1997 ANNUAL MEETING PROGRAM

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1997 Annual Meeting
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DEVELOPING THE ACADEMIC SURGEON SYMPOSIUM

1997

AATS

Postgraduate

Course

Congenital Heart Disease

Sunday, May 4, 1997

8:00 a.m. - 4:30 p.m.

Washington Ballroom Sheraton Washington Hotel
Washington, DC

Objectives

The 1997 Postgraduate Course in Congenital Heart Disease will address the following topics: Complete common atrioventricular (AV) canal defects including management of the common atrioventricular valve, the problem of the small left ventricle in complete and partia AV canal, the decision-making process regarding single ventricle or two-ventricle repair in unbalanced AV canal and long-term outcome and reoperation rates. In addition, various topics related to postoperative care, including management strategies for single ventricle palliation in hypoplastic left heart syndrome, the management of postoperative arrhythmias, and strategies for improved postoperative care including early extubation and use of care paths in congenital heart surgery will be covered. Finally, a comprehensive discussion of approaches to the management of pulmonary atresia with ventricular septal defect and aortopulmonary colaterals will include video presentations of surgical techniques. The current status of knowledge of the molecular basis of congenital heart disease will be presented by a national expert. The course will provide attendees the opportunity to interact with recognized experts involved in 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. At the completion of the course, participants should have an enhanced knowledge of the diagnosis and management options in patients with complex congenital heart anomalies.

	Registration The registration fee is $100 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

Sheraton Washington Hotel - Washington Ballroom

7:00 a.m. **REGISTRATION AND CONTINENTAL BREAKFAST**

8:00 a.m. **INTRODUCTION**

Thomas L. Spray, M.D., Course Chairman

Session I ATRIOVENTRICULAR CANAL DEFECTS

Moderators: Thomas L. Spray, M.D.

Marc R. de Leval, M.D.

8:05 a.m. **Repair of CAVC in Infancy - Current Results**

Thomas L. Spray, M.D., Philadelphia, PA

8:25 a.m. **Partial AVC and the Problem of the Small LV**

Peter Manning, M.D., Cincinnati, OH

8:45 a.m. **"Unbalanced" AVC - Repair vs. Fontan**

Marc R. de Leval, M.D., London, UK

9:05 a.m. **Reoperation after Repair of AVC**

Francisco J. Puga, M.D., Rochester, MN

9:25 a.m. **Discussion**

9:45 a.m. **Refreshment Break**

Session II POSTOPERATIVE MANAGEMENT ISSUES

Moderator: Erie H. Austin, III, M.D.

10:30 a.m. **Critical Care Pathways for Congenital Heart Surgery**

John E. Mayer, Jr., M.D., Boston, MA

10:50 a.m. **Early Extubation After Cardiac Surgery in Neonates and Small Infants**

Lawrence S. Fox, M.D., Fort Worth, TX

11:10 a.m. **Modified Ultrafiltration - Effects on Morbidity**

J. William Gaynor, M.D., Philadelphia, PA

11:30 a.m. **Postoperative Management Strategies after Norwood Operation**

Erie H. Austin, III, M.D., Louisville, KY

11:50 a.m. **Current Management of Postoperative Arrhythmias - JET**

Larry Rhodes, M.D., Philadelphia, PA

12:10 p.m. **Discussion**

12:30 p.m. **Luncheon**

Session III MANAGEMENT OF TETRALOGY OF FALLOT WITH PULMONARY ATRESIA

Moderators: Frank L. Hanley, M.D.

Thomas L. Spray, M.D.

1:45 p.m. **Initial Iterventions (Video)**

Roger B.B. Mee, M.D., Cleveland, OH

2:05 p.m. **Staged Unifocalization (Video)**

Hillel Laks, M.D., Los Angeles, CA

2:25 p.m. **One-Stage Repair (Video)**

Frank L. Hanley, M.D., San Francisco, CA

2:50 p.m. **Homograft Reconstruction with Catheter Interventions**

Richard A. Jonas, M.D., Boston, MA

3:10 p.m. **Discussion and Case Presentations**

Session IV BASIC SCIENCE LECTURE

3:40 p.m. **Towards a Molecular Understanding of Congenital Heart Disease**

Arnold W. Strauss, M.D., St. Louis, MO.

5:00 p.m. **RECEPTION IN EXHIBIT HHALL**

1997 AATS General Thoracic Surgery Symposium *Sponsored in cooperation with The General Thoracic Surgical Club*	**Objective** The 1997 General Thoracic Surgery Symposium will begin with a discussion of complex esophageal problems The management of T3N1 esophageal cancer will include two presentations: surgery alone versus preoperative adjuvant therapy prior to resection and chemoradiation alone. A panel discussion with audience participation will follow. The Palliation of unresectable esophageal cancer will then be discussed. Management of esophagea perforation will complete the program on esophageal problems. The morning session will conclude with a review of surgery for metastatic disease to the lung. Session II will be devoted to pleural space problems: following infection (empyema), post-lobectomy, and post-pneumonectomy empyema. Again, a panel discussion will facilitate audience participation.

Sunday, May 4, 1997 **8:00 a.m. - 5:00 p.m.** **North Sheraton Ballroom,** **Sheraton Washington** **Hotel** ***Washington, DC***	Session III will cover controversies in pulmonary surgery: postoperative adjuvant therapy for lung cancer, chest trauma, and thoracoscopic lobectomy. Session IV will feature issues for the thoracic surgeon in the managed care environment. This symposium is designed for the practicing thoracic surgeon and provides attendees the opportunity to interact with individuals experienced in the management of difficult problems, some of which are infrequently seen. Topics will be illustrated by using clinical cases, and management issues will be stressed. Finally, an appraisal of our common preoperative, hospital, and postoperative practices will be reviewed from the managed care perspective. At the completion of the symposium, participants should have an enhanced knowledge of the diagnosis and management of complex esophageal problems, a better understanding of the intricacies of pleural space problems, and current information on several areas of controversy in thoracic surgery. In addition, participants will be better able to practice their specialty in the current as well as forthcoming managed care environment. **Registration** The registration fee is $100 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

Sheraton Washington Hotel - North Sheraton Ballroom

7:00 a.m. **REGISTRATION AND CONTINENTAL BREAKFAST**

8:00 a.m**. INTRODUCTION AND WELCOME**

Larry R. Kaiser, M.D., Co-Chair

Carolyn E. Reed, M.D., Co-Chair

Session I MANAGEMENT OF T3N1 ESOPHAGEAL CANCER

Moderator: Carolyn E. Reed, M.D.

8:05 a.m. **Surgery vs. Adjuvant Therapy/Surgery**

Richard F. Heitmiller, M.D., Baltimore, MD

8:25 a.m. **Combined Modality Therapy of Esophageal Cancer**

David J. Sugarbaker, M.D., Boston, MA

8:45 a.m. **PANEL DISCUSSION**

9:00 a.m. **Palliation of Unresectable Esophageal Cancer**

Darroch W.O. Moores, M.D., Albany, NY

9:30 a.m. **Managing the Patient with Esophageal Perforation**

Alex G. Little, M.D., Las Vegas, NV

10:00 a.m. **COFFEE BREAK**

10:30 a.m. **Surgery for Metastatic Disease to the Lung**

Joe B. Putnam, Jr., M.D., Houston, TX

Session II SPACE PROBLEMS

Moderator: Larry R. Kaiser, M.D.

11:00 a.m. **Management of Empyema**

Douglas E. Wood, M.D., Seattle, WA

11:20 p.m. **Prolong Air Leak and Post-Lobectomy Space Problems**

Jean DesLauriers, M.D., Sainte-Foy, Quebec, Canad

11:40 p.m. **Post-Pneumonectomy Empyema**

Willard A. Fry, III, M.D., Evanston, IL

12:00 noon **PANEL DISCUSSION**

12:30 p.m. **LUNCHEON**

Session III

Moderator: Larry R. Kaiser, M.D.

2:00 p.m. **Adjuvant Therapy of N1 and N2 Lung Cancer Following Resection**

Steven M. Keller, M.D., New York, NY

2:30 p.m. **Chest Trauma/Flail Chest/Airway Disruption/Lung Contusion**

Kenneth L. Mattox, M.D., Houston, TX

3:00 p.m. **Thoracoscopic Lobectomy vs. Muscle-Sparing Lobectomy**

Malcolm DeCamp, Jr., M.D., Boston, MA

3:30 p.m. **REFRESHMENT BREAK**

Session IV THORACIC SURGERY IN THE MANAGED CARE ENVIRONMENT: COST EFFECTIVENESS & OUTCOMES

Moderator: Carolyn E. Reed, M.D.

4:00 p.m. **Pre-Operative Staging of Lung Cancer: What Tests are Necessary?**

Gerard A. Silvestri, M.D., Charleston, SC

4:20 p.m. **Providing Quality Care in a Cost-Constrained Environment**

J. Sanford Schwartz, M.D., Philadelphia, PA

4:40 p.m. **Thoracic Surgeryin the Managed Care Environment: Follow-up Management**

Leslie J. Kohman, M.D., Syracuse, NY

5:00 p.m. **RECEPTION IN EXHIBIT HALL**

1997 AATS Adult Cardiac Surgery Symposium

Frontiers in Cardiac Surgery

Sunday, May 4, 1997

8:00 a.m. - 5:00 p.m.

South Sheraton Ballroom Sheraton Washington

Hotel

Washington, DC

Objective

The 1997 Adult Cardiac Surgery Symposium will feature the evolving frontiers in cardiac surgery covering the following specific topics: minimally invasive aortic valve and mitral valve procedures as well as minimally invasive coronary revascularization with and without cardiopulmonary bypass.

In addition, the afternoon session will cover the specific topics of arterial revascularization with emphasis on utilization of the radial artery, gastroepiploic artery, and strategies for total arterial revascularization in patients undergoing reoperation (including interior epigastric artery utilization). Finally, techniques in ventricular remodeling for chronic left ventricular failure will be presented.

This symposium is designed for the practicing cardiac surgeon. At the completion of this symposium, participants should have an enhanced knowledge of the procedures using state-of-the-art techniques for minimally invasive aortic valve and mitral valve procedures, minimally invasive coronary revascularization with and without cardiopulmonary bypass, arterial revascularization with and without coronary bypass, arterial revascularization with emphasis on utilization of the radial artery, gastroepiploic artery, total arterial revascularization in patients undergoing reoperation (including interior epigastric artery utilization), and ventricular remodeling for chronic left ventricular failure.

Registration

The registration fee is $100 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

Sheraton Washington Hotel - South Sheraton Ballroom

7:00 a.m. **REGISTRATION AND CONTINENTAL BREAKFAST**

Session I MINIMALLY INVASIVE AORTIC VALVE PROCEDURE

8:00 a.m. **Right Paramedian Approach**

Lawrence H. Cohn, M.D., Boston, MA

8:15 a.m. **Mini Sternotomy Approach to Aortic Valve Repair/ Replacement**

Steven R. Gundry, M.D., Loma Linda, CA

8:30 a.m. **Critique: Limitations and Pitfalls**

Frank C. Spencer, M.D., New York, NY

Session II MINIMALLY INVASIVE MITRAL VALVE PROCEDURE

8:45 a.m. **Minithoracotomy**

Delos M. Cosgrove, M.D., Cleveland, OH

9:00 a.m. **Minithoracotomy with Port Access**

Steven B. Colvin, M.D., New York, NY

9:15 a.m. **Critique: Limitations and Pitfalls**

Albert Starr, Portland, OR

9:30 a.m. **PANEL DISCUSSION - QUESTIONS AND ANSWERS Minimally Invasive Valve Procedures**

10:00 a.m. **REFRESHMENT BREAK**

Session III INITIAL MINIMALLY INVASIVE CORONARY REVASCULARIZATION

10:30 a.m. **Thoractomy Without Pump**

Valavanur A. Subramanian, M.D., New York, NY

10:45 a.m. **Ministernotomy Without Pump**

Kit V. Arom, M.D., Minneapolis, MN

11:00 a.m. **Revascularization With Port Access**

Thomas A. Burdon, M.D., Stanford, CA

11:15 a.m. **Critique: Limitations and Pitfalls**

Denton A. Cooky, M.D., Houston, TX

11:30 a.m. **PANEL DISCUSSION - QUESTIONS AND ANSWERS**

Minimally Invasive Coronary Revascularization

12:00 noon **LUNCHEON**

Session IV SURGERY FOR ISCHEMIC HEART DISEASE

1:00 p.m. **Rationale for Total or Partial Arterial Revascularization Including Internal Mammary**

Noel L. Mills, M.D., New Orleans, LA

1:15 p.m. **Radial Artery - Harvesting, Preparation and Use**

Hendrick B. Earner, M.D., St. Louis, MO

1:30 p.m. **Gastroepiploic Artery - Harvesting, Preparation and Use**

John Pym, M.B., F.R.C.S., Kingston, Ontario, Canada

1:45 p.m. **Strategies for Total Arterial Revascularization Including Inferior Epigastric in Patients Undergoing Reoperation for Coronary Disease**

Bruce W. Lytle, M.D., Cleveland, OH

2:00 p.m. **Critique: Limitations and Pitfalls**

O. Wayne horn, M.D., New York, NY

2:15 p.m. **PANEL DISCUSSION - QUESTIONS AND ANSWERS**

Surgery for Ischemic Heart Disease

2:45 p.m. **REFRESHMENT BREAK**

3:15 p.m. **Ventricular Remodeling for Left Ventricular Failure**

Randes J. Batista, M.D., Curitiba, Brazil

3:45 p.m. **Critique: Limitations and Pitfalls of Remodeling Including Alternative Surgical Procedures**

Craig R. Smith, M.D., New York, NY

4:15 p.m. **PANEL DISCUSSION - QUESTIONS AND ANSWERS**

5:00 p.m. **RECEPTION IN EXHIBIT HALL**

The American Association
For Thoracic Surgery
77TH ANNUAL MEETING
May 4-7, 1997
Sheraton Washington Hotel
Washington, DC

MONDAY, MAY 5, 1997

8:00 a.m. BUSINESS SESSION (Limited to Members) Sheraton Ballroom
8:15 a.m. PLENARY SCIENTIFIC SESSION

Sheraton Ballroom

Moderators: David B. Skinner, M.D.

James L. Cox, M.D.

1. OUTFLOW OBSTRUCTION AFTER THE ARTERIAL SWITCH OPERATION: A MULTI-INSTITUTIONAL STUDY.

William G. Williams, M.D., Jan M. Quaegebeur, M.D., John W. Kirklin, M.D. and Eugene H. Blackstone, M.D.

Toronto, Ontario, Canada; New York, New York and Birmingham, Alabama

Discussant: Frank L. Hanley, M.D.

Whether or not right-sided and left-sided outflow obstruction immutably accompanies the arterial switch operation to some degree is unknown, as are factors that may decrease its prevalence. This was studied in 514 neonates undergoing an arterial switch operation for simple transposition or transposition with ventricular septal defect entering 23 institutions before 15 days of age between January 1, 1985 and March 1, 1989. Each patient has been followed yearly.
The time-related freedom from percutaneous or surgical intervention for obstruction across time is shown in the first figure. The results of a multi-variable analysis of right-sided events is shown in the table. The influence of the coronary pattern and the improvement with date of operation are shown in the second figure. The "base prevalence" predicted at the end of the experience in the best subset of patients is contrasted with those receiving coronary excision away from the transection site in the third figure.

Inferences (derived assumptions): 1) There is a "base valence" (5%-10%) of the need for reintervention for right-sided obstruction, which is predominately late postoperatively. *2)* When the enlargement of the base of the pulmonary trunk (PT) effected by the operation is less (for example, when the coronary explant is away from the transection site or when the left coronary artery comes from sinus 2), the prevalence is increased. *3)* Apparently mild and often overlooked congenital variability of the right ventricular outflow tract and "outflow valve" may occasionally yield a morphology which increases the prevalence (albeit more proximal). *4)* Apparently mild and often overlooked variability in the "LeCompte maneuver" (performed in all but 20 patients in this study, with 2 right-sided events) may increase the prevalence (variability less when the LeCompte is not done), albeit more distal in the pulmonary artery. *5)* Inexperience and operator variability may result in a "less than optimal" PT reconstruction which increases this prevalence (therefore, a date of operation and institution variables in the analysis). *6)* These same types of variability probably affect the aortic root, but its native characteristics plus higher distending pressure make the basic prevalence considerably less than that for the "right side".

Incremental Risk Factors for Intervention Right-Sided Obstruction	**Hazard phase**	
	Early	**Late**
Left coronary artery arising from sinus 2	*P* = .002	
Coronary explant away from transection site		*P* =.01
Institution X	*P* = .0003	
Institution Y	*P* = .0002	

Institution Z		***P* = .01**
Earlier date of arterial switch	*P* = .01	

*By invitation

§2. VALVE REPAIR VERSUS REPLACEMENT FOR MITRAL INSUFFICIENCY: WHEN IS A MECHANICAL VALVE STILL INDICATED?

Eugene A. Grossi, M.D.*, Aubrey C. Galloway, M.D., Greg H. Ribakove, M.D.*, Alfred T. Culliford, M.D., Rick Esposito, M.D.*, Julie Delianides, M.A.*, Patricia Buttenheim, M.A.*, F. Gregory Baumann, Ph.D.*, Frank C. Spencer, M.D. and Stephen B. Colvin, M.D.*

New York, New York

Discussant: Gary W. Akins, M.D.

While many advantages of mitral valve reconstruction are well known, the specific subgroup of patients in which mechanical valve replacement offers superior long term results remains uncertain. This study addressed this issue by examining the long term results of mitral valve surgery in patients (pts) with mitral insufficiency who received either a St. Jude valve **(SJV)**(n = 516) or a mitral reconstruction with ring annuloplasty **(MVR)** (n = 725) between 1980 and 1995. Overall operative mortality was 7.2% in the **SJV** pts and 5.4% in the **MVR** pts (NS); isolated mortality was 2.5% in the **SJV** pts and 2.2% in the **MVR** pts (NS). Three hundred and forty pts had a follow-up interval > 5 yrs; 51 pts had a follow-up interval > 10 yrs (mean follow-up = 39.8 months; 98.5% complete). Actuarial analysis of freedom from late cardiac death **(LCD)**, reoperation **(REOP),** and all valve-related complications **(AVC)** is shown below:

Freedom from:	5 Years (%)		8 Years (%)		p
	SJV	MVR	SJV	MVR	
All Patients					
LCD	87.8	90.1	86.8	84.1	NS
LCD & REOP	84.2	82.9	81.5	73.1	NS
LCD, REOP, & AVC	84.0	74.4	79.9	64.8	NS
Isolated NonRheumatic MVR					
LCD	91.7	98.1	91.7	95.4	<.05
LCD & REOP	85.4	93.6	85.4	88.3	.08
LCD, REOP, & AVC	85.0	87.5	82.2	82.1	NS
Isolated Rheumatic MVR					
LCD	91.7	98.1	91.7	98.1	.13
LCD & REOP	85.4	84.0	85.4	84.0	NS
LCD, REOP, & AVC	85.0	71.5	82.2	71.5	.04
Concomitant Valve					
LCD	89.1	80.0	89.1	60.5	NS
LCD & REOP	88.2	72.2	88.2	51.0	<.01

LCD, REOP, & AVC	88.0	67.2	83.7	50.8	<.001	Cox

multivariate analysis in isolated, non-rheumatic mitral valve pts revealed that **MVR** was independently associated with increased survival from late cardiac death (p = .04) irrespective of preoperative NYHA class. In rheumatic and multiple valve pts **SJV** offered better freedom from late cardiac death, reoperation and all valve-related complications. **MVR** is preferred for isolated, non-rheumatic pts, whereas **SJV** gives improved late results in rheumatic and multiple valve pts.

§Authors have a relationship with Baxter, St. Jude Medical and Medtronics

*By invitation

3. REOPERATIVE TRACHEAL RESECTION AND RECONSTRUCTION FOR FAILED REPAIR OF POSTINTUBATION STENOSIS.

Dean M. Donahue, M.D.*, Hermes C. Grillo, M.D., John C. Wain, M.D.*, Cameron D. Wright, M.D.* and Douglas J. Mathisen, M.D.

Boston, Massachusetts

Discussant: F. Griffith Pearson, M.D.

Primary tracheal resection and reconstruction for postintubation stenosis restores airway continuity and avoids life-long tracheostomy. Success can be expected in over 90% (408/450) of cases with a low incidence of morbidity (15.1%, 68/450) and mortality (2.2%, 10/450). When primary resection fails, there is still an opportunity for restoration of the airway, but the operation is of much greater complexity with the potential for added morbidity and mortality. Timing of surgery, airway management, and attention to technical detail are critical to successful reoperation.

We have had experience with 69 patients undergoing reoperation for tracheal stenosis following failed primary repair. Temporary airway management was accomplished with T-tubes in 18 and tracheostomy in 14. The amount of trachea removed at the initial operation was 3.5 cm (range 1.0 to 5.5). The average amount of trachea resected at the reoperation was 3.4 cm (range 1.0 to 6.0). A release maneuver (laryngeal = 17, hilar = 1) was employed in 18 patients (26.1%), compared to 7.1% (32/450) in our series of intial repairs. There were 14 major complications (20%) and 15 minor complications (22%). The major complications were restenosis (N = 3), anastomotic granulations (N = 3), sternal infection (N = 3), dehiscence (N = 2), pneumonia (N = 2), and temporary vocal cord paralysis (N=1). There were four failures (5.8%) requiring permanent T-tube or tracheostomy. Two patients required a second reoperation for restenosis (2.8%), both with good long term results. There were two deaths in the series (2.8%). Successful reconstruction of the airway was achieved in 88.4% (good = 51, satisfactory = 10) at a mean follow-up of over 3 years. Reconstruction following failed repair for tracheal stenosis is possible by adhering to certain principles and attention to the technical details of the operation.
*By invitation

4. SUSTAINED RELIEF TO THE LEFT VENTRICLE IN

HYPERTROPHIC OBSTRUCTIVE CARDIOMYOPATHY BY EXTENDED MYECTOMY AND RECONSTRUCTION OF THE SUBVALVULAR MITRAL APPARATUS.

Friedrich A. Schoendube, M.D., M.S.*, Heinrich G. Klues, M.D.*, Sebastian Reith, M.D.*, Andreas Franke, M.D.*, Frank A. Flachskampf, M.D.*, Peter Hanrath, M.D.* and Bruno J. Messmer, M.D.

Aachen, Germany

Discussant: Robert B. Wallace, M.D.

Background: Classical myotomy-myectomy for patients with severely symptomatic hypertrophic obstructive cardiomyopathy (HOCM) represents inherent risks of ventricular septal defect or incomplete relief. A modified surgical technique with extended myectomy and reconstruction of the subvalvular apparatus was developed to allow safe and lasting relief of the left ventricular outflow tract obstruction.

Material and Methods: Between 1979 and 1996, 74 patients (45 male), age 49 (15-73) years were operated upon HOCM. All patients were severely symptomatic despite adequate medication. Pre-operative echocardiographic studies showed moderate mitral regurgitation in 27% and severe mitral regurgitation in 5% of the patients. All patients had a significant SAM (3-4+). Concommittant surgical procedures were performed in 17 patients: CABG 11/74 (15%), MV-Repair: 2/74 (3%), ICD: 1/74 (1.5%), Ao. asc. aneurysm: 1/74 (1.5%), RV-myectomy: 2/74 (3%).

Results: Perioperative mortality (30 days) was 0%, hospital mortality concerned one patient with septic multi-organ failure (1.3%). Peri-operative non-fatal complications included one transient cerebral attack with full recovery, three patients required permanent pacemaker therapy. No ventricular septal defect occurred in the whole series. Long-term follow-up (73/74 patients / 98%) comprised a total of 434 patient-years (py) (mean 84, 1-204 months). Linear mortality was 1.4%/py (7/73), ten year survival was 86 ± 7% (5-year 94%). No sudden cardiac death occurred during follow-up. Echoes were performed for 65/66 patients at latest follow-up (1996). Normal LVEDD (46 ± 6 mm) and LVESD (32 ± 8 mm) were shown, septal thickness was particularly small (13.4 ± 4 mm) for HOCM patients (pre-op: 25 ± 5 mm, $p < 0.05$). None of the patients showed at follow-up relevant systolic anterior movement of the mitral valve (SAM) and no significant mitral regurgitation was observed. Eleven patients had pre- and post-operative 3-D reconstruction from multiplane transesophageal echoes and showed an increase of the minimal cross-sectional area of the LVOT from 1.1 ± 1.0 Cm^2 pre-operatively to 4.4 ± 2.7cm^2 postoperatively ($p < 0.05$). Maximal deviation of the mitral leaflets fell from 15 + 7 mm pre- to 7 ± 8 mm postoperatively ($p < 0.05$) as consequence of subvalvular reconstruction. Functional capacity of the patients at long-term follow-up is still excellent for the majority of patients (65%) in NYHA class I or II. Patients being in class III (NYHA) are all in their 8th decade of life and nobody was in class IV.

Conclusion: Transaortic extended myectomy and reconstruction of the subvalvular mitral apparatus has proven to be a highly effective therapy for patients with severely symptomatic hypertrophic obstructive cardiomyopathy because: 1) obstruction to left ventricular outflow tract is reliably eliminated, 2) long-term results show an excellent functional and hemodynamic status of the patients, 3) annual mortality rate is low and no sudden cardiac death occurred during follow-up.

9:40 a.m. EVARTS A. GRAHAM MEMORIAL TRAVELING FELLOW PRESENTATION

Monica Robotin-Johnson, M.D., Sydney, Australia

9:45 a.m. INTERMISSION - VISIT EXHIBITS

*By invitation

10:30 a.m. PLENARY SCIENTIFIC SESSION

Sheraton Ballroom
Moderators: Floyd D. Loop, M.D.

James L. Cox, M.D.

5. LATE RESULTS OF 151 AORTIC VALVE PRESERVING OPERATIONS IN PATIENTS WITH ANEURYSMS OF THE ASCENDING AORTA AND ROOT.

Petra J. Gehle, M.D.*. Rosemary C. Radley-Smith, F.R.C.P., M.B.B.S.* and Magdi H. Yacoub, F.R.C.S., Ph.D.

London, England

Discussant: Tirone E. David, M.D.

A technique of excising aneurysms of the ascending aorta and root with resuspension of the aortic valve and implantation of the coronary ostia into a dacron graft was devised in 1979 and used by one surgeon whenever possible thereafter. The aortic sinuses are excised to within one millimeter of the aortic anulus. A dacron tube of the appropriate size is fashioned to have three tongue-shaped processes to match the three reconstituted sinuses. To date 151 patients (70% of all patients undergoing resection of aneurysm of the ascending aorta) were operated on using this technique. Their ages ranged from 2 to 77 years (mean 43); 46 patients were female, 105 male; 64 patients had skeletal manifestations of Marfan syndrome. Nearly one third (49 patients) presented with acute or chronic type A dissection. Emergency surgery was required in 32 patients. Additional procedures such as partial or complete arch replacement, coronary artery revascularisation or mitral valve repair were performed in 53 patients. In all there were eight (5.3%) early deaths (1.3% for elective and 12.2% for emergency operations and dissections), and 13 late deaths during a follow-up period varying from 1 to 209 months (mean 79). There were no early deaths in the 109 electively operated patients since 1986. The actuarial survival at 5, 10 and 15 years was 92.4%, 87.2% and 70.4%, respectively. Nine patients (7 of whom are Marfan patients) required re-operation (aortic valve replacement or re-do repair) 4 months to 12 years (mean 5.4) after operation. There were no early deaths in this group. The probability of freedom from re-operation at 5 and 10 years was 95.7% and 90.7%, respectively.

There were no instances of infective endocarditis or thromboembolic complications. No anticoagulants were used. Echocardiography showed reduction in left ventricular end systolic and end diastolic dimensions which was maintained throughout the period of follow-up except in those patients who required re-operation. Mild or no aortic regurgitation was demonstrated in 93%, moderate in 5.1% and moderate to severe in one patient who is currently awaiting re-operation.

It is concluded that valve preserving operations are possible in a large proportion of patients with aneurysms of the ascending aorta and that the medium and "long" term results are encouraging.
*By invitation

6. IS RETURN OF ANGINA AFTER CABG IMMUTABLE ?

Paul T. Sergeant, M.D., Eugene H. Blackstone, M.D., Bart Meyns, M.D.*

Leuven, Belgium and Birmingham, Alabama

Discussant: Bruce W. Lytle, M.D.

Since today survival after either surgery or angioplasty seem similar for a wide spectrum of coronary patients, the efficacy of surgery in long term relief of angina assumes higher priority. Therefore time-related return of angina, without infarct or death the same day, was studied in a multivariable parametric analysis of a consecutive series of 9600 patients after primary isolated CABG (Jan./71-Jan./92). The common closing date (Jan./93) follow-up was 99.9 % complete. Extensive arterial revascularisation was used since 1972 with different prevalence over time.

The 1-yr., 5-yr., 10-yr., 15-yr. and 20-yr. freedom from angina was 94%, 82%, 61%, 38% and 21% respectively. A two-phase hazard function was identified. Early return of angina, rapidly declining after two months, was influenced by demographic variables, preoperative anginal status, distribution of coronary disease, vascular comorbidity, but more strongly by procedural (e.g. extensive arterial revascularisation) and institutional variables. Late return of angina, rising after two years and for the whole extent of the follow-up, was influenced by demographic variables, anginal status, left ventricular function, distribution of coronary disease, very strongly by coexisting cardiac and non-cardiac comorbidity (such as obesity, diabetes and preoperative lipid levels), but, in contrast with early return of angina, moderately by procedural variables. The 2-yr freedom from angina for a median patient with 4 distals was 94.7%, 95.5%, 96.0% and 96.3% with 0, 1, 2 and 3 arterial anastomoses. The 15-yr freedom from angina for a median patient with 4 distals was 38.1%, 42.0%, 45.7% and 49.2% with 0, 1, 2 and 3 arterial anastomoses.

Thus early return of angina is minimized by use of procedural techniques such as arterial grafts, but reduction of late angina return requires control of non-cardiac comorbidity.

11:15 a.m. PRESIDENTIAL ADDRESS

Shaping the Revolution: Thoracic Surgeons and Something More.

David B. Skinner, M.D., New York, New York.

12:00 p.m. ADJOURN FOR LUNCH - VISIT EXHIBITS

*By invitation

MONDAY AFTERNOON, MAY 5, 1997

1:30 p.m. PLENARY SCIENTIFIC SESSION

Sheraton Ballroom

Moderators: Mortimer J. Buckley, M.D.

Andrew S. Wechsler, M.D.

7. PEDIATRIC AND ADULT LUNG TRANSPLANTATION FOR CYSTIC FIBROSIS.

Eric N. Mendeloff, M.D.*, Charles B. Huddleston, M.D.*, George B. Mallory, M.D.*, Alan Cohen, M.D.*, Stewart Sweet, M.D.*, Burt P. Trulock, M.D.*, Sudhir Sundaresan, M.D.*, Joel D. Cooper, M.D. and G. Alec Patterson, M.D.

St. Louis, Missouri

Discussant: Frank C. Detterbeck, M.D.

It has been suggested that lung transplantation for cystic fibrosis is fraught with increased morbidity and mortality. Since 1989, we have performed 103 bilateral sequential lung transplants in patients with cystic fibrosis (46 pediatric, 48 adult) including 9 redo transplants. Mean age at transplantation for the entire population was 21 ± 10 years (13 ± 3 years in the pediatric population and 29 ± 8 years in the adult group) and mean weight was 43 ± 15 Kg (32 ± 10 Kg in the pediatric and 52 ± 12 in the adult group). Average waiting periods from time of listing to time of transplantation were 231 ± 175 days and 362 ± 210 days in the pediatric and adult populations, respectively. All transplants except one in the pediatric age group were performed using cardiopulmonary bypass while this modality was employed selectively in the adults (23%). Hospital mortality for the entire population was 2.1%, with both early deaths occurring in the adult age group. Bronchial anastomotic complications requiring dilation, stent placement or surgery took place with equal frequency in the pediatric and the adult population occurring in 15 of 206 (7.3%) anastomoses at risk. At an average length of follow-up of 2.1 ± 1.6 years. The one and three year actuarial survival for the entire group were 80% and 59%, with no significant difference between the pediatric and adult age groups. Mean forced expiratory volume in 1 second at the time of listing for transplant was 25 ± 9% for the entire population, while average values at 1 month and 1 year post-transplantation improved to 54 ± 17% and 79 ± 35%, respectively. There was an average of 1 episode of acute rejection per patient-year, with the majority occurring in the first 6 months post-transplant. Actuarial freedom from bronchiolitis obliterans (biopsy proven or by clinical criteria) was 63% at 3 years. The subset who underwent redo transplantation consisted entirely of patients in the pediatric age range (<18 years old), and the mean time from the initial transplant to re-transplantation was 56 ± 44 weeks. The combined early and late mortality in this group was 44%. Eight living related donor transplants have been performed (4 as primary transplants and 4 as redo transplants) with an early survival of 87.5%. Lung transplantation in patients with end stage cystic fibrosis can be performed with low peri-operative mortality and with complication rates similar to those seen in pulmonary transplantation for other disease entities.
*By invitation

8. MID-TERM RESULTS AFTER MINIMALLY INVASIVE CORONARY SURGERY (LAST OPERATION).

Antonio M. Calafiore, M.D.*, Giovanni Teodori, M.D.*, Gabriele Di Giammarco, M.D.*, Giuseppe Vitolla, M.D.*, Angela IacÃ³, M.D.*, Teresa lovino, M.D.* and Sergio Cirmeni, M.D.*

Chieti, Italy

Sponsored by: Tomas Antonio Salerno, M.D., Buffalo, New York

Discussant: Steven R. Gundry, M.D.

Background. Left internal mammary artery (LIMA) to left anterior descending (LAD) artery via a left anterior small thoracotomy (LAST) is a recently proposed procedure specially designed to be effective, reproducible and to increase patients' comfort. We reviewed our experience to evaluate if these goals could be considered achieved.

Methods. From November 1994 to October 1996 366 patients (pts) underwent LIMA to LAD grafting via a LAST. One hundred eighty-two pts (49.8%) had a single LAD disease, in 184 LAD lesion was part of multiple vessel disease. High risk factors for, cardiopulmonary bypass were present in 51 pts (13.9%). Intravenous diltiazem was infused during the operation. LIMA was harvested for the length enough to reach the LAD.

Results. One hundred forty-seven pts (40.1%) were extubated in the OR or in the 1st hour. Mean ICU stay was 4.2 h; mean postoperative in hospital stay was 53 h; 30 day mortality was 0.8% (3 pts); late mortality was 1.1% (4 pts). All pts who died but one had a patent anastomosis. Eighteen pts were reoperated on early (< 30 d) and 7 late (> 30 d) due to conduit or anastomotic malfunction; 4 were reoperated on with patent anastomosis for progression of disease (3) or pericarditis (1). Four pts had angina, 3 due to anastomotic stenosis (spontaneously reversed in 2) and 1 due to progression of disease. A 5th pt had dispnea; LIMA was patent but apical dyskinesia, was present. A patent and well functioning anastomosis, checked by angiography or stress doppler flow assessment, was obtained in 340 pts (92.9%). Twenty-three months after surgery, actuarial survival was 98.0% (100% in 1-v disease and 96.4% in 2/3-v disease, p = ns); event free was 87.9% (90.9% in 1-v disease and 85.8 in 2/3-v disease, p = 0.006). In the last 130 pts (from April 1, 1996), with increased experience and better instruments, a patent well functioning anastomosis was obtained in 128 pts (98.5%); 7 month survival was 99.1% (100% in 1-v disease and 97.9% in 2/3-v disease, p = ns) and event free survival was 93.5% (95.1% in 1-v disease and 91.5% in 2/3-v disease, p = ns).

Comment. LAST Operation is a safe operation that gives good midterm results. The great majority of events happened in the first four months, and are, at our opinion, due to technical factors or selection of the patients. However for single LAD lesion our experience compares favourably with stent PTCA procedures on LAD.

*By invitation

9. SURVIVAL AFTER PHOTODYNAMIC THERAPY FOR ENDOBRONCHIAL MALIGNANCY: A 14 YEAR STUDY.

James S. McCaughan, Jr., M.D.* and Thomas E. Williams, M.D. *Columbus, Ohio*

Discussant: Douglas E. Wood, M.D.

Background: After being injected intravenously, the photosensitizer dihematoporphyrin ether is selectively retained in the tumor cells. The photosensitizer absorbs 630 nanometer wavelength light (red) energy delivered from a laser and produces a singlet oxygen which destroys the tumor. A limiting factor in the effectiveness of PDT is the fact that the light only penetrates 5 to 10 mm. The bronchi, however, have a maximum diameter of 9 mm and therefore photodynamic therapy is ideally suited to relieving obstruction due to endobronchial tumors. Photodynamic therapy (PDT) was performed using 630 nm light generated by an argon dye laser system delivered through cylinder diffusing tip quartz fibers passed through the biopsy channel of a flexible endoscope.

