460	5.0 ± 0.1	108 ± 40†	4.9 ± 0.1	21.6 ± 4.1
Allo(n=10)	2534 ± 409*	4.9 ± 0.1*	2633 ± 371*	5.5 ± 0.2*	213 ± 37*	5.2 ± 0.1*	37.7 ± 15.8*
Allo+AG (n=9)	1687 ± 110	4.7 ± 0.1	1692 ± 482	5.1 ± 0.1	96 ± 20	4.9 ± 0.1	20 ± 4.3

[VP = Î¼g plasma*g tissue^{-1}*min^{-1}; Mean±SD; (*)=P<0.005 vs. Con, Iso, AG; (†) = P<0.005 vs. Con by ANOVA]

*By invitation

F24. BASAL NITRIC OXIDE EXPRESSES ENDOGENOUS CARDIOPROTECTION DURING REPERFUSION BY INHIBITION OF NEUTROPHIL-MEDIATED DAMAGE AFTER SURGICAL REVASCULARIZATION.

Hiroki Sato, M.D.*, Zhi-Qing Zhao, M.D, Ph.D.*, James E. Jordan, B.S.*, James C. Todd, B.S.*, Ping Li, Ph.D.*, John W. Mammon, Jr., M.D. and Jakob Vinten-Johansen, Ph.D.*

Winston-Salem, North Carolina

Ischemia-reperfusion damages endothelium and impairs basal production of nitric oxide (NO). Basally released NO is cardioprotective by inhibiting neutrophil activities. Loss of endogenous NO with endothelial injury may occur during two phases: cardioplegic ischemia or reperfusion (aortic declamping). This study tested the hypothesis that inhibition of endogenously released NO in hearts subjected to regional ischemia, cardioplegic arrest and reperfusion: 1) restricts endogenous cardioprotection and permits neutrophil (PMN)-mediated damage, and 2) expresses damage primarily during the reperfusion phase. L-nitro-arginine (L-NA) was used to block basal NO production. In 22 anesthetized dogs, the LAD was ligated for 90 min followed by 1 hr arrest with cold multidose (q. 20 min) blood cardioplegia (BCP). Dogs were divided into 3 groups: SBCP, n=8: standard blood cardioplegia (BCP); LNA-BR, n=7: L-NA was administered during both phases of potential injury, as additive to BCP (1 mM) and infusion during reperfusion (34 mg/kg); LNA-R, n=7: L-NA was administered only at reperfusion. The LAD ligature was released during the second infusion of cardioplegia. Infarct size (TTC stain) was increased in LNA-R compared to SBCP (49 ± 6%* vs 34 ± 2%), but was not further extended in LNA-BR (56 ± 3%), suggesting primarily a reperfusion process. PMN-specific myeloperoxidase in the area at risk was elevated comparably in LNA-BR and LNA-R (2.9 ±0.5*, 3.9 ±1.0* U/g tissue) vs SBCP (1.7 ± 0.3), suggesting PMN accumulation during reperfusion. PMN adherence in experimental ischemic-reperfused LAD segments was comparably greater in LNA-BR (195 ±21* PMN/mm^2 LAD) and LNA-R (224 ± 20*) relative to SBCP (108 ± 19). There was no significant adherence to nonischemic circumflex arteries. We conclude that blockade of endogenous NO augments postischemic injury mediated by PMN, and this damage is expressed primarily during the reperfusion phase. These data imply that: 1) basal NO participates in endogenous cardioprotection during the reperfusion phase of surgical revascularization; 2) loss of NO production secondary to endothelial injury may be detrimental; and 3) augmentation of basal NO by L-arginine precursor may increase protection. *$p<0.05$ vs. SBCP.

*By invitation

F25. MATURATION ALTERS THE PULMONARY ARTERIAL RESPONSE TO HYPOXIA AND INHALED NITRIC OXIDE IN THE PRESENCE OF ENDOTHELIAL DYSFUNCTION.

Jeff L. Myers, M.D.*, Joseph J. Wizorek, B.S.*, Adam K. Myers, Ph.D.*, Michael O'Donoghue, M.S.*, Peter C. Kouretas, M.D.*, Heidi J. Dalton, M.D.*, Yi-Ning Wang, M.D.* and Richard A. Hopkins, M.D.

Washington, DC

These studies test the hypothesis that maturational changes in the newborn period alter the response to hypoxia and subsequent NO inhalation in a model of endothelial dysfunction. Eight 48-hour-old piglets and eight 14-day-old piglets underwent high fidelity instrumentation to record pulmonary artery pressure (PA), flow (PAF), and main pulmonary artery radius (Ro). Following treatment with the NO-synthase inhibitor L-NA (20 mg/kg, IV bolus), hemodynamic measurements were performed at baseline, during 10 minutes of hypoxia (F_iO_2=0.1) and during 10 minutes of NO inhalation (100 ppm). Fourier analysis of the waveforms produced values for input mean impedance (Zm) and characteristic impedance (Zo), reflecting opposition to flow in the distal arteriolar bed and larger proximal pulmonary arteries respectively. Vessel wall elasticity (Ey) was calculated from Zo and Ro.

	PAP	PAF	Zm§	Zo§	Ey	Ro
48-hr baseline	17.4 ± 1.5	5.8 ± 0.9	4826 ± 272	1171 ± 76	$1.0E^{07}$ ± LIE^{07}	3.82 ± 0.21
hypoxia	30.6 ± 1.5*	5.3 ± 0.7	8744 ± 488**	1337 ± 74	$1.48E^{07}$ ± $3.7E^{06}$	3.97 ± 0.13
nitric oxide	17.4 ± 1.5**	5.2 ± 0.9	4825 ± 213**	1146 ± 70	$1.14E^{07}$ ± $5.8E^{06}$	391 ± 0.14
14-day baseline	12.6 ± 0.7	7.0 ± 0.5	3129 ± 73	419 ± 15	$1.92E^{06}$ ± $32E^{"5}$	452 ± 0.21
hypoxia	28.0 ±0.9**	6.6 ± 0.5	6000 ± 134**	797 ± 20**	1 $05E^{07}$ ± $3.9E^{05}$**	4.94 ± 0.20**
nitric oxide	16 5 ± 1.0**	7.3 ± 0.6*	2449 ± 54**	375 ± 13**	$2.2E^{06}$ ± $3.1E^{05}$**	4.89 ± 0.20
*p<0.05 and *p<0.01 v. preceding intervention §dyne*cm*sec^{-5}						

Both groups underwent severe distal arteriolar vasoconstriction with hypoxia evidenced by the large increases in PAP and Zm. NO relieved this effect. The younger animals underwent no alteration of Zo, Ey, or Ro, indicating the larger proximal vessels were unaffected by either hypoxia or nitric oxide. In contrast, the radius actually increased in 14-day-old animals under the higher transmural pressure (increased PAP), but the vessel wall became stiffer by a factor of almost 10 (increased Ey). The net effect was an increase in Zo that represents a significant increase in the opposition to flow from the right ventricle in animals with a dysfunctional endothelium. In conclusion, NO synthase inhibition, as might be seen in endothelial dysfunction, results in profound vasoconstriction at the distal arteriolar level in both age groups. The lower resting tone in the older piglets (lower baseline Zo and Ey) allows a proximal hypoxic vasoconstrictor effect that is not evident in the younger animals. Also, in the face of endothelial dysfunction, nitric oxide is still effective in relieving hypoxic pulmonary arterial vasoconstriction in both age groups.
*By invitation

F26. INTRAOPERATIVE IDENTIFICATION OF SPINAL CORD BLOOD SUPPLY DURING DESCENDING AND THORACOABDOMINAL AORTIC REPAIRS.

Lars G. Svensson, M.D., Ph.D.*

Burlington, Massachusetts

Sponsored by: David M. Shahian, M.D., Burlington, Massachusetts

Previous animal and human studies have shown that the intercostal and lumbar segmental arteries are critical for maintaining adequate spinal cord (SC) perfusion and that failure to reattach segmental arteries may result in postoperative paraparesis or paraplegia. Porcine experimental studies have shown that the vessels supplying the SC can be accurately identified by using hydrogen and a platinum electrode alongside the SC.
METHODS: After obtaining institutional review board approval, informed consent was obtained from 14 patients. Under local anesthetic, a specially constructed catheter with a platinum electrode was placed intrathecally by lumbar puncture alongside the SC and control radiographs obtained to check the positioning of the catheter. Intraoperatively, after crossclamping the aorta, hydrogen in a saline solution was injected into the occluded segment of the aorta and if it was shown that the segment supplied the SC with blood, then in addition, those identified intercostal arteries were injected with hydrogen as necessary. Five patients had descending aortic repairs, three had Type I thoracoabdominal aneurysm repairs, and six had Type II repairs. Postopera-tively, patients underwent highly selective angiography of the reattached intercostal arteries to identify the radicular arteries supplying the SC.
RESULTS: The average aortic crossclamp time was 46.6 minutes (range 20-85 minutes) with an average time for testing of 4.46 minutes (range 0-15 minutes). Postoperative angiography revealed that the radicular arteries supplying the SC had been accurately identified. Five SC perfusion patterns were noted: 1) direct; 2) collateral; 3) no direct supply from segment tested; 4) from atrio-femoral bypass; and 5) occluded reattached intercostals. One patient had permanent paraparesis

(1/14, 7%) following intraoperative cardiopulmonary resuscitation for 15 minutes and two patients had transient paraparesis. One patient died postoperatively.
CONCLUSION: The intraoperative use of hydrogen and an intrathecal platinum electrode identifying vessels supplying the spinal cord is safe and accurate, and the additional aortic crossclamp time required is minimal.
*By invitation

F27. THE SUPERIORITY OF HYPOCALCEMIC VS NORMOCALCEMIC BLOOD CARDOPLEGIA IN NEONATAL MYOCARDIAL PROTECTION.

Kirk Bolling, M.D., MPH*, Michael Kronen, M.D.*, Bradley S. Allen, M.D.*, Shaikh Rahman, M.S.*, Tingrong Wang, M.D.*, Renee S. Hartz, M.D. and Harold Feinberg, Ph.D.*

Chicago, Illinois

In newborns, the ideal cardioplegic calcium (Ca^{2+}) concentration continues to be debated. However, most studies examining cardioplegia calcium concentrations were done using a non-clinical model (isolated heart preparation), which (1) may not be clinically applicable and (2) did not examine the effect of calcium concentration in a clinically relevant "stressed" (hypoxic) heart. We therefore attempted to determine the ideal calcium concentration using an in vivo (clinical) model in non hypoxic (uninjured) and "stressed" (hypoxic) hearts.

Twenty neonatal piglets, 5-18 days old, were placed on cardiopulmonary bypass, and their aorta cross clamped for 70 min with either hypocalcemia or normocalcemic multidose blood cardioplegic infusions. Group 1: (n=5, low Ca^{2+}, 0.2-0.3 mM), and Group 2: (n=5, normal Ca^{2+}, 1.0-1.3 mM) were nonhypoxic (uninjured) hearts. Ten other piglets were first ventilated at an FiO_2 of 8-10% (O_2 saturation 65-70%) for 60 minutes (hypoxia) and then reoxygenated at an FiO_2 of 100% utilizing cardiopulmonary bypass. This has been shown to produce a clinically relevant "stress" injury resulting in myocardial depression. They underwent cardioplegic arrest (as above) with either a hypocalcemic (n=5, Group 3) or normocalcemic (n=5, Group 4) blood cardioplegic solution. Myocardial function was assessed using pressure volume loops, and expressed as a percentage of control. Coronary vascular resistance was measured during each cardioplegic infusion. In nonhypoxic hearts (Group 1 and 2), good myocardial protection was achieved at either concentration of cardioplegia calcium as demonstrated by preservation of postbypass systolic function (EES 104% vs 99%), diastolic compliance (152% vs 162%), no increase in myocardial edema (78.9% vs 78.7%), and no change in ATP levels or coronary vascular resistance. Low calcium blood cardioplegia solution repaired the hypoxic/reoxygenation injury in "stressed" hearts (Group 3) resulting in no statistical difference in myocardial function, coronary vascular resistance or ATP levels compared to nonhypoxic hearts (Group 1 and 2). Conversely, when a normocalcemic cardioplegia solution was used in "stressed" (hypoxic) hearts (Group 4), there was marked reduction in postbypass systolic function (EES 49% ± 4%)* loss of diastolic compliance (276% ± 9%)*, increase in myocardial edema (79.7% ± 0.2%)*, rise in coronary vascular resistance*, and no change in ATP levels compared to Groups 1, 2 and 3.

In conclusion, this study demonstrates that: 1) in the clinically relevant, intact, animal model, good myocardial protection is independent of cardioplegia calcium concentration in nonhypoxic (noninjured) hearts; 2) "stressed" (hypoxic) hearts are extremely sensitive to the cardioplegic calcium concentration; and 3) normocalcemic cardioplegia is detrimental to neonatal myocardium subjected to a preoperative hypoxic stress. Optimal myocardial protection in infants undergoing repair of cyanotic congenital defects is therefore provided by a hypocalcemic cardioplegia solution.

*p<0.05 mean ± S.E.

*By invitation

F28. COST AND EFFICACY OF SURGICAL LIGATION VERSUS TRANSCATHETER COIL OCCLUSION OF PATENT DUCTUS ARTERIOSUS.

John A. Hawkins, M.D.*, L. LuAnn Minich, M.D.*, Lloyd Y. Tani, M.D.*, Jane E. Sturtevant, B.S.N.*, Garth S. Orsmond, M.D.* and Edwin C. McGough, M.D.

