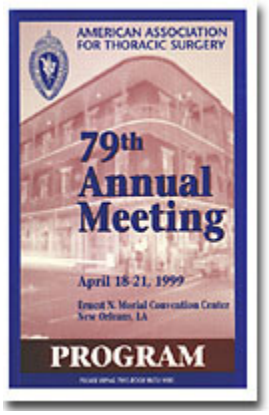
# 1999 ANNUAL MEETING PROGRAM

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# 1999 ANNUAL MEETING

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# DEVELOPING THE ACADEMIC SURGEON SYMPOSIUM

***Saturday, April 17, 1999 1:00 p.m. - 6:00 p.m.***

***Ernest N. Mortal Convention Center - Room 211/212***

## Objective

The 1999 Academic Surgeon's Symposium is a new symposium designed to help develop the Academic Cardiothoracic Surgeon. This is a new effort by the American Association for Thoracic Surgery to help provide a specific educational conference for potential and active academic cardiothoracic surgeons. The present Symposium will focus on several areas including time management, developing surgical techniques, research, administrative skills and mentorship.

This Symposium is designed for Residents interested in a career in academic cardiothoracic surgery, junior Faculty in academic institutions, as well as senior Faculty including Division and Department Heads. It is intended that at the completion of this Symposium, participants should have better knowledge regarding developing and teaching academic skills.

## 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 4.5 credit hours in Category 1 of the Physicians Recognition Award of the American Medical Association. Each physician should claim only those hours he/she spent in the continuing medical education program.

# Program

**12:00 p.m. LUNCH - Room 213**

**1:00 p.m. GETTING STARTED**

Edward D. Verrier, M.D.

University of Washington, Seattle, Washington

**1:30 p.m. CLINICAL RESEARCH**

Frederick L. Grover, M.D.

University of Colorado Health Sciences Center

and Denver VAMC, Denver, Colorado

**2:00 p.m. TEACHING SURGERY**

William A. Baumgartner, M.D.

Johns Hopkins Hospital, Baltimore, Maryland

**2:30 p.m. GRANT GETTING**

Irving L. Kron, M.D.

Univ. of Virginia Health Sciences Center,

Charlottesville, Virginia

**3:00 p.m. DEALING WITH INDUSTRY**

Larry R. Kaiser, M.D. Hospital of Univ. of Pennsylvania,

Philadelphia, Pennsylvania

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

**4:00 p.m. DEVELOPING ADMINISTRATIVE SKILLS**

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

Medical University of South Carolina,

Charleston, South Carolina

**4:30 p.m. SURGICAL INNOVATION**

Tirone E. David, M.D.

Toronto General Hospital,

Toronto, Ontario, Canada

**5:00 p.m. BECOMING A SURGICAL LEADER**

Lawrence H. Cohn, M.D. Brigham & Womens Hospital,

Boston, Massachusetts

**5:30 p.m. MENTORSHIP**

Floyd D. Loop, M.D.

Cleveland Clinic, Cleveland, Ohio

**6:00 pm - 7:30 p.m. RECEPTION - Room 213**

# CONGENITAL HEART DISEASE SYMPOSIUM

***SUNDAY, April 18, 1999 8:00 a.m. - 5:00 p.m.***

***Ernest N. Mortal Convention Center - Room 208/209***

## Objective

The 1999 AATS Congenital Heart Disease Symposium will address six specific topics, each carefully selected for its controversial nature or evolving status. This year the format of the program will be substantially expanded. Each topic will be specifically addressed under a different format to enhance the particular issues related to that topic. Two of the topics will be presented as "in-depth sessions" each consisting of three separate presentations. These presentations will address surgical technique, decision making and timing of management, and evolving understanding of pathophysiology. The two topics which will be presented using the "in-depth sessions" format will be:

1. Management of the right ventricular outflow tract following prior Tetralogy of Fallot repair, and

2. Management of anomalous coronary artery arising from the pulmonary artery.

Three other topics will be presented using a point- counter point format. In each of these sessions two carefully chosen experts will present opposite sides of the controversial issue, followed by an expanded moderator-led discussion period between the audience and the two speakers. The topics chosen for point- counter point presentations will include:

1. The use and efficacy of deep hypothermic circulatory arrest,

2. Management of critical neonatal aortic stenosis, and

3. Surgical management of complex congenital heart lesions which have two ventricles and two atrio- ventricular valves.

The final topic will be presented as a panel discussion. In this session each of five carefully chosen experts will present a five minute overview of the topic from their particular perspective and then an expanded moderator led panel discussion among the experts, with audience involvement, will follow. The subject of this session will be the use of ultrafiltration in Pediatric Cardiac Surgery. At the completion of the symposium participants should have an enhanced understanding of these evolving and controversial areas of congenital heart disease with respect to pathophysiology, management options, and timing of intervention.