Objectives: Determine factors affecting survival rates, benefits and complications of patients with endobronchial cancer treated with photodynamic therapy.

Methods: All patients had already received, refused, or were ineligible for other modalities; none was refused PDT because of a low performance status; and some were on a respirator when first treated. All signed informed consents approved by the Institutional Review Board. From 1982 to May, 1996 photo-dynamic therapy was performed on 175 patients with endobronchial and endo-tracheal tumors (158 squamous, 17 adeno). All were clinically staged at the time of PDT. Sixteen

were Stage I, 9 Stage II, 42 Stage IIIA, 64 Stage IIIB, and 44 Stage IV. All patients were followed until death or November, 1996.

Results: Multivariate analysis of survival using a model of the effects of age, sex, race, histology, Karnofsky Performance Status (KPS) and clinical stage showed the clinical stage ($p<.0001$) was the only statistically significant factor. Sixteen Stage I patients had a 93% five-year disease related estimated survival. Median (months) survivals after PDT were: Stage I = not reached; Stage II = 22.5; Stage IIIA = 5.7; Stage IIIB = 5.5; Stage IV = 5.0. KPS does become significant when it reaches 50 but is not significant for Stages I or II. Within Stages III and IV a Karnofsky Performance Status (KPS) ‰¥ 50 had a significant effect. For Stage IIIA the median survival was 8.2 months when the KPS was ‰¥ 50 and 2.0 for a KPS < 50 . For Stage IIIB the median survival was 7.2 months when the KPS was ‰¥ 50 and 4.0 for a KPS < 50. For Stage IV survival was 6.5 months when the KPS was ‰¥ 50 and 2.6 for a KPS < 50.

Conclusions: Photodynamic therapy may be considered as an alternative treatment for patients under consideration for surgery for Stage I carcinoma who are high surgical risks. The length of palliation for "non-curative" patients was equal to or better than that reported historically for most other treatment regimens.

2:30 p.m. BASIC SCIENCE LECTURE

Implications for Gene Therapy in Treating Coronary Artery Disease and Lung Cancer.
Ronald G. Crystal, M.D., New York, New York

3:15 p.m. INTERMISSION - VISIT EXHIBITS

*By invitation

4:00 p.m. PLENARY SCIENTIFIC SESSION

Sheraton Ballroom

Moderators: Mortimer J. Buckley, M.D.

Andrew S. Wechsler, M.D.

10. LONG-TERM RESULTS, OVER 10 YEARS, OF CONSERVATIVE SURGERY OF CONGENITAL MITRAL VALVE INSUFFICIENCY.

Sylvain Chauvaud, M.D.*, Jean-Francois Fuzellier, M.D.*, Remi Houel, M.D.*, Alain Berrebi, M.D.*, Serban Mihaileanu, M.D.* and Alain F. Carpentier, M.D., Ph.D.

Paris, France

Discussant: Richard A. Hopkins, M.D.

Background: Previous publications from various authors have stressed the benefits of mitral valve repair over mitral valve replacement in children. Very few communications have reported the long-term results and none with follow-up over 10 years. This paper reports our results in a series of 141 patients (pts) operated on for congenital mitral valve insufficiency (MVI) using the same technique (Carpentier technique) in the same center.

Patients and Methods: Between 1970 and 1995, 141 patients (pts) younger than 12 years underwent surgery for congenital MVI. Mean age was 5.8 ± 3.1 years ranging from 0.5 to 12 years. According to Carpentier classification, 30 pts mitral dysfunction were classified type I (normal leaflet motion), 77 classified type II (leaflet prolapse), 34 classified type III (restricted leaflet motion), 14 with normal papillary muscle and 20 with abnormal papillary muscle (hammock or parachute valve). Associated lesions were present in 38 pts (27%). Conservative surgery was possible in 134 pts (95%). Among them, 66 pts required a prosthetic annuloplasty and 10 valve extension with patch. Valve replacement was necessary in 7 pts (5%).

Results: In-hospital mortality was 5.6% (8 pts). No early death was observed in the group of pts who underwent valvular replacement. In-hospital mortality in type I was 10%, in type II, 3.9%, in type III, 5.9% (p : NS). Only 2 of these pts had an associated lesion (ventricular septal defect). Early reoperation was required in 3 pts for recurrent MVI.

Mean follow up was 7.2 ± 6 years (0.4 to 25 years), available in 129 pts (97%). There were 5 late deaths. Actuarial survival was 89.2% ± 6.4% at 15 years and respectively 90.8% and 68.6% in pts who underwent mitral valve repair and in pts who underwent mitral replacement. Late reoperation was required in 6% (8 pts) : 5.5% (7 pts) in pts who had undergone mitral repair and 14.3% (1 pt) in pts with valve replacement. Causes of reoperation were recurrent MVI (6 pts), mitral stenosis (1 pt) and bioprosthesis degenerescence (1 pt). Actuarial freedom from reoperation was 85.9 ± 11.4% at 15 years and a linearized rate of pts exposed to reoperation was 0.9% pts-year. No thromboembolic event was observed.
Conclusion: Congenital MVI can be repaired in infancy with low mortality. Conservative surgery using Carpentier techniques is feasible in the majority of cases of congenital MVI. This technique offers stable long-terms results with a low rate of reoperation.

*By invitation

11. EFFICACY OF ENDOVENTRICULAR PATCH PLASTY REPAIR IN LARGE POST-INFARCTION AKINETIC SCAR AND SEVERE LV DYSFUNCTION. COMPARISON WITH A SERIES OF LARGE DYSKINETIC SCAR.

Vincent Dor, M.D., Marisa Di Donate, M.D.*, Michel Sabatier, M.D.*, Anna Toso, M.D.*, Fran9oise Montiglio, M.D.* and Mauro Maioli, M.D.*

Monte Carlo, Monaco and Florence, Italy

Discussant: Michael K. Pasque, M.D.

In previous studies we have demonstrated that endoventricular circular patch plasty repair (EVCCP) for post-infarction anterior LV aneurysm improves early and late clinical and haemodynamic status. Since 1984 more than 700 pts with different degree and type of post-infarction asynergies have been proposed in our Center for EVCPP, associated coronary grafting and cryotherapy (when indicated for ventricular arrhythmias). Large akinetic scars, associated with severely depressed pump function, are more difficult to treat by surgery as the limit between scar and sound tissue is not as clear as in pure dyskinetic aneurysm. Therefore the technique of the patch anchorage slightly differs, the site of the patch inside the left ventricle depending on the size of the chamber that is « worthy » to leave. The present report concerns 49 pts (60 ± 8 yrs) with large akinetic scar and compares to 40 pts (61 ± 9 yrs) with large dyskinetic scar proposed for EVCPP and coronary grafting. Pts were selected if EF ‰¤ 30% and A% (A is the extent of left ventricular perimeter involved by the asynergy) ‰¥ 60%. Regional wall motion was quantitatively evaluated with the centerline method before and after surgery. All pts have had an anterior myocardial infarction (Ml), groups were comparable for

symptoms, indication for surgery, delay from MI and other clinical variables. Heart failure was the major indication for surgery in both groups, 72% of pts were in NYHA class III/IV, 13 pts were operated on emergency. Ventricular tachycardia (VT) was inducible in 43% (group 1) and 48% (group 2).

	AKINETIC (1)		DYSKINETIC (2)	
	Pre-op	**Post-op**	**Pre-op**	**Post-op**
EF%	23 ± 6	38 ± 11*	23 ± 4	42 ± 8*
Contractile EF%	33 ± 9		38 ± 10	
EDVI (ml/m^2)	248 ± 79	107 ± 47*#	211 ± 79	79 ± 22*
CWP (mmHg)	19 ± 9	12 ± 7*	15 ± 8	12 ± 5*
PAP (mmHg)	28 ± 10	21 ± 9*	23 ± 12	18 ± 6*
CI(ml/min/m)	2, 6 ± .7	2, 6 ± .8	2, 5 ± .6	2, 6 ± .5

**p<= 0.001 vs basal. # vs dyskinetic.*

The mortality rate was 10, 2% (5/49) in akinetic and 17% in dyskinetic (7/40) -ns-. Associated procedures were mitral repair or replacement in 10 pts (5 in group 1 and 5 in group 2) and cryotherapy in 22/49 (group 1) and 20/40 (group 2). Coronary grafting was performed in 98% of pts (LIMA in 89%); the mean number of bypass was 1, 9 ± 0, 4. VT was still inducible in 7/33 pts of group 1; no pt in group 2 had inducible VT after surgery. Results show that the surgical outcome of EVCPP does not depend on the presence or absence of dyskinesia pts with large akinetic scar have even worse pre-operative hemodynamics nevertheless, they benefit from a surgical technique previously reserved only for dyskinetic aneurysms. The reduction of wall tension and oxygen demand due to the marked decrease in volume and the increase in oxygen supply due to complete myocardial revascularization play the major role in improving pump function in either akinetic or dyskinetic post-infarction scars with severely depressed pump function. Therefore, EVCPP can be considered as an alternative to heart transplantation in pts with end stage ischemic cardiomyopathy and predominant akinesia.

*By invitation

12. FIRST RESULTS WITH VIDEO-ASSISTED MINIMALLY INVASIVE MITRAL VALVE REPAIR USING THE PORT-ACCESS-SYSTEM.

Friedrich W. Mohr, M.D., Ph.D.*, Volkmar Falk, M.D.*, Anno Diegeler, M.D.*, Thomas Walther, Ph.D.*, Jaques A.M. van Son, M.D., Ph.D.* and Rudiger Autschbach, M.D., Ph.D.*

Leipzip, Germany

Sponsored by: Hans G. Borst, M.D., Hannover, Germany

Discussant: W. Randolph Chitwood, Jr., M.D.

Background: This study was performed to evaluate the application of the Port-Access-System (Heartport, Redwood, CA) for video assisted minimally invasive mitral valve repair. As yet this is the largest series using this technique worldwide.

Patients and Methods: After approval by the local ethical committee 18 consecutive patients (mean age 65.1 ± 9.4 years, 16 female, 2 male, LVEF 58 ± 12%) were included in the study. Mitral insufficiency (MI) grade III-IV° was present in 14 patients while 4 had predominant mitral stenosis

(MS). Patients (pts) were placed on femoro-femoral bypass and an aortic endoclamp (Heartport) was inflated in the ascending aorta under flouroscopy and TEE control. A minithoracotomy was performed in the 5th intercostal space (length of incision 3.8 to 6.5 cm). Cardiac arrest was induced by antegrade crystalloid cardioplegia via the distal lumen of the endoclamp. The left atrium was opened and a stereoscope inserted through a separate port. The heart was vented through a transvenously placed endopulmonary vent (Heartport).

Results: With videoscopic assistance quadrangular resection and ring implantation was performed in 7 pts. In 4 pts. commissurotomy alone (n = 2) or in combination with a ring annuloplasty (n = 2) and additional chordal replacement (n = 2) was performed. In 5 pts. partial or complete ring annuloplasty was performed. One pt. had persistent MI 11° after quadrangular resection, chordal replacement, and ring implantation and consequently underwent mitral valve replacement using the same approach. Mean duration of operation, cardiopulmonary bypass, and crossclamp time were 180 ± 33, 114 ± 21, and 71 ± 16 min, respectively. Intubation time was 25.6 hours (range 5 to 123 hours). Postoperative pain index (day 2) was low averaging 1.1 ± 0.8 on a 0-10 scale. Duration of ICU treatment and hospital stay were 2 days (1-11 days) and 12 days (10-20 days), respectively. There was one non cardiac related postoperative death. Two pts. required reexploration for bleeding (intercostal artery n = 1, port site n = 1). In 2 pts. transient psychosis most likely due to incomplete deairing was noted. At a mean follow up of 10 ± 6 weeks all patients are in a NYHA class I or II. Echocardiography revealed excellent results of mitral valve repair in all patients with only trivial regurgigation (equal or less MI I°) in 5 pts.

Conclusion: Using the Port-Access-System even complex mitral valve repairs can be performed minimally invasively with good results. The stereoscope allows visualization of all valvular structures in great detail and facilitates repair. With gaining experience operation time decreased to less than 2.5 hours making this new approach a valuable alternative to conventional mitral valve repair.

*By invitation

§13. PARTIAL LIQUID VENTILATION MINIMIZES PULMONARY PARENCHYMAL AND VASCULAR INJURY AND IMPROVES CARDIAC OUTPUT IN A NEONATAL SWINE MODEL OF CARDIOPULMONARY BYPASS.

Ira M. Chaifetz, M.D.*, Michael L. Cannon, M.D.*, Damian M. Craig, B.S.*, George Quick*, Ross M. Ungerleider, M.D., Peter K. Smith, M.D. and Jon N. Meliones, M.D.*

Durham, North Carolina

Discussant: John E. Mayer, M.D.

During cardiopulmonary bypass (CPB) organ protective strategies are traditionally directed at the myocardium and brain. Without a strategy for lung protection, the pulmonary parenchyma and vasculature may suffer severe injury after CPB, especially in neonates. CPB may result in a hypoxic/ ischemic injury, reperfusion injury, surfactant dysfunction, and immune system/complement activation. These processes often result in decreased pulmonary compliance, increased pulmonary vascular resistance, and, potentially, decreased cardiac output (CO). Partial liquid ventilation (PLV) has been shown to be beneficial in both clinical and animal evaluations of acute lung injury. The beneficial effects of PLV result from its oxygen carrying capability, surfactant function, alveolar distending properties, and anti-inflammatory properties. Thus, we hypothesized that PLV might minimize the pulmonary parenchymal and vascular injuries seen in neonates after CPB.

Methods: Twenty neonatal swine (2.0-3.4 kg) were randomized to receive CPB with (n = 9) or without (n = 11) PLV. In the liquid ventilated group, a single dose of perflubron (LiquiVent, Alliance Pharmaceutical Corp.) was administered to functional residual capacity prior to CPB. The control group (CTL) was ventilated conventionally. Each animal was placed on non-pulsatile CPB at 125 mL/kg/min and cooled to a nasopharyngeal temperature of 18°C over 20 minutes. Low-flow CPB (35 mL/kg/min) was then performed for 60 minutes. The flow rate was returned to 125 mL/kg/min, and the animals warmed to 37°C. The animals were removed from CPB, and data were obtained at 30, 60, and 90 minutes after CPB.

Results: The pre-CPB data (mean + sem) for each group were compared to the data 30 min. after CPB by paired t-tests (# $p<0.05$ vs. pre-CPB). The post-CPB data (30, 60, and 90 min.) were compared between the 2 groups using a linear regression model of analysis of variance with repeated measures (*$p<0.05$vs. CTL).

Time	Group	CO (mL/min)	Rin (d-s/cm^5)	Zo (d-s/cm^5)	Cstat (mL/mcH, O/kg)
pre-CPB	CTL	229 ± 29	4434 ± 527	794 ± 78	1.16 ± 0.09
	PLV	223 ± 22	4294 ± 537	801 ± 113	1.36 ± 0.15
30 min.	CTL	140 ± 18#	22, 522 ± 4713#	1339 ± 219#	0.88 ± 0.06#
	PLV	215 ± 18*	1 0, 850 ± 805#*	913 ± 59*	1.11 ± 0.11#*
60 min.	CTL	153 ± 19	17, 865 ± 2517	1228 ± 113	0.83 ± 0.07
	PLV	190 ± 21*	12, 045 ± 1896*	744 ± 52 *	1.10 ± 0.11*
90 min.	CTL	146 ± 15	18, 738 ± 2790	1114 ± 125	0.78 ± 0.05
	PLV	170 ± 12*	13, 793 ± 1852*	922 ± 136*	1.04 ± 0.11*

Rin, input pulmonary vascular resistance; Zo, characteristic impedance; Cstat, statis pulmonary compliance.

Conclusions: The lung protection strategy of partial liquid ventilation minimized the pulmonary parenchymal and vascular injuries associated with neonatal CPB while increasing cardiac output. PLV may become an important technique for protecting the lungs from the deleterious effects of CPB. The morbidity associated with CPB as well as the cost of post-operative care may be significantly reduced if the pulmonary sequelae of CPB can be diminished.

§Authors have a relationship with Alliance Pharmaceutical Corp.
*By invitation

TUESDAY MORNING, MAY 6, 1997

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

Sheraton Ballroom
Moderators: Edward D. Verrier, M.D.

Pedro J. Del Nido, M.D.

F1. CREATION OF VIABLE PULMONARY ARTERY AUTOGRAFTS THROUGH TISSUE ENGINEERING.

John E. Mayer, Jr., M.D., Toshiharu Shinoka, M.D.*, Dominique Shum-Tim, M.D.*, Peter X. Ma, Ph.D.*, Ronn E. Tanel, M.D.*, Noritaka Isogai, M.D.*, Robert Langer, M.D.* and Joseph P. Vacanti, M.D.*

Boston and Cambridge, Massachusetts

Background. "Repair" of many congenital cardiac defects requires use of conduits to establish right ventricle to pulmonary artery continuity. Currently available homografts or prosthetic conduits lack growth potential and can become obstructed by tissue ingrowth or calcification leading to multiple conduit replacements. Tissue engineering (TE) is an approach where cells are grown in vitro onto biodegradable polymers to create "tissues" for implantation. A TE approach has recently been used to construct cardiac valve leaflets from autologous cells. This study assessed the feasibility of a TE approach to constructing pulmonary artery conduits.

Materials and Methods: Ovine artery (Grp A, N = 4) or vein (Grp V, N = 3) segments were harvested, separated into individual cells, expanded in tissue culture, and seeded onto synthetic biodegradable (polyglactin/polyglycolic acid) tubular scaffolds (20 mm long x 15 mm diameter). After 7 days in vitro culture the autologous cell/polymer vascular constructs were used to replace a 2 cm segment of main pulmonary artery in lambs (age = 68.4 ± 15.5d, weight = 18.7 ± 2.0 kg). One other animal received an acellular polymer tube sealed with fibrin glue. Animals were sacrificed at intervals of 11 to 23 weeks (mean follow-up = 125.4 30.8 days, mean weight 38.6 ± 13.0 kg) after echocardiographic and angiographic studies. Explanted TE conduits were assayed for collagen (4-hydroxyprolene) and calcium content, and a tissue DNA assay (bis-benzimide dye) was used to estimate number of cell nuclei. Mechanical tensile strength was evaluated with a vitrodyne V-1000 device.

Results: The acellular control (polymer only) graft developed progressive obstruction and thrombosis, but all 7 TE grafts were patent and demonstrated increase in diameter (Grp A = 18.3 ± 1.3 mm = 95.3% of native PA. Grp V = 17.1 ± 1.2 mm = 86.8% of native PA). None of the biodegradable polymer scaffold remained in any TE graft histologically. Collagen content in TE graft was 73.9 ± 8.0% of adjacent native PA. Tensile strength was 1.115 MPa (native PA = 0.583 MPa). Histologically elastin fiberswere present in the TE vessel wall and Factor VIII (specific for endothelium) was present on the luminal surface. DNA assay showed decreasing numbers of cell nuclei leftover 11 and 23 weeks suggesting an ongoing tissue remodeling. TE grafts calcium content was elevated (A= 7.95 ± 5.09, V= 13.2 ±5.48, native PA= 1.2 ±0.8 mg/g dry wt), but no macroscopic calcification was found.

Conclusion: In growing lambs vascular grafts engineered from autologous cells and biodegradable polymers functioned well in the pulmonary circulation and demonstrated increase in diameter and development of an extracellular matrix and an endothelial lining.

This tissue engineering approach may ultimately allow the development of viable vascular grafts for clinical use.

*By invitation

§F2. COMPARISON OF SURGICAL AND CATHETER-BASED TECHNIQUES OF VEGF DELIVERY ON MYOCARDIAL PERFUSION AND ENDOTHELIUM-DEPENDENT RELAXATION.

Frank W. Sellke, M.D., Motohisa Tofukuji, M.D., Ph.D.*, Roger Laham, M.D.*, Jianyi Li, M.B., M.S.*, Mukesh D. Hariawala, M.D.*, Stuart Bunting, M.D.*, and Michael Simons, M.D.*

Boston, Massachusetts and San Francisco, California

Previous studies have found that the administration of vascular endothelial growth factor (VEGF) in models of chronic myocardial ischemia significantly increases myocardial contractile function, in addition to increasing myocardial perfusion and coronary vascular endothelium-dependent relaxation, two major determinants in the development of unstable angina. In order to determine if surgically or catheter-based techniques of the administration of VEGF are superior at restoring myocardial perfusion and microvascular endothelium-dependent relaxation, ameroid occluders were placed around the left circumflex artery (LCx) of pigs. After 6 weeks, coronary angiography confirmed total LCx occlusion. VEGF was then administered to the epicardial surface of the LCx area with an implanted (thoracotomy) osmotic pump (20 mg over 3 weeks), via intracoronary (1C) injection (20 mg single bolus) through a LCx catheter, or via transvascular LCx injection (20 mg single injection). 1C injection of saline served as a control.

Myocardial blood flow (ml/min/gram tissue) in the collateral-dependent LCx territory and normally-perfused left anterior descending (LAD) artery territory was determined with colored microspheres. Arterioles (130 μm) were isolated from the LCx and LAD territories and examined in vitro with videomicroscopy. Arteriolar relaxations to the endothelium-dependent agonist adenosine 5' diphosphate (ADP) and the endothelium-independent vasodilator sodium nitroprusside (SNP) were studied in precontracted microvessels. Responses = % relaxation of U46619-induced contraction. *p<0.05 vs Control, †p<0.05 vs respective LCx value (2 way ANOVA and Fisher's test), n = 6 in each group. [Drug] = 10 uM in all cases.

	LCx flow	LAD flow	ADP LCx	ADP LAD	SNP LCx
Control	0.57 ± .05	1.00 ± .13†	48 ± 6	74 ± 7†	58 ± 7
Surgical pump	1.10 ± .41*	1.09 ± .36	41 ± 5	69 ± 6†	64 ± 5
Catheter-perivascular	1.21 ± .33*	1.04 ± .14	63 ± 8*	65 ± 6	55 ± 5
Catheter-intracoronary	1.16±.17*	1.07 ± .15	62 ± 7*	65 ± 5	59 ± 8

While delivery of VEGF by a surgically implanted pump was associated with a return of myocardial perfusion to normal levels, it did not affect the impaired endothelium-dependent relaxation in the collateral-dependent LCx territory. Delivery of VEGF by either transvascular injection or intracoronary infusion was associated with improved myocardial blood flow, but also normalization of endothelium-dependent relaxation in the collateral-dependent territory. In conclusion, chronic myocardial ischemia is associated with decreased myocardial blood flow and reduced endothelium-dependent relaxation in the collateral-dependent coronary circulation compared to that in the normally perfused myocardium. Myocardial perfusion is restored with either surgically or catheter-based methods of growth factor delivery, while vascular reactivity is best restored with intravascular, catheter-based techniques.

§Authors have a relationship with Genentech

*By invitation

F3. NON-ANTICOAGULANT HEPARIN PRESERVES

REGIONAL MYOCARDIAL CONTRACTILITY AFTER ISCHEMIA-REPERFUSION INJURY: ROLE OF NITRIC OXIDE.

Peter C. Kouretas, M.D.*, Adam K. Myers, M.D.*, Young D. Kin, M.D.*, Jeff L. Myers, M.D., Ph.D.*, Yi-Ning Wang, M.D.*, Robert B. Wallace, M.D. and Robert L. Hannan, M.D.*,

Washington, DC

Prevention of myocardial dysfunction after ischemia-reperfusion (IR) injury remains a formidable challenge. We hypothesized that heparin may protect the myocardium from IR by a mechanism independent of its anticoagulant properties. Fifteen anesthetized dogs were subjected to 15 minutes ischemia followed by 120 minutes reperfusion and pre-treated with either saline (control, n = 5), heparin (6.0 mg/kg, n = 5) or N-acetylheparin (6.0 mg/kg, n = 5), a heparin derivative without anti-coagulant properties. The left anterior descending (LAD) artery was instrumented with an occluder and a pair of sonomicrometry crystals were placed in the myocardium for measurement of regional systolic shortening, a measure of myocardial contractility. Drugs or vehicle were administered after instrumentation and prior to LAD occlusion. The LAD was occluded for 15 minutes and functional recovery of myocardial performance was assessed at 15, 60 and 120 minutes reperfusion. In order to elucidate the role of the nitric oxide (NO) pathway, a specific NO inhibitor (nitro-L-arginine-1.5 mg/kg, n = 5) was given prior to heparin administration.

Regional Myocardial Function Calculated as Systolic Shortening (SS)

	Pre-Ischemia	5 Min Reperfusion	60 Min Reperfusion	120 Min Reperfusion
Control-IR alone	12.2 ± 1.8	11.0 ± 2.9	6.9 ± 1.4*	6.1 ± 1.6*
Heparin	11.6 ± 0.7	11. 8 ± 1.5	11.4 ± 1.0	11.3 ± 1.7
N-Acetylheparin	12.0 ± 1.5	12.8 ± 0.6	12.1 ± 1.3	12.5 ± 1.8
Heparin + LNNA	13.2 ± 1.0	11.0 ± 1.2	10.1 ± 1.3 *	9.71 ± 1.7*

Value Mean ± SEM *p<0.05 Compared to Pre-Ischemia Using Analysis of Variance With Repealed Measures

Systolic shortening calculated from SS = End Diastolic Length (EDL) - End Systolic Length □ EDL x 100

Systolic shortening was significantly depressed in the control group at 60 and 120 minutes reperfusion. Heparin and N-acetylheparin treated dogs, however, showed preservation of systolic shortening throughout reperfusion. Administration of the nitric oxide inhibitor nitro-L-arginine significantly attenuated heparin's protective effect on myocardial contractility during reperfusion. Activated clotting times were significantly elevated in the heparin and were normal in the N-acetylheparin and control groups. These results confirm the hypothesis that heparin preserves myocardial contractility after ischemia- reperfusion injury independent of its anticoagulant properties. Furthermore, the protective mechanism of heparin during ischemia-reperfusion injury appears to be regulated through the nitric oxide pathway. Administration of heparin derivatives may have important clinical implications in the prevention of myocardial injury without the adverse sequelae of bleeding.

*By invitation

F4. HYPOXIC INDUCTION OF TISSUE FACTOR PROMOTES ENHANCED ENDOTHELIAL CELL PRO-COAGULANT ACTIVITY.

Edward M. Boyle, Jr., M.D.*, Nigel Mackman, Ph.D.*, Timothy Pohlman, M.D.*, Timothy G. Canty, Jr., M.D.*, Owen Lawrence, Ph.D.* and Edward D. Verrier, M.D.

Seattle, Washington and La Jolla, California

The goal of treatment of myocardial ischemia is reperfusion, however, at times microcirculatory no-reflow contributes to ongoing ischemia once blood flow has been reestablished. Hypoxic endothelial cell activation may contribute to the no-reflow phenomenon, yet the mechanisms of impaired post-ischemic flow are poorly characterized. Because microthrombosis seems to play a role in this phenomenon we hypothesized that hypoxically activated endothelial cells would express the extrinsic pathway of coagulation activator, tissue factor (TF). TF is the most potent initiator of clotting known, and if expressed on the surface of ischemic endothelial cells it could result in impaired micro-circulatory blood flow upon reperfusion.

Methods: Cultured human umbilical vein endothelial cells (HUVEC) were exposed to normoxic (N) conditions (21% O_2) or hypoxia (H) in a controlled environmental chamber containing 2-3% oxygen for 2-24 hours. Additional HUVEC were exposed to H for 2 hours followed by 2-24 hours of reoxygenation in a normoxic environment (HR). N, H and HR cell lysates were assayed for TF promoter transcriptional activation by luciferase induction and TF protein production by Western blot analysis. The ability of these conditions to promote coagulation was assessed by exposing HUVECs treated with N, H, or HR to citrated human plasma in the presence of CaCU and recording the time to visible fibrin strand formation. TF activity per 10^6 cells was plotted on a log-log curve against a standard curve constructed with various known concentrations of soluble tissue factor. The ability of a monospecific antibody to TF (□TF-ab) to inhibit this procoagulant activity is assessed to establish this response as secondary to TF.

Results: HUVECs treated with N alone do not make TF, as evidenced by a lack of constitutive promoter activity or TF protein on Western blots, however, following H or HR there is a 3-fold induction of TF promoter activity and a marked increase in TF protein manufactured by HUVECs. Functionally, there is a dramatic increase in procoagulant activity that peaks at 8 hours of H. (136 ± 54 vs. 8.4 ± 3.0) This response is markedly accenuated when cells were exposed to HR. (796 ± 511) Addition of □TF-ab completely abolishes the procoagulant responses to both H and H/R.

Conclusions: This work provides the first direct evidence that exposure of cultured HUVECs to H and HR increases the transcription, translation and surface expression of TF. Furthermore, addition of □TF-ab completely abolishes the potent procoaglulant response to H. Because of the extremely potent procoagulant response of human serum to TF it is conceivable that the expression of TF in vivo could contribute to impaired microcirculation after ischemia and reperfusion. The improved understanding of the role of the endothelial procoagulant response to ischemia/reperfusion should lead to more directed therapies to attenuate the post ischemic no-reflow phenomenon that contributes clinically to tissue injury and impaired myocardial function.

*By invitation

F5. ENDOTHELIAL DYSFUNCTION IN CEREBRAL MICROCIRCULATION DURING HYPOTHERMIC CARDIOPULMONARY BYPASS.

Pierantonio Russo, M.D.*, L. Craig Wagerle, Ph.D.* and Deborah A. Davis, M.D.*

Philadelphia, Pennsylvania

Sponsored by: Stanley K. Brockman, M.D., Philadelphia, Pennsylvania

Inflammatory stimuli and/or mechanical stresses associated with HCPB could potentially impair cerebrovascular function resulting in inadequate cerebral perfusion. We hypothesize that HCPB is associated with endothelial and/or vascular smooth muscle dysfunction and associated cerebral hypoperfusion. Therefore, we studied the cerebrovascular response to endothelium-dependent vasodilator, acetylcholine (Ach), endothelium-independent nitric oxide donor, sodium nitroprusside (SNP), and vasoactive amine, serotonin, in newborn lambs undergoing HCPB. Studies were performed on seven lambs equipped with a closed cranial window and cerebral arteriolar caliber (169 ± 22 μm diameter) was monitored using video microscopy. Topical application of Ach caused dose-dependent increase in diameter. This vasodilator response to Ach was absent in animals undergoing HCPB *(left panel).* HCPB did not alter the vasodilation in response to SNP *(right panel).* Furthermore, the contractile response to serotonin (10^{-5} M) was fully expressed during HCPB (Adiameter = -29 ± 2 vs -30 ± 8%). The specific loss of Ach-induced vasodilation suggests endothelial cell dysfunction rather than impaired ability of vascular smooth muscle response to nitric oxide. It is speculated that loss of endothelium-epended regulatory factors in the cerebral microcirculation during HCPB may enhance vasoconstriction and impaired cerebrovascular function may be a basis for associated neurological injury during or following HCPB.

*By invitation

F6. FLOW-INDUCED RELEASE OF EDRF DURING PULSATILE BYPASS: EXPERIMENTAL STUDY IN THE FETAL LAMB.

Gerard L. Champsaur, M.D., Catherine Vedrinne, M.D.*, Stephane Martinet, M.V.D.*, Franpois Tronc, M.D.*, Jacques Robin, M.D.* and Michel Franck, M.V.D.*

Lyon, France

Previous experimental studies have shown that when compared to continuous flow (CF) during fetal bypass, pulsatile flow (PF) enhances organs perfusion, particularly the placenta, through a diminution of vascular resistances. This study was initiated to test the hypothesis that fetal hemodynamic changes in this setting might be related to the release of endothelium-derived relaxing factor (EDRF) through oscillating shear stress and flow changes, as demonstrated in some isolated organ preparations.

Normothermic bypass was instituted in utero in 21 pre-term fetal lambs after maternal general anesthesia and usual hemodynamic instrumentation. In the fetus partially exposed through cesarean section, fetal bypass was established for a one-hour period after fetal sternotomy through right atrial and main pulmonary artery cannulations. Ultrasonic flowmeters were positioned around the post-ductal descending aorta and the umbilical artery. The circuit was primed with fresh blood and consisted of an oxygenator and a specific centrifugal pump set to either CF (n = 7) or PF (n = 7). Pump flow was monitored by an ultrasonic flowmeter placed around the pump outflow and was

adjusted to maintain a physiological fetal main arterial pressure of 50 mmHg. EDRF blockade was carried out in seven animals (PBF) after 30 minutes of PF using a specific EDRF competitive inhibitor (N‰-nitro--arginine) as a bolus followed by a continuous venous fetal infusion. Flows in ml/min expressed as mean ± SD were the following in each group after respectively 30 and 60 minutes of bypass.

Group	Pump flow		Aortic flow		Umbilical flow	
	30'	60'	30'	60'	30'	60'
CF	612 ± 144*	530 ± 54	224 ± 132*	198 ± 72	61 ± 24*	66 ± 23
PF	907 ± 153	941 ± 228*	458 ± 213	405 ± 177*	181 ± 71	208 ± 90*
PBF	987 ± 228	607 ± 117	421 ± 123	187 ± 88	132 ± 70	50 ± 22

**p<0. 05 between groups at a given time*

Changes in systemic vascular resistances were similar, being significantly lower in Groups PF and PBF than in Group CF (550 ± 106 versus 821 ± 212 dynes/sec/cm"). However, after EDRF blockade in Group PBF, resistances increased gradually to reach the level of that of group CF at the end of the bypass time (943 ± 77 versus 556 ± 143 dynes/sec/cm-5 in the non-blocked PF Group). In conclusion, EDRF blockade during 30 minutes returns fetal hemodynamics back to CF conditions. The specific EDRF inhbition agent used in this experiment suggests that nitric oxide may be released by fetal vascular endothelium during pulsatile bypass.

*By invitation

§F7. COMPLEMENT INHIBITION WITH SOLUBLE COMPLEMENT RECEPTOR TYPE I LIMITS ISCHEMIC DAMAGE DURING REVASCULARIZATION OF ACUTELY ISCHEMIC MYOCARDIUM.

Harold L. Lazar, M.D., Yusheng Bao, M.D.*, Samuel Rivers, B.S.*, TakafUmi Hamasaki, M.D.*, Sheilah Bernard, M.D.* and Richard J. Shemin, M.D.

Boston, Massachusetts

The increased inflammatory response resulting from complement activation during cardiopulmonary bypass (CPB) may contribute to myocardial damage during the revascularization of acutely ischemic myocardium. Soluble human complement receptor type 1 (SCR_1) is a recombinant form of human complement receptor which is a potent inhibitor of complement activation. This study was therefore undertaken to determine whether alteration of complement activation with SCR_1 would reduce myocardial dysfunction during the revascularization of acutely ischemic myocardium. In 20 pigs, the second and third diagonal coronary arteries were occluded for 90 minutes. Animals were then placed on CPB followed by 45 minutes of cold, antegrade, blood cardioplegic arrest and 180 minutes of reperfusion with the coronary snares released. In 10 pigs, SCR_1 (10 mg/kg) was intravenously infused over 30 minutes during the period of coronary occlusion; 10 other pigs received no SCR_1 (Unmodified). Total hemolytic complement activity (CH_{50}) was measured prior to ischemia, during coronary occlusion and reperfusion and expressed as the percent of preischemic values. Ischemic damage in the area at risk was assessed by measuring the change in myocardial tissue pH (ApH) from preischemic values; Wall Motion Scores (WMS) using transthoracic echo-cardiography (4 = normal to -1 = dyskinesia) and infarct size (Area of Necrosis/Area at Risk; AN/AR) using histochemical staining. Data is expressed as the Mean ± Standard Error.

		SCR_1	UNMODIFIED	P Value
CH_{50} (%)	90 min occlusion	2.2 ± 1.3	30.2 ± 3.0	<0.0001
	180 min reperfusion	1.2 ± 1.2	7.8 ± .5	<0.002
□ pH	90 min occlusion	-.44 ± .06	-.85 ± .03	<0.0001
	180 min reperfusion	-.41 ± .03	-.72 ± .02	<0.0001
WMS	90 min occlusion	2.70 ± .13	1.80 ± .16	<0.0001
	180 min reperfusion	3. 10 ± .09	1.67 ± .16	<0.0001
AN/AR(%)		24.6 ± 2.0	41.0 ± 1.3	<0.0001

We conclude that complement inhibition with SCR_1 significantly limits ischemic damage during the revascularization of acutely ischemic myocardium.
§ Presenter has a relationship with T Cell Sciences, Inc.