Salt Lake City, Utah

Transcatheter closure of the patent ductus arteriosus (PDA) using coil occlusion (CO) has emerged as primary therapy for the small PDA because of avoidance of surgery and a presumed lower cost. In July 1994, we began a study to compare surgical closure of PDA using new critical pathway methodology with outpatient transcatheter CO of PDA in non-neonatal patients. Selection for transcatheter CO was based on anatomic feasility and patient preference, while surgical closure was performed in remaining patients. Surgical techniques included a small transaxillary, muscle-sparing thoracotomy, triple ligation of the PDA, no chest tube, placement of a small catheter for intermittent intercostal 0.25% bupivicaine, and discharge within 24 hours using critical pathway methods.
From July 1994 until October 1995, 15 patients underwent transcatheter CO of a PDA and 14 patients underwent surgical closure of the PDA. Duration of hospitalization was significantly less for the CO patients (12 ± 8 hours, X ± SD) as compared to the surgical ligation patients (26 ± 6 hours, $p<0.05$). However, the duration of the procedure was significantly less for surgical ligation (44 ± 6 minutes) as compared to transcatheter CO patients (91 ± 29 min, $p<0.05$). Total costs, including all hospital and professional fees, were slightly less for surgical ligation ($7,035 ± 403) as compared to CO ($7,298 ± 885, $p>0.05$). Morbidity in the transcatheter CO included inability to occlude the PDA in 1 patient (1/15, 6.7%) and residual patency in 2 patients (2/15, 13%) on late color flow Doppler echocardiography. Morbidity in the surgical ligation group included nausea and vomiting requiring hospitalization >36 hours in 1 patient (1/14, 7%) and transient left recurrent laryngeal nerve palsy in 1 patient (1/14, 7%). There were no instances of residual PDA patency in the surgical group by echocardiography with color flow Doppler.
Transaxillary thoracotomy without tube thoracostomy and critical pathway methodology allows safe and effective ligation of the PDA with early hospital discharge. This surgical method has slightly lower overall cost, higher efficacy rate, and applicability in all patients as compared to newer transcatheter CO techniques for occlusion of the PDA. These findings are important for future cost-benefit considerations in the context of managed care.
*By invitation

9:30 a.m. SIMULTANEOUS SCIENTIFIC SESSION D -

ADULT CARDIAC SURGERY
Room 6A/B, San Diego Convention Center
Moderators: Robert B. Wallace, M.D.

Tirone E. David, M.D.

41. CLINICAL SIGNIFICANCE OF PERIOPERATIVE Q WAVE MYOCARDIAL INFARCTION IN THE EMORY ANGIOPLASTY VERSUS SURGERY TRIAL.

George T. Hodakowski, M.D.*, Joseph M. Graver, M.D., Ellis L. Jones, M.D., Spencer B. King, M.D.* and Robert A. Guyton, M.D.

Atlanta, Georgia

Discussant: Bruce W. Lytle, M.D.

The primary end point of the Emory Angioplasty versus Surgery Trial (EAST) was a composite of three events: death, Q-wave infarction (QMI), and a new large defect on three-year postoperative thallium scan. A QMI was identified on predischarge electrocardiograms read by blinded, experienced electrocardiographers. This study examines the clinical significance of QMI in the surgical cohort (194 patients) of EAST. Twenty QMI patients were identified (10.3%) with a large proportion, 13 patients, identified with inferior QMIs. Seven had anterior, lateral, septal, or posterior QMIs (anterior QMIs). In the inferior QMI group, postoperative catheterization (at 1 year or 3 years) in eleven patients revealed normal ejection fraction (EF greater than 55%) in ten (91%), no wall motion abnormalities in ten (91%), and all grafts patent in ten (91%). In the anterior QMI group, postoperative catherization in six patients revealed normal EF in five (83%), no wall motion abnormality in three (50%), and all grafts patent in three (50%). Average peak postoperative CKMB levels (IU) were: no QMI (n=174) 37 ± 43, inferior QMI 43 ±31, anterior QMI 58 ± 38. Mortality in the 20 patients with QMI was 5% (1/20) at three years and was 6.2% (11/174) in patients without QMI. Of 17 QMI patients who had postoperative catheterization, 11 (69%) had a normal ejection fraction, normal wall motion, and all grafts patent with an uneventful three-year postoperative course. The core lab screening of postoperative electrocardiograms, particularly in the case of inferior QMI, appears to identify a number of patients as having a QMI who have minimal and clinically insignificant postoperative electrocardiographic changes. QMI identified in the postoperative period seems to be a weak end point without prognostic importance and therefore not valuable for future randomized trials.

*By invitation

42. CORONARY-CORONARY BYPASS GRAFT:

AN ARTERIAL CONDUIT SPARING PROCEDURE.

Remi Nottin, M.D.*, Jean Michel Grinda, M.D.*, Sami Anidjar, M.D.*, Thierry Folliguet, M.D.* and Marc Detroux, M.D.*

Paris, France

Sponsored by: Claude Planche, Paris, France
Discussant: Noel L. Mills, M.D.

Only a few cases of coronary-coronary bypass grafting (CCBG) have been previously reported. However, CCBG appears as an interesting additive technique for coronary artery bypass grafting. Proximal and distal anastomosis can be achieved either between two segments of the same coronary artery or between two different coronary arteries.
From December 1989 to December 1994, 120 patients underwent myocardial revascularization using one CCBG (117 patients) or two CCBG (3 patients) in addition to pediculed IMA, Y graft and aorto-coronary bypass for a total of 381 distal anastomoses (3.17 ± 0.79 per patient). Presence of a suitable proximal coronary segment was mandatory. Indications for CCBG were: arterial conduit sparing procedure, inadequate length of the graft, calcified ascending aorta, avoidance of aortic implantation of arterial graft, stenosed or occluded subclavian arteries. Graft conduits used

for CCBG were 90 arterial grafts (90/123; 73.2%) including 75 RIMA, 14 LIMA and one radial artery (RA), and 33 saphenous vein (SV) (33/123; 26.8%). CCBG were performed on the right coronary artery (RC) in 113 cases (91.8%), on the circumflex artery (CX) in six (4.8%) and on the left anterior descending coronary artery (LAD) in two (1.6%). In two cases (1.6%), a CCBG was performed between two different coronary arteries, the RC and the LAD.
Two patients (1.6%) died from myocardial infarction during the postoperative period. Early postoperative control angiogram was performed in 63 patients. One month patency rate was 98.5% for 66 CCBG controlled. During the follow-up period of 27 ± 16 months, three patients died and one underwent a reoperation. After two months, 97.5% of tested patients (83/120) had a normal exercise testing. A one year and three year exercise testing was normal in 96.1% and 94.1% of patients, respectively.
In conclusion, CCBG provides good results with a variety of conduits. CCBG could promote an expanded use of arterial grafts, particularly the IMA graft. In addition, CCBG could lead to a sparing of arterial graft material, and allow complex myocardial revascularization with a liberal use of both IMAs.
*By invitation

43. RISK FACTORS FOR STROKE IN PATIENTS UNDERGOING AORTOCORONARY BYPASS SURGERY.

Lynda L. Mickleborough, M.D., Paul M. Walker, M.D.*, Yasushi Takagi, M.D.*, Masanori Ohashi, M.D.*, Joan Ivanov, MSc.* and Miguel Tamariz, BSc.*

Toronto, Ontario, Canada

Discussant: Gary W. Akins, M.D.

To determine predictors of stroke in patients undergoing first time aortocoronary bypass surgery, data were prospectively collected on 1631 consecutive patients. Those with a history of CNS symptoms (119) and/or carotid bruits (185) underwent carotid Doppler evaluation. Patients with symptomatic unilateral disease (>70% stenosis) or significant bilateral disease (77% stenosis) were referred for combined CABG and carotid endarterectomy (21). Postop patients with neurologic symptoms were assessed by a neurologist. Events were classified as reversible (TIA) or (RIND) or irreversible (stroke). Results of sureerv are as follows:

	n	Stroke	Hospital mortality
Total population	1631	(19) 1.2%	(20) 1.2%
Asymptomatic bruit	68	(3) 1.5%	(0) 0%
Total carotid occlusion	22	(6) 27.3%	(0) 0%
Diseased ascending aorta at OR	39	(4) 10.3%	(1) 2.6%

By stepwise logistic regression analysis, three variables were identified as risk factors for stroke. The most important predictor was unilateral carotid occlusion with or without contralateral stenosis (odds ratio = 23.8). In this group, 4/5 strokes occurred on the occluded side. The other two risk factors were presence of ascending aortic disease at time of surgery (OR = 13.1) and previous history of stroke (OR = 4.4).
Conclusion: In patients undergoing CABG, risk factors for perioperative stroke have been identified. Asymptomatic patients with carotid bruits are at low risk. Patients with carotid occlusion are at risk for a stroke on the occluded side.
*By invitation

44. PORT-ACCESS BILATERAL INTERNAL MAMMARY ARTERY GRAFTING FOR LEFT MAIN CORONARY ARTERY DISEASE: CANINE FEASIBILITY STUDY.

Thomas A. Burden, M.D.*, William S. Peters, M.B., Ch.B.*, Mario F. Pompili, M.D.*, John H. Stevens, M.D.*, Lawrence C. Siegel, M.D.*, Frederick G. St. Goar, M.D.* and Bruce A. Reitz, M.D.

Stanford and Palo Alto, California

Discussant: Valavanur A. Subramanian, M.D.

Minimally invasive surgical techniques have been applied to cardiac surgery. We used an endovascular cardiopulmonary bypass system that allows cardioplegia and cardiac venting to extend the applications of minimal access cardiac surgery to bilateral internal mammary artery (IMA) bypass grafting. We report the results of a series of six dogs (22-27 kg). The left IMA was taken down thoracoscopically from three left lateral chest ports, followed by the right IMA from the right side. One left sided port was extended medially 5 cm with or without rib resection, to expose the pericardium. Following heparin administration, both IMAs were divided and exteriorized through the left anterior mediastinotomy. Flow and pedicle length was satisfactory in all cases. Animals were placed on femoral-femoral bypass and the heart arrested with antegrade delivery of cardioplegic solution via the central lumen of a balloon catheter (Endoaortic Clamp; Heartport; Redwood City, CA) inflated in the ascending aorta. All grafts were made through the mediastinotomy under direct vision. In five studies the RIMA was attached to the LAD and the LIMA to the circumflex, and in one study the RIMA was tunneled through the transverse sinus to the circumflex and the LIMA anastomosed to the LAD. All animals were weaned in sinus rhythm without ionotropes. CPB time was 108 ± 27 minutes (mean ± SD) and the mean clamp time was 54 ± 10 minutes. Preoperative and postoperative cardiac outputs were 2.9 ± 0.68 1/min and 2.4 ± 0.30 1/min respectively (p=NS). Preoperative and postoperative pulmonary capillary wedge pressures were 5 ± 1.5 1/min and 5.6 ± 1.6 1/min respectively (p=NS). All 12 grafts were demonstrated to be fully patent by angiography. At postmortem, all target coronary vessels had been correctly grafted, and pedicles were well aligned. We conclude the canine model demonstrates the potential for a less invasive approach to the surgical management of left main coronary artery disease in humans.

10:50 a.m. INTERMISSION

*By invitation

11:10 a.m. SIMULTANEOUS SCIENTIFIC SESSION D -

ADULT CARDIAC SURGERY
Room 6A/B, San Diego Convention Center

Moderators: Robert B. Wallace, M.D.

Tirone E. David, M.D.

45. HUMAN AORTIC VALVE ALLOGRAFTS ELICIT A DONOR-SPECIFIC IMMUNE RESPONSE.

Patrick G. Hogan, BSc., Ph.D., FRACP, FRCPA*, Marjorie Green, Ph.D.*, Lynette Duplock, MSc.*, Susan E. Smith, BSc.*, Ian H. Frazer, M.D., FRCP, FRCPA*, Kenneth L. Gall, BAppSc.* and Marie F. O'Brien, FRCS, FRACS

Brisbane, Queensland, Australia

Discussant: MarkF. Lupinetti, M.D.

Human aortic valve allografts (AVA), cryopreserved within 2-8 hr of collection and containing viable cells, provided the model for a quantitative analysis of the donor-specific immune response in recipients. It is unclear precisely to what extent and in what form AVA antigens survive cryopreservation. Therefore, 10 recipients of AVA were investigated for anti-donor antibody and T cell-mediated responses against the same donor splenic or peripheral blood mononuclear cells collected at the same time as the AVA. Anti-donor immune responses were measured quantitatively in recipients at 7, 30, 90 and 365 days after AVA implantation using flow cytometric and lymphocytotoxic cross-match assays and mixed lymphocyte cultures (MLCs).

By day 30 after surgery, all 10 recipients developed significant litres of IgG antibodies which were directed against both class I and II major histocompati-bility (MHC) antigens present in the donor tissue. In addition, all recipients developed lower levels of multispecific antibodies which cross-reacted with third party non-donor MHC antigens. Anti-donor antibodies persisted for at least 365 days after surgery. In MLCs, most recipients (6 of 7) had significantly increased levels of donor-specific T cell proliferative responses compared with those to third party lymphocytes at days 30 and 90 after implantation.