### 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. Each physician should claim only those hours he/she spent in the continuing medical education program.

## Program

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

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

Frank L. Hanley, M.D., Chairman

**SESSION LATE REOPERATION ON THE RIGHT VENTIRCULAR OUTFLOW TRACT IN REPAIRED TETRALOGY OF FALLOT**

*Moderator: Edward L. Bove, M.D.*

**8:05 a.m. CRITERIA AND TIMING FOR RE-INTERVENTION**

William G.Williams, M.D.

Hospital for Sick Children and Toronto Congenital Cardiac Center for Adults, Univ. of Toronto,

Toronto, ON, Canada

**8:25 a.m. RIGHT VENTRICULAR PHYSIOLOGY IN REPAIRED TETRALOGY OF FALLOT**

Andrew Redington, M.D.

Royal Brompton & Harefield NHS Trust, London, United Kingdom

**8:45 a.m. SURGICAL TECHNIQUES FOR REVISING THE RIGHT VENTRICULAR OUTFLOW TRACT**

Edward L. Bove, M.D. University of Michigan Hospital,

Ann Arbor, Michigan

**9:05 a.m. DISCUSSION PERIOD**

**SESSION II MANAGEMENT OF ANOMALOUS CORONARY ARTERIES ARISING FROM PULMONARY ARTERY**

*Moderator: Hillel Laks, M.D.*

**9:30 a.m. THE PHYSIOLOGY OF STUNNED AND HIBERNATING MYOCARDIUM**

Shahbudin Rahimtoola, M.D. University of Southern California

Los Angeles, California

**9:50 a.m. SURGICAL OPTIONS FOR REPAIR AND TECHNIQUE OF MYOCARDIAL PROTECTION**

Hillel Laks, M.D.

University of California at Los Angeles, Los Angeles, California

**10:10 a.m. LONG TERM OUTCOME AND VENTRICULAR FUNCTION FOLLOWING REPAIR**

Tom R. Karl, M.D. Royal Childrens Hospital Melbourne, Australia

**10:30 a.m. DISCUSSION PERIOD**

**10:50 a.m. COFFEE BREAK**

**SESSION III POINT-COUNTERPOINT PANEL DEEP HYPOTHERMIC CIRCULATORY ARREST**

*Moderator: Marc R. de Leval, M.D.*

**11:15 a.m. Deep Hypothermic Circulatory Arrest Remains Useful as a Routine in Pediatric Heart Surgery**

Thomas L. Spray, M.D.

The Children's Hospital of Philadelphia, University

of Pennsylvania, Philadelphia, Pennsylvania

**11:30 a.m. Deep Hypothermic Circulatory Arrest Can and Should be Avoided Completely in Pediatric Heart Surgery**

V. Mohan Reddy, M.D.

University of California at San Francisco

San Francisco, California

**11:45 a.m. DISCUSSION AND DEBATE**

**12:15 p.m. LUNCHEON**

**SESSION IV POINT-COUNTERPOINT PANEL SURGICAL MANAGEMENT OF COMPLEX CONGENITAL HEART DISEASE WITH TWO VENTRICLES AND TWO AV VALVES**

*Moderator: V. Mohan Reddy, M.D.*

**1:30 p.m. The Fontan Operation is the Procedure of Choice**

Marc R. de Leval, M.D. Hospital for Sick Children London, United Kingdom

**1:45 p.m. Complex Two Ventricle Repair is the Procedure of Choice**

Michel N. Ilbawi, M.D.