*By invitation

F8. EXTRACELLULAR SUPEROXIDE DISMUTASE TRANSGENE OVEREXPRESSION SIGNIFICANTLY IMPROVES PRESERVATION OF MYOCARDIAL FUNCTION FOLLOWING ISCHEMIA AND REPERFUSION INJURY.

Edward P. Chen, M.D.*, Hartmuth B. Bittner, M.D., Ph.D.*, R. Duane Davis, M.D.*, Peter Van Trigt, M.D. and Rodney J. Folz, M.D., Ph.D.*

Durham and Greensboro, North Carolina

Myocardial injury after ischemia and reperfusion injury may be mediated, at least in part, by oxygen-derived free radicals and is supported by the observation that significant quantities of these radicals are generated during post-ischemic reperfusion. To directly assess the protective effect of the extracellular superoxide dismutase (EC-SOD), a controlled prospective, double-blinded experimental study was performed to evaluate myocardial function in the hearts of transgenic mice overexpressing human EC-SOD to levels 3.5x greater than controls. Heterozygous (EC-SOD, n = 6, 22-26 g) and nonheterozygous litter mate controls (CTL, n = 8, 22-26 g) were analyzed by PCR analysis of tail DNA. An isolated work-performing murine heart preparation was used to evaluate preload-dependent cardiac output (CO), contractility (dP/dt), stroke work (SW), stroke volume (SV), and heart rate (HR) before (Pre-I) and after (Post-I) a 6 minute period of normothermic ischemia. Results are expressed as mean ± SEM (ANOVA, paired/unpaired t-test). There was no significant difference between EC-SOD and CTL in any parameter of myocardial function Pre-I. The average Pre-I HR for CTL and EC-SOD was 438 ± 19 beats/min and 482 ± 16 beats/min. There was an 87% recovery in post-I HR in CTL and a 94% recovery in post-I HR in EC-SOD ($p<0.05$). Pre-I SW/SV/ dP/dt in CTL were 674 + 79 dyne*cm/12.7± 1.6 µl/2305± 157 mmHg/s, while Pre-I EC-SOD SW/SV/ dP/dt were 593 ± 28 dyne*cm/10.6 ± 0.6 µl/ 2127 ± 104 mmHg/s. Post-I SW/SV/ dP/dt in CTL recovered by 55%/54%/80%, while Post-I EC-SOD SW/SV/ dP/dt recovered 77%/78%/90% ($p<0.001$). The table displays the preload dependent Frank-Starling relationships in CO (mL/min) in both groups Pre-I and Post-I (*=$p<0.01$ Pre-I vs Post-I; t = $P<0.05$ EC-SOD vs CTL, SEM in parentheses):

Pre-I CO	**5 mm Hg**	**10 mm Hg**	**15 mm Hg**	**20 mm Hg**	**25 mm Hg**
CTL	3.78 (0.53)	5.57 (0.60)	6.01 (0.60)	5.87 (0.60)	5.78 (0.57)

EC-SOD	3.30(0.49)	5.24(0.25)	5.66(0.25)	5.64(0.20)	5.70(0.18)
Post-I CO	**5 mm Hg**	**10 mm Hg**	**15 mm Hg**	**20 mm Hg**	**25 mm Hg**
CTL	1.22(0.21)*	2.44(0.29)*	3.11(0.36)*	3.50(0.34)*	3.34(0.30)*
EC-SOD	2.21 (0.36)†	3.82 (0.29)*†	4.29 (0.33)*†	4.23 (0.30)*	4.34 (0.38)*†

Conclusions: EC-SOD transgene overexpression does not affect baseline myo-cardial function compared to CTL hearts. Following global normothermic ischemia and subsequent reperfusion, significant decreases in cardiac function were observed in both EC-SOD and CTL, however, a significantly higher percentage of recovery was observed in EC-SOD overexpressed hearts. These data suggest that EC-SOD transgene overexpression significantly improves preservation of myocardial function following ischemia and reperfusion injury.

*By invitation

9:00 a.m. PLENARY SCIENTIFIC SESSION

Sheraton Ballroom
Moderators: David B. Skinner, M.D.

James L. Cox, M.D.

14. OPERATIVE OUTCOME AND HOSPITAL COST.

Victor A. Ferraris, M.D., Ph.D., Suellen P. Ferraris, Ph.D.* and Amandeep Singh*

Albany, New York

Discussant: Floyd D. Loop, M.D.

Introduction: Health care costs are increasing at an alarming rate and cardiac procedures contribute to this increase. It is likely that patient risk factors contribute to this cost increase since operative interventions are being performed on high risk patients with greater frequency, but the exact relationship of patient risk factors to hospital cost is poorly understood. Because of this knowledge deficit and because of the possibility that modification of patient risk factors might lead to decreased cost, we undertook a study to identify patient risk factors associated with increased hospital cost and to evaluate the relationship of increased cost to serious hospital morbidity and mortality.

Methods: More than 100 patient variables were collected in a prospective manner in 1221 patients undergoing cardiac procedures. Simultaneously, patient hospital cost was computed from the cost-to-charge ratio after validation of the individual departmental ratios. Univariate statistics were used to explore the relationship between hospital cost and patient outcomes. Multivariate regression models using logistic regression, Cox proportional hazards regression, and stepwise linear regression identified independent patient risks for mortality, hospital morbidity (inferred from length-of-stay) and hospital cost, respectively.

Results: The greatest cost occurred in 31 patients who did not survive operation ($74, 466 ± 19, 393 95% CI). This was significantly greater than the cost in 120 patients who suffered serious non-fatal morbidity ($60, 335 ± 6, 248 95% CI, $p = 0.02$) and the cost in 1070 patients who survived operation without complication ($31, 459 ± 711 95% CI, $p<0.01$). Hospital cost was not directly related to length of stay (LOS), although the increased cost in operative fatalities was associated with increased length of stay compared to uncomplicated procedures (14.2 ± 7.4 95% CI vs. 8.3 ±

0.3 95% CI days, p = 0.02). Breakdown of the components of hospital cost in fatalities and in patients with non-fatal complications revealed that the greatest contribution to cost was in the anesthesia and operating room costs as well as pharmacy costs, two components not directly related to LOS. Significant independent risks for mortality, morbidity (LOS), and hospital cost identified by multivariate regression are shown in the table:

Mortality		Length of stay		Cost	
Risk factor	Significance	Risk factor	Significance	Risk factor	Significance
CHF	<0.0001	Age/RBC volume	<0.0001	CHF	<0.0001
Cath crash	0.001	OR type	< 0.0001	NYS mortality risk	<0.0001
OR type	0.004	Renal dysfunction	< 0.0001	Renal dysfunction	<0.0001
NYS mortality risk	0.04	NYS mortality risk	< 0.0001	OR type	<0.0001
		CHF	0.003	Age/RBC volume	0.003
		Hypertension	0.032	Priority	0.007
		COPD	0.073	Redo procedure	0.03
		Previous stroke	0.082	Preop IABP	0.08

Abbreviations: NYS mortality risk = sum of variables related to serious co-morbidity and compromised ventricular function; RBC volume = HCT times estimated blood volume; OR type is either CABG, valve, valve/CABG or other.

Conclusions: We conclude that: 1) operative death is the most costly outcome, 2) LOS is not an accurate indicator of hospital cost, 3) ventricular dysfunction associated with operations for other than coronary disease are significantly associated with increased cost, and 4) patient factors that are amenable to preoperative intervention to reduce costs are correction of preoperative anemia (i.e. increase RBC volume) and treatment of CHF. These results suggest a high-risk patient profile that should be a target for cost reduction strategies.

*By invitation

15. EN BLOC ESOPHAGECTOMY IMPROVES SURVIVAL FOR STAGE III ESOPHAGEAL CANCER.

Nasser K. Altorki, M.D., Leonard Girardi, M.D.* and David B. Skinner, M.D.

New York, New York and Houston, Texas

Discussant: Victor F. Trastek, M.D.

The role of enbloc esophagectomy in patients with locally advanced esophageal cancer is not well defined. Between January 1988 and June 1992 we continued our selective surgical approach whereby patients with favorable disease (Stage I and II) were treated by enbloc resection while patients with suspected Stage III disease underwent resection by standard techniques. Since the mortality and morbidity of both techniques appeared similar we adopted enbloc esophagectomy more liberally in all stages since 1992. The purpose of this study was to examine the influence of this strategy on the survival of resected Stage III patients.

Between January 1988 and September 1996, 128 patients underwent esophagectomy by an enbloc technique (n = 78) or a standard esophagectomy (n = 50, 46 transthoracic, 4 transhiatal). There were 101 males and 27 females with a median age of 62 (range 34-87). Squamous cell cancer was present in 38 and adenocarcinoma in 90 patients. Hospital mortality was 5.4% (7/128) and morbidity 54% and was not influenced by the type of procedure. Enbloc resection

was done in 100% of Stage 0 and Stage I patients (18), 66% of Stage II patients and 60% of Stage III patients. Two and three year survival, median survival and p values are shown below for all stages excluding Stage IV (n = 23).

	n	Enbloc		Median	Standard		Median	p-value
		2 yr	3 yr		2 yr	3 yr		
Stage 0	5		100.0%	-	-	-	-	-
Stage I	13		71.9%	not reached				
Stage II	33	67.0%	49.0%	38m	40.0%	13.0%	20.6	0.02
Stage III	54	48.0%	36.0%	22m	16.0%	10.0%	12.0	0.02

Eighty-six patients had positive nodes (68%). Two and three year survival for patients with Nl disease treated by enbloc resection was 59% and 35% respectively (median 23 m) versus 13% and 6% (median 12.6 m) for patients treated by standard resections, (p = 0.007).
Based on a significant improvement in 2 year and 3 year survival and median survival we conclude that enbloc resection improves survival in patients with Stage III cancer of the esophagus and that excision of nodal disease may have a positive survival benefit.

*By invitation

16. GROWTH POTENTIAL AFTER BIVENTRICULAR REPAIR IN CHILDREN WITH SMALL BUT NOT HYPOPLASTIC LEFT HEART.

†Alain E. Serraf, M.D., Nicolas Bonnet, M.D.*, FranÃ§ois Lacour-Gayet, M.D.*, Dominique Piot, M.D.*, Anita Touchot, M.D.*, Jacqueline Bruniaux, M.D.* and Claude PlanchÃ©, M.D.
LePlessis-Robinson, France
Discussant: Thomas L. Spray, M.D.

Because of the lack of strict criterias, there is still no agreement whether to perform uni or biventricular repair in children with small left heart structures. Thirty six children with small left heart sizes underwent biventricular repair at our Institution. Preoperative echocardiographic assessment allowed to record the diameter of the mitral valve, end-diastolic (EDLVD) and end-systolic (ESLVD) left ventricular diameters, aortic and subaortic root diameters. Left ventricular volumes (EDLVV and ESLVV) were estimated according to the corrected formula and stroke volumes (SV) were then calculated. All measurements were standardized to normal by the Z-value method. All the pts presented with small but normal left ventricular anatomy, those with profound structural anomalies of the left ventricle (LV) (hypoplastic left heart syndrome, complete AV canals) were excluded from this work. There were 19 males and 17 females. The median age at the first operation was: 13 days and the mean weight was 3.5 ± 0.9 kg. Three groups could be distinguished: Group I (n = 15) with high left to right atrial shunt and insufficient preload of the LV, Group II (n = 9) with elevated left ventricular afterload and Group III (n = 12) which combined both anomalies of pre and afterload. Sixteen underwent single stage complete repair and 20 had an incomplete or palliative procedure. Early reoperation was necessary in 12 pts because of the inability of the LV to sustain systemic output. Two were converted to univentricular first stage palliation and the other underwent closure of atrial shunts to preload the LV. There were 6 early deaths, 5 of whom could be attributed to the inability of the LV to sustain a systemic output. All the survivors demonstrated a rapid growth of left heart structures already at discharge from hospital.

Group I Group II Group III

Z-Values	Before	After	Before	After	Before	After
Mitral diameter	-1.9	-0.6	-1.9	-1.4	-2.4	-0.8
Aortic root diameter	-2	-1.5	-4.5	-3.6	-5.5	-0.4
EDLVD	-5	-0.9	-4.9	-3.5	-5.5	-1.7
ESLVD	-6.2	-2.8	-5.5	-7	-6.15	-2.6
EDLVV	-1.4	+0.4	-1.3	+1.2	-1.6	+0.3
ESLVV	-2.2	-0.9	-3	-2.5	-1	-0.8
SV	-1.3	+4	-1.25	+2	-1.6	+0.5

Statistical analysis demonstrated that non survivors had a smaller aortic root diameter (p=0.01) Eleven patients underwent 20 late reoperations for closure of residual ventricular shunts (n = 6), subaortic stenosis (n = 3) and for mitral valve stenosis (n = 2) with 2 deaths. At the time of reoperations, the sizes of left heart structures were within normal ranges in all but 2 patients. A median follow-up of 40 months (Ranges: 6-120 months) was achieved in all survivors. They were all in NYHA classes HI with normal sizes of left heart structures. Actuarial survival and freedom from reoperation rates at 8 years were 60.6 ± 15.9% and 25.2 ± 11.2%. In conclusion, biventricular repair promotes rapid growth of the left ventricular structures in children with small but not hypoplastic left heart and appears to be a good surgical option. It remains however difficult to determine clear predictive criterias for either biventricular or univentricular repair.

10:00 a.m. INTERMISSION - VISIT EXHIBITS

†1993-94 AATS Graham Fellow

*By invitation

10:45 a.m. PLENARY SCIENTIFIC SESSION

Sheraton Ballroom

Moderators: David B. Skinner, M.D.

James L. Cox, M.D.

17. INDUCTION THERAPY FOR ESOPHAGEAL CANCER WITH PACLITAXEL (TAXOL®) AND HYPER-FRACTIONATED RADIOTHERAPY: A PHASE I/II STUDY.

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

Boston, Massachusetts

Discussant: Michael E. Burt, M.D.

Induction chemoradiation followed by surgery is a promising approach to treatment for esophageal cancer. Previous reports emphasize the importance of a high pathologic complete response rate as these patients have enhanced survival. Paclitaxel (Taxol) is a new agent with high response rates

in metastatic esophageal cancer. However, the use of Paclitaxel has not been reported in induction regimens. Twenty-seven patients with esophageal cancer were enrolled in a Phase I/II trial of induction chemoradiation followed by esophagectomy beginning in May 1995. The chemotherapy consisted of paclitaxel at 3 dose levels (75, 125 and 100 mg/m^2), cisplatin and 5-fluorouracil. The radiotherapy was concurrent and hyperfractionated and delivered 42 Gray to the mediastinum with a 16.5 Gray boost to the tumor. The mean age of the patients was 60 and 21 (78%) had adenocarcinoma. Pretreatment staging was by computed tomography and endoscopic ultrasonography. Nine patients were T2NO, 1 T2N1, 11 T3NO, 5 T3N1, and 1 was T4NO. Patients were hospitalized for a mean of 15.8 days for chemotherapy or complications of induction treatment. The number of patients who had severe (Grade 4) esophagitis at each dose level of paclitaxel was as follows: 75 mg/m^2-50%, 125 mg/m^2-75%, and 100 mg/m^2-47%. One patient died during induction therapy at home presumably from sepsis. Twenty-six patients underwent esophagectomy with a mean hospital stay of 13.7 days. The average time to surgery from the initiation of treatment was 77 days. There was 1 postoperative death due to an aorto-esophageal fistula. Eleven of 26 patients (42%) had a complete pathologic response in the resected specimen. Nineteen of 26 patients (73%) had no tumor in the resected nodes. Twelve of 26 patients (46%) had no tumor in the resected esophagus. Two patients have recurred with distant disease and died. Twenty-three patients have no evidence of disease with a mean follow-up of 10.3 months. In this regimen, paclitaxel at a dose of 100 mg/m^2appears to have acceptable toxicity. Previous reports suggest a pathologic complete response rate up to 25% with most induction regimens. The relatively high pathologic complete response rate (42%) with this regimen is encouraging but survival data are not yet available to confirm increased survival.

*By invitation

18. EARLY RESULTS WITH PARTIAL LEFT VENTRICULECTOMY.

Patrick M. McCarthy, M.D., Randall C. Starling, M.D.*, Gregory M. Scalia, M.B.B.S.*, James D. Thomas, M.D.*, Nicholas G. Smedira, M.D.* and James B. Young, M.D.*

Cleveland, Ohio

Discussant: D. Craig Miller, M.D.

Partial left ventriculectomy (PLV), the Batista procedure, has demonstrated significant clinical improvement in some patients (pts) with dilated cardiomyopathy (DCM). Since May 1996 we have performed PLV in 30 patients, initially in heart transplant (Tx) candidates, and more recently in non-Tx candidates. The mean age of the pts was 54 years (range 34 to 72); 60% were Class IV and 40% Class III. Preoperatively all pts were thought to have idiopathic DCM. As our experience has accrued we have increased the extent of left ventriculectomy and more complex mitral valve (MV) repairs. For only one pt was MV replacement performed (rheumatic MV disease). For 29 pts the anterior and posterior MV leaflets were approximated (Alfieri repair); 24 pts also had ring posterior valvuloplasty. The lateral wall (circumflex territory) between the papillary muscles was the location for ventriculectomy in 29 pts. In 4 pts the posterior papillary muscle was divided, additional posterior wall was resected, and the papillary muscle heads reimplanted. The extent of resection was gauged by a formula using the LV internal diameter (LVID) and interpapillary muscle distance (IPD) to predict post resection diameter:

$$LVID_{post}=LVID_{pre}- (IPD / \square)$$

All ventriculotomies were closed with soft felt or bovine pericardium. Intraoperative hemodynamic and echocardiographic changes for 17 pts operated before October are in the Table. Initial pressure area loops have shown decreased stroke work, filling pressures, and filling volumes; with preserved stroke volume.

	Pre	Post	p value
LVID (cm)	8.1	6.0	<0.001
MR (0-4+)	2.6	0	<0.001
Ejection fraction	16%	36%	<0.001
Cardiac index (L/min/m^2)	2.0	2.7	0.006
Left atrial pressure (mmHg)	23	13	0.01

There were no in-hospital deaths; there was one reoperation for bleeding. Three pts required HeartMate left ventricular assist devices postoperatively; two are being weaned as the heart recovers. Three pts have been relisted for Tx. There was one death at 3 months from cerebral edema. Of 20 discharged pts 80% are subjectively Class I or II.

Conclusion Ninety-seven percent of pts with severe LV dysfunction and mitral regurgitation are alive following PLV and valve repair; most are clinically improved. The clinical outcome is not always predictable however, and therefore longer follow-up and further studies are required to optimize pt selection and surgical techniques.

11:25 a.m. ADDRESS BY HONORED SPEAKER

Esophageal Surgery at the End of the Millenium.

Antoon E.M.R. Lerut, M.D., Leuven, Belgium

12:10 p.m. ADJOURN FOR LUNCH - VISIT EXHIBITS

12:10 p.m. CARDIOTHORACIC RESIDENTS' LUNCHEON

*By invitation

TUESDAY AFTERNOON, MAY 6, 1997

1:45 p.m. SIMULTANEOUS SCIENTIFIC SESSION A - ADULT CARDIAC SURGERY

South Sheraton Ballroom

Moderators: Karl H. Krieger, M.D.

Marko I. Turina, M.D.

19. IMPROVED EVENT FREE SURVIVAL FOLLOWING TRANSMYOCARDIAL LASER REVASCULARIZATION VERSUS MEDICAL MANAGEMENT IN PATIENTS WITH UNRECONSTRUCTED CORONARY ARTERY DISEASE.

Robert J. March, M.D.*, Sara Aranki, M.D.*, Steven Boyce, M.D.*, Lawrence H. Cohn, M.D., Denton A. Cooley, M.D., John R. Crew, M.D., Gregory Fontana, M.D.*, O. Howard Frazier, M.D., Hartley P. Griffith, M.D, Kevin P. Landolfo, M.D.*, Allan Lansing, M.D, James Lowe, M.D, Bruce W. Lytle, M.D, Mahmood Mirhoseini, M.D. and Craig Smith, M.D.

Chicago, Illinois; Boston, Massachusetts; Washington, DC, Houston, Texas; San Francisco and Los Angeles, California; Pittsburgh, Pennsylvania; Durham, North Carolina; Louisville, Kentucky and Cleveland, Ohio

Discussant: Ralph J. Damiano, Jr., M.D.

To evaluate the efficacy of Transmyocardial Laser Revascularization (TMR) in the treatment of symptomatic, end-stage coronary artery disease (CAD), 160 patients from 12 U.S. centers were enrolled in a 1:1 randomized, prospective study comparing TMR to continued Medical Management (MM). Of the 77 patients initially randomized to TMR using a CO_2 Laser (PLC Medical Systems, Inc.), 72% improved by at least 2 anginal classes. In the 83 patients randomized to the MM group, the angina class remained unchanged in 69% and worsened in 31%. Event free survival for death, unstable or Class IV angina at six months, was 73% for the TMR group versus 12% for the MM patients (p=0.0001). Quality of life indices increased an average of 127% for patients undergoing TMR compared to no change in the MM group. Twenty-six patients (31 %) crossed over from MM to TMR due to worsening angina or the development of unstable symptoms. There was a 27% perioperative mortality for this crossover group compared to a 1% perioperative mortality for patients having TMR initially. The study mortality was 16% for patients randomized to MM and 6% for those randomized to TMR. Event free survival, angina class and quality of life appear improved by TMR compared to MM in patients with symptomatic, end-stage CAD. Continued MM associated with a worsening clinical status significantly increases the risk of TMR, thereby supporting the early application of this treatment modality.

*By invitation

§20. TRANSMYOCARDIAL LASER TREATMENT DENERVATES CANINE MYOCARDIUM.

King F. Kwong, M.D.*, Georgios K. Kanellopoulos, M.D.*, Joshua C. Nickols*, Stephen Pogwizd, M.D.*, Jeffrey E. Saffitz, M.D., Ph.D.*, Richard B. Schuessler, Ph.D.* and †Thoralf M. Sundt, III, M.D.*

St. Louis, Missouri

Sponsored by: William A. Gay, Jr., M.D., St. Louis, Missouri

Discussant: Gerald D. Buckberg, M.D.

Transmyocardial laser treatment (TML) reduces angina clinically. The objective of our study was to test the hypothesis that TML alters the cardiac nerve fibers which convey the pain of angina pectoris. ***Methods:*** Left thoracotomy was performed in sixteen adult mongrel dogs that were

divided into three treatment groups: laser (n = 5), phenol (n = 5), and sham (n = 6). A portion of the anterior left ventricule (LV) was subjected to the creation of transmyocardial channels with a Holmium:YAG laser, phenol application on the epicardium (which chemically destroys cardiac afferent nerve fibers), or no treatment. Cardiac afferent nerves were stimulated with topical epicardial bradykinin (300 ug/150 ul), a potent algesic, before any treatment and again at two weeks after operation; the resulting central nervous system mediated reflex decrease in systemic mean arterial blood pressure (MAP) was measured. Immunoblot analysis was performed on treated and untreated LV myocardium of each dog using antibody for tyrosine hydroxylase, a neural-specific enzyme. ***Results:*** Reflex systemic arterial pressure changes were seen with all dogs upon bradykinin stimulation prior to treatment. At two weeks post-operatively, LV areas treated with laser or topical phenol failed to show any response to bradykinin but untreated LV regions in the same dogs remained responsive. All sham dogs were responsive to repeat bradykinin stimulation (see table). Immunoblots demonstrated loss of tyrosine hydroxylase immunoreactivity only in the treated regions of phenol and laser dogs.

Percentage Bradykinin-Evoked Decrease from Baseline MAP

	untreated LV wall		treated LV wall	
	pie	post	pie	post
Sham	34 ± 11	31 ± 15	22 + 9	22 ± 8
Phenol	39 + 9	32 + 13	15 ± 6	2 ± 2
Laser	40 ± 10	44 + 25	23 ± 8	0 ± 0

Values given as mean ± SD, *p < 0.001 (pre versus post treatment)

Conclusion: Transmyocardial laser treatment destroys cardiac nerve fibers, which may contribute to the reduction of angina pectoris seen clinically.

†1994-96 Robert E. Gross AATS Scholar

§Author has a relationship with CardioGenesis Corp.

*By invitation

21. SEVEN-YEAR FOLLOW-UP OF CORONARY ARTERY BYPASSES PERFORMED WITH AND WITHOUT CARDIOPULMONARY BYPASS.

Steven R. Gundry, M.D., Matthew Romano*, Howard Shattuck*, Anees J. Razzouk, M.D.* and Leonard L. Bailey, M.D.

Loma Linda, California

Discussant: Federico J. Benetti, M.D.

There has been resurgent interest in coronary revascularization performed on the beating heart (BHCABG). The advantages of shortened hospital stay, reduced costs, lessened patient discomfort and elimination of the negative effects of cardiopulmonary bypass must be weighed against the untoward effects of constructing bypasses on a moving field and the potential of limiting full revascularization. Heretofore, there has been no long term followup or any comparison of this technique to traditional coronary artery bypass with cardioplegia (CABG). From June 1989 to July 1990, all patients presenting for coronary revascularization to three surgeons were considered for BHCABG: 107 underwent successful BHCABG while 112 were felt unsuitable and underwent

revascularization on bypass with cardioplegia (CABG). Mean ages (65 ± 10 yrs) and risk factors were identical. BHCABG pts had 2.4 ± 0.9 grafts versus 3.3 ± 1.1 for CABG pts. At 7 year followup, the following results were obtained:

	Alive	**Cardiac deaths**	**Recathed**	**PTCA or redo CABG**
BHCABG	86/107 (80%)	13/107 (12%)	31/107(30%)*	21/107 (20%)*
CABG	88/112(79%)	10/112(9%)	18/112(16%)	5/112(4%)

*$p < 0.05$ compared to CABG

No CABG pt required reoperation while the majority of reinterventions on the BHCABG group were PTCA's (15/21 (71%)).

In conclusion, despite one less graft/pi, at 7 year followup, survival and cardiac death rates were identical between pts whose grafts were performed off bypass and those on bypass. In contrast, twice as many BHCABG pts required recath (30% vs 16%) and 20% of BHCABG pts needed a second intervention vs only 4% of on bypass pts. These results demonstrate that limited revascularization of the beating heart provides excellent long term results compared to full revascularization on bypass but will require approximately a five fold increase in re-interventions to achieve these results.

*By invitation

2:45 p.m. INTERMISSION - VISIT EXHIBITS

3:15 p.m. SIMULTANEOUS SCIENTIFIC SESSION A -

ADULT CARDIAC SURGERY
South Sheraton Ballroom

Moderators: Karl H. Krieger, M.D.

Marko I. Turina, M.D.

22. FIVE-YEAR RESULTS OF CORONARY BYPASS GRAFTING USING THE RADIAL ARTERY.

Christophe Acar, M.D.*, Ahmad Ramshey, M.D.*, Jean Yves Pagny, M.D.*, Bernard Beyssen, M.D.*, Jean Noel Fabiani, M.D.*, Alain Deloche, M.D.* and Alain F. Carpentier, M.D., Ph.D.

Paris, France

Discussant: Hendrick B. Earner, M.D.

Since its revival in 1989, the radial artery (RA) was used as a conduit in 783 patients undergoing coronary artery bypass. The aim of this study was to assess the long term clinical results as well as the late patency rate of the RA grafts.

A complete follow-up was obtained for the first one hundred consecutive patients surviving the operation. Patients were reviewed after a mean follow-up of 5.4 years (4 to 7 years postoperatively). Age ranged from 38 to 84 years (mean: 68 ± 8 yrs). The mean number of grafts was 2.6 ± 0.8 including RA (n = 128). left internal mammary artery (IMA) (n = 94), right IMA (n=18), free IMA (n=11) and vein (n=12). RA grafts were anastomosed to the circumflex (51%), right coronary

(29%), diagonal (16%) and left anterior descending coronary artery (LAD) (4%) and the left IMA grafts were anastomosed to the LAD (94%) or circumflex coronary artery (6%).

Eleven patients died during the period of follow-up (cardiac related death: 3, non-cardiac: 8). The other 89 patients were asymptomatic (81%) or had occasional angina (8%). Two patients had congestive heart failure (CHF) (NYHAIII/IV). Pharmacological treatment of patients was as follows: calcium channel blockers (53%), beta blockers (34%), nitrates (46%), aspirin (70%), coumadin (14%). Three patients underwent percutaneous transluminal angioplasty during the period of follow-up and there was one reoperation for aortic valve stenosis. There was no other morbidity at the site of RA removal than a mild dysesthesia of the thumb in 12 cases.

An EKG stess test was obtained in all but four cases (patients > 80 years of age, CHF) (n = 85). Tests were performed for 78 ± 5% of the predicted maximal heart rate. Stress test was negative in 71 cases and positive in 14 cases (EKG changes alone (12) or with chest pain (2)). A routine control angiogram was perfomed in 50 cases (including all symptomatic patients) at a mean follow-up of 5.6 years. The patency rate of the RA grafts was 84.2% (excellent result: 47/57, stenosis 1/57, string or occlusion: 9/57).
Haemodynamic factors that could compromise graft function were noted in 5/9 occluded RA grafts: no stenosis on the native coronary vessel (n = 4), progression of atheroma on the distal run-off (n = 1). The patency rate of the left IMA grafts was 89.8% (excellent result: 42/49, stenosis: .2/49, string or occlusion: 5/49).

Conclusion: the use of the RA for coronary bypass grafting provides excellent clinical and angiographic results at 5 years.

*By invitation

23. DOUBLE VALVE REPLACEMENT WITH RECONSTRUCTION OF THE FIBROUS BODY BETWEEN THE AORTIC AND MITRAL ANNULI.

James Kuo, M.D.*, Tirone E. David, M.D., Susan Armstrong, M.Sc.* and Joan Ivanov, M.Sc.*

Toronto, Ontario, Canada

Discussant: Alain F. Carpentier, M.D.

The fibrous body between the aortic and mitral annuli may be destroyed by infection, calcification or multiple previous mitral valve replacement, making double valve replacement difficult. A solution for this problem is to excise the aortic and mitral valve and the diseased fibrous body between them by extending the aortotomy into the dome of the left atrium and then into the mitral valve. Once this is done the base of the left ventricle is widely exposed and the mitral and aortic orifices become a single orifice. If only the fibrous tissue between the mitral and aortic annuli was excised, reconstruction is accomplished by a triangular shaped patch of glutaraldehyde bovine pericardium sutured to the lateral and medial fibrous trigones and to the aortic root. If the entire mitral annulus was debrided because of infection or extensive calcification, a large oval shaped patch of bovine pericardium is sutured to the endocardium of the left ventricle posteriorly and to the base of the aortic root superiorly; an opening is made in this patch to create a mitral annulus and secure a mitral valve prosthesis. With either procedure, the roof of the left atrium is closed with a separate triangular shaped patch before an aortic valve prosthesis is implanted. This operation is also useful to enlarge both the mitral and aortic annuli.

This operation was performed in 43 pts because of multiple previous valve replacement (17 pts), infective endocarditis of the aortic and mitral valve with abscess (15 pts), extensive calcification of the base of the heart (6 pts), and enlargement of the aortic and mitral annuli (5 pts). There were 20 men and 23 women with a mean age of 59 years, range 33 to 81. Thirty-one pts had had at least one previous valve replacement. All pts were in NYHA functional classes III and IV, and 9 pts were moribund at the time of surgery. There were 7 operative deaths (16%). Two pts required reoperation, one for acute prosthetic valve endocarditis and one for paravalvular leak. Pts have been followed for a mean of 38 ± 30 months. There have been 6 late deaths. The actuarial survival at 5 years was 56% ± 6%. There has been no late patch or prosthetic valve dehiscence.

This operation has provided satisfactory results in pts with extremely complex aortic and mitral valve pathology.

*By invitation

24. CLINICAL AND HEMODYNAMIC RESULTS OF 174 AORTIC VALVE REPLACEMENTS WITH A STENTLESS PORCINE VALVE.

Francis D. Ferdinand, M.D.*, John R. Pepper, F.R.C.S.*, Asghar Khaghani, F.R.C.S.*, Sue Edwards, R.G.N.* and Magdi H. Yacoub, F.R.C.S., Ph.D.

London and Harefield, England

Discussant: Edward D. Verrier, M.D.

A stentless valve has the potential advantages of better post-operative haemodynamics, long-term function and overall quality of life. Newer xenograft devices may mimic homografts in this respect; and in addition, offer the advantage of uniform availability. We report our results in 174 patients from July 1992 to July 1996 with the Toronto SPV valve. The mean follow-up was 16 months (range 0-38 months) and the percent follow-up was 97%. The average age was 68.1 years (range 35 to 89) and 66% were male. Pre-operatively, 2% were in NYHA Functional Class I, 43% in Class II, 50% in Class III and 5% in Class IV. Valve sizes implanted ranged from 20 to 29 mm. Forty-four point nine percent of patients had concomitant procedures of which 42% were CABG. Mean cardiopulmonary bypass time (mean ± SD) was 119.6 ± 35.8 minutes and cross clamp time was 91.4 ± 24.3 minutes. Post-operatively, at mean follow-up of 16 months, 79% of patients were in NYHA Functional Class I, 20% in Class II, 1% in Class III and 0% in Class IV. Echocardiographic analysis at follow-up revealed a mean systolic gradient ranging from 4.2 to 9.5 mmHg with no significant differences between valve sizes implanted. There was none or trivial aortic insufficiency in 85% of patients, 5% had mild, and 4% had moderate. There was no progression of aortic insufficiency during follow-up. There was a 5.7% early (‰¤ 30 d) mortality: one was valve-related and 9 were non valve-related deaths. There were four late deaths, one of which was valve-related. There were one early and 2 late cases of prosthetic valve endocarditis. At 2 years, freedom from death (%, 95% CL) was 90.9, 86.3-95.5; freedom from endocarditis was 97.3, 94.2-100; and freedom from thromboembolism was 95.2, 91.1-99.2.

It is concluded that the Toronto SPV stentless valve offers predictable performance and is associated with good early and intermediate results. Further experience is required to define the long-term performance particularly with regards to thromboembolism and endocarditis.

*By invitation

25. OVER 60 MINUTES OF DEEP HYPOTHERMIC CIRCULATORY ARREST WITH RETROGRADE CEREBRAL PERFUSION IS NOT A RISK FACTOR FOR MORTALITY AND STROKE IN AORTIC ARCH SURGERY.

Yutaka Okita, M.D.*, Shinichi Takamoto, M.D.*, Motomi Ando, M.D.*, Tetsuro Morota, M.D.*, Ritsu Matsukawa, M.D.* and Yasunaru Kawashima, M.D.

Osaka, Japan

Discussant: M. Arisan Ergin, M.D.

[Purpose] To investigate perioperative variables that affect mortality and cerebral outcomes in patients with aneurysm of the aortic arch.
[Patients] From May 1993 until September 1996, 148 patients with aneurysm of the aortic arch underwent surgery using DHCA combined with RGCP technique. Age at operation was 63.9 ± 11.6 years and 52 patients were over 70 years old. Of 70 patients with aortic dissection, 28 had acute dissection. Twelve patients had ruptured aneurysms. Fourteen patients had previous surgery on the thoracic aorta. Etiologies of the aneurysm were atherosclerosis in 123 patients and others in 25. Preoperative complications consisted with AAA in 24 patients, coronary arterial disease in 19, cerebrovascular lesions in 17, valvular heart disease in 15, COLD in 9, renal failure (RF) in 5, and peripheral vascular obstruction in 5.
[Methods] Median sternotomy was used in 92 patients and left thoracotomy in 56. Twenty-eight patients underwent replacement of the ascending aorta, including proximal arch, 70 had total arch replacement, 38 had replacement of the distal arch, and 12 had simultaneous replacement of the distal arch and the descending aorta or thoracoabdominal aorta. Arterial cannula was inserted in the ascending aorta in 50 patients and in the femoral artery or descending aorta in 78. Concomitant surgery was performed in 22 patients. [Results] There were 15 (10.1%) early deaths. Postoperative stroke was found in 6 (4.0%) patients and transient delirium in 34 (22.9%). Duration of the bypass, cardiac arrest and DHCA+RGCP was 187 ± 78 minutes, 79 ± 38 minutes and 49 ± 17 minutes respectively. Duration of the bypass was over 180 minutes in 16 patients, cardiac arrest over 120 minutes in 24, and DHCA+RGCP over 60 minutes in 35 (95 minutes at maximum). Postoperative wake-up was noticed at 7.5 ± 8.2 hours and extubation was obtained at 47.1 ± 107.8 hours after operation. Major complications consisted with respiratory problems in 22 patients, bleeding in 18, low cardiac output in 10, RF in 8, septicemia in 3, and DIG in 2. Stepwise logistic regression analysis of 31 perioperative variables demonstrated that the significant risk factors for mortality were ruptured aneurysm, preoperative COLD, and perioperative stroke. Risk factors for stroke were ruptured aneurysm and replacement of the distal arch. Risk factors for delirium were age over 70 years and atherosclerotic etiology. Risk factors for delayed awakening (over 24 hours)was ruptured aneurysm and for delayed extubation (over 36 hours) were emergency surgery and postoperative respiratory complications. Duration of DHCA+RGCP did not correlate to postoperative wake-up time ($r=0.03$, $p=0.80$), extubation time ($r=0.12$, $p=0.92$), and hospital stay ($r=0.05$, $p=0.69$). The difference for the incidence of mortality ($p=0.91$), stroke ($p=0.86$), and delirium ($p=0.47$) were not significant between two groups of patients, one with over 60 minutes of DHCA+RGCP and other.