These data indicate that MHC antigens in AVA survive our current early cryopreservation method and elicit a donor-specific antibody and T cell-mediated response. This alloreactivity is likely to be responsible for the pathological changes of hypocellularity, valve thickening, fibrosis and calcification with stenosis observed in failed AVA, particularly in younger recipients. Therefore, suppression of the anti-donor response in AVA recipients may reduce degeneration of the AVA and thus enhance its performance. Further studies of the immune response to AVA are in progress so that the precise immunological mechanisms responsible for AVA injury can be identified and then targeted in future trials of selective immune suppression. The effectiveness and/or duration of immuno-suppression can be monitored quantitatively with this model, which therefore serves as a unique system for future analyses.

*By invitation

46. WHAT IS THE BEST PERFUSION TEMPERATURE FOR CORONARY REVASCULARIZATION?

Richard M. Engelman, M.D., A. Bernard Fleet, M.D.*, John A. Rousou, M.D., Joseph

E. Flack, III, M.D.*, David W. Deaton, M.D.*, Cheryl A. Gregory, R.N.* and

Penelope S. Pekow, Ph.D.*

Springfield and Amherst, Massachusetts

Discussant: Robert A. Guyton, M.D.

An NIH funded prospective randomized trial was begun in 1994 to study the effect of perfusate temperature on recovery after coronary revascularization. Criteria for entry into the study demanded an ejection fraction (EF) >30%, the performance of at least 3 bypass grafts and age ‰¤ 75 years. This is a review of the first 125 patients to have completed a one-month follow-up. All patients were perfused either cold (C) at 20°C (n=37), tepid (T) at 32°C (n=48) or warm (W) at 37°C (n=39). There were no significant differences between the three groups in any preoperative (81% male, 52% EF) operative (3.7-4.0 grafts/patient, 134-144 min pump time) characteristics and thus, the baseline data were comparable. Antegrade-retrograde blood cardioplegia was used at appropriate temperatures (C=8°C, T=32°C, and W=37°C) for each group. There was no perioperative mortality, and only one of the 124 were reoperated upon for bleeding.

Data collected specific to this report concerned bleeding and coagulation parameters, rapidity of recovery, length of postop hospital stay, neurologic function (at 4 days and 1 month postop) and incidence of postop arrhythmias. Mean ± SEM data are presented:

* significant difference between groups by Kruskal Wallis 1 way ANOVA; ** significant difference by Fisher's Exact Text; [+]difference not significant by Fisher's Exact Text (all 5 CVA patients recovered complete function by 3 mos.); [++]significant difference between C and both T&W by pairwise t-tests.

Conclusion: l)Perfusion temperature is a factor in recovery from CBG; 2) Cold is associated with a longer duration of intubation and hospitalization, less blood loss and the least activation of fibrinolysis. It is not protective against CVA; 3) Warm is associated with the shortest duration of extubation and the most fibrinolysis. It is not associated with the highest stroke rate; and 4) Tepid is the best perfusion temperature, the shortest LOS, no strokes, acceptable levels of fibrinolysis, and the lowest level of blood products transfused. The commonly employed technique of allowing the perfusion temperature to drift is most consistent with tepid perfusion.

*By invitation

47. PRECONDITIONING IN CORONARY ARTERY BYPASS SURGERY: A WORD OF CAUTION.

Philippe MenaschÃ©, M.D., Ph.D., Louis P. Perrault, M.D.*, Alain Bel, M.D.*, Thierry de Chaumaray, M.D.*, Jacqueline Peynet, M.D.*, Adrian Mondry, Ph.D.* and Jean-Marie Moalic, Ph.D.*

Paris, France

Discussant: Steven F. Bolling, M.D.

Objective: Ischemic preconditioning (PC) is now established as an effective means of reducing infarct size. Use of PC before coronary artery bypass grafting (CAPB) under normothermic ventricular fibrillation has led to the preservation of myocardial levels of high-energy phosphates. However, it is not yet established whether PC can improve the myocardial protection afforded by cardioplegia. The present study was designed to address this issue.

Methods: Twenty patients undergoing CABG with the use of retrograde continuous warm blood cardioplegia were studied. After the institution of cardiopulmonary bypass (CPB), 10 patients were preconditioned with 3 minutes of aortic crossclamping followed by 2 minutes of reperfusion before the onset of cardioplegic arrest. Ten case-matched patients served as controls. Blood samples were simultaneously drawn from the radial artery and the coronary sinus before CPB, at the end of the 5-minute PC protocol or after 5 minutes of CPB in control patients and at the end of cardioplegic arrest. These samples were assayed for CK-MB and lactate. Right atrial biopsy specimens were taken at the same time points and processed by Northern blotting for the expression of the mRNAs of *c-fos,* a proto-oncogene which induces changes in cardiac gene expression in response to ischemic stimuli, and of heat shock protein (HSP) 70, a stress-induced cardioprotective protein. The blots were hybridized with probes specific for *c-fos* and HSP 70 mRNAs and for 18S ribosomal RNA. The quantification of *c-fos* and HSP 70 mRNA was calculated as the ratio between each of these two signals and that yielded by 18S RNA. Results are presented as the percentage of the signal given by rat cardiac RNA used as an internal standard (% I.S.).

Results: There were no differences in pre- or intraoperative variables between the two groups. At the end of arrest (72 ± 6 minutes and 64 ± 5 minutes in control and preconditioned patients,

respectively [mean ± SEM]), the release of CK-MB from the myocardium (calculated as the difference between coronary sinus and radial artery values) was markedly greater in preconditioned patients than in the controls (5.7 ± 1.7 ng/mL vs. 1.9 ± 1.1 ng/mL, p=0.05). The trans-myocardial lactate gradient was shifted towards production in the PC group (+0.22 ± 0.13 mmol/L) and towards extraction in the control group (-0.06 ± 0.21 mmol/L). Molecular biology data summarized below did not suggest a protective effect of PC.

	c-*fos* (%I.S.)			HSP 70 (% I.S.)		
		End of PC/5			End of PC/5	
Group	PreCPB	min of CPB	End-arrest	PreCPB	min of CPB	End-arrest
Control	8 ± 4	52 ± 20	92 ± 23*	104 ± 41	128 ± 55	119 ± 53
PC	9 ± 5	87 ± 9	118 ± 30**	87 ± 32	93 ± 16	143 ± 54

*p<0.01 vs. PreCPB

**p<0.02 vs. PreCPB and 5 min of CPB

There were no PC-related clinical adverse events.

Conclusion: PC does not enhance cardioplegic protection and might even be deleterious. These results do not dismiss the therapeutic exploitation of the mechanisms of endogenous myocardial protection in cardiac surgery. They rather emphasize the need for identifying pharmacologic mediators that could safely and effectively duplicate the cardioprotective effects of ischemic PC.

12:10 p.m. ADJOURN

*By invitation

9:30 a.m. SIMULTANEOUS SCIENTIFIC SESSION E - GENERAL THORACIC SURGERY

Room 6C/F, San Diego Convention Center
Moderators: Andre C.H. Duranceau, M.D.

Alex G. Little, M.D.

48. LONG-TERM RESULTS AFTER ENBLOC DOUBLE-LUNG TRANSPLANTATION WITH BRONCHIAL ARTERIAL REVASCULARIZATION.

Eugene M. Baudet, M.D.*, Claire Dromer, M.D.*, Jean Dubrez, M.D.*, Jacques Jougon, M.D.*, Xavier Roques, M.D.*, Jean-FranÃ§ois Velly, M.D.*, Claude Deville, M.D.* and Louis Couraud, M.D.

Pessac, France

Discussant: Thomas R.J. Todd, M.D.

Background: Since May 1990, bronchial arterial revascularization (BAR) was associated with *enbloc* double-lung transplantation and tracheal anastomosis in order to improve airway healing. At the same time, it was also suggested that BAR could play a role in preventing the occurrence of obliterative bronchiolitis (OB).

Objective: This study was undertaken to evaluate the long-term results of this technique, and to assess whether BAR could improve tracheal healing and reduce OB incidence.

Material and Methods: Between May, 1990 and January, 1994, 18 patients (pts), 13 males and 15 females, underwent *enbloc* double-lung transplantation with BAR using a saphenous vein graft interposed between the orifices of bronchial arteries and the recipient's ascending aorta. Follow-up ranged from 22 to 54 months. The results of this combined technique were assessed according to tracheal healing, functional status, OB incidence, and infection and lung rejection episodes.

Results: There was neither operative death nor re-exploration for BAR-related bleeding. According to the criteria defined in a previous staging study, tracheal healing was qualified as grade I in 7 pts, grade IIa in 8, grade IIb in 2, and grade III in only one pt. Angiographic studies, between postoperative day 15 to 30, have shown a patent venous graft in 12 of the 15 controlled pts. Eleven pts are currently alive, with a mean follow-up of 43 months. In all 15 pts surviving more than one year, 5 have developed a documented bronchiolitis obliterans syndrome (BOS). Long-term functional results are excellent in the others, with a mean FEV_1 over 80% of predicted value. The 5 pts who have developed a BOS had an early or late documented vein graft thrombosis. On the contrary, in pts free from BOS and alive, all but one have to date a patent venous graft with effective BAR, two years and over after transplantation.

Conclusion: *Enbloc* double-lung transplantation with BAR appears to be a safe, available, and quite effective procedure allowing primary tracheal healing. Moreover, these first results could suggest that long-term BAR patency does have an impact on OB incidence; improved mucosal trophicity and mucociliary clearance could contribute to prevention of obliterative bronchiolitis.

*By invitation

49. BILATERAL VS SINGLE LUNG TRANSPLANTATION FOR CHRONIC OBSTRUCTIVE PULMONARY DISEASE.

Joseph E. Bavaria, M.D.*, Robert Kotloff, M.D.*, Harold Palevsky, M.D.*, Bruce Rosengard, M.D.*, John R. Roberts, M.D.* and Larry R. Kaiser, M.D.

Philadelphia, Pennsylvania

Discussant: Thomas J. Kirby, M.D.

Traditionally, despite VQ mismatch, single lung transplantation (SLT) has been the mainstay for end-stage COPD. We tested the hypothesis that bilateral sequential lung transplantation (BLT) has superior short and intermediate term results compared to SLT for COPD.

Methods: One hundred twelve consecutive lung transplants have been performed from 11/91 to 10/95. Sixty-three have been transplanted for COPD. The diagnosis of COPD includes emphysema (82.5%), Alpha-1 antrypsin deficiency (11.1%) and LAM (6.4%). Twenty-three transplants have been bilateral (BLT Group) and 40 have been single (SLT Group). MEAN age was *55* for SLT and 50 for BLT (p=ns). The distribution of the diagnoses was similar between the two groups except for Alpha-1 antrypsin deficiency which tended towards BLT. There were 19 survivors of SLT and 14 survivors of BLT out at least 6 months with complete data for evaluation. FEV_1, and 6-minute walk test (6MWT) were evaluated at a mean of 16 months and 12 months postoperative, respectively.

Results: 60-day mortality was 22.5% for SLT vs only 4.3% for BLT (p=0.05). Additionally, Kaplan-Meier analysis reveals 1- and 2-year survival of 71% and 60% for SLT vs 92% and 85% for BLT, respectively. Functional results are summarized in the table. Both 6MWT and FEV_1 were improved from baseline by SLT and BLT (p=0.001).

	Preop FEV_1(L)	Postop FEV_1(L)	% Change	Preop 6MWT(ft)	Postop 6MWT(ft)	% Change
SLT (=19)	0.55	1.53	+178%	632	1238	+96%
BLT (=14)	0.54	2.59	+380%	675	1677	+148%

Conclusion: BLT improves FEV_1 significantly over SLT ($p<0.01$). BLT improves 6MWT over SLT but does not reach significance. Both peri-operative mortality and Kaplan-Meier survival (to 2 yrs) is significantly improved using BLT vs SLT for COPD in our series.

*By invitation

50. PULMONARY RETRANSPLANTATION: DOES THE INDICATION FOR SURGERY INFLUENCE POSTOPERATIVE LUNG FUNCTION?

Richard J. Novick, M.D., Larry W. Stitt, MSc.*, Hans Joachim Schafers, M.D.*, Bernard AndrÃ©assian, M.D.*, Jean Pierre Duchatelle, M.D.*, Walter Klepetko, M.D.*, Robert L. Hardesty, M.D., Adaani E. Frost, M.D.* and G. Alexander Patterson, M.D.

London, Ontario, Canada; Homberg, Germany;

Clichy, France; Vienna, Austria; Pittsburgh, Pennsylvania;

Houston, Texas and St. Louis, Missouri

Discussant: Bruce A. Reitz, M.D.