University of Illinois at Chicago, Chicago, Illinois

**2:00 p.m. DISCUSSION AND DEBATE**

**SESSION V POINT-COUNTERPOINT PANEL MANAGEMENT OF CRITICAL NEONATAL AORTIC STENOSIS**

*Moderator: Thomas L Spray, M.D.*

**2:30 p.m. Balloon Valvotomy is the Procedure of Choice**

Robert H. Beekman, in, M.D. Children's Hospital Medical Center Cincinnati, Ohio

**2:45 p.m. The Ross Operation is the Procedure of Choice**

Frank L. Hanley, M.D.

University of California at San Francisco

San Francisco, California

**3:00 p.m. DISCUSSION AND DEBATE**

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

**SESSION VI ULTRAFILTRATION IN PEDIATRIC CARDIAC SURGERY PANEL DISCUSSION**

*Moderator: Frank L Hanley, M.D.*

Panelists:

Martin J. Elliott, M.D.

Hospital for Sick Children, London, United Kingdom

Hanni Hennein, M.D.

Loyola University Medical Center, Maywood, Illinois

Richard A. Jonas, M.D.

Children's Hospital, Boston, Massachusetts

LeNardo Thompson, M.D.

University of California at San Francisco,

San Francisco, California

Ross M. Ungerleider, M.D.

Duke University Medical Center,

Durham, North Carolina

**5:00 p.m. ADJOURN**

**5:00 p.m. WELCOMING RECEPTION-**

**EXHIBIT HALL**

# GENERAL THORACIC SURGERY

# SYMPOSIUM

**Sponsored in cooperation with The General**

**Thoracic Surgical Club**

***SUNDAY, April 18, 1999 8:00 a.m. - 5:30 p.m.***

***Ernest N. Mortal Convention Center - Room 210***

## Objective:

The intent of this course is to present the practicing thoracic surgeon with an array of exciting new technologies that will shape the care of thoracic patients as we begin the next millennium. Topics include advances in the application of minimally invasive surgery such as Nissen fundoplication, myotomy, photodynamic therapy and volume reduction, as well as multimodality therapies for lung cancer and esophageal disease. Great care has been taken to evaluate and select topics that appear to be those new therapies that will soon be in the therapeutic armamentarium of the practicing thoracic surgeon. It is hoped that by attending this course and gaining an exposure to these new technologies and therapies, thoracic surgeons will incorporate these approaches into their surgical practice in tandem with other medical specialists. The course is envisioned to give the thoracic surgical community as "heads up" regarding these emerging and promising approaches. These topics will be presented in an interactive format in order to maximize the educational experience of course registrants. An expanded syllabus will accompany the course as well.

The Faculty has been selected based on their leadership position in each of the areas to be discussed as well as their track record in incorporating these new approaches into their clinical practice. Interactions with faculty by course registrants will be maximized during this one-day course.

## 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 7.5 credit hours in Category 1 of the Physicians Recognition Award of the American Medical Association. Each physician should claim only those hours he/she spent in the continuing medical education program.

## Program

**8:00 a.m. INTRODUCTION: David J. Sugarbaker, M.D., Chairman**

**SESSION I MINIMALLY INVASIVE THORACIC SURGERY**
*Moderator: Paul F. Waters, M.D.*

**8:15 a.m. INTERACTIVE CASE PRESENTATION - ESOPHAGUS**

[†]John R. Roberts, M.D.

Vanderbilt University Hospital,

Nashville, Tennessee

**8:25 a.m. LAPROSCOPIC NISSEN FUNDOPLICATION**

Keith S. Naunheim, M.D.

St. Louis University Medical Center,

St. Louis, Missouri

**8:50 a.m. ENDOSCOPIC MYOTOMY IN ACHALASIA**

Tom R. DeMeester, M.D.

USC School of Medicine,

Los Angeles, California

**9:15 a.m. PHOTODYNAMIC THERAPY IN BARRETTS ESOPHAGUS**

‡Jacques Van Dam, M.D., Ph.D.

Brigham & Women's Hospital,

Boston, Massachusetts

**9:40 a.m. THORACOSCOPIC LVRS**

Robert J. Keenan, M.D.

University of Pittsburgh Medical Center,

Pittsburgh, Pennsylvania

**10:05 a.m. PANEL DISCUSSION**

**10:25 a.m. COFFEE BREAK**

**SESSION II THORACIC ONCOLOGY**

*Moderator: Thomas R. J. Todd, M.D.*

**11:00 a.m. INTERACTIVE CASE PRESENTATION - STAGE I LUNG CANCER**

Michael Liptay, M.D.