[Conclusion] Over 60 minutes duration of DHCA+RGCP was not a risk factor for early mortality, stroke, and delirium in patients who underwent surgery for aneurysms of the aortic arch.

4:35 p.m. EXECUTIVE SESSION (Limited to Members)

6:30 p.m. MEMBER RECEPTION

*By invitation

1:45 p.m. SIMULTANEOUS SCIENTIFIC SESSION B - GENERAL THORACIC SURGERY

North Sheraton Ballroom

Moderators: Victor F. Trastek, M.D.

Douglas J. Mathisen, M.D.

26. EXPERIENCE WITH LIVING LOBAR TRANSPLANTATION FOR NON - CYSTIC FIBROSIS INDICATIONS.

Vaughn A. Starnes, M.D., Mark L. Barr, M.D.*, Felicia A. Schenkel, R.N.*, Monica V. Horn, R.N.*, Robbin G. Cohen, M.D.*, Jeffrey A. Hagen, M.D.* and Winfield J. Wells, M.D.*

Los Angeles, California

Discussant: G. Alec Patterson, M.D.

Since developing a living donor bilateral lobar transplantation protocol for cystic fibrosis patients (n = 37, 1 year survival of 73%), our indications have expanded to include recipients with other diagnoses. We report on our experience in 6 non-cystic fibrosis patients with primary pulmonary hypertension (PPH, n = 3), viral obliterative bronchiolitis (VOB, n=1), post-chemotherapy pulmonary fibrosis (PF, n=1), and bronchopulmonary dysplasia (BPD, n = 1). The average age of the 6 patients was 16.6 (range 9 - 32), all were female, 2 were on preoperative steroids, 2 were in-hospital, and the mean preoperative PCO_2 was 62 mmHg (range 36 - 120). The 1 patient with PF was intubated on high frequency jet ventilation. Each recipient received a right lower lobe (n = 5) or a right middle lobe (n = 1), and a left lower lobe (n = 6) from a total of 12 donors representing various combinations of the recipients' family, (mothers n = 5, fathers n = 4, brother n = 1, sister n = 1, cousin n = 1). The average recipient height was 59.5 inches (range 53 - 63) and the mean weight was 87.6 pounds (range 66 - 142). The mean donor height was 66 inches (range 59 - 73) and the weight was 160 pounds (range 100 - 213). Average number of HLA matches was 3.0 (range 1 - 6) and mismatches was 2.4 range (0 - 4). With an average follow up of 1 year the overall survival is 83%. The 1 death occurred in the VOB patient with recurrence of viral pneumonia at one month. Of the remaining five patients there have been no rejections and no other infections. For the 4 patients followed at least 6 months, mean FVC was 69% predicted (range 52 - 87), FEV, 69% predicted (range 54 - 83), FEF 25/75 73% predicted (range 47 - 97), DLCO/VA 76% predicted (range 66 - 85). For those patients with PPH, preoperative hemodynamics revealed mean pressures (in mmHg): RA 8 (range 2-13), PA 69 (range 60-77), PCWP 8 (range 7-10), C.I. 4.5 $L/min/m^2$ (range 3.3-4.0) and PVRI 16.8 Wood units indexed to BSA (range 16.2 - 17.4). Postoperative hemodynamics revealed a mean RA 2 (range 0 - 4), PA 18 (range 17 - 20), PCWP 6 (range 5 - 7), C.I. 5 (range 4 - 6) and PVRI 2.6 (range 2.2 - 3). Early results of living bilateral lobar transplantation for diseases other than cystic fibrosis have resulted in satisfactory survival and pulmonary function. Additionally, patients with severe PPH have had dramatic normalization of their hemodynamics despite the limited amount of lung tissue transplanted. The one year survival, incidence of rejections and infections appear to be superior to the results of CF patients in this small cohort experience.

*By invitation

27. POSTOPERATIVE CHEST X-RAYS: OPTIMUM USE IN THORACIC SURGERY.

Thomas W. Rice, M.D, Ruffin J. Graham, M.D.*, Nancy A. Obuchowski, Ph.D.*, Thirugnanam Agasthian, M.D.*, Neil A. Christie, M.D.*, Kathleen Gaebelein, M.S.N.* and Moulay A. Meziane, M.D.*

Cleveland, Ohio

Discussant: Claude Deschamps, M.D.

Daily portable chest x-rays are routinely ordered following thoracic surgery. To assess the efficacy and cost of this practice and to determine the optimum use of postoperative x-rays, a prospective review of all portable chest x-rays following 100 consecutive elective thoracotomies (DRG 75) was conducted. Each x-ray initiated a three-part survey. First, the surgeon listed whether or not the x-ray was routine and the anticipated management if the x-ray were not available. The radiologist then interpreted and scored the x-ray as either: A, expected findings requiring no intervention; B, minor findings requiring intervention; or C, major findings requiring intervention. Finally, the x-ray and the interpretation were returned to the surgeon. Any interventions necessitated by the x-ray were recorded.

In 6 months, 99 patients underwent 84 pulmonary resections and 16 other major procedures. Postoperatively, 769 portable chest x-rays were ordered, median 5 per patient (range 2-49). Of these, 731 (95%) were routine and 38 (5%), non-routine. Severity scores were:

Severity	A	B	C
All x-rays	664 (86.3%)	59 (7.7%)	46 (6.4%)
Routine x-rays	631 (86.3%)	56 (7.7%)	44 (6.0%)
Non-routine x-rays	33 (86.8%)	3 (7.9%)	2 (5.3%)

X-ray findings altered mangement in 43 of 769 x-rays (5.6%), in 33 routine (4.5%), in 10 non-routine (26.3%), in 13 A (2.0%), in 22 B (37.3%), and in 8 C (17.4%).

These results demomstrate that routine daily portable chest x-rays minimally impact management. It is, in fact, non-routine x-rays that more often alter management. If routine portable chest x-rays that cost $114 each in our institution were limited to one immediately after operation, only 133 x-rays (100 routine and 33 non-routine) would have been needed in the care of these patients. Elimination of 636 (82.7%) x-rays reduces the cost of care by $725 per patient ($286, 000 annually).

We conclude that for major thoracic procedures, it is safe, efficacious, and cost effective to initiate a protocol of one immediate postoperative portable chest x-ray as the standard order and additional portable x-rays only when clinically indicated.
*By invitation

28. GENE THERAPY FOR MALIGNANT MESOTHELIOMA: PRECLINICAL TOXICITY STUDIES LEADING TO A HUMAN CLINICAL TRIAL.

W. Roy Smythe, M.D.*, John H. Kucharczuk, M.D.*, Daniel H. Sterman, M.D.*, Ashraf E. Elshami, M.D.*, Leslie A. Litzky, M.D.*, Steven M. Albelda, M.D.* and Larry R. Kaiser, M.D.

Philadelphia, Pennsylvania

Discussant: David J. Sugarbaker, M.D.

We previously demonstrated and reported promising efficacy utilizing adenoviral vector transfer of the herpes simplex thymidine kinase gene (HSVtk) followed by ganciclovir (GCV) administration with eradication of malignant mesothelioma in animal models. We now report results of animal toxicity studies and preliminary data from a Phase I human clinical trial.

Eighty Fischer rats received either $1x10^{10}$ pfu of intrapleural H5.01ORSVTK (adenovirus carrying HSVtk) followed by 14 days of intraperitoneal (IP) GCV. Serial blood samples and necropsies were obtained. No hematologic abnormalities were noted. Mild pleuritis, epicarditis, and pneumonitis were found at necropsy. Additional animals received $1.4x10^{6}$to $1.4x10^{10}$ pfu of H5.01ORSVTK to evaluate "dose-effect." Pleural and epicardial changes were more pronounced in the higher dose groups. H5.110CBlacZ, a vector carrying a marker gene, resulted in less pronounced inflammation. HSVtk DNA was noted by PCR in spleen, liver, and kidney without pathologic change.

Three baboons were treated with $1x10^{12}$ pfu of intrapleural H5.01ORSVTK followed by IP (infusion pump) GCV. No hematologic or radiographic toxicity was noted. Only mild pleuritis was noted at necropsy.

Fourteen patients with MM have been treated with intrapleural H5.01ORSVTK at escalating doses up to $3.2x10^{11}$ pfu followed by systemic GCV. Minimal toxicity has included fever, anemia, transient liver function abnormality, and vesicular skin rash.

In summary, HSVtk/GCV gene therapy for MM produced minimal toxicity in animals when given in the same schema as proposed for a human trial. Early results from a clinical trial confirm the safety of the approach.
*By invitation

2:45 p.m. INTERMISSION - VISIT EXHIBITS

3:15 p.m. SIMULTANEOUS SCIENTIFIC SESSION B - GENERAL THORACIC SURGERY

North Sheraton Ballroom

Moderators: Victor F. Trastek, M.D.

Douglas J. Mathisen, M.D.

29. MINIMALLY INVASIVE SURGICAL STAGING IS SUPERIOR TO ENDOSCOPIC ULTRASOUND IN DETECTING LYMPH NODE METASTASES IN ESOPHAGEAL CANCER.

James D. Luketich, M.D.*, Richard Kim, M.D.*, Philip R. Schauer, M.D.*, Rodney J. Landreneau, M.D., Edmund S. Kassis*, Kathleen Urso*, Peter F. Person, M.D.* and Robert J. Keenan, M.D.* *Pittsburgh, Pennsylvania*

Discussant: Thomas W. Rice, M.D.

Purpose: Endoscopic ultrasound (EUS) is widely used to assess the loco-regional extent of esophageal cancer, but few studies exist to validate its accuracy in nodal staging. Our objective was

to compare EUS to video-assisted thoracoscopy (VATS) and laparoscopy (LAP) in evaluating the locoregional extent of esophageal cancer.

Methods: Twenty-six patients with esophageal cancer were identified over a 15-month period as having resectable disease by conventional non-invasive staging. EUS was performed followed by LAP/VATS. Tumor penetration (T) and nodal status (N) were recorded by EUS, LAP/VATS allowed lymph node sampling and limited T evaluation to rule out T4 involvement.

Results: In 8 patients EUS evaluation was NO, but LAP/VATS provided histologic confirmation of Nl in 6 of these patients. In 5/26 (19%) an obstructing lesion prevented EUS, and 3 of these had Nl by LAP/VATS. In 13 patients N1 disease was suspected by EUS, and 12/13 (92%) patients were histologically confirmed as N1 by LAP/VATS. The sensitivity and specificity of EUS for nodal evaluation were 65% and 66% respectively. The sensitivity of EUS dropped further (44%) when small nodal metastases (< 1 cm) were present and confirmed by LAP/VATS. LAP/VATS concurred with EUS evaluation of T-status in all cases. EUS was negative for metastatic disease in all 26 patients, but in 4/26 (15%), LAP identified liver metastases. In 3 patients CT suggested metastatic disease but LAP/VATS biopsies were benign. No deaths occurred following surgical staging, and the average hospital stay was less than 72 hours.

Conclusions: The overall accuracy of EUS in the diagnosis of nodal metastases in esophageal cancer was only 65% when compared to LAP/VATS. EUS was 100% accurate in evaluating T-status. EUS was severely limited in identifying small (< 1 cm) metastatic periesophageal lymph nodes (44% sensitivity). LAP/VATS improved the accuracy of staging locoregional involvement in esophageal cancer, and can be performed in the setting of high-grade obstructing lesions. LAP/VATS has the advantage of evaluating the thoracic and abdominal cavities for possible metastases.

*By invitation

30. RESECTION FOR BARRETT'S MUCOSA WITH HIGH GRADE DYSPLASIA - IMPLICATIONS FOR PROPHYLACTIC PHOTODYNAMIC THERAPY.

†Mark K. Ferguson, M.D. and Keith S. Naunheim, M.D.

Chicago, Illinois and St. Louis, Missouri
Discussant: Nasser K. Altorki, M.D.

Background Photodynamic therapy has recently been introduced as a technique for eradicating Barrett's mucosa with high grade dysplasia (HGD). However, distinguishing among HGD, carcinoma-in-situ, and invasive cancer, even with esophageal ultrasonography, is difficult. We sought to determine the incidence of invasive cancer and surgical and long-term outcomes after resection for HGD.

Methods We performed a retrospective review of patients who underwent esophagectomy for Barrett's esophagus from 1985 to 1996. Operative outcome, pathologic findings, and long-term survival were recorded.

Results During the study period 94 patients with Barrett's esophagus underwent resection. Of these, 14 were operated on for a preoperative diagnosis of HGD. There were 12 men and 2 women with a mean age of 64 years (range 35 to 76). The 14 patients rarely reported important dysphagia, and the mean weight loss was less than 2 kg. No patient received preoperative radiotherapy or chemotherapy. The operation was performed using a transhiatal approach in 9, through a left thoracotomy with an intrathoracic anastomosis in 3, through a left thoracotomy with a cervical

anastomosis in 1, and using a modified Ivor Lewis approach in 1. Reconstruction was accomplished with the stomach in 12 patients and with a colon interposition in 2. The mean blood loss was 730 ml, and patients were transfused an average of 1.4 units of blood in the perioperative period. There was no operative mortality. Four patients suffered anastomotic leaks, 3 had pulmonary complications, and 3 had cardiovascular complications. The median length of stay was 14 days. The final pathology demonstrated dysplasia in 4 patients, carcinoma-in-situ in 1, and invasive carcinoma in 9 patients (64%). All patients with invasive carcinoma had T1NOMO Stage I disease. Follow-up is complete in all patients through October, 1996, for a mean duration of 33 months (median 22 months). All patients are alive and none of the patients with invasive cancer has recurrent disease.

Conclusions A substantial percentage of patients with Barrett's mucosa containing foci of HGD have invasive carcinoma at the time of diagnosis. The use of prophylactic photodynamic therapy for these patients may expose them to the unnecessary risk of harboring an untreated cancer in tissue layers too deep to permit the photoactive compound to be activated by laser energy. Surgical management of HGD, which is accompanied by a low operative risk and achieves an excellent long-term outcome, should remain the standard therapy for Barrett's esophagus with high grade dysplasia.

†1986-88 Edward D. Churchill AATS Research Scholar

*By invitation

31. ESOPHAGECTOMY FOR FAILED ANTIREFLUX SURGERY.

Jeffrey A. Hagen, M.D.*, Michael Gadenstatter, M.D.*, Manfred P. Ritter, M.D.*, Tom R. DeMeester, M.D., Jeffrey H. Peters, M.D.*, Rodney J. Mason, M.D.* and Peter F. Crookes, M.D.*

Los Angeles, California

Discussant: Mark B. Orringer, M.D.

Introduction: Although most patients with gastroesophageal reflux disease (GERD) respond well to antireflux surgery errors in technique or procedure selection may result in failure. Most of these can be salvaged by a remedial antireflux procedure but in some esophageal function has deteriorated to the level where esophagectomy is a reasonable option. The aim of this study was to evaluate the indications and clinical outcome of esophageal resection in this setting.

Methods: Seventeen patients (M:F ratio 9:8, median age 54 years, range 27-66) who had failed previous antireflux procedures and presented with poor esophageal function and/or undilatable strictures had esophageal replacement over a 16 year period. Reflux symptoms were present for a median of nine years (range 1-30) prior to resection. All but one patient had severe dysphagia. Eight patients (47%) had one, five (29%) two and four (24%) three or more previous antireflux operations. Colon was used for replacement in 15 patients, jejunum in two. In seven patients the esophagectomy included a partial or total gastrectomy. Symptomatic improvement, meal capacity and weight change were assessed in 16 of the 17 patients at a median of seven years following surgery.

Results: Endstage disease was reflected by global loss of motility in 13 and an undilatable stricture in four patients. The former was identified by the presence of **BOTH** 40% or more simultaneous wave forms and contraction amplitudes less than 25 mmHg in the distal two thirds of the esophagus. Complications occurred in four patients and there was no mortality. Two patients required remedial

surgery for delayed gastric emptying or bile reflux. The median hospital stay was 15 days (range 12-24). All patients stated that their preoperative symptoms were cured (6/16) or improved (10/16). Thirteen patients (81%) were able to eat three meals a day and twelve (75%) enjoyed an unrestricted diet. Six patients gained or maintained weight (median gain 6 lb.) and ten lost weight (median loss 12 lb.). Two thirds of the patients were at or above their ideal body weight when asked. Fourteen patients (88%) were fully satisfied with the results of the operation, while two would not undergo the same procedure again if they had to make the decision. Conclusions: Patients with endstage disease who have had failed antireflux procedures can be salvaged by esophageal resection with a high expectation of success. Indications include global loss of motility with or without undilatable strictures. The operation can be performed safely, restores the ability to eat and maintains nutritional status.

*By invitation

32. MASSIVE HIATUS HERNIA: EVALUATION AND SURGICAL MANAGEMENT.

Donna E. Maziak, M.D.C.M.*, Tom R.J. Todd, M.D. and F. Griffith Pearson, M.D.

Toronto, Ontario, Canada

Discussant: MarkS. Allen, M.D.

Between 1960-1996 ninety-four patients with massive, incarcerated hiatus hernia were seen at our institution. The mean age was 64 years (39-85 years) with a male:female ratio of 1:1.8. Organoaxial volvulus was present in 61%. Clinical presentation in these patients included: dysphagia in 48%; chronic iron deficiency anemia in 38%; aspiration in 29%; and post-prandial pain in 56%. Symptomatic reflux, either present or remote, was seen in 83%. All patients had endoscopy. The esophagogastric junction was at a level above the diaphragmatic hiatus, denoting a sliding type of hiatus hernia, in all but 3 patients. Gross, endoscopic peptic esophagitis was observed in 36% of patients: ulcerative esophagitis in 22% and peptic esophagitis with stricture in 14%. Preoperative esophageal motility was done in all 42 patients since 1980. It was possible to advance the catheter beyond the esophagus into the stomach in 32 patients. Of these 32 complete manometric studies, the lower sphincter was hypotensive in 18 patients, and the amplitude of peristalsis in the distal esophagus was diminished in 20 patients. These are both features of significant gastroesophageal reflux disease (GERD). In 13 recent patients the distance between the upper and lower esophageal sphincters was measured during manometry. The average distance was 15.4 cm (11-20 cm), which is consistent with acquired short esophagus. The normal distance is > 18 cm. All 94 patients were treated surgically: 96% had a transthoracic repair with fundoplication, and a gastroplasty was added in 79% because of clearly defined or presumed short esophagus. There were 2 operative deaths. The mean follow-up was 70 months. Of the 88 patients followed-up, 80% are asymptomatic (excellent result); 15% have inconsequential symptoms requiring no therapy (good result); and 5 patients (4%) are improved but with significant symptoms (fair result). Two patients, neither of whom had the addition of gastroplasty, had poor results due to recurrent hernia and severe reflux. Both were successfully managed by reoperation and the addition of gastroplasty.

In summary, the majority of these 94 patients had symptoms, along with endoscopic, manometric and operative findings consistent with a sliding hernia, and a high incidence of reflux esophagitis and acquired short esophagus. True paraesophageal hernias appear rare when accurate endoscopy and motility are used. These observations support our choice of a transthoracic approach for repairs in most patients.

4:35 p.m. EXECUTIVE SESSION (Limited to Members)

6:30 p.m. MEMBER RECEPTION

*By invitation

1:45 p.m. SIMULTANEOUS SCIENTIFIC SESSION C -CONGENITAL HEART DISEASE

Washington Ballroom

Moderators: Thomas L. Spray, M.D.

Richard A. Hopkins, M.D.

33. CONOTRUNCAL REPAIR FOR TETRALOGY OF FALLOT: MID-TERM RESULT.

Hiromi Kurosawa, M.D.*, Kiyozo Morita, M.D.*, Masaaki Yamagishi, M.D.*, Anton E. Becker, M.D.* and Robert H. Anderson, M.D.*

Tokyo, Japan, Amsterdam, The Netherlands and London, England

Sponsored by: Edward L. Bove, M.D., Ann Arbor, Michigan

Discussant: Gary Lofland, M.D.

Because of the left sided main conduction bundle, the membranous flap can be safely used for closure of the VSD in tetralogy of Fallot.

Conotruncal repair consists of 1) a closure of VSD using the membranous flap or a muscle bar and not using the tricuspid septal leaflet, and 2) a short outflow patch with a wide monocusp. Elimination of fixation of the tricuspid septal leaflet could avoid dysfunction of the tricuspid valve and right ventricle.

Two hundred twenty-eight consecutive patients of tetralogy of Fallot underwent Conotruncal repair. Forty-four percent of patients were under two years old and 11% were less than twelve months old. Eighty-five percent of patients had a membranous flap at the postero-inferior border of VSD, 13% had a muscle bar between VSD and the tricuspid valve and only 2% had neither membranous flap nor muscle bar. Twenty cases had pulmonary atresia. Xenopericardial monocusp was used in the first 100 and PTFE monocusp has been used in the recent 128 patients. There were no early deaths and only 2 late deaths occurred (Late mortality = 0.08%) over a mean follow up period of 6.8 years. One death was due to pneumonia after reoperation for residual partial anomalous pulmonary venous return (PAPVR) and the other from heart failure due to coronary injury during operation. No patient required reoperation except for two with residual PAPVR. All patients had sinus rhythm and 55% had right bundle branch block. No patient has congestive heart failure or significant residual VSD. Most patients had no or trivial heart murmur indicating absence of residual VSD or outflow obstruction. Good coaptation of PTFE monocusp was revealed by echocardiogram up to five years after surgery. A consecutive group of 20 patients under 2 years-old underwent catheterization after surgery and showed CVP less than 10 mmHg, 0.46 of right/left ventricular pressure, normal right and left ventricular end-diastolic volumes, and normal election fractions of both ventricles.

In conclusion, Conotruncal repair for tetralogy of Fallot provides good quality of life, no significant hemodynamic residue, sinus rhythm, and low CVP.

*By invitation

34. THE SURGICAL MANAGEMENT OF MULTIPLE VENTRICULAR SEPTAL DEFECTS.

Lucian A. Durham, M.D., Ph.D.*, Tetsuya Kitagawa, M.D., Ph.D.*, Ralph S. Mosca, M.D.* and Edward L. Bove, M.D.

Tokushima, Japan and Ann Arbor, Michigan

Discussant: John W. Brown, M.D.

The management of patients with multiple VSD's remains controversial. Primary closure, interventional catheter techniques, and palliative surgery all may have a role and specific management guidelines remain undefined. We reviewed the records of all 33 patients with multiple VSD's undergoing repair between 1/88 and 10/96. Pulmonary artery hypertension was present in 21 patients (group 1), while the pulmonary vascular bed was protected in the remaining 12 patients (group 2). Among group 1 patients, the mean age at repair was 5.9 ± 0.9 months and all but one were less than 12 months. VSD location was perimembranous (11), posterior muscular (4), midmuscular (4), anterior muscular (5), apical (8), and subpulmonary (2). Among the 24 perimembranous and muscular VSD's, closure was accomplished from a right atriotomy alone in 22, while 2 underwent a right ventriculotomy. Apical VSD's were closed from a limited apical left ventriculotomy and subpulmonary VSD's from a pulmonary artery approach. Major associated anomalies included coarctation (n = 6) and straddling tricuspid valve (n = 1). Reoperation was performed in 4 (immediately following bypass in 2) for additional VSD not diagnosed preoperatively. There were no early or late deaths, no heart block, and no significant residual VSD's. All patients remain free of significant residual conditions at a mean of 23.4 ± 5.1 months. Among group 2 patients, the mean age at repair was 6.6 ± 3.2 years and only one was < 1 year. VSD location was perimembranous (6), posterior muscular (4), midmuscular (6), anterior muscular (2), apical (1), and subpulmonary (1). Major associated anomalies included tetralogy of Fallot (2), valvar and/or infundibular pulmonary stenosis (4), DORV with hypoplastic LV (1), and isolated LV hypoplasia (1). Unsuccessful percutaneous device closure was attempted in 2 patients. There was 1 early death (DORV and LV hypoplasia). One patient required reoperation for residual VSD and 2 underwent cardiac transplantation for LV hypoplasia or dysfunction. There were no late deaths. Six patients remain alive without significant residual or transplantation at a mean of 36.2 ± 8.0 months.

This experience indicates that primary repair for infants with multiple VSD's is associated with good late outcomes. The right atrial approach is satisfactory for all muscular defects. Limited apical left ventriculotomy for apical defects was not a risk factor. Pulmonary artery banding should be limited to patients with complex associated defects.
*By invitation

35. FATE OF RIGHT VENTRICLE TO PULMONARY ARTERY HOMOGRAFT CONDUITS: DETERMINANT FACTORS OF LATE OBSTRUCTIONS.

Jaroslav F. Stark, M.D., Kate Bull, M.D.*, Mila Stajevic, M.D.*, Muthu Jothi, M.D.*, Martin J. Elliott, M.D.* and †Marc R. de Leval, M.D.

London, England

Discussant: F. Mark Lupinetti, M.D.

Materials and Methods: The factors determining the longevity of homograft conduits (HC) remain unclear and controversial. We have reviewed records of 425 patients who survived 30 days after

placement of the pulmonary (P) ventricle to pulmonary artery HC. There were 329 aortic (A), 91 pulmonary and 5 unknown HC. The date of conduit failure was defined by the date of conduit replacement (88), balloon intervention (11), or death of the patient with the conduit in place (24). The following variables were entered into a Cox proportional hazards model: aortic versus pulmonary HC, antibiotics versus cryopreservation, ABO and Rh compatibility, type of material used for HC extension, age at operation, conduit number (reoperations). Because the prevalence of reoperations as well as of homograft type and preservation methods varied across the series, "conduit number" (1-425) was included in the multivariate models.

Results: First conduits and those inserted earlier in the series appeared to last longer than second conduits or those inserted later in the series ($p = 0.0002$ and 0.0005 respectively). Actuarial survival of first conduits was 83% at 5 and 55% at 10 years. For second, third and fourth conduits, the corresponding figures were 67% at 5 and 30% at 10 years. Regarded univariately, PHC did not perform better than AHC, even when conduit number was taken into account. Longevity did not appear to be influenced by the underlying diagnosis, age at operation, preservation technique, material used for conduit extension, or ABO and Rh matching.

Conclusions: The homograft longevity was not influenced by the use of AHC or PHC, nor by the homograft preservation technique. The most striking finding was that the second and third conduits did not last as long as the first conduits. A possible explanation for this disturbing finding is that it is more difficult at reoperation to optimise the flow dynamics through these conduits.

2:45 p.m. INTERMISSION - VISIT EXHIBITS

†1973-74 AATS Graham Fellow

*By invitation

3:15 p.m. SIMULTANEOUS SCIENTIFIC SESSION C -CONGENITAL HEART DISEASE

Washington Ballroom

Moderators: Thomas L. Spray, M.D.

Richard A. Hopkins, M.D.

36. ATRIOVENTRICULAR VALVE FUNCTION AFTER SINGLE PATCH REPAIR OF ATRIO VENTRICULAR CANAL DEFECT IN INFANCY: HOW EARLY SHOULD WE REPAIR?

V. Mohan Reddy, M.D.*, Doff B. McElhinney, M.S.*, Andrew J. Parry, M.D.*, Michael M. Brook, M.D.* and Frank L. Hanley, M.D. *San Francisco, California*

Discussant: James A. Alexander, M.D.

Patients (pts) with complete atrioventricular canal defects (CAVCD) are generally managed with medical therapy in very early infancy, largely due to technical concerns about the fragility of the atrioventricular valve (AW) tissue. We have taken the approach of completely repairing these pts earlier in infancy rather than continuing with prolonged medical management. From 7/92 to 9/96, 68 infants (<1 yr) underwent primary repair of CAVCD. Median age was 3.9 months (mo). Forty percent of pts were <3 mo of age and 80% were <6 mo. Significant associated lesions included tetralogy of Fallot or valvar pulmonary stenosis (7) , double-orifice left AVV (5), single left papillary muscle (2), aortic coarctation (3), and left superior vena cava (12). Preoperative common AVV insufficiency was severe in 2 pts, moderate in 15, mild in 32, and none in 19. Primary

complete repair was performed in all pts except for 3 who had undergone neonatal coarctation repair between 1 and 4 weeks earlier. A single patch technique with division of the bridging leaflets was employed in all pts. The left AVV cleft was closed completely (57) or partially (10) in all but 1 pt with single left papillary muscle. Annuloplasty procedures were performed on the left AVV in 14 pts and on the right AVV in 2 pts. In 10 pts Transesophageal echocardiography demonstrated inadequate valve repair requiring a return to bypass for revision. There was 1 early death (1.5%), in a pt with single papillary muscle who underwent surgery at 45 days of age. None required reoperation in the early postoperative period, and only 1 pt had greater than mild left AVV insufficiency at discharge (moderate). During a median follow-up of 20 mo (1-49 mo), 3 pts had undergone late reoperation: 1 for left AVV replacement due to severe stenosis (5 mo post-repair) and 2 for subaortic membrane resection 22 and 23 mo post-repair. Follow-up left AVV regurgitation was moderate in 1 pt, mild in 25 pts and none in the rest. Twelve pts had mild right AVV insufficiency. Age (as a continuous variable) at surgery had no relation to early or late AVV function. There was no difference in the incidence and severity of AW insufficiency between pts younger than vs older than 3 mo of age.
Conclusions. Despite concerns about AW tissue fragility in very young pts, with proper techniques excellent results can be achieved. From neonates to older infants, age of repair appears not to influence AW function. Early surgical repair rather than aggressive medical therapy is preferable to avoid prolonged morbidity associated with waiting.

*By invitation

37. DOES AORTIC-PULMONARY ANNULUS MISMATCH PREDICT AORTIC INSUFFICIENCY AFTER THE ROSS PROCEDURE IN CHILDREN?

Frank L. Hanley, M.D., V. Mohan Reddy, M.D.*, Doff B. McElhinney, M.S.*, Colin K. Phoon, M.D.* and Michael M. Brook, M.D.*

San Francisco, California

Discussant: Jan M. Quaegebeur, M.D.

Valvar insufficiency after pulmonary autograft aortic valve replacement (PAVR) can be due to a number of factors, including geometric mismatch between the native aortic and pulmonary annuli, previous or concomitant left ventricular outflow tract (LVOT) procedures, implantation techniques, and morphology of the pulmonary valve (PV). To identify factors predictive of PAVR insufficiency, we retrospectively analyzed morphologic and operative data in 38 children patients (age 4 d to 18 yrs; median 7.7 yrs) who have undergone PAVR since July 1992. One or more operations on the LVOT had previously been performed in 23 pts, including surgical valvotomy (18), subaortic resection (3), AV replacement (1), and a Konno procedure (1). No abnormal PV morphology was observed. The diameter of the PV was greater than that of the AV in 24 cases, equal in 3 cases, and less in 11 cases. The median difference between the PV and AV (PV-AV) was +3mm and ranged from +10 to -12mm. Thirteen pts had additional LVOT procedures performed at the time of PAVR (Konno =11, myectomy = 5), and a sinus obliteration technique was performed in 11 pts. PV-AV in pts undergoing Konno procedure ranged from +3 to +10mm. In these pts, geometric mismatch was corrected by ventriculoplasty. In all other pts the mismatch was corrected by gradual adjustment along the entire circumference of the autograft implant rather than by tailoring procedures at the commissures. Follow-up echocardiography at a median of 22 mo (1 to 38 mo) revealed no or trace aortic insufficiency (AI) in 28 pts, mild AI in 8 pts, and moderate AI in 2 pts. Distal aortic obstruction was present in 2 pts, 1 of whom had resulting moderate AI and underwent reoperation for arch augmentation. One other pt underwent aortic valve replacement 2 yrs post-PAVR for AI. There was no correlation between follow-up AI (‰¥miled) and age, PV-AV mismatch, previous or concurrent LVOT procedures, or sinus obliteration. Subtle

variation in techniques of PAVR are probably more important determinants of post-PAVR AI than the other factors analyzed.
*By invitation

38. NEONATAL THYMECTOMY: DOES IT EFFECT IMMUNE FUNCTION?

Winfield J. Wells, M.D.*, Robertson Parkman, M.D.* and Vaughn A. Starnes, M.D.

Los Angeles, California

Discussant: Steven R. Gundry, M.D.

Background. Thymectomy is frequently performed to improve exposure for complex congenital heart repair in the neonate. The impact on immune function has not been extensively investigated.

Methods. Nineteen neonates (<30 days of age) who had thymectomy at their operation for congenital heart repair were prospectively entered into a study to determine their subsequent immune function. Tests of immune competence included: T lymphocyte count and immunophenotype analysis (CD2, CD3, CD4, and CDS); Lymphocyte blastogenesis to mitogen (PHA), and antigen (tetanus toxoid). Antibody liters to tetanus were also determined. Samples were obtained pre-operatively, following immunization (~3 months), and at 1 year. At follow-up patients were asked about infections.

Results.

Immunophenotype: The percentage of lymphocytes expressing CD3 (all T-lymphocytes), CD4 (helper T-cells), and CDS (suppressor T-cells) are reported in Table 1. Measurements were made prior to thymectomy, at about 3 months (following immunizations), and at one year.

Table 1

Time	CD3 %	CD4%	CDS %
Pre-op	64.8 ± 16	46.6 + 12	19.8 + 7.3
~3 months	52.5 ± 12.6	37.8 ± 9.9	13.8 ± 4.5
1 year	*48.4 ± 12	*30.0 ± 11	*15.9 ± 6.8

***p = <0.05**

Although there was a significant decrease in the percentage of T lymphocytes, this decrease did not reach a level that has been associated with clinical immunodeficiency. The CD4:CD8 ratio was within normal limits.

Blastogenic Response to PHA: Blastogenesis to PHA was normal prior to thymectomy, and continued to be so at the two subsequent study times (85903.2 ± 47304.2, 103974.1 ± 64296.8, 122544.7 ± 57220.6 CPM; p = N.S.). Responses to Tetanus: Blastogenesis to tetanus toxoid following immunization was normal in all except 3 patients, and one of these three was the only patient to show a low antibody liter to tetanus toxoid with a level less than 0.1 IU/ml.

Clinical Course: No study patient required re-admission for infection over the one year of follow-up. There were a reasonably normal number of infections including bronchitis, otitis, sinusitis, and conjunctivitis which were treated with antibiotics (mean 3.5 ± 3.4 events/patient), but there was no correlation with lymphocyte number or immune function.

Conclusions. Neonatal thymectomy results in a modest decrease in T-lymphocyte level, but there is no compromise in important immune function.

*By invitation

39. PEDIATRIC HEART TRANSPLANTATION FOLLOWING THE FONT AN OR GLENN PROCEDURE.

Jan M. Quaegebeur, M.D., Mark E. Galantowicz, M.D.*, Robert E. Michler, M.D., Craig R. Smith, M.D., Eric A. Rose, M.D., Maryanne R. Kichuck, M.D.*, Linda J. Addonizio, M.D.* and Daphne T. Hsu, M.D.*

New York, New York

Discussant: Charles B. Huddleston, M.D.

At our institution, 134 pediatric heart transplants have been performed. Twenty patients had previously undergone a Fontan (17) or Glenn (3) procedure. Not only do these patients represent a significant operative challenge but they typically have multiple medical issues confounding their perioperative management. Co-morbid states present at the time of transplant are listed below. These surgical and medical issues are manifest by longer bypass times, 241 ± 67 vs. 147 + 64 minutes, longer hospital stays, 37 ± 37 vs. 27 ± 20 days, and higher thirty day mortality, 87% vs. 69%, in this high risk group as compared to the other 114 patients. However, all the patients in this cohort discharged from the hospital are still alive heralding a long-term survival equivalent to the non-Fontan group.