An international series of pulmonary retransplantation was updated in order to determine factors associated with pulmonary function, Bronchiolitis Obliterans Syndrome (BOS) stage and survival in the intermediate-term postoperatively. The study cohort included 160 patients who underwent retransplantation in 35 centers in North America and Europe from 1985 to 1995; follow-up was 100% complete. Survivors were followed for a median of 780 days (versus 630 days in our previous report), with 62 patients alive at 1 year, 39 at 2 years, 27 at 3 years and 13 at four years after retransplantation. Actuarial survival was 45 ±4% (± = SEM), 41 ± 4% and 33 ± 4% at 1, 2 and 3 years, respectively. Nonetheless, in the 89 three-month postoperative survivors, actuarial survival at 2 years was 72 ± 5%. On multivariate analysis, the only predictor of 3-month survival was preoperative ambulatory status ($p=0.005$, odds ratio 2.97 in favor of ambulatory recipients), whereas individual center experience with at least 5 pulmonary retransplants was the sole predictor of 2-year survival ($p=0.04$, odds ratio 2.52). Analysis of the forced expiratory volume in 1 second (FEV_1) and BOS data revealed that the overall prevalence of stage 3 (severe) BOS was 12% at 1 year, 15% at 2 years and 32% at 3 years after retransplantation, similar to that reported after primary lung transplantation. Sixty-three percent of retransplant recipients were free of BOS (stages 1-3) at 2 years and 56% at 3 years. The FEV_1 value at 2 years was associated with subsequent survival at 3 ($p=0.002$) and 4 years ($p=0.01$). Furthermore, retransplant recipients in BOS stage 3 at 1 and 2 years had a significantly worse actuarial survival than those in BOS stages 0 to 2 *($p<0.01$*for both years). By 3 years after retransplantation, FEV_1 was significantly lower in patients reoperated for obliterative bronchiolitis (OB, 1.10 ± 0.15 L/sec) than in those retransplanted for acute graft failure or an airway complication (1.95 ± 0.29 L/sec, $p=0.02$*);* this difference was not apparent in our previous report. Only 31% of patients retransplanted for OB were in BOS stage 0 at 3 years versus 83% of patients retransplanted for other indications ($p=0.02$). Subset analyses revealed that the interval between transplant procedures in patients with OB did not significantly influence the

prevalence of BOS at 2 *(p*=1.0*)* and 3 years *(p*=0.53*)*. We conclude that patients undergoing retransplantation for OB develop more significant pulmonary dysfunction after 3 years of follow-up than those undergoing retransplantation for other indications. Preoperative ambulatory status predicts early survival and individual center experience predicts intermediate-term outcome after retransplantation. Improved management strategies are necessary to prevent the development of progressive draft dysfunction after retransplantation for OB.

*By invitation

51. AEROSOL CYCLOSPORINE LEADS TO IMPROVEMENT IN PULMONARY FUNCTION AND GRAFT HISTOLOGY IN LUNG TRANSPLANT RECIPIENTS WITH PERSISTENT ACUTE REJECTION.

Robert J. Keenan, M.D.*, Aldo T. Iacono, M.D.*, James H. Dauber, M.D.*, Samuel A. Yousem, M.D.*, Hartley P. Griffith, M.D. and Akihiko Kawai, M.D.*

Pittsburgh, Pennsylvania

Discussant: Sara J. Shumway, M.D.

We continued to be plagued by loss of pulmonary allografts from refractory acute or chronic rejection. The present study was undertaken to determine the effectiveness of aerosolized cyclosporine (CsA) for the treatment of persistent acute cellular rejection (ACR). Nine patients (pts) with persistent ACR, documented by transbronchial biopsy, were treated after having failed at least three courses of systemic steroids and/or cytolytic therapy (ATGAM). Patients remained on maintenance immunotherapy consisting of tacrolimus or cyclosporine, azathioprine and prednisone. Only pts with rejection grade >2 were included. Patients were given 300 mg of aerosolized CsA/day (in propylene glycol) 3 times weekly. Serial pulmonary function testing and biopsies were repeated at 6-8 week intervals. Seven of the 9 pts completely resolved their rejection 9-105 days (mean 54 days) after institution of aerosolized CsA therapy. One recipient improved from moderate (grade 3) to minimal (grade 1) ACR while 1 pt remained with persistent mild (grade 2) ACR. Pulmonary function (FEV_1), which had declined post-transplant from a best value of 2.02 ± 0.59 L to 1.58 ± 0.58 L (p=0.001) immediately prior to aerosol therapy, improved over 3-4 months to 1.90 ±0.75 L (p=0.006 post-aerosol vs pre-aerosol). As the figure demonstrates, this improvement is in distinct contrast to the decline experienced by a group of 23 historic controls matched for rejection history. No nephrotoxicity or hepatotoxicity was observed.

Aerosolized cyclosporine is an effective rescue therapy for lung transplant patients suffering from persistent acute rejection. A prospective randomized control trial is presently underway.

10:50 a.m. INTERMISSION

*By invitation

11:10 a.m. SIMULTANEOUS SCIENTIFIC SESSION E - GENERAL THORACIC SURGERY

Room 6C/F, San Diego Convention Center

Moderators: Andre C.H. Duranceau, M.D.

Alex G. Little, M.D.

52. RESULTS OF VATS THYMECTOMY IN PATIENTS WITH MY ASTHENIA GRAVIS.

Michael J. Mack, M.D., Rodney J. Landreneau, M.D., Anthony P. Yim, M.D.*, Steven R. Hazelrigg, M.D.* and Granger R. Scruggs*

Dallas, Texas, Pittsburgh, Pennsylvania,

Hong Kong, Hong Kong, and Springfield, Illinois

Discussant: Paul A. Kirschner, M.D.

Although video-assisted thoracic surgery (VATS) has a role in the management of a number of intrathoracic diseases, the efficacy of the procedure for thymectomy in patients with myasthenia gravis has not been examined. Thirty-three consecutive patients in 4 institutions underwent total thymectomy by VATS between March, 1992 and October, 1995. There were 13 males and 20 females with a mean age of 38.61 ± 17.68 years (range 9 to 84 years). The procedures were performed by either a right of left thoracoscopic approach and all anterior mediastinal tissue was removed to ensure a complete thymectomy. There was no perioperative mortality nor long-term morbidity. One patient required conversion to a lateral thoracotomy due to bleeding. All patients were extubated immediately except one patient who required postoperative ventilation. The mean hospital stay was 4.12 ± 6.46 days (range 1 to 37 days), with the median stay 3 days. Mean follow-up is 15.2 ± 5.93 months (range 1 to 41 months). Clinical improvement was seen in 69.6% (23/33) of the patients with 2 or 2 (100%) in Stage I, 14 of 19 (73.6%) in Stage IIA, 4 of 9 (44.4%) in Stage IIB and 3 of 3 (100%) as Stage III. We conclude that VATS thymectomy is as effective in the traditional open surgical approaches for performance of thymectomy in the management of patients with myasthenia gravis. In addition, the better cosmesis of the VATs approach makes thymectomy more acceptable to the patient and neurologist.
*By invitation

53. CHYLOTHORAX AFTER THORACOTOMY.

Robert J. Cerfolio, M.D.*, Mark S. Allen, M.D.*, Claude Deschamps, M.D.*, Victor F. Trastek, M.D. and Peter C. Pairolero, M.D.

Rochester, Minnesota

Discussant: George T. Christakis, M.D.

Between July 1987 and May 1995, there were 11,315 general thoracic surgical procedures performed at our institution, and 47 patients (0.42%) developed a postoperative chylothorax. There were 32 men and 15 women whose median age was 65 years (range 21-88). Initial operation was for esophageal disease in 27, pulmonary disease in 13, mediastinal mass in six, and thoracic aortic aneurysm in one. All patients were initially treated with hyperalimentation, cessation of oral intake, or medium chain triglyceride diet. Nonoperative therapy was successful in 13 patients (27.7%), and oral intake was resumed a median of seven days postoperatively (range 2-15). Reoperation was required in the remaining 34 patients. 88.9% of the patients who had an esophageal procedure required reoperation, whereas only 38.5% of the patients who had a pulmonary resection required reoperation. Preoperative lymphangiogram was performed in 16 patients and identified the site of the leak which was usually at the level of the azygous vein. To control the fistula, the thoracic duct was ligated at the diaphragm in 13 patients, at the level of the fistula in 12, at both levels in 7, and by mechanical pleurodesis and fibrin glue in 2. Initial reoperation was successful in 31 patients (91.2%). Three patients required a second reoperation to control the fistula. Combined operative mortality was 2.1%, and complications occurred in four patients (8.5%). Factors that predicted reoperation were esophageal surgery and an average daily drainage of >1000 cc/day for the first

five days postoperatively. We conclude that postoperative chylothorax is an unusual complication which can occasionally be treated nonoperatively; however, when drainage is >1000 cc/day or when it occurs after an esophageal operation, reoperation will usually be necessary. Preoperative lymphoangiogram can help identify the anatomy of the thoracic duct and localize the site of injury. The fistula can usually be controlled by ligation of the duct at the site of the leak or where it enters the thoracic cavity.
*By invitation

54. POSTPNEUMONECTOMY BRONCHOPLEURAL FISTULA FOLLOWING SUTURED BRONCHIAL CLOSURE: INCIDENCE, RISK FACTORS AND MANAGEMENT.

Cameron D. Wright, M.D.*, John C. Wain, M.D.*, Douglas J. Mathisen, M.D. and Hermes C. Grille, M.D.

Boston, Massachusetts

Discussant: Robert J. Ginsberg, M.D.

Postpneumonectomy bronchopleural fistula (BPF) remains a morbid complication following pneumonectomy. Over a 15-year period (1980-1995), 256 consecutive patients underwent pneumonectomy with a standardized suture closure of the bronchus. The bronchus was then covered by autologous tissue. The indications for pneumonectomy were lung cancer (198), other malignancy (20) and benign causes (38). Possible risk factors for BPF in these patients included preoperative radiotherapy (30), preoperative pleuropulmonary infection (37), completion pneumonectomy (35), and postoperative ventilation (31). One hundred three patients underwent right pneumonectomy and 6 (6%) developed BPFs. Left pneumonectomy was performed in 153 patients and 2 (1%) developed BPFs. The only risk factors that were significant for BPF were the need for the postoperative ventilation ($p<0.0001$) and right pneumonectomy ($p=0.04$). Two (25%) of the eight patients who developed BPFs died. Five patients developed BPF due to pneumonia requiring ventilation while the cause for 3 patients appeared to be technical. Re-closure was successful in 5 patients (mean postoperative day 12) while 1 patient healed a pinhole fistula by drainage alone. Two patients were treated by drainage alone because of severe adult respiratory distress syndrome and both died. Careful sutured closure of the main bronchus following pneumonectomy yields excellent results. The primary risk factor for BPF is the need for postoperative ventilation. Re-closure can be successful even if performed late.
12:10 p.m. ADJOURN

*By invitation

9:30 a.m. SIMULTANEOUS SCIENTIFIC SESSION F - CONGENITAL HEART DISEASE

Room 6D/E, San Diego Convention Center
Moderators: Richard A. Jonas, M.D.

Thomas L. Spray, M.D.

55. HYPERTROPHIC OBSTRUCTIVE CARDIOMYOPATHY IN PEDIATRIC PATIENTS: RESULTS OF SURGICAL TREATMENT.

David A. Theodoro, M.D.* and Gordon K. Danielson, M.D.

Rochester, Minnesota

Discussant: Michel N. Ilbawi, M.D.

Between April 1975 and May 1995, 25 pediatric patients on one hospital service underwent left ventricular septal myectomy for hypertrophic obstructive cardiomyopathy (HOCM). Ages ranged from 2 months to 20 years (mean=11 years). Associated cardiac diagnoses included mitral valve insufficiency (n=18), aortic valve insufficiency (n=3), atrial septal defect (n=2), history of ventricular arrhythmias (n=2), and bicuspid aortic valve (n=l). Symptoms included dyspnea (n=18), chest pain (n=12), syncope (n=5), and palpitations (n=2). Five patients were in NYHA class I, 10 were in class II, and 10 were in class III/IV. Preoperative medications included beta blockers (n=17), calcium channel blockers (n=4), disopyramide (n=l), or a combination (n=3), and length of treatment ranged from 2.6 months to 7.2 years (mean=2.2 years). One patient underwent dual-chamber pacemaker implantation without improvement of symptoms or left ventricular outflow tract (LVOT) gradient. Gradients ranged from 50 to 154 mmHg (mean=97.4) as determined by echocardiography (n=17) or cardiac catheterization (n=8). Concomitant cardiac procedures included mitral valve repair (n=3), AICD implantation (n=3), and closure of ASD (n=l). Intraoperative pre-myectomy LVOT gradients ranged from 20 to 117 mmHg (mean=82.3) and post-myectomy gradients ranged from 0 to 41 mmHg (mean=8.6). Post-myectomy mitral insufficiency, determined by double-sampling dye curves, was reduced to 0 to 12% regurgitant fraction (n=20), and no patient required mitral valve replacement. Aortic cross-clamp time ranged from 11 to 53 min (mean=35.3). Postoperative complications included ventricular arrhythmia (n=2), intra-aortic balloon pump support (n=l), and temporary complete heart block (n=l). There was no early mortality and no instances of aortic or mitral valvular damage or surgically created ventricular septal defect. One patient required placement of a pacemaker prior to hospital discharge for complete heart block; however, on follow-up at one year, normal sinus rhythm had returned. Follow-up ranged from 5 months to 15.8 years (mean=5.6 years). There were no late deaths. The mean LVOT gradient by echocardiography was 16 mmHg. Mild mitral valve insufficiency was present in 8 patients, mild aortic insufficiency was present in 3, and all patients were in normal sinus rhythm. Reoperation for recurrent LVOT obstruction was required in two patients at 3.2 years and 12.4 years after initial myectomy, respectively. At 5 years, freedom from reoperation for recurrent LVOT obstruction was 94.4 ±5.4%. We conclude that septal myectomy is a safe and effective means of relieving LVOT obstruction and cardiac symptoms in pediatric patients with hemodynamically important HOCM, and late survivorship compares extremely favorably with that of the natural history of nonoperative management in the pediatric population.
*By invitation

56. FACTORS IN THE EARLY FAILURE OF CRYO-PRESERVED HOMOGRAFT PULMONARY VALVES IN CHILDREN: PRESERVED IMMUNOGENICITY?

Roger J. Baskett, M.A.*, David B. Ross, M.D.*, Maurice A. Nanton, M.B.* and

David A. Murphy, M.D.