Evanston Hospital, Evanston, Illinois

**11:10 a.m. LIFE BRONCHOSCOPY**

*Stephen Lam, M.D.

University of British Columbia,

Vancouver, BC, Canada

**11:35 a.m. MOLECULAR SUB-STAGING IN STAGE I LUNG CANCER**

Malcolm M. DeCamp, Jr., M.D.

Cleveland Clinic Foundation, Cleveland, Ohio

**12:05 p.m. INDUCTION THERAPY IN STAGE I NSCLC**

Paul Bum, M.D.

University of Colorado Cancer Center,

Denver, Colorado

**12:35 p.m. PANEL DISCUSSION**

**1:00 p.m. LUNCHEON**

**SESSION III ESOPHAGEAL CANCER**

*Moderators: Harold C. Urschel, Jr., M.D. and*

*Scott J. Swanson, M.D.*

**2:00 p.m. INTERACTIVE CASE PRESENTATION - ESOPHAGEAL CANCER**

Kemp Kernstine, M.D., Ph.D.

The University of Iowa,

Iowa City, Iowa

**2:10 p.m. PRE-RESECTION STAGING IN ESOPHAGEAL CANCER**

Mark J. Krasna, M.D.

University of Maryland, Baltimore, Maryland

**2:35 p.m. INDUCTION THERAPY-WHO BENEFITS?**

Robert J. Mayer, M.D.

Dana-Farber Cancer Institute

Boston, Massachusetts

**3:05 p.m. VATS ESOPHAGECTOMY -DEAD OR ALIVE?**

David Harpole, M.D.

Duke University Medical Center,

Durham, North Carolina

**3:30 p.m. PANEL DISCUSSION**

**4:00 p.m. COFFEE BREAK**

**SESSION IV FUTURE THERAPIES IN THORACIC ONCOLOGY**

*Moderator: Larry R. Kaiser, M.D.*

**4:30 p.m. Stage III B LUNG CANCER METASTATIC TO THE PLEURA**

Joseph S. Friedberg, M.D.

University of Pennsylvania Medical Center,

Philadelphia, Pennsylvania

**4:40 p.m. GENE REPLACEMENT FOR LUNG CANCER**

[+]JackA.Roth,M.D.

M.D. Anderson Cancer Center, Houston, Texas

**5:05 p.m. ROBOTICS**

[#]Michael J. Mack, M.D.

Medical City Dallas Hospital,

Dallas, Texas

**5:30 p.m. QUESTIONS & ANSWERS**

**5:45 p.m. ADJOURN**

**7:00 p.m. WELCOMING RECEPTION IN EXHIBIT HALL**

[†] Author has a relationship with Bristol-Meyers Squibb

[‡] Author has a relationship with Sanofi Pharmaceuticals, Inc

***** Author has a relationship with LBFE-Lung-Xillix Technologies

[+] Author has a relationship with Intragen Therapeutics

[#] Author has a relationship with Computer Motion

# ADULT CARDIAC SURGERY

# SYMPOSIUM

***SUNDAY, APRIL 18, 1999 8:00 a.m. - 5:00 p.m.***

***Ernest N. Mortal Convention Center - Ballroom***

## Objective:

The 1999 Adult Cardiac Surgery Symposium will focus on new areas in cardiac surgery. It is divided into two sessions. The first session will pertain to both brain and spinal cord protection in various surgical settings including minimally invasive surgery, valvular heart surgery, coronary bypass surgery, and surgery on the thoracic aorta. The second session will relate to various types of cardiac surgical remodelling to treat congestive heart failure.

The Symposium is designed for the practicing cardiac surgeon. At the completion of the symposium, the participants should have knowledge of mechanisms of neurologic injury during cardiac surgery as well as treatment options to reduce these complications. They should also have a better understanding of the various techniques available to restore the left ventricle in patients with congestive heart failure.

## 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. Each physician should claim only those hours he/she spent in the continuing medical education program.

## Program

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

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

Irving L. Kron, M.D., Chairman

**SESSION I PREVENTION OF NEUROLOGIC INJURY DURING CARDIAC SURGERY**
**8:00 a.m. Mechanisms and Potential Solutions of Neurologic Injury**

John W. Hammon, M.D.