Co-Morbidities	
Low cardiac output	14
Protein-losing enteropathy	6
Recurrent pleural effusions	6
Severe growth retardation	6
Intractable arrhythmia	5
Cyanosis	4
Ventricular failure	3
ECMO	2
Severe A-V valve regurgitation	1

Operative strategies required flexibility in techniques to reconstruct the recipients' anatomy to afford an orthotopic heart transplant. The use of donor tissue, especially extra-length on the great vessels and pericardium, was characteristic of these reconstructions. Residual anatomic defects were manifest in 10 patients post-operatively: aortopulmonary collaterals (6), pulmonary A-V fistulae (3), branch pulmonary artery stenoses (2), coarctation (1). Pulmonary A-V fistulae all regressed spontaneously post-transplantation. All other defects were effectively managed in the invasive catheterization suite through embolization or dilatation. Although this patient cohort presents significant challenges operatively and during the immediate post-operative period they enjoy similar long-term survival as pediatric patients with less complex anatomy receiving heart transplants.

4:35 p.m. EXECUTIVE SESSION (Limited to Members)

6:30 p.m. MEMBER RECEPTION

WEDNESDAY MORNING, MAY 7, 1997

7:00 a.m. FORUM SESSION II -

GENERAL THORACIC SURGERY

South Sheraton Ballroom

Moderators: Nasser K. Altorki, M.D.

Larry R. Kaiser, M.D.

F9. TOTAL RESPIRATORY SUPPORT FROM SWINE PULMONARY XENOGRAFTS IN PRIMATES.

Casey W. Daggett, M.D.*, Mark Yeatman, F.R.C.S.*, Edward P. Chen, M.D.*, Andrew J. Lodge, M.D.*, Carmelo Gulotto, B.B.A.*, Shu S. Lin, M.D.*, Jeffery L. Platt, M.D.* and Robert D. Davis, M.D.*

Durham, North Carolina

Sponsored by: Ross M. Underleider, M.D., Durham, North Carolina

Background. The use of nonhuman lung donors, such as swine, has the potential to provide an unlimited supply of organs. However, a major barrier to pulmonary xenotransplantation is hyperacute rejection. *Objective.* To test the hypothesis that pre-transplant swine lung perfusion will deplete xenoreactive antibody, prevent hyperacute swine-to-primate pulmonary xenograft rejection and allow for a functional swine pulmonary xenograft. *Methods.* Six baboons (12-15 kg) underwent left pneumonectomy followed by left orthotopic swine lung transplantation. Three baboons (group I) received antibody depletion by perfusion with swine lungs prior to transplantation, and three received no treatment prior to transplantation (group II). *Results.* Perfusion of baboon blood through swine lungs for 120 minutes achieved an 85 ± 5% reduction in xenoreative IgM levels. Following transplantation, group I pulmonary xenografts had a blood flow of 613.3 ± 122.1 ml/min and a pulmonary vascular resistance (PVR) of 29.4 ± 14.4 mm Hg/L/min at 60 minutes of reperfusion, compared to group II, which had a pulmonary blood flow of 26.7 ± 12.1 ml/min ($p<0.05$) and a PVR of 440.4 ±196.1 mm Hg/L/min ($p<0.05$). While group II lungs lost all pulmonary blood flow by three hours of reperfusion, group I pulmonary xenografts continued to function well for the duration of the study and at eleven hours of reperfusion maintained a pulmonary blood flow of 440.0 ± 40.7 ml/min with a PVR of 51.5 ± 23.7 mm Hg/L/min. After 60 minutes of reperfusion, two of three group I animals also tolerated complete occlusion of the right pulmonary artery, having the baboon rely completely on the swine pulmonary xenograft for respiratory function over eleven hours. Group II animals did not tolerate occlusion of the right pulmonary artery and displayed cardiovascular failure within 20 seconds of occlusion. Pathologic analysis of group I lungs displayed little histologic evidence of injury in biopsies taken at one and eleven hours of reperfusion. However, group II lung biopsies taken at one hour of reperfusion showed alveolar edema and hemorrhage with small vessel thrombosis. The function of group I pulmonary xenografts during occlusion of the right pulmonary artery were measured by arterial blood gas analysis, illustrated in the table below. (Data are given as the mean (± SEM).

Reperfusion time (hours)	1	3	5	7	9	11
PaO_2(mmHg)	368(90)	511(49)	342(171)	410(12)	457(105)	476(71)
pCO_2(mmHg)	34(11)	34(5)	33(4)	33(4)	31(1)	29(3)

pH	7.4(0.1)	7.5(0.1)	7.4(0.1)	7.4(0.1)	7.4(0.1)	7.4(0.1)

Conclusion. Pre-transplant perfusion with swine lungs is effective in removing xenoreactive antibodies and prevents hyperacute pulmonary xenograft rejection. Swine pulmonary xenografts can provide complete respiratory support in primate recipients.

*By invitation

F10. CONTROLLED REPERFUSION AFTER LUNG ISCHEMIA: IMPLICATIONS FOR IMPROVED FUNCTION AFTER LUNG TRANSPLANTATION.

Bradley S. Allen, M.D.*, Ari Halldorsson, M.D.*, Michael Kronen, M.D.*, Kirk S. Boiling, M.D., M.P.H.*, Tingrong Wang, M.D.*, Shaikh Ramon, M.S.* and Harold Feinberg, Ph.D.*

Chicago, Illinois

Sponsored by: Renee S. Hartz, M.D., Chicago, Illinois

Despite improvements in tissue preservation, reperfusion injury remains a major source of morbidity and mortality following lung transplantation. Although controlling the conditions of reperfusion and the composition of the reperfusate have been shown to modify the reperfusion injury in the myocardium, these principals have not been investigated following lung ischemia. Twenty adult pigs underwent 2 hours of warm left lung ischemia by cross clamping the left bronchus and pulmonary artery. In 5 (Group 1) the cross clamp was simply removed (unmodified reperfusion). Fifteen other pigs underwent modified reperfusion using blood from the femoral artery to perfuse the lung via the pulmonary artery (pressure <50mm Hg) for 10 minutes prior to removing the clamps. In 5 (Group 2) blood was mixed with crystalloid using a BCD resulting in a substrate enriched, hypocalcemic, hyperosmolar, alkaline solution, in 5 (Group 3) the blood was circulated through a leukocyte depleting filter, and 5 (Group 4) underwent reperfusion with a WBC filter and modified solution. Left lung function was assessed 60 minutes after reperfusion and expressed as percentage of control, and a biopsy taken for lung water and myloperoxidase (us/me protein).

	Compliance	PVR	a/A ratio	Lung water	Myloperoxidase
Group 1	77 ± 1%	198 ± 1%	27 ± 2%	84.3 ± .2%	0.35 ± .02
Group 2	86 ± 1%*	153 ± 2%*	51 ± 1%*	83.4 ± .2%*	0.21 ± .03*
Group 3	91 ± 1%*	133 ± 1%*	77 ± 2%*	83.3 ± .2%*	0.17 ± .01*
Group 4	98 ± 1%**	106 ± 1%**	97 ± 2%**	82.1 ± .4%**	0.1 8 ± .02*
Mean ± S.E., *p<0 05 vs. Group 1 (control), **<0.05 vs. all groups					

In conclusion, after 2 hours of pulmonary ischemia: 1) a severe lung injury occurs following uncontrolled (unmodified blood) reperfusion, 2) controlled reperfusion with either a modified reperfusion solution or WBC filter limits, but does not avoid, a lung reperfusion injury, 3) reperfusion using both a modified reperfusate and WBC filter results in complete preservation of pulmonary function. We therefore believe surgeons should control the reperfusate following lung transplantation to improve postoperative pulmonary function.

*By invitation

F11. PROGNOSTIC SIGNIFICANCE OF p27 AND p53 IN PATIENTS WITH ADENOCARCINOMA IN BARRETT'S ESOPHAGUS.

Surendra P. Singh, M.D.*, Jennifer Lipman, M.D.*, M. Giulia Cangi, Ph.D.*, Laura Aizenman*, F. Henry Ellis, Jr., M.D. and Massimo Loda, M.D.*

Boston, Massachusetts

BACKGROUND. Loss of cell cycle control is believed to be an important step in human tumor development. The Cyclin-dependent kinase inhibitors (Ckis) p21 and p27 mediate cell cycle arrest by preventing cells from entering S phase. p21 is induced by tumor suppressor p53 in response to DNA damage, while p27 is induced in quiescent/terminally differentiated cells.

HYPOTHESIS. We hypothesize that loss of p27 in invasive carcinomas may be associated with disease progression. Finally, overexpression of p53, indicative of mutation of this gene with subsequent failure to induce the Cki p21, may also be associated with cancer progression.

METHODS. We previously evaluated the expression of p27 in formalin-fixed and paraffin-embedded sections from 49 cases of invasive adenocarcinoma, 4 carcinoma in situ, in Barrett's esophagus (BE) by immunohistochemistry with a monoclonal anti-p27kip1 antibody. In this study we also examined the expression of p53 as another cell cycle regulator. Twenty-three of these cases had BE-associated dysplasia. As previously described, cases with ‰¥50% and ‰¥10% positive nuclear staining were considered positive for p27 and p53, respectively.

RESULTS. As expected, normal mucosa and non-dysplastic BE showed no p53 immunoreactivity while p27 was expressed in the nuclei of superficial differentiated cells. In contrast, p27 was expressed in the base of the pits of all cases of dysplasia, presumably to counteract enhanced proliferative activity. p53 was also overexpressed in 74% of dysplasias. Tumor progression was associated with loss of p27 and p53 overexpression: 82% of invasive cancers had low p27 expression while p53 was overexpressed in 55%. p27 expression correlated with patient survival (p=0.0007), presence of lymph node metastasis (p=0.0014) and histopathologic differentiation (p=0.0001) whereas p53 correlated only with histopathologic differentiation (p=0.0035). There was no correlation between p27 and p53.

CONCLUSIONS. 1) p27 overexpression in dysplastic cells of BE may be a physiological response to genetically damaged cells. 2) p27 and p53 may be used as immunohistochemical markers of dysplasia in patients with BE. 3) Loss of p27 expression, but not p53 overexpression, is a negative prognostic factor in patients with adenocarcinoma in BE.
*By invitation

F12. INHALED NITRIC OXIDE ATTENUATES ISCHEMIA-REPERFUSION INJURY AFTER NON-HEART-BEATING-DONOR LUNG TRANSPLANTATION.

Emile A. Bacha, M.D.*, Shinya Murakami, M.D.*, Paolo Machiarini, M.D.*, Guy-Michel Mazmanian, M.D.*, Alain R. Chapelier, M.D.*, Philippe Herve, M.D.* and Philippe G. Dartevelle, M.D.

Boston, Massachusetts and Le Plessis-Robinson, France

Nitric oxide inhibits polymorphonuclear neutrophils (PMN) activation and attenuates pulmonary IR injury. We studied the effect of inhaled NO on IR injury after NHBD lung transplantation by

measuring lung function, recipient survival, graft PMN sequestration as well as adherence of recipient circulating PMN to cultured pulmonary artery endothelial cells (PAEC).

Methods: Pigs were assigned to a NO (30 ppm) vs control group (n = 9). Cadavers were ventilated. After 3 hours of postmortem in situ warm ischemia, and 2 hours of cold ischemia, left allotransplantation was performed. The right PA was ligated one hour after reperfusion. Hemodynamic and gas exchange data were recorded hourly for 9 hours. Circulating PNM adherence to tumor necrosis factor-alpha (TNF)- and calcium ionophore (Cal)-stimulated PAEC was measured after reperfusion. Lung PMN sequestration was determined by measuring myeloperoxidase activity.

Results: After PA ligation, NO-treated animals exhibited significantly (two-way analysis of variance) lowered pulmonary vascular resistance ($p<0.01$), improved oxygenation ($p<0.01$), and survival ($p<0.05$). Adhesion of PMN to PAEC was significantly inhibited in the NO group (22 ± 3 vs 33 ± 3% after Cal stimulation; 20 ± 3 vs $52 + 3$% after TNF stimulation, $p<0.0001$). PNM sequestration was significantly reduced by NO (0.12 ± 0.06 vs 0.25 ± 0.04 U/100mg tissue, $p<0.05$).

Conclusions: Inhaled NO attenuates IR injury after NHBD lung transplantation. This is likely the result of a dual action by inhaled NO: 1) prevention of IR-induced pulmonary vasoconstriction, and 2) direct action on PMN resulting in inhibition of adherence to endothelium.

*By invitation

F13. IN VIVO AND EX VIVO GENE TRANSFER IN RAT LUNG ISOGRAFTS.

Carlos H. Boasquevisque, M.D.*, Bassem N. Mora, M.D.*, Teng C. Lee, B.S.*, Ronald K. Scheule, Ph.D.*, Joel D. Cooper, M.D, Mitchell D. Botney, M.D.* and G. Alec Patterson, M.D.

St. Louis, Missouri and Framingham, Massachusetts

Background: In transplantation, gene transfer to the donor organ prior to transplant offers potential for targeted therapy directed at specific post-operative complications, such as ischemia-reperfusion injury and rejection. It is important that the vector be non-toxic and the transferred gene be expressed at the time of implantation. The aim of this study was to achieve transgene expression in transplanted lung grafts using a cationic lipid complexed to a reporter gene.

Methods: cDNA encoding for chloramphenicol acetyl transferase (CAT) was complexed to a cationic lipid, Lipid #67 (Genzyme Corporation, Framingham, MA), and was injected into Fischer rats. Successful transfection was assessed by the CAT assay. The distribution and type of transfected cells were evaluated by *in situ* hybridization. Lung toxicity was assessed by measuring arterial oxygenation (P_{aO2}), the host inflammatory response (by H&E staining and EDI immunohistochemistry) and TNF-Î± levels. Animals were divided into three major groups. In Group 1 (non-transplant setting), the CAT-Lipid #67 complex was injected intravenously via the left external jugular vein. Lungs were harvested at various time points later: 2 hours, 6 hours, 12 hours, 1, 2, 3, 5, 8 and 21 days (n = 3). In Group 2 (transplant setting - *in vivo* graft transfection), rats were divided into 3 sub-groups (n = 5). In sub-group 1, animals were intravenously injected with the CAT-Lipid #67 complex 4 hours prior to left lung harvest and orthotopic implantation in recipient animals, which were sacrificed 44 hours later. In sub-groups 2 and 3, lungs were harvested 4 hours and 48 hours after intravenous injection and served as controls. In Group 3 (transplant setting - *ex vivo* graft transfection) the CAT-Lipid #67 complex was infused

retrograde via the left pulmonary vein after graft harvest and flush (n = 6). Grafts were then kept at room temperature for 4 hours prior to implantation. Recipients were sacrificed 44 hours later.

Results: Gene expression was detected as early as 2 hours. High levels of gene expression were present from 6 hours to 8 days. By 21 days, gene expression was greatly attenuated. Transgene expression was observed in all treated animals and was homogeneously distributed throughout the lung. *In situ* hybridization localized CAT mRNA to endothelial cells, macrophages and interstitial cells. Lung gas exchange was not significantly different in treated and untreated animals (P_{aO2}, mmHg: 501.18 ± 40.89, 537.17 ± 71.24, and 523.56 ± 18.4 for transplanted treated animals, non-transplanted treated animals and untreated normal rats, respectively, p=0.8). Inflammatory infiltrate was minimal, although TNF-a levels increased seven-fold in treated animals.

Conclusion: *In vivo* and *ex vivo* cationic-lipid-mediated gene transfer to lungs isografts is possible and allows significant transgene expression without impairment in graft function.

*By invitation

F14. AEROSOL CYCLOSPORINE PREVENTS ACUTE ALLOGRAFT REJECTION IN EXPERIMENTAL PULMONARY TRANSPLANTATION.

Surindra N. Mitruka, M.D.*, Si M. Pham, M.D.*, Adrianna Zeevi, Ph.D.*, Sen Li, M.D.*, Jane Cai, M.D.*, Gilbert J. Burckart, Pharm.D.*, Samuel A. Yousem, M.D.*, Robert J. Keenan, M.D.* and Bartley P. Griffith, M.D.
Pittsburgh, Pennsylvania

BACKGROUND: The incidence of acute rejection and the morbidity associated with systemic cyclosporine (CsA) following pulmonary transplantation is significant. Recent evidence suggests that the lung allograft locally initiates and modulates the immune mechanisms involved in acute rejection. The purpose of this study was to determine if regional immunosuppression with aerosolized cyclosporine would prevent acute lung rejection, achieve high intra-graft concentration with low systemic delivery, and effect production of the pro-inflammatory cytokines involved in the acute rejection response.

METHODS: Unilateral orthotopic left lung transplantation was performed in 18 rats (ACI to Lewis) across major and minor histocompatibility barriers. The rats were divided into two groups : allogeneic control (n = 6) and aerosolized (3 mg/kg/day) cyclosporine (n = 12). Rats were sacrificed on POD 2, 4, and 6, and the transplanted lung, native lung, spleen, and blood collected. Histology, HPLC for CsA concentrations, and RT-PCR for cytokine gene expression was performed. Low dose (2 mg/kg/day) and high dose (10 mg/kg/day) systemic CsA groups (previous data) were used for comparison.

RESULTS:

	Rejection POD 6	Blood CsA cone mean POD 2-6	Graft CsA cone mean POD 2-6
allo control	4	0	0
low dose IM	3	232	3688
high dose IM	2	2046	14519
aerosol CsA	1	725	12824

Aerosol CsA controlled rejection with a significantly less blood concentration (725 ng/ml vs. 2046 ng/ml) and a similar tissue concentration (12, 824 ng/mg vs. 14, 519 ng/mg) compared to the high dose systemic group. The pro-inflammatory cytokines increased continuously in untreated animals from POD 2 to POD 6 at which time rejection was complete. Aerosol CsA treated animals initially expressed IL-6 and IFN-g on POD 2 but none thereafter, and iNOS production was completely attenuated; similar to results obtained in the high dose systemic CsA group.

CONCLUSION: Local delivery of CsA by aerosol inhalation effectively prevented acute rejection of the rat lung allograft. Moderate dose aerosolized CsA achieved high graft concentrations with low systemic delivery. The gene expression of pro-inflammatory cytokines involved in acute rejection was suppressed by aerosol CsA therapy.

*By invitation

F15. EXPRESSION OF ACIDIC FIBROBLAST GROWTH FACTOR CONTRIBUTES TO MALIGNANT TRANSFORMATION IN BARRETT'S ESOPHAGUS.

Robert Soslow, M.D.*, Liang Ying, M.D.* and Nasser K. Altorki, M.D.

New York, New York

The process of tumorigenesis involves loss of function of tumor suppressor genes or activation of oncogenes many of which encode for various growth factors. Acidic fibroblast growth factor (aFGF) is a potent mitogen whose RNA transcripts were shown to be overexpressed in Barrett's adenocarcinoma. In this study we investigated aFGF protein expression in 17 esophagectomy specimens from patients with Barrett's adenocarcinoma. Immunostaining was performed on paraffin embedded tissue using a streptavidin-biotin technique with monoclonal antibody against aFGF. In nine cases, the examined sections contained residual Barrett's epithelium (metaplasia 5, low grade dysplasia 3, high grade dysplasia 9). Epithelial cells were considered positive for aFGF if greater than 10% were immunostained.
Results are shown below:

	Positive aFGF	**Negative aFGF**
Control (gastric funds)	2	15
Barrett metasplasia	1	4
Low grade dysplasia	2	1
High grade dysplasia	8	1
Carcinoma	17	0

Immunostaining was very intense (3+) in all carcinoma cases and 8/9 cases with high grade dysplasia. We conclude that increased expression of aFGF plays an important role in tumorigenesis in patients with Barrett's esophagus and further studies should be conducted to evaluate its use as a clinical blomarker.

*By invitation

F16. SUCCESSFUL IN VIVO AND EX VIVO TRANSFECTION OF PULMONARY ARTERY SEGMENT IN LUNG ISOGRAFTS.

Motoki Yano, M.D.*, Carlos H. Boasquevisque, M.D.*, Itaru Nagahiro, M.D.*, Masafiimi Hiratsuka, M.D.*, Bassem N. Mora, M.D.*, Joel D. Cooper, M.D. and G. Alec Patterson, M.D.

St. Louis, Missouri

Background: Gene transfer into donor lung grafts is feasible and may be useful in reducing reperfusion injury and rejection. However using conventional viral vectors whole organ transfection remains inefficient. Yet focal proximal pulmonary artery endothehal transfection may provide satisfactory downstream effects on the whole graft. The aim of this study was to achieve transfection of proximal pulmonary artery segments in left lung isografts.

Methods: Fisher rats (250-280 g) were divided into three groups. In group I (n = 4) and group II (n = 7) intact donor rats were subjected to occlusion of the proximal left pulmonary artery segment for twenty minutes after which flow was restored. In group I, 0.03 ml saline was injected into the pulmonary artery segment via a catheter in the right ventricle. In group II, the pulmonary artery segments were injected with 2-4x10^{10} pfu/ml replication deficient adenovirus type V with LacZ gene encoded Î²-galactosidase. In group III (n = 5) donor lungs were flushed with 20 ml LPDG solution and extracted. The same construct as for group II was instilled ex vivo into the occluded left pulmonary artery segment. After three hours storage (10°C), the grafts were implanted. In all groups, seventy-two hours after reperfusion, heart-lung blocks were flushed with PBS and Bluo-gal and immersed in Bluo-gal for three hours.

Results: The survival rates were 50% (group I), 43% (group II) and 100% (group III). Macroscopically, in all survival animals of group II and III, multiple blue spots were observed on the endothelial surface of the proximal left pulmonary artery indicating successful gene transfection. Microscopically, blue stained endothelial and smooth muscle ells were observed.

Conclusion: A high rate of focal gene transduction was observed in proximal pulmonary artery segments following in vivo and ex vivo exposure. Direct gene transfer to pulmonary artery segments is feasible and may avoid potential complications of systemic transfection strategies.

*By invitation

F17. CHANGES IN PULMONARY PHYSIOLOGY AFTER LUNG VOLUME REDUCTION SURGERY IN A RABBIT MODEL OF OBSTRUCTIVE DIFFUSE EMPHYSEMA.

Joseph Huh, M.D.*, Matthew Brenner, M.D.*, John C. Chen, M.D.*, Edward A. Stemmer, M.D., Benedict Yoong, B.S.*, David Mukai, B.S.* and Jeffrey C. Milliken, M.D.*
Orange, California

Purpose: While surgical treatment of emphysema has recently gained popularity, the mechanism by which lung volume reduction surgery (LVRS) improves respiratory physiology is still incompletely understood. Using an elastase induced purely obstructive emphysema model in New Zealand White rabbit, we studied the effects of LVRS on puhnonary compliance, airway flow, measured lung volume, and diffusion capacity.

Methods: Emphysema was induced in 14 New Zealand white rabbits by aerosolizing 15, 000 units of porcine elastase through an endotracheal tube under general anesthesia. Transpleural pressures were measured at 60, 50, 40, 30, and 20 cc's inflation above functional residual capacity (FRC). Measurements were taken at baseline prior to induction of emphysema, preoperatively at 4 weeks following induction of emphysema, and 1 week postoperatively following LVRS. FEV1, helium dilution lung volume, and single breath DLco were also measured concurrently. Stapled resection of bilateral upper lobes was performed through a midline sternotomy with a standard multirow surgical stapler (Ethicon). Histologic examination was obtained one week postoperatively.

Results: Comparison of compliance curves showed an increase in compliance following induction of emphysema and a decrease in response to LVRS (graph). In like fashion, FEV1 showed improvement in airway flow postoperatively, although this did not reach statistical significance, while FRC decreased following LVRS. DLco did not show a significant change (Table). Histologic examination confirmed presence of severe diffuse emphysema in each animal at necropsy.

LUNG PARAMETERS			(t-test)
	Baseline	Preop	p value
FEV1 (cc)	47.22	21.09	0.006
FRC (cc)	26.63	32.00	0.03
DLco (cc/min/mraHg)	0.62	0.56	0.37
	Preop	Postop	p value
FEV1 (cc)	21.09	31.33	0.28
FRC (cc)	32.00	22.52	0.05
DLco (cc/min/mmHg)	0.56	0.61	0.56

Conclusion: We have developed an animal model of elastase induced diffuse emphysema applicable for LVRS studies. Decreased compliance and increased airway flow following volume reduction surgery parallels findings in human studies and suggests that similar mechanisms of increased elastic recoil and airway support contribute to improvement. Furthermore, helium dilution volumes show a decrease in lung volume postoperatively without significant decrease in diffusion capacity. This model may be useful in assessing surgical techniques in LVRS, and may help identify optimal location and quantity of lung tissue excision in the surgical treatment of emphysema.

Supported by NIH Grant #RR-011-92, DOE Grant #DE-FG03-91ER61, and DOD Grant #N00014-91-C-0134.

*By invitation

F18. THE RELATIONSHIP OF ISCHEMIA REPERFUSION INJURY AND THE EXPRESSION OF MAJOR HISTOCOMPATIBILITY COMPLEX ON HOST LYMPHOCYTES.

Karim A. Qayumi, M.D., Ph.D.*, David V. Godin, Ph.D.*, Maryam Nikbakht-Sangari, B.Sc.*, John C. English, M.D.*, Kathleen J. Horley, Ph.D.*, Seung P. Lim, M.D., Ph.D.*, Shahid Gul, B.Sc.* and Michael S. Koehle, B.Sc.H.*

Vancouver, British Columbia and Toronto, Ontario, Canada; Chung-Ku, Korea

Sponsored by: G. Frank O. Tyers, M.D., Vancouver, British Columbia, Canada

This study was designed to examine the effect of *ex vivo* preservation time on the release of specific inflammatory mediators, and the levels of plasma and tissue antioxidants related to the rejection of the transplanted organ (measured by the expression of MHC HLA-DR-Î² on host lymphocytes). Single lung transplantation was performed on three gropus of domestic swine. Group A (n = 7) and Group B (n = 6) had *ex vivo* preservation times of 4 and 15 hours respectively at 4°C hypothermia. Group C (n = 6) underwent 2 hours of warm ischemia with the left pulmonary artery, vein, and bronchus cross-clamped without explantation. Methods of assessment included: the release of inflammatory mediators-thromboxane B_2 (TxB), interleukin-2 (IL-2), IL-4, IL-10, tumour necrosis factor Î± (TNFÎ±) quantitated by radioimmunoassay and/or enzyme linked immunosorbent assay; the levels of plasma and tissue antioxidants determined by enzyme bioassay, the expression of MHC HLA-DR-P on host lymphocytes by fluorescence intensity; and the mechanics of lung function by measurement of lung compliance, a/A ratio, and lung weight. The results demonstrated increases ($p<0.05$) TxB, IL-2, IL-4, lung weight, O_2 gradient, and HLA-DR-Î² expression on host lymphocytes directly proportional to ischemic time. IL-10, TNFÎ±, lung compliance, a/A ratio, and both plasma and tissue antioxidants were inversely proportional to ischemic time ($p<0.05$). Similar results were observed in Group C which experienced ischemia reperfusion injury without tissue incompability. These results suggest that ischemia reperfusion injury alone without the complication of tissue incompatibility. These results suggest that ischemia reperfusion injury alone without the complication of tissue incompatibility is enough to initiate an acute post-transplantation response. Thus the severity of ischemia reperfusion injury, as measured by the release of inflammatory mediators and the levels of antioxidants, could be directly related to the intensity of rejection of the transplanted organ, as measured by the expression of HLA-DR-Î² on host lymphocytes.

*By invitation

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

North Sheraton Ballroom

Moderators: D. Glenn Pennington, M.D.

Tirone E. David, M.D.

F19. LONG-TERM GENE EXPRESSION AFTER VIRAL TRANSDUCTION OF CARDIAC ISOGRAFTS USING DNA VIRAL VECTORS.

Boulos Asfour, M.D.*, Paul D. Kessler, M.D.*, Ralph H. Hruban, M.D.*, Duke E. Cameron, M.D. and Barry J. Byrne, M.D., Ph.D.*

Baltimore, Maryland

Objectives: Viral transduction of cardiac allografts provides the opportunity to genetically modify graft vasculature and myocardium as well as to achieve graft-specific immunosuppression or possibly tolerance. However, successful gene therapy in cardiac transplantation will require both gene delivery in a clinically applicable manner and long-term gene expression. Adenovirus has been used successfully to achieve short-term transduction in cardiovascular tissue. Adeno-associated virus (AAV) is a parvovirus which we have previously shown to be effective in long-term transduction of skeletal muscle, vascular smooth muscle, and cardiomyocytes *in vitro* and *in vivo.*

Methods: Female adult Sprague-Dawley rats (200-250 gms, n = 20) served as isograft donors and recipients for vascularized heterotopic cardiac transplants. Following cannulation of the right

carotid artery, the graft was perfused with oxygenated cold (15°C) Krebs-Henselite solution and the donor organ harvested. A viral solution containing either, AAV-IacZ (5 x 10^9 particles) or Ad-lacZ (1 x 10^9 pfu), encoding the marker protein, bacterial Î²-galactosidase was then delivered to the coronary vasculature. Perfusion was discontinued to allow for intracardiac recirculation of virus via creation of an atrial septal defect or pulmonary bypass (PA-LA or PA-Ao shunt).

Results: Two weeks to two months following transplantation, grafts were removed for histological analysis of Î²-galactosidase activity. Uniform staining of cardiac and vascular smooth muscle was observed at the highest dose with viral recirculation. The transgene was expressed for up to two months in hearts transduced by AAV-lacZ.

Conclusions: We have shown that AAV-lacZ and Ad-lacZ delivered via the coronary vasculature of cardiac allografts are able to transduce vascular and cardiac tissue under conditions of hypothermic perfusion and storage currently used in clinical cardiac transplantation. This approach will be useful for genetic modification of cardiac allografts in the management of graft vasculopathy and rejection.

*By invitation

F20. HEARTS AND KIDNEYS FROM TRANSGENIC SWINE EXPRESSING HUMAN COMPLEMENT REGULATORY PROTEINS ARE PROTECTED FROM HYPERACUTE REJECTION IN PRIMATES.

Sepehre Naficy, M.D.*, Lisa E. Diamond, Ph.D.*, John S. Logan, Ph.D.* and David H. Adams, M.D.*

Boston, Massachusetts and Princeton, New Jersey

Sponsored by: Lawrence H. Cohn, M.D., Boston, Massachusetts

Unmodified vascularized porcine organs undergo destruction of tissue architecture and organ function by hyperacute rejection within minutes to hours of transplantation into baboon recipients. Recipient complement activation and deposition onto donor endothelium is pivotal in the pathogenesis of hyperacute rejection. Decay accelerating factor (DAF), membrane cofactor protein (MCP), and CD59 are important human complement regulatory proteins (CRP). In order to clarify the role donor CRP play in pig to baboon hyperacute rejection, transgenic swine expressing different human CRP combinations were used as donors. One heart expressing MCP, two hearts expressing CD59/DAF, and two kidneys expressing CD59/DAF were transplanted into five baboon recipients. Cardiac grafts were placed heterotopically in the neck and followed by periodic inspection, echocardiography, and biopsy. Renal grafts were placed heterotopically in the abdomen following bilateral recipient nephrectomies. Recipient blood was tested serially for trough Cyclosporine A levels, CBCs, creatinine, and serum xenoreactive antibody liters. Xenograft samples were examined by light microscopy and immunostained for detection of antibody deposition and complement activation.

Transgenic organs were protected against hyperacute rejection. The MCP heart was explanted with normal function after 1.8 days following sudden death of the baboon. The CD59/DAF hearts survived 3.6 and 5.4 days. One CD59/DAF kidney was explanted after 8.3 days with normal function (creatinine 2.1) from a terminally anemic recipient. The second transplanted kidney is functioning well (creatinine 1.3) after 11 days. Biopsies from functioning grafts were noted to have preserved histologic architecture. Rejected hearts demonstrated regional hemorrhage and interstitial edema, with fibrin plugs in thrombosed vessels. Immunostaining revealed specific and prominent IgM and C4 endothelial deposition and inhibition of terminal membrane attack complex

formation. Kidney samples showed normal histology with positive immunostaining for IgM and C4. Our results demonstrate transgenic swine organs expressing human CRP are protected from hyperacute rejection. Further understanding of ongoing immune events resulting in graft failure is required before pig xenotransplantation can be applied clinically.
*By invitation

F21. EFFICIENCY OF A HIGH-TITRE RETROVIRAL VECTOR IN GENE TRANSFER INTO SKELETAL MYOBLASTS.

Reida M. El Oakley, F.R.C.S., M.D.*, Mark A. Poznansky, M.R.C.P., Ph.D.*, Madeliene C. McMullen*, Gregor M. Adams, B.Sc.*, Nigel J. Brand, Ph.D.*, Paul J.R. Barton, Ph.D.* and Magdi H. Yacoub, F.R.C.S., Ph.D.

London, England

Background: Grafting genetically modified skeletal myoblasts for myocardial repair is dependent on an efficient gene transfer system that integrates the gene(s) of interest into the chromosome of the target cell and its progeny. The aim of this investigation is to evaluate the use of a new retroviral-based gene transfer system for this purpose.

Methods & Results: The retroviral vector MFG, carrying the Î² galactosidase gene (lacZ) with nuclear localisation signal, was used to transduce the skeletal myoblast cell line L6. The MFG-lacZ construct was packaged in a fourth generation, high-litre, split-genome packaging cell line (FLYA4). This cell line produced 10^5-10^6 infectious units per ml. L6 cells were cultured in tissue culture flasks and transduced with MFG-lacZ using filtered supernatant from the packaging cells. Transduced L6 cells were divided into 4 groups. Group I, cells were fixed as myoblasts 3 days after transduction. Group II, cells were allowed to differentiate into myotubes. Group III, cells were split every 3 days for 4 months. Group IV, cells were split as in group III, and allowed to differentiate into myotubes. For each group, un-transduced L6 cells acted as "control". All samples were fixed with 3.7% formaldehyde and stained for lacZ activity. The percentage of cells with successful transgene expression are presented in the following table:

	group I	group II	group III	group IV	Controls
% of positive cells	69.3%	73.6%	68.9%	71.4%	0%

Conclusion: Recombinant MFG retroviral agents packaged in a high-litre, split-genome packaging cells are efficient in gene-transfer into skeletal myoblasts and result in stable transgene expression even after repealed cell division and/or differentiation.

*By invitation

F22. EX-VIVO ADENOVIRAL-MEDIATED GENE TRANSFER TO THE TRANSPLANTED ADULT RAT HEART.

Alan P. Kypson, M.D.*, Karsten Peppel, Ph.D.*, Shahab A. Akhter, M.D.*, R. Eric Lilly, M.D.*, Donald D. Glower, M.D.*, Robert J. Lefkowitz, M.D.* and Walter J. Koch, Ph.D.*

Durham, North Carolina

Sponsored by: Robert W. Anderson, M.D., Durham, North Carolina

The ability to transfer functional genes to adult myocardium represents an area of study with potentially significant therapeutic implications. We investigated the feasibility of adenoviral-mediated transfer of both marker genes *LacZ* and *Luciferase,* as well as the potentially functional gene of the human P_2 adrenergic receptor (P_2-AR) in a heterotopic heart transplant model using adult male Long Evans rats. Donor hearts were arrested with warm cardioplegia and after removal flushed via the aortic root with one milliliter of solution containing 10^{12} total viral particles of recombinant adenovirus encoding one of the three transgenes. Hearts were transplanted into the recipient's abdomen and harvested at five days for hearts injected with the marker genes and at various time points for hearts injected with the $Î²_2$-AR. lacZ-treatcd hearts were assessed by histochemical staining (X-gal). *Luciferase-treated* hearts were assayed for luciferase activity. $Î²_2$-AR-treated hearts underwent radioligand binding assays and immunohistochemistry using an antibody specific for the human P_2-AR. *LacZ* hearts (n = 6) revealed diffuse myocyte staining of both the right and left ventricles, as opposed to no staining within control hearts which received empty adenovirus. *Luciferase* hearts (n = 6) demonstrated a mean activity of 970, 000 ± 220, 000* arbitrary luciferase units as compared to controls which had a mean activity of 500 ± 200 arbitrary luciferase units (*p<0.05 vs. controls). Total P-AR densities (fmol/mg membrane protein) for hearts that received the $Î²_2$-AR transgene, at 3, 5, 7, 10, and 14 days after transfection were as follows; right ventricle - 488.5 ± 126.8, 519.4 ± 81.8*, 477.1 ± 51.8*, 183.0 ± 6.5*, 82.7 ± 19.1; left ventricle - 511.0 ± 167.6, 1206.4 ± 321.8*, 525.3 ± 188.7, 183.5 ± 18.6*, 75.9 ± 15.2 (n = 3 for each group; *p<0.05 as compared to control value of 75.6 ± 6.4). Immunohistochemical analysis with anti-$Î²_2$-AR antibodies revealed diffuse staining of varying intensity within myocardial sarcolemmal membranes. We conclude that global overexpression of two different adenoviral-mediated reporter genes and, a potentially functional gene, the human $Î²_2$-AR, is possible during cardiac transplantation. Furthermore, $Î²_2$-AR overexpression increases with time, peaking at five days, followed by a gradual decline returning to native levels at two weeks. Ultimately, gene transfer during cardiac transplantation may provide a unique opportunity for genetic manipulation of the donor organ, potentially enhancing the function of the heart.