Halifax, Nova Scotia, Canada

Discussant: Richard A. Hopkins, M.D.

Introduction: Most studies addressing the durability of homograft valves in the right ventricular outflow tract (RVOT) use freedom from death or reoperation as the end point. Very few studies have systematically assessed the homograft valve function over time with echocardiography. The

durability of valve function in the RVOT is of increasing importance as more patients are referred for pulmonary valve replacement to protect a failing right ventricle.

Methods: Between 1990 and 1995, 48 homograft valves (15 aortic, 33 pulmonary) cryopreserved on-site were implanted to reconstruct the RVOT in 44 children (mean age: 6 years, range: 3 days to 19 years) with a variety of congenital cardiac lesions. Serial echocardiographic follow-up was performed on all 45 valves in the 41 survivors, with all echocardiograms reviewed in a standardized manner by one blinded pediatric cardiologist. In four cases, the echocardiographic windows were inadequate to reliably comment on valve function and are excluded from this analysis.

Results: Four homograft valves were replaced due to pulmonary insufficiency (3) or stenosis and insufficiency (1) at 2, 7, 14 and 48 months following implantation. Freedom from reoperation is 90% (70% CI, 84-96%) at 4 years. Three valves had >2+ insufficiency at the initial postoperative echocardiogram. Over the follow-up period, a further 15 of the remaining 38 valves (39%) developed progressive pulmonary insufficiency (PR) of at least 2 grades. Three valves developed transvalvular gradients of >50 mmHg, one of which was also insufficient. The freedom from echocardiographic failure (‰¥2+ PR or ‰¥50 mmHg gradient) was only 40% at 4 years (70% CI, 29-51%). Young age (p=0.05), low operative weight (p=0.01), small graft size (p=0.03), and homograft retrieval to cryopreservation time <24 hours (p=0.03) were significantly associated with valve insufficiency. The type of valve, donor age, tissue versus heart beating donor, or blood type mismatch were not associated with failure.

Conclusion: Homografts function well as conduits between the pulmonary ventricle and pulmonary arteries if long-term valve competency is not crucial. However, many rapidly become insufficient. This has important implications for the choice of valve if the indication for valve replacement is to protect a ventricle failing due to pulmonary insufficiency. Short homograft retrieval to cryopreservation times have been shown by others to enhance viability and antigenicity. This may explain the association observed in this study between short homograft retrieval to cryopreservation times and valve failure suggesting an immunological basis for this failure.

*By invitation

57. PULMONARY HYPERTENSION AFTER CONGENITAL OPEN HEART SURGERY: AN ANALYSIS OF RISK FACTORS AND MANAGEMENT.

†Ko Bando, M.D.*, Mark W. Turrentine, M.D.*, Kyung Sun, M.D.*, Thomas G. Sharp, M.D.*, Thomas X. Aufiero, M.D.*, Yasuo Sakine, M.D.* and John W. Brown, M.D.

Indianapolis, Indiana

Discussant: Pedro del Nido, M.D.

Background and Purpose: Pulmonary hypertensive events (PHE) including persistent pulmonary hypertension and pulmonary hypertensive crisis can cause death after cardiac operations for congenital heart defects (CHD). Monitoring and management of these potentially fatal complications have evolved over the last 15 years. Major changes include monitoring of pulmonary arterial pressure and mixed venous saturation (SvO_2 as well as prophylaxis with a-blockers (Chlorpromazine [CP] and/or Prazosin hydro-chloride [PR]) and other vasodilators (nitroglycerin and sodium nitroprusside). The purpose of this study was to identify risk factors for morbidity and mortality of PHE and to determine the impact of postoperative management on outcome.

Method: Two thousand four hundred fifty-four patients with CHD operated on using cardiopulmonary bypass between January 1980 and December 1994 were included in this study. Using univariate and multiple regression analysis, high risk candidates for post-op PHE were identified and risk of morbidity and mortality of PHE was analyzed.

Results: Patients with complete atrioventricular canal (CAVC; n=182), truncus arteriosus (TA; n=47), total anomalous pulmonary venous connection (TAPVC; n=90), transposition of great arteries (TGA; n=67), hypoplastic left heart syndrome (HLHS; n=50) and ventricular septal defect (VSD; n=414) were at high risk for post-op PHE. Fifty-eight percent (36/62) of hospital deaths in the high risk group were associated with PHE. Prevalence and mortality of PHE in each diagnostic group are depicted below.

	CAVC	TA	TAPVC	TGA	HLHS	VSD	total
Prevalence (%; #/at risk pts)	6%	30%	40%	21%	6%	14%	16%
	(11/182)	(14/47)	(36/90)	(14/67)	(3/50)	(60/414)	(138/850)
Mortality (%; #/at risk pts)	64%	57%	25%	21%	100%	10%	26%
	(7/11)	(8/14)	(9/36)	(3/14)	(3/3)	(6/60)	(36/138)

Analysis of these 850 cases divided into three different time frames revealed important institutional trends.

	1980-1984	1985-1989	1990-1994	p value
Prevalence of PHE {%; #/at risk pts)	27%)	21%	9%	$p<0.0001$
	(48/176)	(52/247)	(38/427)	
SvO_2 monitoring (%; #/at risk pts)	0.6%	75%	100%	$p<0.0001$
	(1/176)	(186/247)	(427/427)	
CP/PR prophylaxis (%; #/at risk pts)	0	2%	56%	$p<0.0001$
	(0/176)	(5/247)	(241/427)	

By multiple logistic regression, pre-op PH ($p<0.001$), absence of SvO_2 monitoring ($p<0.001$) and absence of CP/PR prophylaxis ($p=0.002$) were significant risk factors for the development of PHE. Only pre-op PH ($p<0.001$) was a significant risk factor for early death related to PHE. In each diagnostic group except TAPVC, definitive repair at older age was a significant risk factor for postoperative PHE ($p<0.001$) and early death related to PHE ($p<0.05$).

Conclusion: Post-op SvO_2 monitoring and CP/PR prophylaxis significantly reduced the prevalence of PHE. Definitive early repair reduces the postoperative morbidity and mortality from PHE.

†1991-92 Graham Fellow

*By invitation

58. MODIFIED FONTAN PROCEDURE IN 99 CASES WITH ATRIOVENTRICULAR VALVE REGURGITATION.

Yasuharu Imai, M.D., Yoshinori Takanashi, M.D.*

and Shuuichi Hoshino, M.D.*

Tokyo, Japan

Discussant: Hillel Laks, M.D.

Modified Fontan procedure generally carries higher risk in cases associated with atrioventricular valve regurgitation (AVVR). Factors influencing death were analyzed. Since January 1985 to August 1995, of 242 patients undergoing modified Fontan procedure, 99 had AVVR ranging in degrees from 1 to 4 (Sellers) by retrograde cineangiography, for which concomitant repair for AVVR was performed. Degree of AVVR was 1.6 ± 0.7 in average, 49 cases had more than grade 2 regurgitation and those with trivial regurgitation by Doppler echocardiography were classified as having no AVVR. Ages at operation ranged from 1 to 27 (9.9 ± 6.1) years. Anomalies consisted of 32 cases with univentricular heart (UVH) of RV type, 14 with UVH of LV type, 7 with classical tricuspid atresia and 46 other anomalies with biventricular heart. There were 24 cases of left isomerism and 25 had right isomerism. Prior to Fontan procedure, 99 palliative procedures mainly consisting of shunt procedure had been performed in 69 cases. Reparative procedures on the valve included circular annuloplasty in 75 cases, conventional annuloplasty in 11, patch-closure of valve in 4, valvoplasty in 4, and none in 4. Hospital mortality was higher in AVVR (12/99, 12%) than in cases without AVVR (4/143, 3%). Three died late during a mean follow-up period of 3.9 ± 2.9 years. Degree of AVVR showed a significant decrease from 1.64 ± 0.74 to 0.4 ± 0.51 ($p<0.0001$) after operation in survivors. Preoperative factors influencing hospital death and survival were number of parameters exceeding limits of ten commandments 4.92 vs. 3.85 ($p<0.0083$), cardiothoracic ratio 62 vs. 56% ($p<0.0048$), ventricular endo-diastolic volume 282 vs. 237% of normal ($p<0.0455$), pulmonary blood flow 3.5 vs. 5.0 L/minute/m_2 ($p<0.0183$), and Mayo's criteria 3.74 vs. 2.96 ($p<0.0275$). However, ventricular ejection fraction, pulmonary vascular resistance, degree of AVVR (1.75 vs. 1.62), morphological types of systemic ventricle and ventricular end-diastolic pressure were not related to death. In conclusion, cases with AVVR can be treated with reasonable risk, provided proper repair of the valve is performed.

10:50 a.m. INTERMISSION

*By invitation

11:10 a.m. SIMULTANEOUS SCIENTIFIC SESSION F - CONGENITAL HEART DISEASE

Room 6D/E, San Diego Convention Center

Moderators: Richard A. Jonas, M.D.

Thomas L. Spray, M.D.

59. PULMONARY ARTERY GROWTH AFTER BIDIRECTIONAL CAVOPULMONARY SHUNT: IS THERE CAUSE FOR CONCERN?

V. Mohan Reddy, M.D.*, Edwin Petrossian, M.D.*, Doff B. McElhinney, A.B.*,

Phillip Moore, M.D.*, Gary S. Haas, M.D.* and Frank L. Hanley, M.D.

San Francisco, California

Discussant: Thomas R. Karl, M.D.

Since pulmonary artery (PA) growth may be influenced by blood flow, it is thought that leaving patients with Bidirectional Cavopulmonary Shunt (BCPS) for extended periods of time may result in suboptimal PA growth. Between March 1990 and October 1995, 112 patients underwent BCPS at our institution; 28 of these patients have subsequently undergone a modified Fontan procedure (FP) and comprise the study group for the present report. Median age at BCPS among these 28

patients was 2.3 years (range: 3.2 months to 43 years), and median duration between BCPS and FP was 18 months (range: 1 to 47 months). At the time of BCPS, 8 patients underwent bilateral BCPS, 14 patients had PA augmentation, and 18 patients were left with an extra source of pulmonary blood flow (ExPBF). In these patients, the pre-BCPS and pre-FP angiograms were reviewed and right (R) and left (L) PA diameters were measured at their bifurcation, and indexed right (RPAI), and left (LPAI), and total (TPAI) PA cross-sectional areas were calculated. Since many patients underwent branch PA plasty, right lower lobe (RLL) and left lower lobe (LLL) PA diameters were also measured at their origin and R, L, and total lower lobe (ILL) indices were calculated. Aortic diameters immediately distal to the left subclavian artery and at the diaphragm were measured for control purposes. PAIs and hemodynamic variables were compared from pre-BCPS to pre-FP by paired /-test. Analysis of variance and linear regression were carried out to evaluate variables correlating with change in PAIs between BCPS and FP and with morbidity and mortality following FP. Variables analyzed included pre-BCPS PAIs, duration from BCPS to FP, bilateral BCPS, PA plasty at BCPS, ExPBF, early post-BCPS PA pressure, and pre-FP pulmonary hemodynamics (PA pressure), indexed pulmonary blood flow (Qp), pulmonary to systemic blood flow ratio (Qp:Qs). Aortic diameters increased significantly ($p<0.01$), and PA pressure ($p=0.02$), ventricular end-diastolic pressure ($p=0.02$), pulmonary to systemic blood flow ratio (Qp:Qs; $p=0.01$), and indexed pulmonary blood flow (Qp; $p=0.04$) all decreased significantly from pre-BCPS to pre-FP catheterization. There was a significant decrease in TPAI among patients undergoing BCPS with no ExPBF ($p=0.02$). There were also significant differences in TPAI ($p=0.05$), RLL PAI ($p=0.03$), and TLL PAI ($p=0.04$) changes from BCPS to FP between patients with and without ExPBF, with mean PAIs increasing among patients with ExPBF and, decreasing among patients without ExPBF. There were no significant differences in various indices between patients with single or bilateral BCPS. There were no significant correlations between the PAIs, variables analyzed and post FP outcome (mortality, effusions, PA pressure).
Conclusion: Although it appears that central pulmonary artery growth is affected by BCPS and the presence of ExPBF, none of the variables analyzed, including change in PAIs, correlated with post-FP mortality, effusions, or PA hemodynamics. However, it is possible that if BCPS is chosen as a long-term palliation method, pulmonary artery growth may become an important issue.
*By invitation

60. EXTRACARDIAC FONTAN FOR COMPLEX CARDIAC ANOMALIES: SEVEN-YEAR EXPERIENCE.

Antonio Amodeo, M.D.*, Lorenzo Galletti, M.D.*, Salvatore Giannico, M.D.*, Paolo Di Renzi, M.D.* and Carlo Marcelletti, M.D.

Rome, Italy

Discussant: Leonard L. Bailey, M.D.