Wake Forest University

School of Medicine

Winston-Salem, North Carolina

**8:20 a.m. Avoiding Stroke by Avoiding Bypass**

Robert W. Emery, M.D.

Cardiac Surgical Associates

Minneapolis, Minnesota

**8:40 a.m. Avoiding Stroke During Port-Access CABG**

Mark A. Groh,M.D.

Ashville, North Carolina

**9:00 a.m. Avoiding Stroke During Minimally Invasive Valve Surgery**

†W. Randolph Chitwood, Jr., M.D.

East Carolina University

School of Medicine

Greenville, North Carolina

**9:20 a.m. The Case for Combined Carotid and Coronary Artery Surgery**

Gary W.Akins, M.D.

Massachusetts General Hospital

Boston, Massachusetts

**9:40 a.m. PANEL DISCUSSION**

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

**10:30 a.m. Pathophysiologic Mechanisms of Central Nervous System Injury Involved in Hypothermic Circulatory Arrest**

William A. Baumgartner, M.D.

Johns Hopkins Hospital

Baltimore, Maryland

**10:50 a.m. The Atherosclerotic Aorta - The Role of Echocardiography**

Jeffrey P. Gold, M.D.

The Albert Einstein College of Medicine

Bronx, New York

**11:10 a.m. Aortic Dissection**

John A. Elefteriades, M.D.

Yale University School of Medicine

New Haven, Connecticut

**11:30 a.m. PANEL DISCUSSION**

**12:15 a.m. LUNCHEON**

**SESSION II PREVENTION OF SPINAL CORD INJURY IN THORACIC AORTIC ANEURYSM**

**1:00 p.m. Mechanisms of Spinal Cord Injury**

John A. Kern, M.D.

University of Virginia

Charlottesville, Virginia

**1:20 p.m. Left Atrial to Femoral Bypass vs. Circulatory Arrest**

Joseph S. Coselli, M.D.

Baylor College of Medicine,

The Methodist Hospital

Houston, Texas

**1:40 p.m. Is Spinal Cord Injury a Preventable Complication?**

M. Arisan Ergin, M.D.

Mt. Sinai Medical Center

New York, New York

**2:00 p.m. Clamp and Sew**

Irving L. Kron, M.D.

University of Virginia

Health Sciences Center

Charlottesville, Virginia

**2:20 p.m. Central and Spinal Cord Neurological Complications with Endovascular Stent Grafts**

‡D. Craig Miller, M.D.

Stanford University Medical Center

Stanford, California

**2:40 p.m. PANEL DISCUSSION**

**3:00 p.m. COFFEE BREAK**

**SESSION III VENTRICULAR RESTORATION**

**3:20 p.m. Basic Concepts**

Gerald D. Buckberg, M.D.

UCLA Medical Center

Los Angeles, California

**3:40 p.m. The Endoventricular Circular Patch Plasty**

Vincent Dor, M.D.

Centre Cardio-Thoracique

Monaco, Cedex, Monaco

**4:00 p.m. The Batista Procedure**

Patrick M. McCarthy, M.D.

Cleveland Clinic

Cleveland, Ohio

**4:20 p.m. Mitral Repair in Cardiomyopathy**

Steven F. Bolling, M.D.

University of Michigan

Ann Arbor, Michigan

**4:40 p.m. PANEL DISCUSSION**

**5:00 p.m. ADJOURN**

**5:00 p.m.- 7:00 p.m.**
**WELCOMING RECEPTION IN EXHIBIT HALL**

† Author has a relationship with Embolex

‡ Author has a relationship with Meadox Medicals/Boston Scientific Corporation (Boston Scientific Vascular)

# 79TH ANNUAL MEETING

Ernest N. Morial Convention Center

New Orleans, Louisiana - April 18-21, 1999

# PROGRAM

## MONDAY, APRIL 19, 1999

**8:00 a.m. BUSINESS SESSION (Limited to Members)**

**8:15 a.m. SCIENTIFIC SESSION**

Ballroom, Ernest N. Morial Convention Center

*Moderators: Lawrence H. Cohn, M.D.*

*Tirone E. David, M.D.*

**1. Transmyocardial Revascularization Combined with Coronary Artery Bypass Grafting Versus Bypass Grafting Alone: A Prospective, Randomized Multi-Center Trial**