*By invitation

F23. ESTROGEN INHIBITS THE DEVELOPMENT OF TRANSPLANT ARTERIOSCLEROSIS BY PREVENTING INDUCIBLE MHC CLASS II ANTIGEN IN THE EARLY PHASE FOLLOWING THE TRANSPLANTATION.

Satoshi Saito, M.D.*, Noboru Motomura, M.D., Ph.D.*, Hong Lou, M.D.* and Marie L. Foegh, M.D., D.Sc.*

Washington, DC

Sponsored by: Edward A. Lefrak, M.D., Annandale, Virginia

Background: The development of transplant arteriosclerosis (TA) is a major limiting factor for long time survival of cardiac transplants. We find chronic estradiol (E2) treatment inhibited TA. Very recently, we discovered that E2 inhibition of coronary TA in a chronic cardiac transplant model is associated with complete abolition of MHC class II expression. This model employs cyclosporin A (10 mg/kg/day) immunosuppression. We hypothesize that E2 independent of cyclosporin A inhibits inducible MHC class II antigen expression in professional and non-professional antigen presenting cells from the early phase following transplantation. The objective

of this study is to investigate in noncyclosporin requiring TA model that E2 treatment abolish MHC class II antigen expression in the allograft in the early phase following transplantation.

Methods: Orthotopic abdominal aorta allograft transplantation was performed using Brown-Norway rats as donors and Lewis rat as recipients . All recipients were treated with either 20µg/kg/day of estradiol 17 (J (n = 20) or placebo (n = 20) continuously s.c. from 2 days prior to transplantation until sacrifice using an osmotic minipump. The animals were sacrificed on postoperative days 1, 3, 7, and 14 and the grafts were harvested following perfusion fixation and then embedding in paraffin. Cross sections of the allografts were used for computerized morphometric analysis of medial area (M) and intimal thickening. Intimal thickening (I/I + M) was quantitated as the ratio of intimal area (I) over total vascular area (intima + media). Following immunohisto-staining, the expression of MHC class II antigen and macrophage was graded semiquantitatively on a scale from 0 to +3.

Results: Intimal thickening was measureable at day 14 and I/I + M in the allograft from E2 treated recipients was significantly lower than placebo treated recipients. (9.2 + 2.2 % vs. 2.3 + 3.6%, $p<0.01$).

MHC class II antigen expression

	Intima		Media		Adventita	
Day	E2	Placebo	E2	Placebo	E2	Placebo
1	0	0	0	0	0	0
3	0	0	0	0.07 ± 0.05	0	0.50 ± 0.25
7	0	0.11 ± 0.07	0	0.55 ± 0.16	0.06 ± 0.02	2.11 ± 0.34*
14	0	0.83 ± 0.4	0	0.29 ± 0.10	0.32 ± 0.10	2.06 ± 0.38*

Macrophage expression

	Intima		Media		Adventita	
Day	E2	Placebo	E2	Placebo	E2	Placebo
1	0	0	0	0	0	0
3	0	0	0	0.22 ± 0.08	0	0.61 ± 0.11
7		00.43 ± 0.13	0	0.17 ± 0.08	0	0.60 ± 0.12
14	0.07 ± 0.04	040.43 ± 0.17*	0	0.29 ± 0.10	0.21 ± 0.10	0.89 ± 0.24*

Summary: 1. Chronic estrogen treatment inhibits development of transplant arteriosclerosis.

2. Estrogen abolishes inducible MHC class II antigen expression on SMC in the media in the early phase following the transplantation.

3. Estrogen suppresses inducible macrophag MHC class II antigen expression in the allograft vessel wall.

Conclusion: Estrogen may inhibit the development of transplant arteriosclerosis by abolition of inducible MHC class II antigen in the early phase following the transplantation.

*By invitation

F24. AORTIC VALVE GRAFTS IN THE RAT MODEL: EVIDENCE FOR REJECTION.

Ahmad Moustapha, M.D.*, David B. Ross, M.D.*, Bindu Bittira, B.Sc.*, Dick Van Velzen, Ph.D.*, Vivian C. McAlister, M.B.*, Christopher L. Lannon, B.Sc.* and Timothy D. Lee, Ph.D.*

Halifax, Nova Scotia, Canada

Sponsored by: David A. Murphy, M.D., Halifax, Nova Scotia, Canada

Background: The role of immune mediated rejection in the failure of allograft heart valves in man is uncertain. Serial sampling of human allografts is not feasible and grafts are seldom removed shortly after implantation when any such damage may be occurring. The use of heart valve transplants between syngeneic and allogeneic strains of rats permits investigations into the role of immune mediated rejection of these grafts.

Aims: To describe the pathological changes in rats, over time, following transplantation of allogeneic versus syngeneic aortic valve grafts and to identify changes indicative of cell mediated rejection.

Methods: Transplantations were performed placing the donor ascending aorta, valve and rim of subvalvular myocardium in the abdominal aorta of the recipient. Recipients were Lewis (Le) rats; syngeneic donors were Lewis and allogeneic donors were Brown Norway (BN). At sacrifice, the grafts were removed, fixed and serially transacted at right angles. Samples of the aortic graft valve ring and a sample of the distal graft aorta, 2 mm above the valve, were routinely processed in paraffin for microscopical assessment using 4 micron sections, H/E and van Giesson/Elastica stains. Histopathological assessment was performed by two pathologists blinded to the original protocol and study design.

Results:**1) Early Post Transplant:** 9 allogeneic (BN to Le) were transplanted with 3 sacrificed at 2, 5 and 7 days with 3 Le transplanted as syngeneic controls. In both syn- and allogeneic cases a progressive, massive infiltration of neutrophils at the valve base and proximal aorta occurred increasing from day 2 to day 7. There was no difference seen between syngeneic and allogeneic animals. **2)4 Weeks Post Transplant:** 8 syngeneic (Le to Le) and 8 allogeneic (BN to Le) were performed. After initial blinded review, only two types of morphology were found. In one, virtually normal aortic grafts were seen, with minimal reactive changes to valves and minimal irregularity of the endothelium. In the other, severe chronic peri-aortic inflammation was associated with continuous cusp obliterating thrombosis, secondary valve degeneration and multi-focal, often completely full thickness, medial cell necrosis. All cases were attributed to either of the two types, reproducibility was complete. After unblinding, normal morphology was seen to be associated with the syngeneic transplants, chronic inflammation and destructive thrombosis was associated with allogeneic transplants. **3) 8 Weeks Post Transplant, Effects of Cryopreservation:** 12 Le to Le and 12 BN to Le were transplanted with half of the donors cryopreserved using standard tissue bank techniques. Syngeneic transplants showed intact valves with no signs of attachment to aortic wall, degeneration or calcification. Only one (1/12) showed any thrombus in a sinus; the elastin layers were preserved and there was no perivascular inflammation. The allogeneic grafts showed complete obliteration of the sinuses of Valsalva by organized thrombus with early calcification in some. Valve tissue remains were seen in the organised thrombus. Development of a neo-intima with medial cell necrosis was present. A moderate, mainly lymphocytic reactive infiltrate was present in the perivascular tissues (11/12) and occasionally in the confines of the aorta (3/12). The pathology did not differ between the cryopreserved and fresh grafts. Conclusions: The pathology of aortic valve transplantation in the rat model is dominated in the first week by a massive infiltration of neutrophils, probably induced by the necrotic muscle transplanted and is similar for both allogeneic and syngeneic grafts. Thereafter, syngeneic grafts remain intact while allogeneic grafts show progressive obliterative thrombosis of the valve leaflets and lymphocytic infiltration of

the aorta, unaffected by cryopreservation. These changes are characteristic of cell-mediated rejection. Investigations into therapies to modify this response appear justified.

*By invitation

F25. THE RELATIONSHIP BETWEEN CALCIUM AND MAGNESIUM IN PEDIATRIC MYOCARDIAL PROTECTION.

Bradley S. Allen, M.D.*, Michael Kronen, M.D.*, Kirk S. Boiling, M.D.*, Shaikh Ramon, M.S.*, Tingrong Wang, M.D.* and Harold Feinberg, Ph.D.*

Chicago, Illinois

Sponsored by: Renee S. Hartz, M.D., Chicago, Illinois

We have shown that adding magnesium to normocalcemic cardioplegic solutions offsets the detrimental effects of calcium in neonatal hearts by competing with calcium entry. However, it is not known whether magnesium offers any benefit when added to low calcium cardioplegic solutions. Fifteen 5-18 day old neonatal piglets underwent sixty minutes of ventilator hypoxia (FiO_2 8-10%) followed by reoxygenation using cardiopulmonary bypass (FiO_2 100%) for 5 minutes, and 20 minutes of normothermic ischemia by cross clamping the aorta. This produces a severe injury that combines ischemia with hypoxia and reoxygenation. The hearts were then protected for seventy minutes with hypocalcemic (Ca^{2+}0.2-0.4 mM/1) multidose blood cardioplegia. In five (Group 1) no magnesium was added to the blood cardioplegia, in 5 (Group 2) magnesium was added to the cardioplegia to produce a concentration of 5 meq/1, and in the last 5 (Group 3) magnesium was added at a concentration of 10 meq/1. Function was assessed using pressure volume loops and expressed as percentage of control. Coronary vascular resistance (CVR) was measured during each cardioplegic infusion. Despite the use of a hypocalcemic cardioplegia solution, in the absence of magnesium supplementation (Group 1) there was marked post bypass depression of systolic (Ees 38 + 1%) and global myocardial function (40 ± 1%), and a marked rise in diastolic stiffness (238 ± 3%). Conversely, even low dose (5 meq/l, Group 2) magnesium supplemented cardioplegia resulted in complete return of systolic (101% vs 38%)* and global myocardial function (102% vs 40%)*, preserved diastolic compliance (154% vs 238%)*, reduced myocardial edema (79.7% vs 80.6%), maintained ATP levels (15.8 vs 12.2 ug/gm dry wt)* and preserved CVR*, compared to a hypocalcemic cardioplegic solution without magnesium (Group 1). The use of a higher dose of magnesium (10 meq/1) did not result in any further improvement. In conclusion: 1) there is complete functional preservation, even in severely stressed neonatal hearts, when cardioplegia solutions are supplemented with magnesium, 2) this occurs despite even when a hypocalcemic cardioplegic solution is used, and 3) doses as low as 5 meq/1 of magnesium are effective. This study therefore strongly supports adding magnesium to all blood cardioplegia solutions regardless of calcium concentration. *$p<0.05$

*By invitation

F26. EFFECT OF THE COX-MAZE PROCEDURE ON THE SECRETION OF ATRIAL NATRIURETIC PEPTIDE.

Ki-Bong Kim, M.D.*, Chang-Ha Lee, M.D.*, Young-Joo Cha, M.D.* and Cheol-Ho Kim, M.D.*

Seoul, Korea

Sponsored by: James L. Cox, M.D., St. Louis, Missouri

The Cox-Maze procedure (CMP) has been confirmed to be effective in curing atrial fibrillation (AF). Some authors reported severe fluid retention after CMP and suggested decreased secretion of atrial natriuretic peptide (ANP) as a possible mechanism. This study was designed 1) to follow the serial changes in ANP after CMP as compared to after coronary artery bypass grafting (CABG), and 2) to elucidate any differences between ANP levels in patients with transient recurrence of AF after CMP and those without recurrence. Blood samples were drawn from the right atrium (RA) and left atrium (LA) in patients undergoing CMP (n = 19) and from the RA in patients undergoing CABG (n = 6) before and 1, 2, and 3 days after surgery. The plasma samples were prepared by refrigerated centrifugation and stored til radioimmunoassay. In the CMP group, ANP levels in the RA were 629 ± 366, 153 ± 112, 162 ± 112, and 183 ± 97, and in the LA were 276 ± 168, 152 ± 91, 162 ± 111, and 145 ± 80 (pg/ml, mean ± SD) before and 1, 2, and 3 days after surgery, showing a marked decrease in ANP levels after CMP ($p<0.01$). In the CABG group, ANP levels in the RA were 115 ±37, 124 ± 48, 154 ± 54, 156 ± 36 (pg/ml mean±SD) before and 1, 2, and 3 days after surgery, showing no change after surgery. There were no differences in ANP levels between patients with transient recurrence of AF (n = 6) and those without recurrence (n = 13) after CMP. There was no significant correlation between ANP levels and LA or RA pressure after CMP, which suggests that the secretion of ANP from atria was impaired. In summary, we observed a significant decrease in ANP levels after CMP and this might be one of the possible causes of fluid retention after CMP. The decreased ANP levels after CMP may result from the multiple atriotomy incisions of the CMP rather than from the conversion of AF to sinus rhythm.

*By invitation

F27. EXTENDING THE CONCEPT OF AUTOGRAFT FOR COMPLETE REPAIR OF TRANSPOSITION OF THE GREAT ARTERIES WITH VSD AND LEFT VENTRICULAR OUTFLOW TRACT OBSTRUCTION. A REPORT OF 10 CASES OF A MODIFIED PROCEDURE.

Dominique R. Metras, M.D., Bernard Kreitmann, M.D.*, Alberto Riberi, M.D.*, John Yao, M.D.*, Elie El Khoury, M.D.*, FranÃ§ois Wernert, M.D.* and Adrienne Pannetier, M.D.*

Marseille, France

BACKGROUND: Although in most cases of TGA with VSD and LVOTO, a REV (Lecompte) procedure is possible without interposition of a conduit between RV and PA, the anterior location of the PAs after the Lecompte maneuver may be a potential cause for RV outflow obstruction that remains reported in 5 to 10% of cases. We have used a tubular segment of aortic autograft to connect the pulmonary artery, left in orthotopic posterior position (without Lecompte maneuver) to the RV in 10 consecutive cases of TGA VSD LVOTO. Late results up to 3, 4 years, show no obstruction in the RV reconstruction, low RV pressure, and no calcifications of the autograft.

METHODS: Ten consecutive patients aged 9 months to 11 years (mean 32 months) have been corrected with a modified REV-Lecompte operation. Eight had a severe pulmonary stenosis, 2 had a pulmonary atresia, 4 had a restrictive VSD at the time of surgery, One had multiple VSDs. Seven had undergone one (5) or two (2) previous modified Blalock-Taussig shunts. All patients underwent a total correction with LV - Ao intra-ventricular connection (4 needed a VSD enlargement), connection between RV and PAs with a tubular segment of autograft aorta, without Lecompte maneuver (anterior location of the bifurcation of Pas) on the right (5) or the left (4) of the aorta. No valvular device was used for the RV outflow repair.

RESULTS: There was no early or late death. One patient with multiple VSDs needed an early (one month) reoperation for a residual muscular VSD. All patients are currently in NYHA class I, without medications, in sinus rhythm, at a mean follow-up of 2 years. There is no calcification on the chest X-ray, and at the most recent echocardiogram, RV pressures were low (25-40, mean 33 mmHg) and no significant gradient (over 10 mmHg) was found between RV and PA. Left and right ventricular functions were satisfactory.

CONCLUSION: This modification of the REV operation using a segment of autograft allows an excellent early and late result, with no danger of compression of anteriorly placed PAs, no significant RV outflow obstruction, normal aspect of the tubular autograft. In view of laboratory and clinical evidence, normal growth of the autograft can be anticipated. It allows an elective correction of TGA VSD LVOTO without previous BT shunt (3 cases) and correction at a young age (three patients less than one year).

*By invitation

F28. FIRST EXPERIENCE WITH A MODIFIED REPAIR TECHNIQUE FOR TRICUSPID INCOMPETENCE IN EBSTEIN'S MALFORMATION.

Roland Hetzer, M.D., Ph.D., Nicole Nagdymann, M.D.*, Peter Ewert, M.D.*, Vladimir Alexi-Meskhisvili, M.D, Ph.D.*, Yu-Guo Weng, M.D.* Felix Berger, M.D.* and Peter E. Lange, M.D, Ph.D.*

Berlin, Germany

Tricuspid incompetence (TI) in Ebstein's malformation is preferably treated by tricuspid repair (TR) rather than replacement (TVR) because of the characteristic complications of valve prosthesis and the higher incidence of heart block after TVR. Most repair techniques involve plication of the "atrialized" chamber which in some cases of small functional right ventricle may be difficult to achieve or, if attempted, may jeopardize the function of both ventricles.

In October 1988 a concept was instituted to restore tricuspid competence by a technique which leaves the atrialized chamber unplicated, reduces tricuspid orifice circumference at the level of the true annulus and uses the individually most mobile leaflet for closing mechanism. Following this concept among 19 highly symptomatic patients, ages 2 to 52 years (mean 21 years) the following procedures were performed: creation of a double orifice tricuspid valve in 1, closure of posterior part of tricuspid orifice in 10, closure of anterior part in 5 and bilateral annulus plication in 3 cases.

There was no early death. Late death occurred in one patient with recurrent sepsis, after operation in active endocarditis. Intraoperative echocardiography revealed residual TI of grade 0-1 in 11, grade I-II in 8 patients. TI progressed in 2 patients and required repeat TR after 19 and 24 months. At a mean follow-up of 27 months (6 to 96 months) clinical status remains improved to NYHA I

in 3, II in 13 and III in 2 patients. There were 2 cases each of transient and permanent heart block. The atrialized chamber has not gained size in any case so far.

It is concluded that the proposed repair principle may offer an acceptable alternative to other repair techniques for TI in Ebstein's malformation.
*By invitation

9:30 a.m. SIMULTANEOUS SCIENTIFIC SESSION D - ADULT CARDIAC SURGERY

South Sheraton Ballroom

Moderators: Robert L. Hardesty, M.D.

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

40. CORONARY ARTERY BYPASS WITHOUT ROUTINE PULMONARY ARTERY CATHETER USE - ASSESSMENT OF CRITERIA TO REDUCE COST AND PRESERVE QUALITY.

Robert D. Stewart, M.D.*, Christian T. Campos, . M.D.*, Triffin Psyhojos, M.D.*, Stephen J. Lahey, M.D.* and Sidney Levitsky, M.D.

Boston, Massachusetts

Discussant: Jack M. Matloff, M.D.

To reduce the cost and resource utilization of coronary artery bypass surgery (CABG) at our institution, criteria to limit pulmonary artery (PA) catheter use were developed. Patients undergoing isolated, non-emergent, primary CABG with a left ventricular ejection fraction ‰¥ 40%, a creatinine < 2.0 mg/dl, without documented pulmonary hypertension or severe chronic obstructive pulmonary disease, and without unstable angina within 24 hours of surgery requiring the institution of intravenous (IV) heparin or nitroglycerin (TNG) or an increase in the dose of IV TNG or the placement of an intraaortic balloon pump were eligible for CABG with only central venous pressure (CVP) monitoring. The impact of these criteria from their implementation on April 22, 1996 until July 31, 1996 in 77 patients (CVP group) were compared with results in 36 patients who met CVP criteria but had a PA catheter placed due to surgeon or anesthesiologist preference (PA group). These 113 patients represented 65% of the 175 isolated, primary CABG operations performed at our institution during this three-month period.

The CVP and PA patients were well matched in demographics (age, gender, body mass index), severity of disease (ejection fraction, coronary vessel disease extent, priority of operation, and pre-op heparin or TNG use), and the prevalence of co-morbid conditions known to influence CABG outcome including hypertension, peripheral vascular disease, previous MI, and diabetes. Mean graft number, internal thoracic artery use, total cardiopulmonary bypass time, and cross clamp time were identical. Significantly more PA patients left the OR with inotropic support (14% vs. 1%; p=0.01), however, no significant increases in in-hospital mortality, mediastinitis, stroke, re-operation for bleeding, need for re-intubation, renal insufficiency, or need to institute post-op inotropic support were seen with CVP use. One patient had a CVP changed to a PA catheter.

	CVP	PA	P	% change vs. PA
12-hr volume infused (liters)	2.6 ±1.3	3.7 ± 1.1	0.001	-28.8
Post-op day #1 weight gain (kgs)	4.8 ± 2.3	5.7 ± 2.8	0.08	-15.5

Intubation time (hrs)	6.7 ± 3.7	8.3 ± 3.6	0.05	-18.4
ICU length of stay (days)	1 .4 ± 0.8	2.7 ± 6.3	0.06	-50.4
Overall length of stay (days)	5 .7 ± 3 .4	6.8 ± 6.7	0.21	-17.1
Hospital charges (thousand dollars)	23.4 ± 6.3	27.4 ± 18.2	0.09	-14.7

(Data are expressed as the mean ± S.D. P values are calculated by two-tailed Student's t-tests.)

A significant reduction in the volume infused in the first 12 hours following operation was observed in the CVP group. Definite trends toward reductions in all other resource outcome indicators were seen in the CVP group as well. These clinically important trends approached statistical significance (Type II error).

We conclude that these criteria for CVP catheter use, applicable to the majority of patients undergoing isolated primary CABG, resulted in clinically significant reductions in all measures of CABG resource utilization with no increase in morbidity or in-hospital mortality.

*By invitation

41. A "PRIMELESS PUMP" FOR CARDIOPULMONARY BYPASS ENHANCES BLOOD CONSERVATION BY REDUCING THE NEED FOR PERIOPERATIVE BLOOD TRANSFUSIONS IN CORONARY BYPASS OPERATIONS.

John A. Rousou, M.D., Richard M. Engelman, M.D., Joseph E. Flack, III, M.D.*, David W. Deaton, M.D.*, Jane L. Garb, M.S.* and Susannah G. Owen, B.A.*

Springfield, Massachusetts

Discussant: Kenneth M. Taylor, M.D.

Severe hemodilution during cardiopulmonary bypass (CPB) often leads to significant drop in hematocrit (Hct) and coagulation factor (CF) levels requiring blood product transfusions (Tx). A method of removing pump prime prior to CPB, initiated and clinically used at our institution, was noted to limit hemodilution and reduce the need for perioperative Tx. A prospective evaluation of two consecutive series of patients, 52 with prime (control) and 95 without prime (primeless) undergoing coronary bypass operation (CABG) was undertaken to objectively study the method's effectiveness in reducing Tx. Baseline characteristics between the **control** and the **primeless** groups such as body surface area (BSA), pre-CPB Hct, number of redos, use of hemocon-centrator and antifibrinolytics, fluid balance in OR, operative and perfusion technique and transfusion criteria were the same in both series and strictly adhered to in all patients as per protocol. Patients within each group were consecutive and non-selected, regardless of their BSA, Hct or any other factors. Group differences in Hct during operation were analyzed using repeated measures analysis of covariance. The proportion of patients requiring transfusions of red blood cells (RBCs) and/or CF were compared using multiple logistic regression controlling for other contributing factors. The drop in Hct during operation (over time) was significantly less for the "primeless" group ($p<<0.0001$) as shown in table below. Also shown are group differences in RBCs and CF given. There was no mortality in either group and no significant differences in complications. Although not statistically significant in a smaller subgroup of this population, there was a trend for more pronounced effect of "primeless" pump on intraoperative Hct for patients with BSA < 2.

	"Primeless"	Control	p value
% Hct drop from pre-CPB value	19.1 ± 0.8	27.8 ± 1.4	

Post CPB Hct (Includes Tx)	26.5 ± 0.4	25.0 ± 0.6	<0.0001 for change in Hct over time
RBC Tx in OR (cc/pt)	29.9 ± 15. 6	142.5 ± 50.2	p=0.034
Tx RBCs in OR (% pts)	4.2	21.1	p<0.005
Clotting Factors Given Postop (% pts)	3	12	p=0.06

RBC: red blood cells; OR: operating room

Conclusions: The "primeless pump" leads to: 1) Significantly higher intraoperative Hct; 2) Significantly fewer patients requiring RBCs during CABG and (possibly) CF transfusion factors postop; 3) The technique is simple and applicable to all cardiac operations leading to significant cost and risk reductions.

*By invitation

42. WHAT FACTORS TRULY AFFECT WAITING TIME TO TRANSPLANTATION? A MULTIVARIABLE ANALYSIS OF THE ISHLT/UNOS THORACIC REGISTRY.

Jonathan M. Chen, M.D.*, Keith D. Aaronson, M.D.*, Alan D. Weinberg, M.S.*, Berkeley M. Keck, R.N., M.P.H..*, Leah E. Bennett, Ph.D.*, Jeffrey D. Hosenpud, M.D.* and Robert E. Michler, M.D.

New York, New York, Richmond, Virginia and Milwaukee, Wisconsin

Discussant: Margaret D. Allen, M.D.

Because of the present donor organ crisis, the equity of organ distribution continues to be scrutinized. Although patients are currently listed and subsequently matched for cardiac transplantation (CT) according to clinical urgency, blood type, and weight, other variables may have an important impact on overall waiting time. We applied the Cox proportional hazards model to all 7791 CT candidates listed with UNOS from 4/94 to 4/96 in an effort to assess the simultaneous effect of multiple variables on the waiting times of CT candidates. The mean waiting time to CT was 151.2 days (median time 175 days), the mean age was 45.6 years, and the mean time spent waiting as a status I candidate was 20.5 days. At the termination of the study period, 4058 (52.1%) patients had undergone transplantation, 1954 (25.1%) were still waiting, 1216 (15.6%) had died and 563 (7.2%) were removed from the list for other reasons. Variables that were not significantly associated with waiting time included: race, education level, arrhythmias, AICD and amiodarone use. Results of a multivariable analysis are depicted below:

Predictors of Shorter Waiting Times	p-value	Likelihood of CT
Proportion of Days as Status I	p=0.0001	11.7
Weight at Listing	p=0.0001	1.2/10 lb. †“ in weight
Blood Type AB*	p=0.0001	3.9
Blood Type A	p=0.0001	2.2
Blood Type B	p=0.0001	1.7
No Need for a Prospective Cross-Match	p=0.0001	2.0
Non-United States Citizenship	p=0.0006	1.4

Age ‰¤ 2 years ‡	p=0.0001	0.3
Age > 2, and ‰¤ 65 years ‡	p=0.0362	0.9

* Blood types compared with Blood Type O; ‡ Age compared with patients > 65 years

Although preoperative predictors of patients at highest risk for death on the waiting list remain elusive, we describe herein factors that have an impact on the waiting time to transplantation. It is our hope that these data may help to identify certain patients at high risk for longer waiting times for whom other surgical alternatives to transplantation may be indicated.

*By invitation

§43. VIDEO-ASSISTED MINIMALLY INVASIVE MITRAL VALVE SURGERY.

W. Randolph Chitwood, Jr. M.D., Christopher L. Wixon, M.D.*, Joseph R. Elbeery, M.D.* and Jon F. Moran, M.D.*

Greenville, North Carolina

Discussant: Aubrey C. Galloway, Jr., M.D.

Video-assisted minimally invasive mitral valve surgery (VMIMS) may have advantages over conventional operative approaches. Since May of 1996, we have used a 2.5 inch right thoracic incision and video-assisted techniques in repairing (Rep N = 8) or replacing (Rpl N = 5) mitral valves. Ejection fractions ranged between .35 and .60 (.54 ± 2.2 SEM) with ages being 18 to 77 years (55.5 ± 5.2 SEM). Other clinical characteristics were similar. Cardiac arrest was induced either by retrograde (N = 5) or antegrade (N = 6) blood cardioplegia using a new transthoracic cross-clamp to occlude the ascending aorta. In two patients cold ventricular fibrillation was used. Systemic perfusion was maintained at 28°C either by central or peripheral arterio/venous cannulation. Newly designed instruments enabled video-assisted suture placement and knot tying. Superb illumination and visualization were provided by a 10 mm port-access 30° thoracoscopic camera. Mean cardiopulmonary perfusion times were 197 ± 7.8 SEM minutes and arrest times averaged 138 ± 12.8 minutes. Postoperative transesophageal echocardio-graphic studies showed excellent valve function with minimal insufficiency, and all patients had little postoperative pain. There were no operative deaths. One patient developed lower extremity deep venous thrombosis as the only major complication. Other patients were discharged between postoperative day three and five (4.6 ± .4 days SEM). The mean hospital stay for the previous 111 conventional mitral operations (N = 67 Rep, N = 44 Rpl) was 9.2 ± 0.9 days SEM. In the VMIMS patients atrial fibrillation (8% vs 28%), reoperation for bleeding (0% vs 5%), ICU length of stay (1.1 vs 2.0 days), and hospital charges (†“ > 30%) also were significantly less than the conventional cohort. Thus, despite long operative times, these early results are encouraging and suggest that video-assisted minimally invasive mitral valve operations are safe and may benefit patients by minimizing postoperative pain, allowing earlier discharge, and decreasing hospital expenses.

10:50 a.m. INTERMISSION

§Authors have a relationship with Scanlan International, Inc.

*By invitation

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

ADULT CARDIAC SURGERY

South Sheraton Ballroom

Moderators: Robert L. Hardesty, M.D.

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

44. MITRAL VALVE REPAIR WITH AND WITHOUT CHORDAL REPLACEMENT WITH EXPANDED POLYTETRAFLUOROETHYLENE SUTURES -A 10-YEAR EXPERIENCE.

Tirone E. David, M.D., Susan Armstrong, M.Sc.*, Zhao Sun, Ph.D.* and Ahmed Omran, M.D.*

Toronto, Ontario, Canada

Discussant: Lawrence H. Cohn, M.D.

Replacement of chordae tendineae with expanded polytetrafluoroethylene sutures (ePTFE) increases the probability of mitral valve (MV) repair in pts with mitral regurgitation (MR) due to myxomatous disease of the MV. This study compares the results of MV repair with and without chordal replacement with ePTFE. From 1985 to 1995, 324 consecutive pts underwent MV repair: 165 with ePTFE chordae and 159 without. There were no statistical differences between these two groups as far as age (58 ± 14), functional class, left ventricular function and the incidence of coronary artery disease (16%). The differences were in the mechanism of mitral regurgitation and in the degree of myxomatous changes in the leaflets.

	With ePTFE	Without ePTFE
Prolapse of the anterior leaflet	49(30%)	9 (6%)
Prolapse of the posterior leaflet	29(17%)	139(87%)
Prolapse of both leaflets	87(53%)	11 (7%)
Advanced myxomatous changes	43 (26%)	14 (9%)

There were 2 operative deaths, both in the ePTFE group, neither one related to the MV repair. Operative morbidity was similar in both groups. Pts have been followed for a mean of 35 ± 30 months. No pt was lost to follow-up. Every pt had an annual Doppler echocardiographic study. Eight pts developed severe recurrent MR and 2 other developed hemolysis and were reoperated on; 6 of them were from the ePTFE group. All reoperations occurred in the first three years of follow-up. A logistic regression analysis identified that only the combination of prolapse of both leaflets and advanced myxomatous changes was predictive of reoperation in all pts. The latest echocardiographic study showed moderate MR in 9 pts (5 pts ePTFE group), and mild or none in 284. The actuarial freedom from morbid events showed no statistical differences between the two groups.

	With ePTFE	Without ePTFE
Freedom from death at 10 years	75% ± 7%	75% ± 6%
Freedom from stroke at 10 years	94% ± 2%	95% ± 2%
Freedom from reoperation at 10 years	94% ± 3%	97% ± 2%

This experience indicates that replacement of chordae tendineae with ePTFE sutures during MV repair for myxomatous disease of the MV provides as good long-term results as MV repair without ePTFE.

*By invitation

45. TEN-YEAR RESULTS OF CORONARY ARTERY BYPASS GRAFTING IN PATIENTS WITH ADVANCED LEFT VENTRICULAR DYSFUNCTION.

Gregory D. Trachiotis, M.D.*, William S. Weintraub, M.D.*, Thomas S. Johnston, M.D.*, Ellis L. Jones, M.D., Robert A. Guyton, M.D. and Joseph M. Graver, M.D.

Atlanta, Georgia

Discussant: Lynda L. Mickleborough, M.D.

The combination of severe coronary artery disease (CAD) and advanced left ventricular dysfunction carries a poor outlook with medical therapy. Coronary artery bypass grafting (CABG) in this group has often been regarded as high risk. We reviewed patients with CAD and sequentially decreased ejection fraction (EF) compared to those with an EF>50% with 10 year follow-up to determine if CABG can provide long-term symptomatic improvement and survival in patients with severe left ventricular dysfunction (EF<25%). Between 1971-1994, 156 (1.3%) patients with an EF<25% [Group I], 588 (5%) patients with an EF=25-34% [Group II], 2438 (20.6%) patients with an EF=35-49% [Group III], and 8648 (73.1%) patients with an EF>50% [Group IV] underwent CABG. The EF was estimated from the contrast left ventriculogram (either uniplaner or biplaner). For all groups mean age was 60 ± 10 years. Groups l-III compared to Group IV had a higher percentage of patients with men (<.0004); diabetes mellitus (p<.0001); class III-IV angina (p<.0001); heart failure (p<.0001); prior MI (p<.0001); 3 vessel disease (p<.0001); and left main disease (p<.0001). Group I had the highest percentage of patients with men (88%); heart failure (34%); and left main disease (24%). The mean EF's were 19 ±4 in Group I, 29 ± 3 in Group II, 42 ± 4in Group III, and vs 64 ± 9 in Group IV. The results were as follows (<.05 significant by ANOVA for Groups I, II, or III vs IV):

Groups (EF%)	# Grafts	Completely Revascularized	IMA Graft	Q wave MI	Death in Hospital	Length of Stay	Angina during Follow-up
I (<25)	3.3 ± 1.1	107 (69%)	54 (35%)	0 (0%)	6 (3.8%)	9.2 ±5. 8	31 (40%)
II (25-34)	3.5 ± 1.1	445 (76%)	244(41%)	12 (2%)	20 (3.4%)	10.5 ± 10.9	120(36%)
III (34-49)	3.4 ± 1.1	1952 (80%)	1149 (47%)	48 (2%)	72 (3%)	9.1 ±7.8	2277 (33%)
IV (>50)	3.3 ± 1 2	7400 (86%)	3616(42%)	231(27%)	134(16%)	8.4 ±6.4	2277 (33%)
P Value	<.0001	<.0001	<.0001	<.04	<.0001	<.0001	<.06

Despite having a higher percentage of risk factors, poorer functional status, and more complex coronary anatomy, patients with compromised left ventricular function have comparable in-hospital outcome to patients with a normal EF. While long-term there is much higher mortality in patients with comprised LV function, over 60% of patients with EF <25% were alive at 5 years. In addition, patients after CABG with EF <25% have excellent long-term control of angina despite the lower use of IMA grafts and less complete revascularization. These results suggest that in selected patients with ischemic cardiomyopathy, CABG may preserve remaining viable myocardium, provide relief of symptoms and offer survival >60% at >5 years.

*By invitation

46. MYOCARDIAL PERFUSION AND FUNCTION FOLLOWING ADENOVIRUS-MEDIATED TRANSFER OF THE $VEGF_{121}$, cDNA TO ISCHEMIC PORCINE MYOCARDIUM.

Charles A. Mack, M.D.*, Shailen R. Patel, M.D.*, Eric A. Schwarz, M.D.*, Pat Zanzonico, Ph.D.*, Rebecca T. Hahn, M.D.*, Arzu Ilercil, M.D.*, Richard B. Devereux, M.D.*, Stanley J. Goldsmith, M.D.*, Timothy F. Christian, M.D.*, Timothy A. Sanborn, M.D.*, Imre Kovesdi, Ph.D.*, O. Wayne Isom, M.D., Ronald G. Crystal, M.D.* and Todd K. Rosengart, M.D.*

New York, New York and Rockville, Maryland

Discussant: Andrew S. Wechsler, M.D.

Background: A replication-deficient adenovirus vector expressing the cDNA for the angiogenic protein vascular endothelial growth factor$_{121}$ (AdVEGF$_{121}$) induces prolonged VEGF$_{121}$ expression *in vivo.* We therefore hypothesized that direct myocardial injection of AdVEGF$_{121}$ would induce collateral vessel formation and enhance myocardial perfusion and function in ischemic myocardial territories.

Methods: Yorkshire swine (28-30 kg) underwent left thoracotomy and ameroid constrictor placement on the left circumflex coronary artery. Three weeks later, AdVEGF$_{121}$ or the control vector, AdNull (each 10^8 pfu in 100 □l) was injected in the myocardium at 10 sites in the circumflex distribution. Ischemia was assessed by echocardiography and ^{99m}Tc-sestamibi imaging during rest and rapid atrial pacing (‰¥ 200 beats/min) at the time of gene transfer and after 4 weeks, and collateral vessel formation was assessed by *ex vivo* an angiography. In a separate group of animals, VEGF$_{121}$ expression was quantified in the injected myocardium and serum by ELISA following gene transfer.