Between November 1988 and September 1995, 58 patients with complex cardiac anomalies underwent an extracardiac Fontan (EF). The EF consisted in the combination of a bidirectional cavopulmonary anastomosis and an extra-cardiac conduit between the inferior vena cava (IVC) and the pulmonary artery (PA) except in one pt in whom the IVC was directly anastomosed to the PA. In 37 pts the EF followed a preliminary BCPA, associated with modified Damus-Kaye-Stansel in 16. The conduits used were Dacron (34), aortic homografts (3) and PTFE used electively in the last 20 pts. There were 6 hospital deaths (10.3%). Two pts required conduit take-down due to a failing Fontan for a left PA stenosis and severe atrioventricular valve regurgitation respectively; another two pts required a revision of the cavopulmonary anastomosis due to anastomotic stricture. All pts were discharged on oral anticoagulation for six months. In a mean follow-up of 35 months (3-80 mos), there were no late deaths with 54 pts in NYHA class I or II. Two pts required balloon dilatation and/or stents implantation of PA after EF. Late atrial arrhythmias were detected in 6/58 pts: 4 had mild sinus dysfunction and 2 atrial flutter requiring a PMK implantation in 1. Patency of

the Dacron conduits was evaluated in the initial 25 pts by MRI showing a reduction of the conduit internal diameter of 12.7% ± 5.1% in the first month and additional narrowing of 2% over the next 2 years. These data demonstrate that the EF provides excellent early and mid-term results in terms of mortality, morbidity, and incidence of late arrhythmias.
*By invitation

61. COAGULATION FACTOR ABNORMALITIES FOLLOWING THE FONTAN PROCEDURE AND ITS MODIFICATIONS.

Marjan Jahangiri, FRCS*, Daryl Shore, FRCS*, Vijay Kakkar, FRCS*, Elliot Shinebourne, FRCP* and Christopher Lincoln, FRCS

London, United Kingdom

Discussant: John E. Mayer, Jr., M.D.

Recently we reported a high incidence of thromboembolism in patients who underwent the Fontan procedure and its modifications. While haemodynamic factors may well contribute to this, recent evidence suggests that coagulation factor abnormalities may also play a role. We therefore set out to investigate the coagulation status in a group of patients who had undergone the Fontan procedure. The study population consists of 20 children who had undergone the Fontan procedure and its modifications. They were examined for coagulation factor abnormalities and their serum albumin, total protein and liver enzymes were measured. The median age at the time of surgery was 6.2 years with a male to female ratio of 2.3:1, the median time from the Fontan repair was 4.9 years (18-76 months).
Protein C ($p<0.001$), protein S ($p<0.005$) and factor VII ($p<0.001$) were significantly lower than the normal range. The changes in serum albumin and total protein, factors II, VII, IX and X were not significant.
It is possible that deficiency in protein C, protein S and factor VII partly account for the high incidence of thromboembolism following Fontan type repair. The risk of long-term anticoagulation should be weighed against the best palliative procedure for these patients. We suggest that reduced protein C, protein S and factor VII in this group of patients should be regarded as risk factors and that such patients should be anticoagulated.
12:10 p.m. ADJOURN

*By invitation

GEOGRAPHICAL ROSTER

NECROLOGY

Clifford D. Benson, M.D. Grosse Pointe, Michigan
William S. Blakemore, M.D. Manteo, North Carolina
David H. Dillard, M.D. Seattle, Washington
Marcus L. Dillon, M.D. Lexington, Kentucky
Emerson H. Drake, M.D. Portland, Maine
Richard E. Gardner, M.D. San Francisco, California
Alfred Goldman, M.D. Palm Springs, California
Vincent M. Iovine, M.D. Chevy Chase, Maryland
Donald B. Miller, M.D. Burlington, Vermont
Ross Robertson, M.D. West Vancouver, BC, Canada
Vernon C. Thompson, M.D. London, England
Robert H. Wylie, M.D. Baltimore, Maryland

The American Association for Thoracic Surgery

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1995-1996

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Kirklin, John W
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Laks, Hillel
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Longmire, William, Jr
Maloney, James V, Jr
Mandal, Ashis K
Matloff, Jack M
Mulder, Donald G
Stames, Vaughn A
Waters, Paul F
Los Osos
Aronstam, El more M
Menlo Park
Peters, Richard M
Montebello
Lui, Alfred H F
Oakland
Ecker, Roger R
Iverson, Leigh I G
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Gazzaniga, Alan B
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Dart, Charles H, Jr
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Hill, J Donald
Leeds, Sanford E
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Eiseman, Ben
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Grow, John B, Sr
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Pomerantz, Marvin
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Kraeft, Nelson H
Tampa
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Seller, Hawley H
Winter Haven
Maurer, Elmer P R

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Mansour, Kamal A
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Symbas, Panagiotis
Williams, Willis H

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Ellison, Robert G
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Chickamauga
Hall, David P

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Dalton, Martin L, Jr
Sealy, Will C
Van De Water, Joseph M

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Hartz, Renee S
Head, Louis R
Hunter, James A
Karp, Robert B
Kittle, C Frederick
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Michaelis, Lawrence
Montoya, Alvaro
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Nigro, Salvatore L

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Levett, James M
Weinberg, Milton, Jr

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Wellons, Harry A, Jr

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Corner, Ralph A
Phillips, Steven J
Zeff, Robert H

Iowa City
Behrendt, Douglas M
Ehrenhaft, Johann L

LOUISIANA

Alexandria
Knoepp, Louis F

Baton Rouge
Berry, B Eugene
Beskin, Charles A

Metairie
Ochsner, Alton, Jr

New Orleans
Blalock, John B
DeCamp, Paul T
Hewitt, Robert L
Lindsey, Edward S
McFadden, P Michael
Mills, Noel L
Moulder, Peter V
Ochsner, John L
Pearce, Charles W
Schramel, Robert J
Webb, Watts R

Rossi, Nicholas P
Stanford, William

KANSAS

Cunningham
Allbritten, Frank F, Jr
Lawrence
Miller, Don R
Prairie Village
Holder, Thomas M
Shawnee Mission
Adelman, Arthur
Padula, Richard T
Wichita
Tocker, Alfred M

KENTUCKY

Lexington
Crutcher, Richard R
Todd, Edward P
Louisville
Austin, Erie H, III
Gray, Laman A, Jr
Mahaffey, Daniel E
Ransdell, Herbert, Jr

MAINE

Portland
Bredenberg, Carl E
Morton, Jeremy R
Rockport
Swenson, Orvar
Windham
Hiebert, Clement

MARYLAND

Baltimore
Attar, Safuh
Baker, R. Robinson
Baumgartner, William A
Blair, Emil
Cameron, Duke Edward
Dodrill, Forest D
Gott, Vincent L
Haller, J Alex, Jr
McLaughlin, Joseph S
Michelson, Elliott
Salomon, Neal W
Turney, Stephen Z
Watkins, Levi, Jr
Bethesda
Pass, Harvey I
Fort Detrick
Zajtchuk, Rostik
Towson
Brawley, Robert K
Worton
Walkup, Harry E

MASSACHUSETTS

Boston
Akins, Gary W
Austen, W. Gerald
Barsamian, Ernest M
Bougas, James A
Buckley, Mortimer J
Burke, John F
Cohn, Lawrence H
Collins, John J
Daggett, Willard M
Daly, Benedict D T
Ellis, F Henry, Jr
Frank, Howard A
Gaensler, Edward A
Grille, Hermes C
Hilgenberg, Alan D
Johnson, Robert G
Jonas, Richard A
Lazar, Harold L
Levitsky, Sidney
LoCicero, Joseph, III
Mathisen, Douglas J
Mayer, John E
Moncure, Ashby C
Rheinlander, Harold F
Russell, Paul S
Scannell, J Gordon
Sellke, Frank W
Shemin, Richard J
Starkey, George W B
Sugarbaker, David J
Thurer, Robert L
Vlahakes, Gus J
Weintraub, Ronald M
Boylston
Okike, Okike N
Brookline
Madoff, Irving M
Burlington
Shahian, David M
Cambridge
Berger, Robert L
Malcolm, John A
Chestnut Hill
Laforet, Eugene G
Concord
Soutter, Lamar
Dover
Black, Harrison
Falmouth
McElvein, Richard B
Framingham
Bernhard, William F
Schuster, Samuel R
Medford
Desforges, Gerard
Methuen
Wilson, Norman J
North Andover
Cook, William A
Shrewsbury
Moran, John M
Springfield
Engelman, Richard M
Rousou, John A
Vineyard Haven
Malm, James R
Wellesley
Cleveland, Richard J
MacManus, Joseph E
West Newton
Neptune, Wilford B
West Roxbury
Khuri, Shukri F

Westport Harbor
Findlay, Charles W
Williamstown
Wilkins, Earle W
Worcester
Vander Salm, Thomas J

MICHIGAN

Ann Arbor
Bartlett, Robert H
Bolling, Steven F
Bove, Edward L
Deeb, G. Michael
Gago, Otto
Greenfield, Lazar J
Kirsh, Marvin M
Morris, Joe D
Neerken, A John
Orringer, Mark B
Prager, Richard L
Sloan, Herbert E
Bloomfield Township
Timmis, Hilary H
Detroit
Arbulu, Agustin
Silverman, Norman A
Steiger, Zwi
Stephenson, Larry W
Wilson, Robert F
Grand Rapids
Harrison, Robert W
Rasmussen, Richard A
Tomatis, Luis A
St. Joseph
Levine, Frederick H
West Bloomfield
Arciniegas, Eduardo

MINNESOTA

Coon Rapids
Gannon, Paul G
Mendota Heights
Dennis, Clarence
Minneapolis
Arom, Kit V
Bolman, R. Morton, III
Emery, Robert W
Foker, John E
Garamella, Joseph J
Helseth, Hovald K
Kaye, Michael P
Molina, J. Ernesto
Nicoloff, Demetre M
Shumway, Sara J
Rochester
Bematz, Philip E
Danielson, Gordon K.
McGoon, DwightC
McGregor, Christopher G A
Olsen, Arthur M
Orszulak, Thomas A
Pairolero, Peter C
Payne, W Spencer
Puga, Francisco J
Schaff, Hartzell V
Trastek, Victor F
Shorewood
Riser, Joseph C
St. Paul
Lillehei, C Walton
Miller, Fletcher A
Waubun
DeNiord, Richard N

MISSISSIPPI

Carthage
Logan, William D, Jr
Jackson
Johnston, James H
Netterville, Rush E
Madison
Hardy, James D

MISSOURI

Bridgeton
Codd, John E
Chesterfield
Bergmann, Martin
Columbia
Bryant, Lester R
Curtis, Jack J
Silver, Donald
Walls, Joseph T

Kansas City
Ashcraft, Keith W
Benoit, Hector W, Jr
Borkon, A Michael
Killen, Duncan A
Mayer, John H, Jr
Piehler, Jeffrey M
Reed, William A
VanWay, Charles W, III
Mount Vernon
Campbell, Daniel C, Jr
St. Louis
Barner, Hendrick B
Baue, Arthur E
Connors, John P
Cooper, Joel D
Cox, James L
Ferguson, T Bruce, Jr
Ferguson, Thomas B
Flye, M Wayne

NEW HAMPSHIRE

Franconia
Taylor, Warren J
Jaffrey
Woods, Francis M
Lebanon
Sanders, John H, Jr

NEW JERSEY

Alpine
Holswade, George R
Belleville
Gerard, Franklyn P
Browns Mills
Fernandez, Javier
McGrath, Lynn B
Camden
Camishion, Rudolph C
DelRossi, Anthony J
East Orange
Auerbach, Oscar

Gay, William A, Jr
Johnson, Frank E
Kaiser, George C
Kouchoukos, Nicholas T
Lewis, J Eugene, Jr
McBride, Lawrence R
Naunheim, Keith S
Pasque, Michael K
Patterson, G Alexander
Roper, Charles L
Strevey, Tracey E, Jr
Willman, Vallee L

MONTANA

Missoula
Duran, Carlos Gomez
Oury, James H

NEBRASKA

Omaha
Fleming, William H
Schultz, Richard D

NEVADA

Las Vegas
Little, Alex G

Short Hills
Hochberg, Mark S
Tenafly
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
Altai, Lari A
Brodman, Richard F
Fell, Stanley C
Ford, Joseph M
Prater, Robert W M
Hirose, Teruo
Veith, Frank J
Brooklyn
Cunningham, Joseph N, Jr
Levowitz, Bernard S
Sawyer, Philip N
Buffalo
Adler, Richard H
Andersen, Murray N
Bhayana, Joginder N
Hoover, Eddie L
Lajos, Thomas Z
Salerno, Tomas A
Cooperstown
Blumenstock, David A
Fayetteville
Bugden, Walter F
Effler, Donald B
Floral Park
Crastnopol, Philip

Hackensack
Hutchinson, John E, III
Jersey City
Demos, Nicholas J
Millburn
Parsonnet, Victor
Moorestown
Morse, Dryden P
Morristown
Parr, Grant V S
Neptune
Roberts, Arthur J
New Brunswick
Lewis, Ralph J
MacKenzie, James W
Scholz, Peter M
Newark
Donahoo, James
Gielchinsky, Isaac
Swan, Kenneth G
Pittstown
Garzon, Antonio A

Lido Beach
Hines, George L
Loudonvillc
Alley, Ralph D
New Rochelle
Rubin, Morris
New York
Acinapura, Anthony J
Adams, Peter X
Anagnostopoulos, C E
Bains, Manjit S
Beattie, Edward, Jr
Bloomberg, Allan E
Boyd, Arthur D
Bregman, David
Burt, Michael E
Cahan, William G
Clauss, Roy H
Conklin, Edward F
Culliford, Alfred T
Ergin, M Arisan
Friedlander, Ralph
Galloway, Aubrey C, Jr
Ginsberg, Robert J
Gold, Jeffrey P
Green, George E
Griepp, Randall B
Isom, O Wayne
Jaretzki, Alfred, III
King, Thomas C
Kirschner, Paul A
Krieger, Karl H
Litwak, Robert S
Martini, Nael
McCord, Colin W
McCormack, Patricia M
Michler, Robert E
Nealon, Thomas F, Jr
Quaegebeur, Jan M
Redo, S Frank
Reemtsma, Keith
Rose, Eric A
Rusch, Valerie W
Skinner, David B