Keith B. Allen*, Anthony J .Delrossi, Fidel Realyvasquez*, Edward A. Lefrak,

Robert D. Dowling*, Thomas Pfeffer, Tommy Fudge, Mark Mostovych,

Douglas Schuch and Szaboic Szentpetery, Carl J .Shaar*, Indianapolis, IN,

Camden, Redding, CA, Falls Church, VA, Louisville, KY, Los Angeles ,CA,

Houma, LA, Jacksonville, FL, Sacramento, CA, Norfolk, VA

*Discussant: O. Howard Frazier, M.D., Houston,* TX

**OBJECTIVE:** To assess the safety and efficacy of transmyocardial revascularization (TMR) combined with coronary artery bypass grafting (C ABG) in patients not amenable to complete revascularization by C ABG alone.

**METHODS:** Two hundred sixty-six patients at 24 centers whose standard of care was CABG, but who had one or more ischemic areas not amenable to bypass grafting were prospectively randomized. Group A (n=133) received CABG of suitable vessels plus TMR to areas not graftable. Group B (n=133) received CABG alone with non-graftable ischemic areas left unrevascularized. Patient demographics including preoperative ejection fraction (EF), distribution and number of bypasses, number of endarterectomies, cardiopulmonary bypass time and predicted operative mortality of 8.6% and 7.7% (Parsonnet analysis) were similar for both groups.

**RESULTS:** Group A (CABG + TMR) operative mortality was 1.5% (2/133) and significantly ($p=0.02$) lower than Group B (CABG alone) at 7.5% (10/133). Group A operative mortality was significantly lower than predicted ($p<0.0001$). Major 30-day cardiac events (death or myocardial infarction) for Groups A and B were 3% (4/133) and 9% (12/133), ($p=0.05$). Multivariable analysis of treatment arm (CABG + TMR vs CABG alone), EF, age, prior CABG, diabetes and gender determined CABG alone as the sole predictor of operative mortality (odds ratio: 5.45, 95% CI 1.3-26.4; $p=0.03$). At three-months, mortality remained significantly lower for Group A [3% (3/117)] than for Group B [10% (11/115)] with 88% and 86% follow-up, ($p=0.03$).

**CONCLUSIONS:** This prospective, randomized multi-center trial demonstrates that TMR combined with CABG is safe and reduces operative mortality in patients not amenable to complete revascularization by CABG alone.

**By Invitation*

### 2. Eliminating the Cervical Esophagogastric Anastomotic Leak with a Side-to-Side Stapled Anastomosis

Mark B. Orringer and Mark D. Iannettoni*, Ann Arbor, Michigan

*Discussant: Victor F. Trastek, M.D., Rochester, MN*

While the acute postoperative complications associated with a cervical esophagogastric anastomosis (CEGA) are less than those with an intrathoracic esophageal anastomosis, the long-term sequelae of a CEGA leak have not proven to be as minor as initially reported. As many as

50% of CEGA leaks result in an anastomotic stricture, and the subsequent need for chronic dilatations negates the merits of an operation intended to restore comfortable swallowing.

**OBJECTIVE:** This study was undertaken to determine if construction of a side-to-side stapled CEGA after transhiatal esophagectomy (THE) could reliably eliminate the majority of anastomotic leaks.

**METHODS:** During the past 18 months, in 98 consecutive patients undergoing THE, a functional side-to-side CEGA was constructed with the endo-GIA stapler applied directly through the cervical wound. This side-to-side stapled anastomosis (SSSA) has 3 rows of staples. Early postoperative anastomotic morbidity, subsequent need for anastomotic dilatations, and patient satisfaction with swallowing were evaluated.

**RESULTS:** Prior to the use of the SSSA, regardless of the manual suture technique used to construct the CEGA, the anastomotic leak rate in over 1,000 patients undergoing THE varied from 10-15%. Among the 98 patients undergoing THE and a SSSA, there were 2 anastomotic leaks (2%). This dramatically lowered leak-rate has contributed to reduction in the currernt average length of stay after a THE to 7 days and has provided more comfortable swallowing, less need for subsequent esophageal dilatations and greater patient satisfaction.

**CONCLUSIONS:** Construction of the CEGA using a SSSA virtually eliminates anastomotic leaks and reduces the long-term morbidity of anastomotic stricture. The SSSA is a major technical advance in the progression of refinements of THE and a CEGA.