Results: AdVEGF$_{121}$-treated animals demonstrated significant VEGF$_{121}$ expression (9 ng/mg protein) in the myocardium 3 days following vector administration. VEGF$_{121}$ was undetectable in the serum of AdVEGF$_{121}$ -treated animals. An improvement in ventricular perfusion and function in the circumflex territory was suggested by rest versus stress ^{99m}Tc-sestamibi scans and echocardiographic assessment of segmental wall thickening, respectively, in AdVEGF$_{121}$ versus AdNull-treated animals. Angiography in the AdVEGF$_{121}$-treated animals demonstrated a collateral network with apparent reconstitution of the distal circumflex artery.

Conclusions: An adenovirus vector can be used to transfer the VEGF$_{121}$ cDNA to the myocardium. This strategy may be useful in inducing therapeutic angiogenesis and improving perfusion and function in the ischemic myocardium.

12:10 p.m. ADJOURN

*By invitation

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

North Sheraton Ballroom

Moderators: Mark K. Ferguson, M.D.

Andre C.H. Duranceau, M.D.

47. ANGIOGENESIS AS A PREDICTOR OF SURVIVAL FOLLOWING SURGICAL RESECTION FOR STAGE I NON-SMALL CELL LUNG CANCER.

Ignacio G. Duarte, M.D.*, Bradley L. Bufkin, M.D.*, Marion F. Pennington, M.D.*, Anthony A. Gal, M.D.*, Cynthia Cohen, M.D.*, Kamal A. Mansour, M.D. and Joseph I. Miller, M.D.

Atlanta, Georgia

Discussant: Valerie W. Rusch, M.D.

A subset of surgically resected Stage I non-small-cell lung cancer (NSCLC) patients will later develop metastatic disease. A histologic marker of metastatic potential and diminished survival for Stage I NSCLC may help identify this subset of patients. This study evaluates the degree of angiogenic activity as a predictor of cancer-related mortality in patients having undergone surgical resection for Stage I NSCLC. Demographic, surgical, and histopathologic data were reviewed for 107 patients with Stage I NSCLC from 1985-1990. Visual quantitation of Factor VHI-related antigen (FVIII) and CDS 1 immunostained microvessels, 0.74 mm^2 area (200x magnification), in 5μ sections from paraffin blocks defined tumor angiogenesis. Mean microvessel count was 20.7 ± 11.2 for FVIII, and 29.6 ± 18.1 for CD31. Mean follow-up was 5.2 ±3.0 years (8 days - 11.06 years) and 95.3% complete. Lung cancer-related mortality was 23% at 5 years. Kaplan-Meier survival curves revealed FVIII count >20 (p=0.028) and the presence of blood vessel invasion (p=0.034) to be significant predictors of disease-related mortality. Logistic regression analysis identified FVIII quantitation > 20 as the single most significant independent correlate of lung cancer mortality (p=0.021, hazard ratio 2.64). CD31 quantitation did not predict survival in univariate and multivariate analyses and did not correlate with FVIII quantitation (Spearman's rank correlation, r = 0.20). This analysis displays a significant association between tumor neovascularization and cancer-related mortality in patients with Stage I NSCLC. FVIII microvessel quantitation, as an indicator of tumor angiogenesis and metastatic potential, may help identify a subset of patients with Stage I NSCLC who may benefit from adjuvant therapy following surgical resection.

*By invitation

48. THYMIC CARCINOMA: CURRENT STAGING DOES NOT PREDICT PROGNOSIS.

David B. Blumberg, M.D.*, Juan Rosai, M.D.*, Manjit S. Bains, M.D., Robert J. Downey, M.D., Robert J. Ginsberg, M.D., Nael Martini, M.D., Patricia M. McCormack, M.D, Valerie W. Rusch, M.D. and Michael E. Burt, M.D.

New York, New York

Discussant: Paul A. Kirschner, M.D.

Thymic carcinomas are currently staged by Masaoka classification, a staging system for thymomas. We retrospectively evaluated surgical patients with thymic carcinoma to determine factors predicting survival.

Methods: Our computerized tumor registry yielded 118 patients with thymoma. Review of pathologic material revealed 43 cases of thymic carcinoma. Medical charts were reviewed. Follow-up was performed by physician charts and telephone. Analysis by Kaplan-Meier method and Cox proportional hazards.

Results: Between 1949 and 1993, 43 patients underwent surgery for thymic carcinoma. Overall survival was 65% at 5 years with a median survival of 6.7 years. Survival was not dependent on Masaoka stage (p=0.3). There were 3 stage I patients alive at 3.6, 4.1 and 8.2 years. Five year survivals were 58% for stage II (n = 15), 55% for stage III (n = 20) and 100% for stage IV (n = 5) patients. Five year survivals of patients with complete resection (n = 29) and patients with partial resection (n=14) were 68% and 62%, respectively, (p=0.2). Survival of completely resected patients (n = 29) was not dependent on age (p=0.1), sex (p=0.7), tumor size (p=0.7), or Masaoka stage (p=0.4). Six patients had tumors invading the innominate vessels, 4 which were low grade (well/moderately differentiated) and one indeterminate. Tumor invasion of the innominate vessels (n = 6) was associated with a worse survival (p=0.01) with only 40% alive at 2 years, compared to 75% alive at 6 years with no invasion of the innominate vessels (n = 23). Of patients with no invasion of the innominate vessels, there were 19 low grade tumors and 4 high grade tumors (poorly differentiated). Survival of patients with low grade tumors was superior (p=0.03) with 95% alive at 6 yrs. as compared to a 50% 4 yr. survival for high grade tumors. By Cox proportional hazards model, survival was predicted only by grade (p=0.07) and innominate vessel invasion (p=0.05).

Conclusions: 1. Masaoka staging does not predict prognosis of patients with thymic carcinoma. **2.** Patients with low grade tumors without invasion of the innominate vessels have **early stage disease** and long term survival may be achieved with surgical resection. **3.**Despite complete resection, patients with high grade tumors and tumors invading the innominate vessels have a poor survival and these patients should be considered as having**advanced disease. 4.** These results have important implications for design of future adjuvant trials of patients with thymic carcinoma.
*By invitation

49. SAFETY AND EFFICACY OF BRONCHO VASCULAR RECONSTRUCTION AFTER INDUCTION CHEMOTHERAPY FOR LUNG CANCER.

Erino A. Rendina, M.D.*, Federico Venuta, M.D.*, Tiziano De Giacomo, M.D.*, Isac Flaishman, M.D.* and Costante Ricci, M.D.*

Rome, Italy

Sponsored by: Valerie W. Rusch, M.D., New York, New York

Discussant: Jean DesLauriers, M.D.

Desmoplastic reactions secondary to induction chemotherapy and/or residual tumor can make lung resection extremely difficult. In these patients, increased postoperative complications and mortality are reported, owing also to the high incidence of pneumonectomy. Between 1990 and July 1996 we have operated on 68 patients who had received 3 cycles of cisplatin-based induction chemotherapy. In 27 of these we have performed a lobectomy (# 25) or bilobectomy (# 2) associated with reconstruction of the bronchus and/or the Pulmonary Artery (PA). In only 5 additional patients pneumonectomy had to be carried out. Before chemotherapy 14 patients were at stage HIA and 13 at stage IIIB. At thoracotomy, 1 patient had no evidence of tumor, 6 were at stage I, 13 at stage II, 6 at stage IIIA, and 1 at stage IIIB. Fourteen patients had epidermoid carcinoma and 11 had adenocarcinoma. The type of reconstruction is tabulated below.

	#
- Bronchial sleeve resection	16
- PA reconstruction by pericardial patch	2

- PA reconstruction by pericardial conduit	1
- Bronchial sleeve + PA pericardial patch	7
- Bronchial sleeve + PA sleeve	1
	27

In 26 patients resection was radical with histologically negative margins. Bronchial anastomoses were wrapped by the intercostal pedicle flap. No bronchial complications nor mortality occurred. One patient had empyema and 2 had wound infection. Mean chest tube stay was 6 days (3 to 15 days). Twelve patients had additional adjuvant therapy with no problems. After a postoperative follow-up of 5 to 73 months (mean 25 months), 14 patients are alive disease-free, 1 is alive with disease and 12 died. No local recurrence occurred. One and four-year survival is 69% and 38%. Although technically demanding, lobectomy associated with bronchovascular reconstruction is feasible with good immediate and long term results after induction chemotherapy.

*By invitation

50. PREOPERATIVE TUMOR VOLUME PREDICTS OUTCOME IN MALIGNANT PLEURAL MESOTHELIOMA.

Harvey I. Pass, M.D., Karen C. Kranda, R.N.*, Seth M. Steinberg, Ph.D.*, Barbara K. Temeck, M.D.* and Irwin R. Feuerstein, M.D.*

Bethesda, Maryland

Discussant: Larry R. Kaiser, M.D.

The staging systems for malignant pleural mesothelioma (MPM) rely upon postoperative pathologic findings for prognostic determination. Since MPM surgical cytoreduction remains controversial, it would be desirable to predict outcomes from quantitative preoperative data. We prospectively analyzed the impact of preoperative and postresection solid tumor volumes on prognostic variables in 47 of 48 consecutively resected MPM patients. **Methods:** From 7/93 to 6/96, 48 MPM patients had cytoreductive debulking to 5 mm or less residual tumor via extrapleural pneumonectomy (EEP-25) or pleurectomy/ decortication (P/D-23). Three dimensional CT reconstructions of pre- and postresection solid tumor using the Voxell Q™ were prospectively performed. All patients received the same postoperative adjuvant therapy and were staged postoperatively according to the new International Mesothelioma Interest Group (IMIG) staging. Patients were followed by chest and abdomen CT scans every 3 months until death. Prognostic factors were examined by Cox proportional hazards model. **Results:** With a median potential follow-up of 23.1 months, median survival for all patients is 14.4 months (EPP-11 mos, P/D-22 mos, $p_2 = 0.066$). Median survival for preop volume <100 cc was 22 months vs 11 months if >100cc, $p_2 = 0.027$. Median survival for postop volume <9 cc was 25 months vs 9 months if >9 cc, $p_2 = 0.0002$. Thirty-two of 47 (68%) had positive Nl or N2 nodes. Tumor volumes associated with negative node patients were significantly smaller (51 cc) than those with positive nodes (166cc, $p_2 = 0.0099$). Progressively higher stage was associated with higher median preoperative volume: 1-4 cc, II-94 cc, III-143 cc, IV-505 cc, $p_2 = 0.0070$ for I vs II vs III vs IV. Patients with preoperative sizes >52 cc had shorter progression-free intervals (8 mos) than those ‰¤51 cc (11 mos), $p_2 = 0.021$. By the Cox model, male sex, preoperative platelet count >314K, preoperative volume >100cc and postresection volume >9cc were associated with decreased survival. **Conclusions:** Preresection tumor volume is representative of T status in MPM, and CT volumetrics can predict overall and progression-free survival, as well as postoperative IMIG stage.

Large volumes are associated with nodal spread, and postresection residual tumor burden may predict outcome. Future trials should develop a uniform, simple method to quantify pretreatment MPM volume, and to verify its prognostic and therapeutic implications.

10:50 a.m. INTERMISSION

*By invitation

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

North Sheraton Ballroom

Moderators: Mark K. Ferguson, M.D.

Andre C.H. Duranceau, M.D.

51. EFFECT OF VOLUME REDUCTION ON LUNG TRANSPLANT TIMING AND SELECTION FOR COPD.

Joseph E. Bavaria, M.D.*, Alberto Pochettino, M.D.*, Robert Kotloff, M.D.*, Bruce R. Rosengard, M.D.*, Peter M. Wahl, B.A.*, Harold Palevsky, M.D.* and Larry R. Kaiser, M.D.

Philadelphia, Pennsylvania

Discussant: Joel D. Cooper, M.D.

Introduction: End-stage COPD has traditionally been treated with lung transplantation (LTX). For two years, our LTX program has placed patients with appropriate criteria for LTX and Volume Reduction (LVR) into a prospective management algorithm. These patients are offered the LVR option as a means to "bridge" or extend the eventual time to LTX. These data examine the results of this pilot program.

Methods: From 7/7/94 to 10/25/96, 33 patients were evaluated for LTX who also had physiological criteria for LVR (FEV_1 ‰¤25%; RV >200%; significant V/Q heterogeneity). These patients were divided into two groups: 26 patients (Group I) underwent LVR as a "bridge" and were simultaneously listed for LTX. Seven patients (Group II), for various reasons, were offered LVR alone and not listed. All patients completed 6 weeks of pulmonary rehab, and then had baseline pulmonary function (PFTs) and Six Minute Walk (6MW) tests. LVR was performed via video thoracic in 81.8% or sternotomy in 18.2% of the patients. Patients were followed postop with repeat PFTs and 6MW at 3 month intervals.

Results: Nineteen of 26 pts (73.1%) in Group I had satisfactory clinical improvement after LVR. These 19 patients (Group I A) were subsequently delisted (Status 7). The remaining 7 patients in Group I (26.9%) had unsatisfactory results (Group IB) and one died perioperatively. Three of 6 survivors were subsequently transplanted with good outcomes. The other 3 patients are presently awaiting organs.

	Pre-op FEV_1 (1)	Post-op FEV_1 (1)	Pre-op RV(1)	Post-op RV(1)	Pre-op 6MW (ft)	Post-op 6MW (ft)
Group IA	0.67 ± 0.18	0.86 ± 0.28	4.64 ± 0.83	3. 82 ± 1.38	1088 ± 340	1376 ± 209

Group IB	0.78 + 0.15	1.00 ± 0.40	4.54 ± 1.42	4.43 ± 0.57	909 ± 253	1208 ± 167
Group II	0.67 ± 0.27	0.85 ± 0.26	4.78 ± 1.27	4.29 ± 1.19	874 ± 365	1227 ± 225

Interestingly, 2 of the 33 patients had A-l antitrypsin deficiency both of which had poor LVR outcome.

Conclusions: LVR in these low FEV_1 patients is safe. LVR has substantially impacted the practice, timing, and selection of patients for LTX. Our wait list presently has a reduced percentage of patients with a COPD diagnosis compared to 2 years ago. Seventy-three percent of otherwise suitable LTX candidates achieved good LVR results, especially reduction in RV and were deactivated from the list. The majority of patients entering our prospective management algorithm have either significantly delayed or completely avoided LTX after LVR. Our experience suggests that LVR may be limited as a "bridge" in alpha-1 antitrypsin patients.

*By invitation

52. PLEURAL TENTING DURING UPPER LOBECTOMY DECREASES CHEST TUBE TIME AND TOTAL HOSPITALIZATION DAYS.

Lary A. Robinson, M.D. and Dianne Preksto, PA-C*

Tampa, Florida

Discussant: Joseph I. Miller, Jr., M.D.

Purpose: A prolonged air leak following an upper lobectomy is one of the major determinants of postoperative morbidity and hospital stay. The use of stapling devices during pulmonary resections has greatly decreased air leaks, but the problem of sealing small persistent leaks to allow early chest tube removal is still present. Creation of a generous pleural tent following upper lobectomy was employed to investigate whether bringing the parietal pleura down to the lung to obliterate the usual post-op apical space would help seal the air leak and shorten chest tube time.

Methods: From August, 1994 to September, 1996, the records of 43 consecutive patients undergoing an upper lobectomy for a malignancy were reviewed. Twenty-three patients had creation of a pleural tent and 20 patients (undergoing surgery in the first year of the study period) did not. Mean patient age: tented 65.6 ± 1.7 years; non-tented 62.8 ± 3.0 years. Demographic and operative profiles of both groups were not significantly different. Patients excluded from the study were those undergoing concomitant chest wall resection (7), patients requiring post-operative mechanical ventilation (1), and those developing the alcohol withdrawal syndrome (2). All resections were performed by the same surgeon through a muscle-sparing thoracotomy and included a mediastinal lymphadenectomy. Chest tubes were removed when there was no air leak for 48 hours and when the total chest tube drainage was less than 75 ml per 8 hours.

Results: The tented patients had significantly shorter chest tube times and total hospitalizations compared to the non-tented patients, as shown below:

Indicator	Tented (n=23)	Non-tented (n=20)	*p* value
Mean days air teak	1.8 ± 03	3.9 ± 1.2	0.083
Mean days chest tube duration	4.1 ± 0.2	6.6 ± 1.0	0.012
Mean total chest tube drainage (ml)	1636.4 ± 111.4	243 1.4 ± 339.4	0026

Mean hospital stay post-op (days)	6.6 ± 0.41	86 ± 1.0	0.050

There were no deaths. Morbidity was minimal: purulent bronchitis (17% tented, 5% non-tented, p=0.25); wound infections 0%; empyema 0%; reoperation for bleeding 0%; cardiovascular events 0%; venous thrombosis 0%; and transfusion 0%. Time required to create the pleural tent averaged 4 minutes.

Conclusions: 1. Creation of a pleural tent at the time of upper lobectomy significantly reduces the time postoperatively that chest tubes remained in place, resulting in shorter hospital stays. 2. There was no morbidity or mortality associated with this simple, quick procedure. 3. Surgeons should consider routine creation of a pleural tent at the time of upper lobectomy.

*By invitation

53. AGGRESSIVE SURGICAL MANAGEMENT IN LOCALIZED PULMONARY MYCOTIC AND NON-MYCOTIC INFECTIONS FOR NEUTROPENIC PATIENTS WITH ACUTE LEUKEMIA: REPORT OF 18 CASES.

Olivier Baron, M.D.*, Betty Guillaume, M.D.*, Philippe Despins, M.D.*, Patrick Germaud, M.D.*, Philippe Moreau, M.D.*, Anne-Yvonne De Lajartre, M.D.* and Jean Luc Michaud, M.D.*

Nantes, France

Sponsored by: Williard A. Fry, M.D., Evanston, Illinois

Discussant: Marvin Pomerantz, M.D.

Patients treated by chemotherapy or bone marrow transplants for hematologic malignancies are at risk for a variety of infectious complications. During a 8-year period (1988-1996), 18 patients (10 women, 8 men; median age 47 years) were referred to our institution for the surgical management of a suspected localized invasive pulmonary aspergillosis (IPA). Only four times the association of aspergillus at the bronchoscopy and the air crescent sign at the chest CT scan was obtained. In the other cases, the diagnosis was based on clinical features, acute localized pulmonary mass at the CT scan, failure to respond to antibiotic therapy and retrieval of fungi by bronchoalveolar lavage. Five patients had haemoptysis. No patient was known to have active fungal or bacterial infection at the time chemotherapy was performed. The diagnosis of IPA was suspected 28 ± 6 days after the beginning of the chemotherapy. Seventeen patients had an antifungal medical treatment before surgery for 32 ± 6 days. The infection was localized in the upper lobe (n = 15), in the lower lobe (n = 5) and the middle lobe (n = 3) ($p<0.001$). Operative procedures included one pneumonectomy, four bilobectomies, seven lobectomies, six wedge resections and one lobectomy with wedge resection (one patient had two procedures). Twice surgery was performed urgently because the mass was located close to the main pulmonary artery. Sixteen patients were treated with antifungal agents after the operation. There were no perioperative deaths and no complications. The histologic examination of the resected specimens confirmed the diagnosis of IPA in 12 cases where invasion of blood vessels by the fungus leaded to ball pulmonary infarction. This infarcted piece of tissue was often separated from the surrounding lung by phagocytes. In the six other cases, the diagnosis was: one classical aspergilloma, one pneumonia, one pulmonary abcess and three pulmonary abcesses colonized with aspergillus without typical invasion of blood vessels by aspergillus that defines IPA. With univariate analysis, in the non-invasive pulmonary aspercillus group (NIPA) there were less thoracic pain (1/6) than in the IPA group (8/12) ($p<0.05$), a tendency to find less air crescent sign at the CT scan (1/6 in the NIPA group versus 6/12 in the IPA group) and aspergillus was more rarely retrieved by bronchoalveolar lavage (1/6 in the NIPA groupversus 7/12 in the IPA group). Sixteen patients required subsequent hematological therapies. Sixty-six percent of the

patients are alive with a mean follow-up of 29.1 ± 27.8 months (range 2 to 103 months) without any statistical difference between the IPA and the NIPA group. Five patients died with a recurrence of their malignancy at a mean of 17.2 ± 12.5 months (range 2 to 30 months) and one had a cerebral recurrence of aspergillus infection during a bone marrow transplantation three months later. Those good results of operation may be attributed to the relative young age of patients, their good pulmonary function and the brief evolution of the disease before surgery that allows limited operation. Those results encourage an aggressive policy in the management of resistant to medical treatment localized infectious pulmonary mass to prevent life threatening haemoptysis and to allow patients to proceed with further chemotherapy and bone marrow transplantation.

12:10 p.m. ADJOURN

*By invitation

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

Washington Ballroom

Moderators: Frank L. Hanley, M.D.

John A. Waldhausen, M.D.

54. DILUTIONAL AND MODIFIED ULTRAFILTRATION REDUCES PULMONARY HYPERTENSION AFTER OPERATIONS FOR CONGENITAL HEART DISEASE: A PROSPECTIVE RANDOMIZED STUDY.

†Ko Bando, M.D.*, Palaniswamy Vijay, Ph.D.*, Mark W. Turrentine, M.D.*, Scott Purvines, B.S.*, Brian J. LaLone, C.C.P.*, Thomas G. Sharp, M.D.*, Yasuo Sekine, M.D.*, Lynn Means, M.D.*, Eri Sekine, B.S., MPH*, John W. Brown, M.D.

Indianapolis, Indiana

Discussant: Ross Ungerleider, M.D.

Background and Purpose: Pulmonary hypertension (PH) is an important cause of morbidity and mortality after congenital heart surgery (CHS). Studies have shown that a potent endothelium-derived vasoconstrictor, endothelin-1 (ET-1) may initiate the development of PH after CHS. This prospective, randomized clinical study tested the hypothesis that removal of plasma ET-1 using ultrafiltration techniques will reduce PH after CHS with cardio-pulmonary bypass (CPB).

Method: Twenty-four patients with pre-op PH (systolic pulmonary arterial pressure/systemic pressure ratio: Pp/Ps > 0.6) undergoing CHS with CPB were randomized into 2 groups: a **control group (n = 12)** who had conventional ultrafiltration and an **experimental group (n = 12)** who underwent dilutional ultrafiltration (DUF) during CPB and modified ultrafiltration (MUF) after CPB **(DUF/MUFgroup).** DUF was designed to actively reduce liters of ET-1 during CPB. Veno-venous MUF was performed to further minimize ET-1 and remove excess fluid after CPB. Plasma ET-1, nitric oxide metabolites (NO) and cyclic GMP (c-GMP) levels were determined: immediately before CPB, 10 minutes into CPB, immediately after CPB, and 0, 3, 6 and 12 hours after surgery in both groups and immediately after MUF and in the ultrafiltrates for the DUF/MUF group. Both groups received prophylactic a-blockers (Chlorpromazine and/or Prazosin) after CPB based on the same protocol. Perioperative changes in hemodynamics, ET-1, NO, and c-GMP levels as well as the incidence of pulmonary hypertensive crisis (PHC) and the duration of ventilatory support were compared between the groups.

Results: DUF and MUF significantly removed plasma ET-1 (1.81 ± 0.86 pg/ml in the DUF ultrafiltrate, 6.44 ± 1.82 pg/ml in the MUF ultrafiltrate). Post-op plasma ET-1 levels and Pp/Ps ratio were significantly lower in the DUF/MUF groups compared to controls. NO and c-GMP increased in both groups up to 12 hrs post-op, with no significant differences between the groups. Three of 12 controls (25%), but none of the DUF/MUF patients had PHC after CPB (p=0.07). Patients treated with DUF/MUF required significantly shorter durations of ventilatory support (68 ± 47 hr vs 178 ±139 hr for controls, p=0.048).

Conclusions: Higher levels of ET-1 may predispose patients to PH after CHS. DUF/MUF reduces plasma ET-1 and Pp/Ps after CPB and thus may represent an important adjunct for prevention of PH early after operations for congenital heart disease in high risk patients.

†1991-92 AATS Graham Fellow

*By invitation

55. BENEFIT OF NEUROMONITORING FOR PEDIATRIC CARDIAC SURGERY.

Erie H. Austin, III, M.D., Harvey L. Edmonds, Ph.D.*, Vedad Seremet, M.D.*, Gregg Niznik, M.S.*, Aida Sehic, M.D.*, Steve M. Audenaert, M.D.* and Michael K. Sowell, M.D.*

Louisville, Kentucky

Discussant: Richard A. Jonas, M.D.

Purpose: The incidence of neurologic sequelae after PCS may reach 25% (Ferry PC, American Journal Diseases of Childhood 1990; 144:369-73). Therefore, we prospectively examined the potential benefit of interventions based on intraoperative neuromonitoring in decreasing both neurologic events and length of stay as a cost proxy.

Methods: With IRB-approved informed parental consent, 232 PCS patients received intraoperative neuromonitoring which consisted of 4-channel quantitative EEG/evoked potentials (EP), transcranial Doppler (TCD) ultrasonic measurement of middle cerebral artery blood flow velocity, and transcranial near-infrared spectroscopic determination of frontal lobe cerebral venous oxygen saturation (CVOS). Surgeon and anesthesiologist were notified if there were signs of seizure activity, a near-loss of EEG/EP or TCD signal, or a >25% CVOS decline from the prebypass baseline. Monitoring-based interventions consisted of 1) perfusion cannula or clamp repositioning, 2) arterial blood pressure increase, 3) cooling or anesthetic-induced decrease in brain metabolism, 4) resumption of cardiopulmonary bypass, 5) correction of perfusion system malfunction and/or 6) neuroprotection with dexamethasone and phenytoin.

Results: During the first year, 155 patients were monitored. Noteworthy changes in brain function were observed in 64/155 (41%) cases. The changes included two patients with a sudden total loss of the TCD signal and 24 cases with very low (<10cm/s) flow velocities despite normal systemic hemodynamics and oxygenation. Interventions were deemed appropriate in 39/64 (61%) cases. Repair complexity was unrelated to the likelihood of a monitored change or the decision to intervene. Neurologic sequelae, ranging from prolonged delerium or transient EEG-detected seizures to radiographically confirmed cerebral infarcts, occurred in 4/91 (4%) cases without noteworthy change, 4/39 (10%) cases with intervention and 17/25 (68%) without intervention (P<.001). Survivors' median length of stay was 7 days in the no change group, 6 days with intervention and 9 days without intervention. Interim analysis of these results led to more

comprehensive monitoring and increased responsiveness during the second year. In the 77 cases monitored thus far in year two, the notification rate increased to 62% and interventions were made in 92% of these cases. Only 3 patients had neurologic sequelae (i.e. prolonged confusion, transient visual neglect, and choreiform movements following a 54 minute period of deep hypothermic circulatory arrest).
Conclusions: Timely detection and correction of cerebral ischemia/hypoxia through multimodality neuromonitoring appears to improve outcome and decrease the cost of PCS. Although additional studies are needed to confirm and expand these findings, the use of randomized designs incorporating an unmonitorcd control group may raise ethical questions.

*By invitation

56. CLINICAL TRIAL OF pH MANAGEMENT STRATEGY IN INFANTS: PERIOPERATIVE RESULTS.

Richard A. Jonas, M.D., Adre J. du Plessis, M.D.*, David Wypij, Ph.D.*, Christine Plumb, R.N.*, David Farrell, M.A., C.C.P.*, Pedro J. del Nido, M.D.*, John E. Mayer, M.D., and Jane W. Newburger, M.D.*

Boston, Massachusetts

Discussant: Julie A. Swain, M.D.

In a randomized, single-center trial, we compared perioperative outcome in infants undergoing reparative open heart surgery after use of the *a stat* vs *pH stat* strategy during deep hypothermic (< 18°C) cardiopulmonary bypass. Admission criteria included (1) reparative open heart surgery; (2) age ‰¤9 months; (3) birth weight > 2.25 kg; and (4) absence of associated congenital or acquired extracardiac disorder.

Among the 182 study infants, diagnoses included dTGA (n = 92), TOP (n = 50), TOF/PA (n = 6), VSD (n = 20), truncus arteriosus (n = 8), CAVC (n = 4, not Downs), and TAPVR (n = 2). In total, 90 pts were assigned to *a stat* and 92, to *pH stat,* with randomization balanced within diagnosis, surgeon, and age group (< 1 mo, 1 -5 mo, and 5-9 mo). There were no differences between the *a stat* versus *pH stat* groups in the duration of circulatory arrest (22 ±16 vs. 21±17 min, mean ± S.D.) or total support time (129 + 49 vs. 124 ± 39 min). Early mortality (< 30 days) occurred in 4 infants (2%), all in the *a stat* group.

Perfusion strategy was not associated with differences in cardiac index measured in 123 patients at 3 hour intervals in the first 24 hours postoperatively; however, within the TGA subgroup, there was a tendency for those assigned to *pH stat* to have higher cardiac index at 12 (P=.14), 15 (p=.11), and 18 hours (p=.12). Also in the TGA subgroup, patients assigned to the *pH stat* strategy had significantly shorter duration of mechanical ventilation (P=.01) and stay in the intensive care unit (P=.01); however, there were no significant differences in these variables among patients in the other diagnostic groups. Pts assigned to *a stat* tended to have a greater incidence of postoperative hypocalcemia (p=.056) and coagulopathy (p=.056).

Continuous EEG was monitored during the first 48 hours postoperatively in 108 infants; ictal (seizure) activity was present in 5/51 pts (9.8%) assigned to *a stat* and 1/57 pts (1.8%) assigned to *pH stat* (p=.098). Clinical postoperative seizures were observed in 4 infants in the *a stat* group (4.4%) and 2 infants (2.2%) in the *pH stat* group (one later diagnosed with DiGeorge syndrome) (P=NS). First EEG activity tended to return sooner among infants randomized to *pH stat* (p=.068).

CONCLUSION: Use of the *pH stat* strategy in infants undergoing deep hypothermic bypass with or without circulatory arrest was associated with a tendency toward fewer EEG seizures and shorter recovery time to first EEG activity, and, in patients with TGA, shorter duration of intubation and ICU stay.

*By invitation

57. THE DAMUS PROCEDURE IN NEONATES AND INFANTS WITH SINGLE VENTRICLE, SUBAORTIC STENOSIS, AND ARCH OBSTRUCTION: REVISITED.

Doff B. McElhinney, M.S.*, V. Mohan Reddy, M.D.*, Norman H. Silverman, M.D.* and Frank L. Hanley, M.D.

San Francisco, California

Discussant: Thomas L. Spray, M.D.

Background. The Damus procedure (DKS), originally proposed for biven-tricular repair of transposed great arteries, is now routinely used for palliation of functional univentricular heart. In the presence of significant arch obstruction, DKS is typically performed with periods of total circulatory arrest (CA), which may contribute to impaired neurological development. In addition, potential for semilunar valve insufficiency is a concern.

Methods. Since 1990, we have performed DKS in 16 infants (median age 12 d; 5 d to 7 mo) with functional single ventricle and subaortic stenosis, 10 of whom were neonates. Significant arch obstruction was present in 11 pts. Diagnoses were {S, L, L} double-inlet left ventricle (n = 10), {S, D, D} tricuspid atresia (n = 2), and other forms of hypoplastic left ventricle (n = 4). All pts were documented to have (or potential for) subaortic obstruction by either a bulboventricular foramen (BVF) to aortic valve diameter ratio of < 1, a pressure gradient across the ventricular septal defect or BVF, or a left ventricle outflow tract gradient at the subvalvar level. In 14 pts, DKS was performed as a primary palliation, at a median age of 9 days. Various techniques were used for the DKS anastomosis of the pulmonary trunk to the ascending aorta, with emphasis on avoiding any distortion of the semilunar valves. In the most recent 5 pts with significant arch obstruction, arch repair was achieved by performing an end-to-side anastomosis of the descending aorta to the ascending aorta, without CA to the upper body, by cannulating at the base of the innominate artery or the arch with an 8 Fr arterial cannula. In the first 6 pts with arch obstruction, a median of 40 minutes total CA was used. In pts without arch obstruction, whole body perfusion was maintained. In 13 pts a systemic to pulmonary artery shunt was placed, and 1 pt underwent concurrent bidirectional Glenn shunt (BGS).

Results. There were 3 early deaths (19%), all in patients with arch obstruction who underwent periods of complete CA. There were no clinically evident neurologic events. At median follow-up of 24 months (2 to 76 months), there were no late deaths or known neurologic complications. Five pts have undergone subsequent BGS and 2 have undergone Fontan completion. No pt has more than trivial aortic or pulmonic valvar regurgitation at follow-up.

Conclusion. DKS is an effective first stage palliation for pts with univentricular heart and subaortic stenosis, with or without arch obstruction. Arch repair can be achieved without CA to the brain. With proper attention to technical details, semilunar valve insufficiency can be avoided.

10:50 a.m. INTERMISSION

*By invitation

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

Washington Ballroom

Moderators: Frank L. Hanley, M.D.

John A. Waldhausen, M.D.

58. BLOOD CARDIOPLEGIA IS NOT DEMONSTRABLY ADVANTAGEOUS OVER CRYSTALLOID CARDIOPLEGIA IN PEDIATRIC CARDIAC SURGERY.

J. Nilas Young, M.D., Isaac O. Choy, M.D.*, Nolli K. Silva, M.D.* and Derek Y. Obayashi*

Oakland, California

Discussant: Bradley S. Allen, M.D.

The superiority of blood cardioplegia in pediatric cardiac surgery has *not* previously been demonstrated in a controlled clinical trial. We prospectively randomized 138 pediatric patients (median age 12 mos ± 3.9[se]) to receive either blood (4:1 dilution, KCl 15 mEq/L) or crystalloid (Plegisol®) cardioplegia during a variety of congenital heart operations (excluding atrial septal defects). Cold (4°C), antegrade multidose cardioplegia was administered in addition to topical cooling during surgery. Systemic hypothermia perfusion (30°C) was routinely utilized and total circulatory arrest was used in 40 patients (median circ. arrest time: 29.5 mini 5.1). Myocardial recovery and outcome measures were assessed by the following clinical *endpoints:* (1) inotropic support in the first 8h post-op, INT (scale 1-10); (2) echocardiographic assessment of ventricular function in the first 24h post-op, **VF** (scale 1-10); (3) overall complication rate, COMP(%); (4)ICU length of stay (days) and (5) 30-day survival, SV(%). Statistical significance of multivariate associations was evaluated using multiple logistic regression and analysis of variance to investigate which of the following clinical *determinants* were contributory: (1) cardioplegia, **CP** (blood, n = 62 vs. crystalloid, n = 76); (2) urgency of operation, **URG;** (3) aortic cross clamp time, **X-time** (mean 66.7 min ± 2.8) and (4) **AGE** of patient. Population data did not differ between the two cardioplegia groups (p>0.05).

Results:

	Endpoints:				
Determinants	**INT**	**VF**	**COMP**	**ICU**	**SV**
CP: Blood (x±SE)	6.0 ± 0.7	9.4 ± 0.2	35.5%	8.9 ± 1.6	96.8%
Crystalloid (x ± SE)	5.2 ± 0.6	9.1 ± 0.3	33.0%	7.2 ± 1.1	92.1%
CP (p-value)	0.31	0.97	0.88	0.35	0.13
URG (p-value)	0.48	0.48	0.14	**<0.001**	0.14
X-time (p-value)	**0.008**	**<0.001**	**<0.001**	**<0.001**	**0.002**
AGE (p-value)	0.056	0.021	0.55	0.37	0.058

Significant variables: **p<0.05**

There were no statistically significant differences between blood and crystalloid cardioplegia with all measured endpoints. Sub-group cohort analysis of cyanotic lesions (n = 55) also showed no differences between blood and crystalloid cardioplegia. The most important clinical determinant of studied endpoints was the aortic cross clamp time (ischemic interval). Our results suggest no clear clinical advantage of blood cardioplegia during hypothermic cardioplegic arrest in pediatric congenital heart surgery.

*By invitation

59. MODIFIED ULTRAFILTRATION IMPROVES LEFT VENTRICULAR SYSTOLIC FUNCTION IN INFANTS AFTER CARDIOPULMONARY BYPASS.

Michael J. Davies, F.R.C.S.*, Khan Nguyen, M.D.*, J. William Gaynor, M.D.* and Martin J. Elliott, M.D.*

London, England and Philadelphia, Pennsylvania

Sponsored by: Marc R. de Leval, M. D., F. R. C. S., London, England

Discussant: Patricia A. Penkoske, M.D.