Larchmont
Steichen, Felicien M

Spencer, Frank C
Spotnitz, Henry M
Subramanian, Valavanur A
Tice, David A
Tyras, Denis H
Wichem, Walter, Jr
Wolff, William I

Patchogue
Finnerty, James

Pittsburgh
Potter, Robert T

Rochester
Craver, William L
DeWeese, James A
Hicks, George L
Schwartz, Seymour 1
Stewart, Scott

Roslyn
Thomson, Norman B, Jr
Wisoff, George

Saranac Lake
Decker, Alfred M, Jr

Scarsdale
Robinson, George

Scottsville
Emerson, George L

Slingerlands
Kausel, Harvey W

Stony Brook
Soroff, Harry S

Syracuse
Brandt, Berkeley, III
Kohman, Leslie J
Meyer, John A
Parker, Frederick, Jr

Valhalla
Moggio, Richard A
Reed, George E

NORTH CAROLINA

Asheville
Berts, Reeve H
Kroncke, George M
Scott, Stewart M
Takaro, Timothy

Helmsworth, James A
Hiratzka, Loren F
Ivey, Tom D
Wilson, James M
Wright, Creighton B
Yee, Edward S

Cleveland
Ankeney, Jay L
Cosgrove, Delos M
Geha, Alexander S
Groves, Laurence K
Kay, Earle B
Kirby, Thomas J
Loop, Floyd D
Lytle, Brace W
McCarthy, Patrick M
Rice, Thomas W
Snow, Norman J
Smith, Craig R

Chapel Hill
Bowman, Frederick, Jr
Egan, Thomas M
Keagy, Blair A
Starek, Peter J
Wilcox, Benson R

Charlotte
Robicsek, Francis
Selle, Jay G

Durham
Anderson, Robert W
Jones, Robert H
Lowe, James E
Oldham, H Newland.Jr
Sabiston, David C, Jr
Smith, Peter K
Ungerleider, Ross M
Van Trigt, Peter
Wolfe, Walter G
Young, W Glenn, Jr

Greenville
Chitwood, W Randolph, Jr

High Point
Mills, Stephen A

Oriental
Deaton, W Ralph, Jr

Pinehurst
Fischer, Walter W

Sugar Grove
Gentsch, Thomas O

Winston-Salem
Cordell, A Robert
Crosby, Ivan Keith
Hammon, John W, Jr
Hudspeth, Allen S
Meredith, Jesse H
Pennington, D Glenn

OHIO

Chagrin Falls
Cross, Frederick S

Cincinnati
Albers, John E
Callard, George M
Flege, John B, Jr
Gonzalez, Luis L

OREGON

Days Creek
Miller, Arthur C

Portland
Cobanoglu, Adnan
Krause, Albert H
Lemmer, John H, Jr
Okies, J Edward
Poppe, J Karl
Starr, Albert

PENNSYLVANIA

Abington
Frobese, Alfred S

Bethlehem
Snyder, John M

Bristol
Dunn, Jeffrey M

Bryn Mawr

Van Heeckeren, Daniel W

Columbus

Davis, J Terrance
Kakos, Gerard S
Meckstroth, Charles
Myerowitz, P. David
Williams, Thomas E, Jr

Dayton

DeWall, Richard A

Delaware

Clatworthy, H Williams, Jr

Grove City

Oman, James W

OKLAHOMA

Jenks

LeBeck, Martin B

Lawton

Barnhorst, Donald A

Oklahoma City

Elkins, Ronald C
Felton, Warren L, II
Fisher, R Darryl
Greer, Allen E
Munnell, Edward R
Williams, G Rainey
Zuhdi, M Nazih

Haupt, George J

Camp Hill

Pennock, John L

Carlisle

DeMuth, William, Jr

Darby

McKeown, John J, Jr

Hershey

Campbell, David B
Pae, Walter E,Jr
Pierce, William S
Waldhausen, John A

Johnstown

KolfF, Jacob

Lancaster

Bonchek, Lawrence I
Rosemond, George P
Winner, Robert H

Philadelphia

Addonizio, V. Paul
Bowles, L Thompson
Brockman, Stanley K
Diehl, James T
DiSesa, Verdi J
Edie, Richard N
Edmunds, L. Henry, Jr

Fineberg, Charles
Gardner, Timothy J
Goldberg, Melvyn
Kaiser, Larry R
MacVaugh, Horace
Nemir, Paul, Jr
Shochat, Stephen J
Spray, Thomas L
Whitman, Glenn J R

Pittsburgh

Bahnson, Henry T
Clark, Richard E
Griffith, Bartley P
Hardesty, Robert L
Kormos, Robert L
Landreneau, Rodney J
Magovern, George J
Myers, John L
Pontius, Robert G
Rams, James J
Siewers, Ralph O

Rosemont

Sink, James D
Templeton, John, III

Villanova

Mundth, Eldred D

Wayne

Hargrove, W Clark, III
Lemmon, William M

Wyncote

Mendelssohn, Edwin

Wynnewood

Wallace, Herbert W

Yardley

Sommer, George N, Jr

RHODE ISLAND

Providence

Karlson, Karl E
Moulton, Anthony L
Singh, Arun K

SOUTH CAROLINA

Parker, Edward F
Sade, Robert M

Columbia

Almond, Carl H

Hilton Head Island

Humphrey, Edward W

Isle of Palms

Mullen, Donald C

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
McBumey, 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
Merrill, Walter H
Randolph, Judson G
Rankin, J Scon
Sawyers, John L
Scott, Henry W, Jr
Stoney, William S
Thomas, Clarence, Jr

Sparta

Charleston
Bradham, R Randolph
Crawford, Fred A, Jr
Kratz, John M

Austin
Hood, R Maurice
Tyson, Kenneth R T
Burnet
Ross, Raleigh R
Coppell
McPhail, Jasper L
Dallas
Adam, Maurice
Estrera, Aaron S
Holland, Robert H
Lambert, Cary J
Mack, Michael J
Mills, Lawrence J
Paulson, Donald L
Plan, Melvin R
Razzuk, Maruf A
Ring, W Steves
Seybold, William D
Urschel, Harold, Jr
Dilley
Hood, Richard H, Jr
El Paso
Glass, Bertram A
Galveston
Conti, Vincent R
Derrick, John R
Zwischenberger, Joseph B
Houston
Baldwin, John C
Beall, Arthur C, Jr
Burdette, Walter J
Cooley, Denton A
Coselli, Joseph S
DeBakey, Michael E
Frazier, O. Howard
Hallman, Grady L
Henly, Walter S
Jones, James W
Lawrie, Gerald M
Mattox, Kenneth L
Mountain, Clifton F
Ott, David A
Overstreet, John W
Putnam, Joe B, Jr
Reul, George J, Jr

Aylett
Gwathmey, Owen
Charlottesville
Dammann, John F
Daniel, Thomas M
Kron, Irving L
Minor, George R
Muller, William, Jr
Nolan, Stanton P
Spotnitz, William D
Tribble, Curtis G
Fredericksburg
Armitage, John M
Lynchburg
Moore, Richmond L

Labrosse, Claude C

TEXAS

Amarillo
Sutherland, R Duncan

Roth, Jack A
Safi, Hazim J
Walker, William E
Wukasch, Don C
Kemp
Davis, Milton V
Lubbock
Bricker, Donald L
Feola, Mario
Wallsh, Eugene
San Antonio
Cohen, David J
Dooley, Byron N
Heaney, John P
Treasure, Robert L
Trinkle, J Kent
Shepherd
Morris, George C, Jr
Temple
Brindley, G. Valter, Jr
Woodville
Harrison, Albert W

UTAH

Salt Lake City
Doty, Donald B
Liddle, Harold V
McGough, Edwin C
Mortensen, J D
Nelson, Russell M

VERMONT

Richford
Grondin, Claude M
West Dover
Humphreys, George H, II

VIRGINIA

Altavista
Pierucci, Louis, Jr
Annandale
Akl, Bechara F
Burton, Nelson A
Lefrak, Edward A
Arlington
Klepser, Roy G

Merendino, K. Alvin
Miller, Donald W, Jr
Rittenhouse, Edward
Sauvage, Lester R
Thomas, George I
Verrier, Edward D
Spokane
Berg, Ralph, Jr

WEST VIRGINIA

Charlestown
Walker, James H
Huntington
Gonzalez-Lavin, Lorenzo
Morgantown
Graeber, Geoffrey M

McLean
Conrad, Peter W
Mills, Mitchell
Reston
Boyd, Thomas F
Richmond
Bosher, Lewis H, Jr
Brooks, James W
Cole, Dean B
Damiano, Ralph J, Jr
Lower, Richard R
Wechsler, Andrew S

WASHINGTON

Bellingham
Varco, Richard L
Friday Harbor
Lawrence, G Hugh
Issaquah
Jarvis, Fred J
Kirkland
Mills, Waldo O
Poulsbo
Malette, William G
Seattle
Allen, Margaret D
Anderson, Richard P
Hill, Lucius D
Jones, Thomas W
Li, Wei-I
Mannas, Dev R
Mansfield, Peter B
Gustafson, Robert A
Hill, Ronald C
Murray, Gordon F
Warden, Herbert E
Parkersburg
Tamay, Thomas J

WISCONSIN

Eau Claire
McEnany, M Terry
Kenosha
Swain, Julie A
Madison
Chopra, Paramjeet S
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
Litwin, S Bert
Olinger, Gordon N
Tector, Alfred J
West Bend
Gardner, Robert J

WYOMING

Teton Village
Kaunitz, Victor H

Geographical - CANADA
1995-1996

ALBERTA

Calgary
Bharadwaj, Baikunth
Miller, George E
Edmonton
Callaghan, John C
Gelfand, Elliot T
Sterns, Laurence P

BRITISH COLUMBIA

Vancouver
Ashmore, Phillip G
Jamieson, W R Eric
Tyers, G. Frank O
Victoria
Stenstrom, John D

MANITOBA

Winnipeg
Barwinsky, Jaroslaw
Cohen, Morley

NOVA SCOTIA

Halifax
Murphy, David A
Mabou
Thomas, Gordon W

ONTARIO

London
McKenzie, F Neil
Novick, Richard J
North York
Goldman, Bernard S
Nottawa
Key, James A
Oakville
Allen, Peter
Sudbury
Field, Paul
Walker, George R
Toronto
Baird, Ronald J
Bigelow, Wilfred G
Christakis, George T
Coles, John G
David, Tirone E
Fremes, Stephen E
McKneally, Martin F
Mickleborough, Lynda L
Pearson, F Griffith
Scully, Hugh E
Todd, Thomas R J
Trimble, Alan S
Trusler, George A
Weisel, Richard D
Williams, William G
Westbrook
Lynn, R Beverley

QUEBEC

Montreal
Blundell, Peter E
Chartrand, Claude C. C
Chiu, Chu-Jeng (Ray)
Cossette, Robert
Dobell, Anthony R C
Duranceau, Andre C H
MacLean, Lloyd D
Morin, Jean E
Mulder, David S
Pelletier, L Conrad
Scott, Henry J

Ottawa
Keon, Wilbert J
Shennib, Hani
Outremont
Lepage, Gilles
Sainte-Foy
DesLauriers, Jean

Geographical - OTHER COUNTRIES
1995-1996

AFGHANISTAN
Kabul
Hankins, John R
ARGENTINA
Buenos Aires
Favaloro, Rene G
AUSTRALIA
QUEENSLAND
Brisbane
O'Brien, Mark F
SOUTH AUSTRALIA
Stirling
Sutherland, H D'Arcy
VICTORIA
Melbourne
Karl, Tom R
Nossal, Gustav J V
AUSTRIA
Leonding
Bruecke, Peter E
Salzburg
Unger, Felix H
Vienna
Wolner, Ernst
BAHAMAS
Heimbecker, Raymond
BELGIUM
Bertem
Sergeant, Paul T
Leuven
Lerut, Antoon E M R
BRAZIL
Rio de Janeiro
Meier, Milton A
Sao Paulo
Jatene, Adib D

ENGLAND
Bath, Avon
Belsey, Ronald
Cambridge
Kennedy, John H
Herts
Lennox, Stuart C
London
Braimbridge, Mark V
de Leval, Marc R
Lincoln, Christopher R
Ross, Donald N
Stark, Jaroslav F
Taylor, Kenneth M
Yacoub, Magdi
Somerset
Abbey-Smith, R
FINLAND
Helsinki
Manila, Sever! P
FRANCE
Bordeaux
Couraud, Louis
Fontan, Francis M
Le Plessis Robinson
Dartevelle, Philippe G
Marseille
Metros, Dominique R
Montpellier
Thevenet, Andre A
Paris
Binet, Jean-Paul
Blondeau, Philip
Cabrol, Christian E A
Carpentier, Alain F
Loisance, Daniel
Menasche, Philippe
Piwnica, Armand H
Planche, Claude
Weldon, Clarence S

Suresnes
Bachet, Jean E
GERMANY
Hannover
Borst, Hans G
Aachen
Messmer, Bruno J
Munchen
Sebening, Fritz
Neuss
Bircks, Wolfgang H
GUATEMALA
Guatemala City
Herrera-Llerandi, Rodolfo
INDIA
Bikaner
VanAllen, Chester M

Tokyo
Imai, Yasuharu
Koyanagi, Hitoshi
Wada, Juro J
KOREA
Seoul
Cho, Bum-Koo
MONACO
Monte Carlo
Dor, Vincent
NEW ZEALAND
Waiwera HBC
Barratt-Boyes, Brian G
P.R. OF CHINA
Beijing
Ying-Kai, Wu
PORTUGAL

IRELAND

Dublin
O'Malley, Eoin

ITALY

Bergamo
Parenzan, Lucio

Milan
Peracchia, Alberto

Naples
Cotrufo, Maurizio

Pisa
Bortolotti, Uberto

Rome
Marcelletti, Carlo

JAPAN

Kanazawa
Iwa, Takashi

Kitakyushushi
Miyamoto, Alfonso T

Osaka
Kawashima, Yasunaru

Sendai
Mohri, Hitoshi

SWEDEN

Sollentuna
Bjork, Viking

SWITZERLAND

Arzier
Hahn, Charles J

Genolier
Castaneda, Aldo R
Norwood, William I

Pully
Naef, Andreas P

Coimbra
Antunes, Manuel J

Lisbon
Macedo, Manuel E M

ROMANIA

Targu-Mures
Deac, Radu C

RUSSIA

Moscow
Bockeria, Leo A

SAUDI ARABIA

Riyadh
Landymore, Roderick W

SCOTLAND

Edinburgh
Logan, Andrew

Glasgow
Wheatley, David J

SPAIN

Madrid
Rivera, Ramiro

Sandander
Revuelta, Jose Manuel

Zurich
Senning, Ake
Turina, Marko I

U.A.E.