*By *Invitation*

### 3. Effect of Repair Strategy on Cost and Outcome for Infants with Hypoplastic Left Heart Syndrome: Palliation versus Heart Transplant

†Ko Bando , Debby Murphy*, Theresa Flaspohler*, Mark W. Turrentine*, Thomas G. Sharp*, Randall L. Caldwell*, Robert K. Darragh*, Thomas X. Aufiero* and John W. Brown, Indianapolis, Indiana and Osaka, Japan

*Discussant: Leonard L. Bailey, M.D., Loma Linda, CA*

**OBJECTIVE:** Treatment of hypoplastc left heart syndrome (HLHS) remains expensive and controversial. Since 1989 we have performed either staged Norwood operations (Nw) or heart transplantation (Tx) for HLHS. This study sought to assess the effect of repair strategy on cost and outcome for HLHS.

**METHODS:** Preop, operative and follow-up costs (adjusted to 1998 US dollars) were obtained for 90 patients (pts) with HLHS from January 1989 to June 1998. Records were reviewed to determine late outcomes. Functional status was assessed by questionnaire.

**RESULTS:** Three groups were studied. **Tx group (n=18):** 12 before 1993. **Nw Group1 (n=24):** Before 1993, Nw was performed on 24 pts (6 cross-over from Tx). **Nw Group2 (n=48):** Since 1994, 48 pts had Nw (4 cross-over from Tx).

| | Tx Group | Nw Group 1 | Nw Group 2 | P value |
|---|---|---|---|---|
| **Preop hospital days** | 27.4±31.6 | 6.9±4.7 | 3.2±.8.5 | †p<0.001 |
| **Pre-op cost *** | 94.6±72.1 | 32.0±14.7 | 10.4±8.0 | †p<0.001 |

| | | | | |
|---|---|---|---|---|
| **5yr total cost*** | 364.1±149.8Œ | 191.0±39.1' | 138.3±24.1' | †p<0.001 |
| **5yr actuarial survival (%)** | 72.7±0.2 | 37.5±0.2‡ | 71.1±0.2 | ‡p=0.002 |
| **NYHA class (mean)** | 1.2 | 1.2 | 1.2 | NS |

** in $1,000. Œ includes Tx and follow up. ´ includes stages 1, 2, 3 and follow-up.*

*†ANOVA, ‡Nw Group1 vs Tx or Nw Group 2 by log rank*

**CONCLUSIONS:** Both Tx and Nw offer good late functional outcomes, but the total costs for Tx exceed Nw significantly. This plus the fact that late survival has significantly improved for Nw since 1994, make it the procedure of choice at our center.

*†*1992-92 *AATS Graham fellow*

**By Invitation*

### 4. Glucose-Insulin-Potassium Solutions Improve Clinical Outcomes in Diabetic Patients Undergoing Coronary Bypass Grafting

Harold L. Lazar, Stuart Chipkin*, George Philippides*, Yusheng Bao* and Carl Apstein*, Boston, Massachusetts

*Discussant: Richard D. Weisel, M.D., FRCS, Toronto, Ontario, Canada*

**OBJECTIVE:** Patients with diabetes mellitus have significantly higher morbidity following Coronary Artery Bypass Grafting (CABG). Our previous studies have shown that Glucose-Insulin-Potassium (GIK) solutions improve clinical outcomes in non-diabetic patients following urgent CABG. This study was therefore undertaken to determine whether substrate enhancement with GIK would improve myocardial performance and limit morbidity following CABG in diabetic patients.

**METHODS:** Forty consecutive CABG patients with medically treated diabetes mellitus (tablets or insulin) were prospectively randomized into a GIK group: N=20; (500 ml D5W+80 units regular insulin + 40 mEq KCL at 30 ml/ hr) and a NO-GIK group: N=20; (D5W at 30 ml/hr). GIK was instituted upon anesthetic induction and continued for 12 hours post-op.