Cardiopulmonary bypass (CPB) in children is often associated with increased capillary permeability leading to increased total body water (TBW), tissue edema, and organ dysfunction. Modified ultrafiltration (MUF), performed after CPB, has been shown to reduce TBW and reverse hemodilution as well as increase cardiac index and systolic blood pressure. The mechanism of the improvement in hemodynamic parameters following MUF is unclear. This study was designed to test the hypothesis that the use of MUF after CPB improves left ventricular (LV) systolic function. Twenty-one infants undergoing CPB were instrumented with a LV micromanometer and ultrasonic dimension transducers to measure the LV anterior-posterior minor axis diameter. Patients were randomized to MUF (n= 11, age 226 ±355 days, weight 6.7 ± 3.1 kg) or control (n = 10, age 300 ± 240 days, weight 7.0 ± 2.5 kg) (p=NS difference between groups). LV systolic function was assessed using the slope of the preload recruitable stroke work (PRSW) index, a load-insensitive index of LV function. Myocardial cross-sectional area was measured by echocardiography. Data were acquired immediately following separation from CPB, at steady state and during transient vena caval occlusion. In the MUF patients, data acquisition was repeated after 13 ± 5 minutes of MUF. In the control patients, data acquisition was repeated after 12 + 5 minutes (p=NS). Inotropic drug support was the same at both study points. In the MUF group, the filtrate volume was 363 ± 262 ml and the hematocrit increased from 26 ± 2.7% to 37 ± 9.5% after MUF (p=0.018). In the control group, the hematocrit did not change (p=NS). Heart rate, end-diastolic dimension, and end-diastolic pressure were unchanged in both groups (p=NS). 'In the MUF group, mean ejection pressure increased from 58 ± 25 to 71+23 mmHg after MUF (p=0.005), but did not change in the control group (p=NS). Myocardial cross sectional area decreased from 3.72 ± 0.35 to 3.63 ± 0.36 CM^2 after MUF (p=0.01), suggesting a reduction in myocardial edema. Myocardial cross sectional area remained constant in the control group (p=NS). After MUF, the slope of the PRSW index increased from 52.3 ± 52 to 74.2 ± 66 $(10^3$ erg/cm$)^3$ (p=0.02), but did not change in the control group (p=NS). One patient from each group died in the postoperative period. Patients in the MUF group received less inotropic drug support in the first 24 hours following surgery (156.62 + 92.31 µg/kg/24hr) than patients in the control group (865.33 ± 1772.26 µg/kg/24hr, p=0.03). The use of MUF after CPB improves intrinsic LV systolic function, increases blood pressure, and decreases inotropic drug utilization in the early postoperative period.

*By invitation

60. ONE-STAGE MIDLINE UNIFOCALIZATION AND COMPLETE REPAIR IN INFANCY VERSUS MULTIPLE STAGE UNIFOCALIZATION FOLLOWED BY REPAIR FOR COMPLEX HEART DISEASE WITH MAJOR AORTOPULMONARY COLLATERALS.

Christo I. Tchervenkov, M.D.*, Gary Salasidis, M.D.*, Renzo Cecere, M.D.*, Marie J. Beland, M.D.*, Luc Jutras, M.D.*, Marc Paquet, M.D.* and Anthony R.C. Dobell, M.D.

Montreal, Quebec, Canada

Discussant: Erie H. Austin, HI, M.D.

Patients with pulmonary atresia (PA), VSD and major aortopulmonary collaterals (MAPCA's) have traditionally required multiple unifocalization staging operations. In the few large series, complete repair was possible on only between 12-60% of all the patients. Recently, the feasability of a single stage unifocalization and repair was demonstrated by Hanley. We would like to share our recent experience with both these approaches. Since 1989, 11 consecutive patients not previously operated with complex heart disease and MAPCA's have undergone corrective surgery. The first 6 pts underwent staged unifocalizations with 5 achieving complete repair (Group I). The last 5 pts since May 1995 have undergone one stage midline unifocalization and complete repair (Group II). Four of these were infants (2 wks to 9 mos) and one was 13 years old. All pts in Group I had Tetralogy (TOP), PA whereas in Group II, three pts had TOP, PA, one had DORV, PA and one CAVC, TGA and severe PS.

	# MAPCA's	MEDIAN AGE AT 1st OR	MEDIAN AGE AT COMPLETE REPAIR	# OR's	POST-OP RVp/LVp
Gr I	3.6(2-6)	6 mos	3 yrs 3 mos	3.2	.48
Gr II	3.0 (2-4)	6 mos	6 mos	1	.46

Complete repair was achieved in 83% of Gr I and 100% in Gr II. There was one intraoperative death (unrecognized severe mitral stenosis) and one late death in Gr I and all 5 pts in Gr II are alive and well with a mean follow-up of 9 mos (2-17 mos).

We conclude that early intervention with both surgical approaches can lead to complete biventricular repair in almost all patients. Because the single stage midline unifocalization and repair can achieve complete repair and excellent survival in infancy with one operation, it is currently our approach of choice.

12:10 p.m. ADJOURN

*By invitation

GEOGRAPHICAL ROSTER

NECROLOGY

Ralph D. Alley, M.D..... Loudonville, New York

Bruce F. Baisch, M.D..... La Jolla, California

Hector W. Benoit, M.D..... Kansas City, Missouri

Lee B. Brown, M.D..... Phoenix, Arizona

John G. Chesney, M.D..... Miami, Florida

William B. Condon, M.D..... Denver, Colorado

Albert DeMatteis, M.D..... St. Petersburg, Florida

H. Edward Garrett, Sr., M.D..... Memphis, Tennessee

W.A. Hudson, M.D..... Jasper, Arizona

Karl E. Karlson, M.D..... Providence, Rhode Island

Winfield O. Kelley, M.D..... Norwich, Connecticut

Joseph E. McManus, M.D..... Wellesley, Massachusetts

Gilles Lepage, M.D..... Quebec, Canada

Raleigh R. Ross, M.D..... Burnet, Texas

John M. Snyder, M.D..... Bethlehem, Pennsylvania

Lamar Soutter, M.D..... Concord, Massachusetts

Henry Swan, M.D..... Lakewood, Colorado

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(Listed by Countries, States, Provinces and Cities)
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Silver, Arthur W

Kirklin, James K

Kirklin, John W

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Drinkwater, Davis C

Daily, Pat O

Holmes, E Carmack

Kay, Jerome H

Khonsari, Siavosh

Laks, Hillel

Lindesmith, George G

Longmire, William, Jr

Maloney, James V, Jr

Mandal, Ashis K

Matloff, Jack M

Mulder, Donald G

Starnes, Vaughn A

Trento, Alfredo

Waters, Paul F

Los Osos

Aronstam, Elmore M

Montebello

Lui, Alfred H F

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Ecker, Roger R

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Follette, David M

Harlan, Bradley J

Hurley, Edward J

Smeloff, Edward A

San Bernardino

Misbach, Gregory A

San Diego

Baronofsky, Ivan D

Chambers, John S

Robertson, John M

Santa Rosa

Neville, William E

Sausalito

Zaroff, Lawrence I

Sonoma

Richards, Victor

Spring Valley

Long, David M, Jr

St Helena

Dugan, David J

Stanford

Mark, James B D

Miller, D. Craig

Oyer, Philip E

Reitz, Bruce A

Shumway, Norman E

Stinson, Edward B

Tiburon

Heydorn, William H

Torrance

Carey, Joseph S

Cukingnan, Ramon A

Moore, Thomas C

State, David

Valley Village

Davis, Lowell L

Victorville

Jurado, Roy A

COLORADO

Beulah

Bartley, Thomas D.

Colorado Springs

Keith, William J

Stern, Harold

DELAWARE

Newark

Lemole, Gerald M

Wilmington

Pecora, David V

DISTRICT OF COLUMBIA

Washington

Aaron, Benjamin L

Gomes, Mario N

Kate, Nevin M

Keshishian, John M

Lefemine, Armand A

Midgley, Frank M

Denver

Brown, Robert K

Campbell, David N

Clarke, David R

Eiseman, Ben

Grover, Frederick L

Grow, John B, Sr

Harken, Alden H

Hopeman, Alan R

Paton, Bruce C

Pomerantz, Marvin

Rainer, W Gerald

Wright, George W

Englewood

Kovarik, Joseph L

Littleton

Pappas, George

Vail

Fuller, Josiah

CONNECTICUT

Bridgeport

Rose, Daniel M

Essex

Jaretzki, Alfred, JII

Hartford

Kemler, R Leonard

New Haven

Elefteriades, John A

Glenn, William W. L

Hammond, Graeme L

Kopf, Gary S

New Milford

Okinaka, Arthur J

Wilton

Pool, John L

Woodbridge

Lindskog, Gustaf E

Simmons, Robert L

FLORIDA

Atlantic Beach

Stranahan, Allan

Bal Harbour

Grondin, Pierre

Belleair

Lasley, Charles H

Boca Raton

Seley, Gabriel P

Clearwater

Wheat, Myron W., Jr

Coconut Grove

Center, Sol

Coral Gables

Cooke, Francis N

Delray Beach

Shumacker, Harris B, Jr

Gainesville

Alexander, James A

Jacksonville

Edwards, Fred H

Koster, J Kenneth, Jr

Stephenson, Sam, Jr

Jupiter

Gerbasi, Francis S

Lakeland

Brown, Ivan W, Jr

Marathon

Mangiardi, Joseph L

Miami

Bolooki, Hooshang

Daughtry, Dewitt C

Greenberg, Jack J

Jude, James R

Kaiser, Gerard A

MacGregor, David C

Palatianos, George M

Papper, Emanuel M

Ripstein, Charles B

Thurer, Richard J

Wilder, Robert J

Miami Beach

Reis, Robert L

Spear, Harold C

Subramanian, S

Naples

Battersby, James S

Linberg, Eugene J

Smyth, Nicholas P D

Orlando

Scott, Meredith L

Sherman, Paul H

Ponte Vedra Beach

Gilbert, Joseph, Jr

Punta Gorda

Taber, Rodman E

St Petersburg

Daicoff, George R

Tallahassee

Kraeft, Nelson H

Tamarac

Mendelssohn, Edwin

Tampa

Angell, William W

Robinson, Lary A

Seiler, Hawley H

Winter Haven

Maurer, Elmer P R

GEORGIA

Atlanta

Chickamauga

Hall, David P

Macon

Dalton, Martin L, Jr

Sealy, Will C

Van De Water, Joseph M

Savannah

Yeh, Thomas J

St Simons Island

Taylor, Frederick H

HAWAII

Honolulu

Ching, Nathaniel P

Gebauer, Paul W

McNamara, J. Judson

IDAHO

Boise

Herr, Rodney H

ILLINOIS

Burr Ridge

Blakeman, Bradford P

Chicago

Barker, Walter L

Breyer, Robert H

Campbell, Charles D

Ebert, Paul A

Faber, L. Penfield

Ferguson, Mark K

Fullerton, David A

Goldin, Marshall D

Hanlon, C Rollins

Hartz, Renee S

Head, Louis R

Hunter, James A

Graver, Joseph M

Guyton, Robert A

Hatcher, Charles, Jr

Hopkins, William A

Jones, Ellis L

Kanter, Kirk R

King, Richard

Lee, Arthur B, Jr

Mansour, Kamal A

Miller, Joseph I, Jr

Rivkin, Laurence M

Symbas, Panagiotis

Williams, Willis H

Augusta

Ellison, Robert G

Rubin, Joseph W

Karp, Robert B

Kittle, C Frederick

Mavroudis, Constantine

Michaelis, Lawrence

Montoya, Alvaro

Najafi, Hassan

Raffensperger, John

Repiogle, Robert L

Shields, Thomas W

Tatooles, C. J

Thomas, Paul A, Jr

Vanecko, Robert M

Warren, William H

Downers Grove

Leininger, Bernard J

Elk Grove Village

Sullivan, Henry J

Evanston

Fry, Willard A

Glencoe

Rubenstein, L H

Harvey

Norman, John C

Maywood

DeLeon, Serafm Y

Pifarre, Roque

Oak Brook

Amato, Joseph J

Hudson, Theodore R

Ilbawi, Michel N

Javid, Hushang

Jensik, Robert J

Mason, G. Robert

Nigro, Salvatore L

Park Ridge

KANSAS

Cunningham

Allbritten, Frank F, Jr

Lawrence

Miller, Don R

Prairie Village

Holder, Thomas M

Shawnee Mission

Adelman, Arthur

Mayer, John H, Jr

Padula, Richard T

Wichita

Tocker, Alfred M

KENTUCKY

Lexington

Crutcher, Richard R

Mentzer, Robert M, Jr

Swain, Julie A

Baffes, Thomas G

Levett, James M

Weinberg, Milton, Jr

Peoria

DeBord, Robert A

Springfield

Wellons, Harry A, Jr

Winnetka

Mackler, S Allen

INDIANA

Bloomington

O'Neill, Martin J, Jr

Indianapolis

Brown, John W

King, Harold

King, Robert D

Mandelbaum, Isidore

Siderys, Harry

IOWA

Cedar Rapids

Lawrence, Montague S

Council Bluffs

Sellers, Robert D

Des Moines

Dorner, Ralph A

Phillips, Steven J

Zeff, Robert H

Iowa City

Behrendt, Douglas M

Ehrenhaft, Johann L

Richenbacher, Wayne E

Rossi, Nicholas P

Stanford, William

Todd, Edward P

Louisville

Austin, Erie H, III

Gray, Laman A, Jr

Mahaffey, Daniel E

Ransdell, Herbert, Jr

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

MAINE

Portland

Bredenberg, Carl E

Morton, Jeremy R

Rockport

Swenson, Orvar

Windham

Hiebert, Clement

Rheinlander, Harold F

Russell, Paul S

MARYLAND

Baltimore

- Attar, Safiih
- Baker, R. Robinson
- Baumgartner, William A
- Blair, Emil
- Cameron, Duke Edward
- Dodrill, Forest D
- Gott, Vincent L
- Haller, J Alex, Jr
- Hankins, John R
- McLaughlin, Joseph S
- Michelson, Elliott
- Salomon, Neal W
- Turney, Stephen Z
- Watkins, Levi, Jr

Bethesda

- Pass, Harvey I

Fort Detrick

- Zajtchuk, Rostik

Reisterstown

- Heitmiller, Richard F

Worton

- Walkup, Harry E

MASSACHUSETTS

Boston

- Akins, Cary W
- Austen, W. Gerald
- Barsamian, Ernest M
- Bougas, James A
- Buckley, Mortimer J
- Burke, John F
- Cohn, Lawrence H
- Collins, John J, Jr
- Daggett, Willard M
- Daly, Benedict D T
- 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

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

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

Rousou, John A

Vineyard Haven

Malm, James R

Wellesley

Cleveland, Richard J

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

Deschamps, Claude

McGoon, Dwight C

McGregor, Christopher G A

Olsen, Arthur M

Orszulak, Thomas A

Pairolero, Peter C

Payne, W Spencer

Puga, Francisco J

Schaff, Hartzell V

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

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

Mendota Heights

Dennis, Clarence

Minneapolis

Arom, Kit V

Bolman, R. Morton, III

Emery, Robert W

Foker, John E

Gannon, Paul G

Garamella, Joseph J

Helseth, Hovald K

Kaye, Michael P

Molina, J. Ernesto

Nicoloff, Demetre M

Shumway, Sara J

Rochester

Allen, Mark S

Bernatz, Philip E

Danielson, Gordon K

Johnston, J. Harvey , Jr

Netterville, Rush E

Madison

Hardy, James D

Natchez

Bloodwell, Robert 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

Borkon, A Michael

Killen, Duncan A

Piehler, Jeffrey M

Reed, William A

Van Way, Charles W, III

Mount Vernon

Campbell, Daniel C, Jr

St Louis

Earner, Hendrick B

Baue, Arthur E

Connors, John P

Cooper, Joel D

Cox, James L

Ferguson, Thomas B

Flye, M Wayne

Gay, William A, Jr

Johnson, Frank E

Jersey City

Demos, Nicholas J

Millburn

Personnel, Victor

Moorestown

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, Tracy 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

NEW HAMPSHIRE

Franconia

Taylor, Warren J

Jaffrey

Woods, Francis M

Lebanon

Nugent, William C

Sanders, John H, Jr

NEW JERSEY

Alpine

Holswade, George R

Belleville

Gerard, Franklyn P

Browns Mills

Fernandez, Javier

McGrath, Lynn B

Morse, Dryden P

Morristown

Parr, Grant V S

Neptune

Roberts, Arthur J

New Brunswick

Lewis, Ralph J

MacKenzie, James W

Scholz, Peter M

Newark

Bregman, David

Donahoo, James

Gielchinsky, Isaac

Swan, Kenneth G

Pittstown

Garzon, Antonio A

Short Hills

Hochberg, Mark S

Tenafly

Gerst, Paul H

NEW MEXICO

Albuquerque

Edwards, W Sterling

Buena Vista

Thai, Alan P

Santa Fe

Davila, Julio C

Silver City

Waddell, William R

NEW YORK

Albany

Ferraris, Victor A

Foster, Eric D

Bay Shore

Ryan, Bernard J

Bronx

Altai, Lari A

Camden

Camishion, Rudolph C

DelRossi, Anthony J

East Orange

Auerbach, Oscar

Hackensack

Hutchinson, John E, III

Brodman, Richard F

Fell, Stanley C

Ford, Joseph M

Prater, Robert WM

Gold, Jeffrey P

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

Fayerteville

Bugden, Walter F

Effler, Donald B

Floral Park

Crastnopol, Philip

Larchmont

Steichen, Felicien M

Lido Beach

Mines, George L

New Rochelle

Rubin, Morris

New York

Acinapura, Anthony J

Adams, Peter X

Nealon, Thomas F, Jr

Quaegebeur, Jan M

Redo, S Frank

Reemtsma, Keith

Rose, Eric A

Rusch, Valerie W

Skinner, David B

Smith, Craig R

Spencer, Frank C

Spotnitz, Henry M

Subramanian, Valavanur A

Tice, David A

Tyras, Denis H

Wichern, Walter, Jr

Wolff, William I

Patchogue

Finnerty, James

Plattsburgh

Potter, Robert T

Rochester

Graver, William L

DeWeese, James A

Hicks, George L

Schwartz, Seymour I

Stewart, Scott

Altorki, Nasser K

Anagnostopoulos, C E

Bains, Manjit S

Beattie, Edward, Jr

Bloomberg, Allan E

Boyd, Arthur D

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

Green, George E

Griepp, Randall B

Isom, O Wayne

King, Thomas C

Kirschner, Paul A

Krieger, Karl H

Lansman, Steven L

Litwak, Robert S

Martini, Nael

McCord, Colin W

McCormack, Patricia M

Michler, Robert E

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

Chapel Hill

Bowman, Frederick, Jr

Egan, Thomas M

Keagy, Blair A

Starek, Peter J

Wilcox, Benson R

Charlotte

Wilson, James M

Wright, Creighton B

Yee, Edward S

Cleveland

Ankeney, Jay L

Cosgrove, Delos M

Geha, Alexander S

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

Wolfe, Walter G

Young, W. Glenn, Jr

Greensboro

Van Trigt, Peter, III

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

Kon, Neal D

Meredith, Jesse H

Pennington, D. Glenn

OHIO

Chagrin Falls

Cross, Frederick S

Cincinnati

Groves, Laurence K

Kay, Earle B

Kirby, Thomas J

Loop, Floyd D

Lytle, Bruce W

McCarthy, Patrick M

Rice, Thomas W

Snow, Norman J

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

Kilman, 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.MNazih

OREGON

Days Creek

Albers, John E

Callard, George M

Flege, John B, Jr

Gonzalez, Luis L

Helmsworth, James A

Hiratzka, Loren F

Ivey, Tom D

Miller, Arthur C

Portland

Cobanoglu, Adnan

Krause, Albert H

Lemmer, John H, Jr

Okies, J Edward

Poppe, J Karl

Starr, Albert

PENNSYLVANIA

Bristol

Dunn, Jeffrey M

Bryn Mawr

Haupt, George J

Mundth, Eldred D

Camp Hill

Pennock, John L

Carlisle

DeMuth, William, Jr

Darby

McKeown, John J, Jr

Hershey

Campbell, David B

Damiano, Ralph J, Jr

Pae, Walter E, Jr

Pierce, William S

Waldhausen, John A

Johnstown

Kolff, Jacob

Lancaster

Bonchek, Lawrence I

Rosemond, George P

Witmer, Robert H

Philadelphia

Addonizio, V. Paul

Bowles, L Thompson

Myers, John L

Pontius, Robert G

Rams, James J

Siewers, Ralph D

Rosemont

Templeton, John, III

Rydel

Frobese, Alfred S

Wayne

Hargrove, W Clark, III

Lemmon, William M

Wynnewood

Wallace, Herbert W

Yardley

Sommer, George N, Jr

RHODE ISLAND

Providence

Hopkins, Richard A

Moulton, Anthony L

Singh, Arun K

SOUTH CAROLINA

Charleston

Bradham, R Randolph

Crawford, Fred A, Jr

Ferguson, T Bruce, Jr

Kratz, John M

Parker, Edward F

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

Mannion, John D

Nemir, Paul, Jr

Shochat, Stephen J

Sink, James D

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

Reed, Carolyn E

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

Howard, Hector S, Jr

Hughes, Felix A, Jr

McBurney, Robert P

Pate, James W

Robbins, S Gwin, Sr

Rosensweig, Jacob

Skinner, Edward F

Watson, Donald C

Nashville

Alford, WilIiam, Jr

Bender, Harvey W, Jr

Gobbel, Walter G, Jr

Merrill, Walter H

Randolph, Judson G

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

Rankin, J Scott

Sawyers, John L

Scott, H. William, Jr

Stoney, William S

Thomas, Clarence, Jr

Sparta

Labrosse, Claude C

TEXAS

Amarillo

Sutherland, R Duncan

Austin

Hood, R Maurice

Tyson, Kenneth R T

Dallas

Adam, Maurice

Estrera, Aaron S

Holland, Robert H

Lambert, Gary J

Mack, Michael J

Mills, Lawrence J

Paulson, Donald L

Platt, 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

Reul, George J, Jr

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

Calhoon, John H

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

Burdette, Walter J

Cooley, Denton A

Coselli, Joseph S

DeBakey, Michael E

Burton, Nelson A

Lefrak, Edward A

Arlington

Klepser, Roy G

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

McLean

Conrad, Peter W

Mills, Mitchell

Wallace, Robert B

Reston

Boyd, Thomas F

Richmond

Bosher, Lewis H, Jr

Brooks, James W

Cole, Dean B

Lower, Richard R

Wechsler, Andrew S

WASHINGTON

Altavista

Pierucci, Louis, Jr

Annandale

Akl, Bechara F

Jones, Thomas W

Lupinetti, F. Mark

Manhas, Dev R

Mansfield, Peter B

Merendino, K. Alvin

Miller, Donald W, Jr

Rittenhouse, Edward

Sauvage, Lester R

Thomas, George I

Verrier, Edward D

Spokane

Berg, Ralph Jr

WEST VIRGINIA

Charleston

Walker, James H

Huntington

Gonzalez-Lavin, Lorenzo

Morgantown

Graeber, Geoffrey M

Gustafson, Robert A

Hill, Ronald C

Murray, Gordon F

Warden, Herbert E

Parkersburg

Tamay, Thomas J

WISCONSIN

Eau Claire

McEnany, M Terry

Madison

Chopra, Paramjeet S

Young, William P

Marshfield

Bellingham

Varco, Richard L

Friday Harbor

Lawrence, G Hugh

Issaquah

Jarvis, Fred J

Kirkland

Mills, Waldo O

Mercer Island

Li, Wei-I

Poulsbo

Malette, William G

Seattle

Allen, Margaret D

Anderson, Richard P

Hill, Lucius D

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

1996-1997

CANADA

ALBERTA

Calgary

Bharadwaj, Baikunth

Miller, George E

Edmonton

Callaghan, John C

Gelfand, Elliot T

Koshal, Arvind

Sterns, Laurence P

BRITISH COLUMBIA

Vancouver

Ashmore, Phillip G

Jamieson, W R Eric

Tyers, G. Frank O

Victoria

Sudbury

Field, Paul

Walker, George R

Toronto

Baird, Ronald J

Bigelow, Wilfred O

Christakis, George T

Coles, John G

David, Tirone E

Feindel, Christopher M

Fremes, Stephen E

McKneally, Martin F

Mickleborough, Lynda L

Pearson, F Griffith

Scully, Hugh E

Todd, Thomas R J

Stenstrom, John D

MANITOBA

Winnipeg

Barwinsky, Jaroslaw

Cohen, Morley

NOVA SCOTIA

Halifax

Murphy, David A

Mabou

Thomas, Gordon W

ONTARIO

London

Guiraudon, Gerard M

McKenzie, F Neil

Novick, Richard J

North York

Goldman, Bernard S

Nottawa

Key, James A

Oakville

Allen, Peter

Ottawa

Keon, Wilbert 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

Shennib, Hani

Sainte-Foy

DesLauriers, Jean

Geographical - OTHER COUNTRIES

1996-1997

ARGENTINA

Buenos Aires

Favaloro, Rene G

AUSTRALIA

QUEENSLAND

Brisbane

O'Brien, Mark F

SOUTH AUSTRALIA

de Leval, Marc R

Lincoln, Christopher R

Ross, Donald N

Stark, Jaroslav F

Taylor, Kenneth M

Yacoub, Magdi

Somerset

Abbey-Smith, R

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

Abaco

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

Wallwork, John

Herts

Lennox, Stuart C

Liverpool

Donnelly, Raymund J, M.B.

London

FINLAND

Kauniainen

Mattila, Severi P

FRANCE

Bordeaux

Couraud, Louis

Fontan, Francis M

Collonges Au Mt D'Or

Champsaur, Gerard L

Le Plessis Robinson
Binet, Jean-Paul

Dartevelle, Philippe G

Marseille

Metras, Dominique R

Montpellier

Thevenet, Andre A

Paris
Blondeau, Philip

Cabrol, Christian E A

Carpentier, Alain F

Loisance, Daniel

Menasche, Philippe

Piwnica, Armand H

Planche, Claude

Weldon, Clarence S

Suresnes

Sachet, Jean E

GERMANY

Aachen

Messmer, Bruno J

Berlin

Hetzer, Roland

Hannover

Haverich, Axel

Munchen

Borst, Hans G

Sebening, Fritz

Braimbridge, Mark V

Neuss

Bircks, Wolfgang H

GUATEMALA
Guatemala City

Herrera-Llerandi, Rodolfo

INDIA
Bikaner

VanAllen, Chester M

IRELAND
Dublin

O'Malley, Eoin

ITALY

Bergamo

Parenzan, Lucio

Milan

Peracchia, Alberto

Naples
Cotrufo, Maurizio

Rome

Bortolotti, Uberto

Marcelletti, Carlo

JAPAN

Kamakura

Suma, Hisayoshi

Kanazawa
Iwa, Takashi

Kitakyushushi

Miyamoto, Alfonso T

Minoo

Kawashima, Yasunaru

Sendai

Mohri, Hitoshi

Shinjuku

Imai, Yasuharu

Tokyo
Koyanagi, Hitoshi

Wada, Juro J

P.R. OF CHINA
Beijing

Ying-Kai, Wu

PORTUGAL
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
Barcelona

Aris, Alejandro

Madrid

Rivera, Ramiro

Sandander

Revuelta, Jose Manuel

SWEDEN

Sollentuna

Bjork, Viking

SWITZERLAND
Arzier

Harm, Charles J

KOREA
Seoul

Cho, Bum-Koo

MONACO
Monaco

Dor, Vincent

THE NETHERLANDS

Wassenaar

Brom, A Gerard

NEW ZEALAND

Waiwera HBC

Barratt-Boyes, Brian G

Genolier
Castaneda, Aldo R

Norwood, William I

Pully
Naef, Andreas P

Zurich

Senning, Akc

Turina, Marko I

VENEZUELA

Caracas

Tricerri, Fernando E

THE AMERICAN ASSOCIATION FOR THORACIC SURGERY
Charter Members
June 17, 1917

E. Wyllis Andrews

John Auer

Edward R. Baldwin

Walter M. Boothby

William Branower

Harlow Brooks

Lawrason Brown

Kenneth Bulkley

Alexis Carrel

Norman B. Carson

J. Frank Corbett

Armistead C. Crump

Charles N. Dowd

Kennon Dunham

Edmond Melchior Eberts

Max Einhorn

Herman Fischer

Albert H. Garvin

Nathan W. Green

Arthur A. Law

William Lerche

Howard Lilienthal

William H. Luckett

Morris Manges

Walton Martin

Rudolph Matas

E.S. McSweeney

Samuel J. Metzler

Willy Meyer (Founder)

James Alexander Miller

Robert T. Miller

Fred J. Murphy

Leo S. Peterson

Eugene H. Pool

Walter I. Rathbun

Martin Rehling

B. Merrill Ricketts

Samuel Robinson

John R. Hartwell	Charles I. Scudder
George J. Heuer	William H. Stewart
Chevalier Jackson	Franz Torek
H.H. Janeway	Martin W. Ware
James H. Kenyon	Abraham O. Wilensky
Adrian V. S. Lambert	Sidney Yankauer

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 shiill 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 nine 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 or all members of this Committee may be reappointed to succeed themselves. The Council may, if it so desires, appoint one of its own members to serve as Chairman of this Committee. The duties of the Necrology Committee shall be to prepare suitable resolutions and memorials upon all deaths of members of the Association and to report such deaths at every annual meeting.

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

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

Section 7. The Evarts A. Graham Memorial Traveling Fellowship Committee shall consist of 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. Miscelleneous 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 preceeding 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

1996-San Diego..... President, Mortimer J. Buckley

GRAHAM EDUCATION AND RESEARCH FOUNDATION

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

President James L. Cox, M.D., St. Louis, Missouri
Vice President Andrew S. Wechsler, M.D., Richmond, Virginia
Secretary-Treasurer William T. Maloney, Manchester, Massachusetts
Director John C. Baldwin, M.D., Houston, Texas

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 Winnepeg, Manitoba, CANADA
2nd	1953-54	W.B. Ferguson Newcastle-upon-tyne, ENGLAND
3rd	1954-55	Lance L. Bromley London, ENGLAND
4th	1955-56	Raymond L. Hurt Radlett Herts, ENGLAND
5th	1956-57	Mathias Paneth

		London, ENGLAND
6th	1957-58	Peter L. Brunnen
		Aberdeen, SCOTLAND
7th	1958-59	N.G. Meyne
		Amsterdam, HOLLAND
8th	1960-61	Godrej S. Karai
		Calcutta, INDIA
9th	1961-62	Fritz Helmer
		Vienna AUSTRIA
10th	1962-63	Theodor M. Scheinin
		Helsinki, FINLAND
11th	1963-64	Masahiro Saigusa
		Tokyo, JAPAN
12th	1963-64	Adar J. Hallen
		Uppsala, SWEDEN
13th	1964-65	Stuart C. Lennox
		London, ENGLAND
14th	1964-65	Elias Carapistolis
		Thessaloniki, GREECE
15th	1965-66	Gerhard Friehs
		Graz, AUSTRIA
16th	1965-66	Ary Blesovsky
		London, ENGLAND
17th	1966-67	C. Peter Clarke
		Fitzroy, AUSTRALIA
18th	1966-67	G.B. Parulkar
		Bombay, INDIA
19th	1967-68	Claus Jessen
		Copenhagen, DENMARK
20th	1969-70	Peter Brueke
		Linz-Puchenau, AUSTRIA
21st	1970-71	Michel S. Slim
		New York, New York USA
22nd	1971-72	Severi Pellervo, Mattila
		Kaunianen, FINLAND
23rd	1972-73	Yasuyuki Fujiwara
		Tokyo, JAPAN
24th	1973-74	Marc Roger de Leval
		London, ENGLAND
25th	1974-75	J. J. DeWet Lubbe
		Cape Town, REPUBLIC OF SOUTH AFRICA
26th	1975-76	Mieczyslaw Trenkner
		Gdansk, POLAND
27th	1976-77	Bum Koo Cho
		Seoul, KOREA
28th	1977-78	Alan William Gale
		Sydney, AUSTRALIA
29th	1978-79	Eduardo Otero Goto
		Valencia, SPAIN
30th	1980-81	Richard K. Firmin
		Leicester, ENGLAND
31st	1981-82	Claudio A. Salles
		Belo Horizonte MG, BRAZIL
32nd	1982-83	Yasuhisa Shimazaki
		Osaka, JAPAN
33rd	1983-84	Georg S. Kobinia

		Klagenfurt, AUSTRIA
34th	1984-85	Aram Smolinsky Tel Hashomer, ISREAL
35th	1985-86	Florentine J. Varga Buenos Aires, ARGENTINA
36th	1986-87	Ari L. J. Harjula Helsinki, FINLAND
37th	1987-88	Byung-Chul Chang Seoul, KOREA
38th	1988-89	Wang Cheng Beijing, PEOPLE'S REPUBLIC OF CHINA
39th	1989-90	Christopher John Knott-Craig Cape Town, SOUTH AFRICA
40th	1991-92	Ko Bando Okayama, JAPAN
41st	1992-93	Timothy E. Oaks Hershey, PA, USA
42nd	1993-94	Alain E. Serraf Le Plessis Robinson, FRANCE
43rd	1995-96	Cornelius McKown Dyke Richmond, VA, USA
44th	1996-97	Monica Robotin-Johnson Sydney, AUSTRALIA
45th	1997-98	Jun Wang Beijing, PEOPLE'S REPUBLIC OF CHINA

THE THORACIC SURGERY FOUNDATION AWARDS

* Individual Research Investigator Grants

* Research Fellowship Awards

* Career Development Awards

* Alley-Sheridan Scholarships

Note: Recipients of the AATS Graham Education and Research Foundation are listed on page 305.

THE THORACIC SURGERY FOUNDATION RESEARCH FELLOWSHIP

Edward M. Boyle, Jr., MD

The University of Washington

Seth Force, MD

The University of Pennsylvania

THE THORACIC SURGERY FOUNDATION RESEARCH GRANT

Si M. Pham, MD

The University of Pittsburgh

Todd K. Rosengart, MD

The New York Hospital - Cornell Medical Center

David S. Schrump, MD

The University of Texas - MD Anderson Cancer

Nina S. Braunwald Career Development Award

Patricia A. Thistlethwatie, MD

The University of Pittsburgh Medical Center

PREVIOUS RESEARCH AWARD RECIPIENTS

THE THORACIC SURGERY FOUNDATION RESEARCH FELLOWSHIP

Julie R. Glasson, MD

Stanford University School of Medicine

1994-1996

Joseph H. Gorman, III, MD

Hospital of the University of Pennsylvania

1995-1996

Robert S. Poston, Jr., MD

Stanford University Medical Center

1996-1997

Andrew J. Sherman, MD
Northwestern University Medical School

1996-1997

THE THORACIC SURGERY FOUNDATION RESEARCH GRANT

Richard P. Embrey, MD
The Medical College of Virginia 1995-1996

Joren C. Madsen, MD

Massachusetts General Hospital

1995-1996

John D. Mannion, MD

Thomas Jefferson University

1995-1996

Si M. Pham, MD

University of Pittsburgh 1996-1997

NINA S. BRAUNWALD CAREER DEVELOPMENT AWARD

Margaret D. Allen, MD

University of Washington School of Medicine

1995-1997

Mary C. Mancini, MD

Louisiana State University Medical Center

1996-1998

NINA S. BRAUNWALD RESEARCH FELLOWSHIP

Elaine E. Tseng, MD

Johns Hopkins Hospital

1995-1997

Jennifer Dale Walker, MD

Medical University of South Carolina

1993-1995

PREVIOUS EDUCATION AWARD RECIPIENTS

HARVARD MPA SCHOLAR-IN-RESIDENCE

Paul N. Uhlig, MD

Wichita, Kansas

MAY. 1996 ALLEY-SHERIDAN SCHOLARS

E. Pendleton Alexander, MD

Washington, DC/VA Medical Center

Richard P. Embrey, MD

Medical College of Virginia Hospitals

Timothy J. Gardner, MD

Hospital of the University of Pennsylvania

Keith S. Naunheim, MD

St. Louis University School of Medicine

Anthony Louis Picone, MD

SUNY Health Science Center

Keith Eric Sommers, MD

University of Pittsburgh

Clifford H. Van Meter, MD

Ochsner Medical Institutions

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
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
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
Duke University Medical Center

ALTON OCHSNER RESEARCH SCHOLARSHIP

"Experimental Cardiac Graft Arteriosclerosis: Pathogenesis and Prevention of Lesion Development"
1992-1994 David H. Adams
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, Columbia-Presbyterian Medical Center
"Development of a Model of Chronic Pulmonary Rejection in Miniature Swine"
1994-1996 Toralf Mauritz Sundt, III
Washington University School of Medicine

JOHN ALEXANDER RESEARCH SCHOLARSHIP

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

ANDREW G. MORROW RESEARCH SCHOLARSHIP

"The Detection of Telomerase Activity in Patients with Non-Small Cell Lung Cancer"

1997-1999 Stephen C. Yang
Johns Hopkins University School of Medicine