Abu Dhabi
Brom, A Gerard

VENEZUELA

Caracas
Tricerri, Fernando E

THE AMERICAN ASSOCIATION FOR THORACIC SURGERY
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Edmond Melchior Eberts
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Nathan W. Green
John R. Hartwell
George J. Heuer
Chevalier Jackson
H.H. Janeway
Arthur A. Law
William Lerche
Howard Lilienthal
William H. Luckett
Morris Manges
Walton Martin
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Samuel J. Metzler
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James Alexander Miller
Robert T. Miller
Fred J. Murphy
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Eugene H. Pool
Walter I. Rathbun
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William H. Stewart
Franz Torek
Martin W. Ware

James H. Kenyon
Adrian V. S. Lambert
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 three classes of members: Honorary, Senior, and Active. 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. Honorary members shall have the privilege of voting but shall not be eligible to hold office.

Section 2. Honorary Membership shall be reserved for such distinguished persons as may be deemed worthy of this honor by the Council with 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 8 following. All new members shall be elected directly to Honorary or Active status.

Section 6. Candidates for membership in this Association must be formally nominated and seconded, in an approved manner, by not less than three Active, Senior or Honorary 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 future 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 the Association who has failed of election three times shall automatically cease to be a candidate and may not be renominated until after a lapse of three years.

Section 7. 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; members dropped from the rolls of the Association.

Section 8. Membership may be voluntarily terminated at any time by members in good standing. The Council, acting as 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 9. The Council shall recommend that any Active 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 10. Notwithstanding Section 9, 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, five Councilors and the Editor of the Association shall be a member ex-officio without vote. 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 8.

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 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 IV, give status as officers of the Association to the individual members of an *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 for not more than four additional terms.

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 an interim 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.

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 without vote.

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 twelve members: the President, the Vice President, the Secretary and at least six members-at-large, three representing each of the areas of adult cardiac, pediatric cardiac and general thoracic surgery. Three of these members-at-large shall be appointed each year by the President for a three-year term. Additional committee members shall be appointed for one-year terms. The Editor shall serve as an ex-officio member of the Committee without vote. 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)r 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 his 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 seven members: the President, Secretary, and Treasurer of the Association and four members-at-large, one member being appointed by the President each year to serve a term of four year. The Chairman shall be the member-at-large serving his fourth 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 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 Thoracic Surgical Workforce Committee 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.

Section 11. The Committee on Graduate Education in Thoracic Surgery shall consist of five members appointed by the Council. One member shall be appointed each year for a five-year term. The committee shall review graduate medical education in thoracic surgery and makes its recommendations to the Council to assist in meeting the educational mission of the Association.

Section 12. The Committee on Publications shall consist of the Secretary, the Treasurer, and the Executive Secretary. The Committee shall oversee the business relationships between the Association and the publisher of its journal and maintain liaison among the publisher, the editor, and the Council.

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 to current expenses and shall be placed in a special fund, to be invested and reinvested in legal securities, to be held intact.

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 the 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 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 proceeding meetings of the Association and the Council.
2. Report of the Treasurer of 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 of 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 as 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 consideration 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 $200.00 and shall include a year's subscription to THE JOURNAL OF THORACIC AND CARDIOVASCULAR SURGERY.

Section 3. Senior Members are exempt from dues.

Section 4. The initiation fee for those elected directly to Active Membership shall be $100.00.

Section 5. 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, April, 1995

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
1991-Washington, D.C..... President, Keith Reemtsma
1992-Los Angeles..... President, John A. Waldhausen
1993-Chicago..... President, John L. Ochsner
1994-New York..... President, Aldo R. Castaneda
1995-Boston..... President, Robert B. Wallace

GRAHAM EDUCATION AND RESEARCH FOUNDATION
13 Elm Street, Manchester, Massachusetts 01944, (508) 526-8330

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Vice President Andrew S. Wechsler, M.D., Richmond, Virginia
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EVARTS A. GRAHAMMEMORIAL TRAVELING FELLOWSHIP

The Evarts A. Graham Memorial Traveling Fellowship was established in 1951 by The American Association for Thoracic Surgery. Administered through the Graham Education and Research Foundation, it provides grants to young surgeons from abroad who have completed their formal training in general, thoracic, and cardiovascular surgery. The award allows the recipient to study a year 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 thoracic surgeons internationally. Awards are made to surgeons of unique promise who have been regarded as having potential for later international thoracic surgical leadership. Since the inception of the Graham Fellowship, 43 young surgeons from 23 countries have completed their training at thoracic surgical centers.

1st	1951-52	L.L.Whytehead, M.D., F.R.C.S Winnepeg, Manitoba, CANADA
2nd	1953-54	W.B. Ferguson, M.B., F.R.C.S. Newcastle-upon-tyne, ENGLAND
3rd	1954-55	Lance L. Bromley, M.Chir., F.R.C.S. London, ENGLAND
4th	1955-56	Raymond L. Hurt, F.R.C.S. Radlett Herts, ENGLAND
5th	1956-57	Mathias Paneth, F.R.C.S. London, ENGLAND
6th	1957-58	Peter L. Brunnen, F.R.C.S. Aberdeen, SCOTLAND
7th	1958-59	N.G. Meyne, M.D. Amsterdam, HOLLAND
8th	1960-61	Godrej S. Karai, M.D. Calcutta, INDIA
9th	1961-62	Fritz Helmer, M.D. Vienna AUSTRIA
10th	1962-63	Theodor M. Scheinin, M.D. Helsinki, FINLAND
11th	1963-64	Masahiro Saigusa, M.D. Tokyo, JAPAN
12th	1963-64	Adar J. Hallen, M.D. Uppsala, SWEDEN
13th	1964-65	Stuart C. Lennox, M.D. London, ENGLAND
14th	1964-65	Elias Carapistolis, M.D., F.A.C.S. Thessaloniki, GREECE
15th	1965-66	Gerhard Friehs, M.D. Graz, AUSTRIA
16th	1965-66	Ary Blesovsky, M.D. London, ENGLAND
17th	1966-67	C. Peter Clarke, F.R.A.C.S Fitzroy, AUSTRALIA
18th	1966-67	G.B. Parulkar, M.D. Bombay, INDIA
19th	1967-68	Claus Jessen, M.D.

		Copenhagen, DENMARK
20th	1969-70	Peter Brueke, M.D. Linz-Puchenau, AUSTRIA
21st	1970-71	Michel S. Slim, M.D. New York, New York USA
22nd	1971 -72	Severi Pellervo, Manila, M.D. Kaunianen, FINLAND
23rd	1972-73	Yasuyuki Fujiwara, M.D. Tokyo, JAPAN
24th	1973-74	Marc Roger de Leval, M.D. London, ENGLAND
25th	1974-75	J. J. DeWet Lubbe, M.D. Cape Town, REPUBLIC OF SOUTH AFRICA
26th	1975-76	Mieczyslaw Trenkner, M.D. Gdansk, POLAND
27th	1976-77	Bum Koo Cho, M.D. Seoul, KOREA
28th	1977-78	Alan William Gale, M.D., FRACP, FRACS Sydney, AUSTRALIA
29th	1978-79	Eduardo Otero Goto, M.D. Valencia, SPAIN
30th	1980-81	Richard K. Firmin, M.D. Leicester, ENGLAND
31st	1981 -82	Claudio A. Salles, M.D. Belo Horizonte MG, BRAZIL
32nd	1982-83	Yasuhisa Shimazaki, M.D. Osaka, JAPAN
33rd	1983-84	Georg S. Kobinia, M.D. Klagenfurt, AUSTRIA
34th	1984-85	Aram Smolinsky, M.D. Tel Hashomer, ISREAL
35th	1985-86	Florentine J. Vargas, M.D. Buenos Aires, ARGENTINA
36th	1986-87	Ari L. J. Harjula, M.D. Helsinki, FINLAND
37th	1987-88	Byung-Chul Chang, M.D. Seoul, KOREA
38th	1988-89	Wang Cheng, M.D. Beijing, PEOPLE'S REPUBLIC OF CHINA
39th	1989-90	Christopher John Knott-Craig, M.D. Cape Town, SOUTH AFRICA
40th	1991-92	Ko Bando, M.D., Ph.D. Okayama, JAPAN
41st	1992-93	Timothy E. Oaks, M.D. Hershey, PA, USA
42nd	1993-94	Alain E. Serraf, M.D. Le Plessis Robinson, FRANCE
43rd	1995-96	Cornelius McKown Dyke, M.D. Richmond, VA, USA
44th	1996-97	Monica Robotin-Johnson, M.D. Paris, FRANCE

THE THORACIC SURGERY FOUNDATION FOR RESEARCH AND EDUCATION
ORGANIZATION

The Thoracic Surgery Foundation for Research and Education was established in December of 1989 to identify, encourage and provide for the education and research needs in thoracic surgery. The Foundation is entirely supported through private donations

The American Association for Thoracic Surgery, The Society of Thoracic Surgeons, the Southern Thoracic Surgical Association and The Western Thoracic Surgical Association fully endorse and encourage the work of the Foundation. The sixteen-member Board of Directors is comprised of representatives nominated by these groups.

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GRANTS AND AWARDS

Evaluation of grant and award applications to The Foundation is based on meticulous peer review procedures. Both the merit of the proposal and the leadership promise of the applicant are rated by recognized authorities committed to impartiality.

Research Committee

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Irving L. Kron, M.D. Vaughn A. Starnes, M.D.
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Lynda L. Mickleborough, M.D.

Funding programs of The Foundation are:
I. Individual Research Investigator Grants
II. Research Fellowship Awards
III. Career Development Awards
IV. Alley-Sheridan Scholarships

Thoracic Surgery Foundation 1996 Awardees

Robert S. Poston, Jr., M.D., Stanford University Medical Center
Generation of Allograft Specific Tolerance with Combination Ex Vivo Antisense ICAM-1 and Systemic Anti-LFA MAB.

The Thoracic Surgery Foundation Research Fellowship Award

Si M. Pham, M.D., University of Pittsburgh
Nitric Oxide In Allograft Vasculopathy.

The Thoracic Surgery Foundation Research Investigator Grant
Mary C. Mancini, M.D., Louisiana State University Medical Center
The Role of Platelet Derived Growth Factor in Allograft Vascular Disease.

The Nina S. Braunwald Career Development Award
Andrew Sherman, M.D., Northwestern University Medical School
Gene Expression of Sarcoplasmic Reticular Regulatory Proteins in Myocardial Hibernation

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 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

ALFRED BLALOCK RESEARCH SCHOLARSHIP

"Efficacy and Toxicity of a New Blood Substitute: Polymerized, Ultra-Pure, Stroma-Free Bovine Hemoglobin"
1988-1990 Gus J. Vlahakes, M.D.
Massachusetts General Hospital and Harvard Medical School

JOHN H. GIBBON, JR., RESEARCH SCHOLARSHIP

"Load-Independent Assessment of Cardiac Performance by Noninvasive Means"
1990-1992 Donald D. Glover, M.D.
Duke University Medical Center

ALTON OCHSNER RESEARCH SCHOLARSHIP

"Experimental Cardiac Graft Arteriosclerosis: Pathogenesis and Prevention of Lesion Development"
1992-1994 David H. Adams, M.D.
Brigham and Women's Hospital

ROBERT E. GROSS RESEARCH SCHOLARSHIP

"Role of Intra-Cellular Messenger cAMP and cGMP in Organ Preservation"
1994-1996 Melmet C. Oz, M.D., Columbia-Presbyterian Medical Center
"Development of a Model of Chronic Pulmonary Rejection in Miniature Swine"
1994-1996 Toralf Mauritz Sundt, III, M.D.
Washington University School of Medicine

JOHN ALEXANDER RESEARCH SCHOLARSHIP

"Strategies to Prevent Hyperacute Rejection of the Pig Lung by Human Blood"
1996-1998 Richard Norris Pierson, III
Vanderbilt University Medical Center