**RESULTS:** The two groups did not differ statistically in gender, number of vessels bypassed, angina class, ejection fraction, pre-op cardiac index, preop serum glucose, crossclamp or cardiopulmonary bypass times. There were no mortalities in either group. Values on table represent the Mean ± standard error.

| | **GIK(N=20)** | **NO GIK(N=20)** | **P Value** |
|---|---|---|---|
| Postop Weight Gain (Ib) | 5.8±.8 | 13.8±1.4 | <0.0001 |
| Cardiac Index - 12 hours postop (L/Min/M2) | 2.86±0.11 | 2.19±.09 | <0.0001 |
| Serum Glucose - 12 hours postop (mg/ml) | 135.8±8.1 | 238.1±9.7 | <0.0001 |
| Inotropic Score | 0.40±.15 | 1.25±.32 | <0.0001 |
| (0=no inotropes) | | | |
| Time on Ventilator (hours) | 8.35±.58 | 13.45±1.63 | <0.0001 |
| Atrial Fibrillation (%) | 3(15%) | 12(60%) | <0.0001 |

| | | | |
|---|---|---|---|
| Postop Hospital Stay (days) | 6.70±.34 | 10.20±1.48 | <0.0001 |

**CONCLUSIONS:** Substrate enhancement with GIK in diabetic patients improves myocardial performance, limits weight gain, reduces inotropic dependency and the need for ventilatory support, decreases the incidence of atrial fibrillation and results in faster recovery following CABG surgery.

**9:35 a.m. John Alexander Research Scholar Presentation**

Richard Norris Pierson, III, Nashville, TN

**9:40 a.m. Evarts A. Graham Memorial Traveling Fellowship Presentation**
Christian Kreutzer, Buenos Aires, Argentina

**9:45 a.m. INTERMISSION - VISIT EXHIBITS**

**By Invitation*

**10:30 a.m. SCIENTIFIC SESSION**

Ballroom, Ernest N. Morial Convention Center

*Moderators: Delos M. Cosgrove, M.D.*

*Andrew S. Wechsler M.D.*

**5. Effects of Lung Volume Reduction Surgery on Pulmonary Function and Survival in Patients with End-Stage Emphysema: A Four Year Update**

Michael Argenziano*, Byron Thomashow*, Patricia A. Jellen*, Lyall A. Gorenstein*, Eric A. Rose, Kenneth M. Steinglass*, Alan D. Weinberg* and Mark E. Ginsburg*, New York, New York
*Discussant: David J. Sugarbaker, M.D., Boston, MA*

**OBJECTIVE(s):** The short-term efficacy of lung volume reduction surgery (LVRS) in the palliation of end-stage emphysema has been reported. It is not clear, however, whether these effects are long-lived or whether LVRS prolongs life expectancy. In medically managed patients with FEV1 < 30% of predicted, 3-year survival has been estimated at 40%.

**METHODS:** Over 4 years, LVRS was performed in 144 patients with severe emphysema. Mean age was 63 ± 8 years. Preoperative PFT revealed FEV1 of 587 ± 229 cc (23 ± 8% of predicted) and dyspnea index (DI) of 3.9 ± 0.9 (range of 0-5). Operation was unilateral (n = 54) or bilateral (n = 90).

**RESULTS:** There were 9 perioperative deaths (6%) and 25 late deaths (2 to 34 months post-operatively). Mean followup was 29 months (range 7 to 47 months). Six months of followup was available in 133 patients, 12 months in 104 patients, 24 months in 73 patients, and 36 months in 31 patients. FEV1 and DI were improved significantly 12 and 24 months postoperatively (table), and actuarial survival at 12, 24, 36, and 42 months was 85, 74, 73, and 73%, respectively.

| | **FEV1 (cc)** | **FEV1 (% pred)** | **Dyspnea Index** |
|---|---|---|---|
| preop | 587±229 | 23±8 | 3.9±0.9 |
| 12 months | 778±439* | 30±14** | 1.9±1.2*** |
| 24 months | 784±384** | 36±13* | 2.0±1.9*** |

*p<0.05; ***p<0.01;*****p<0.0001

**CONCLUSIONS:** LVRS improves assessments of dyspnea (DI) and objective measures of pulmonary function (FEV1), and these benefits are preserved for at least two years postoperatively. Actuarial survival over 42 months after LVRS appears to be superior to reported survival for patients with end-stage emphysema managed with medical therapy alone.

**By Invitation*

### 6. Dilation of the Pulmonary Autograft Following the Ross Procedure

Tirone E. David, Mauro P.I. De Sa*, Ahmed Omran*, Brian Sonnenberg*, Susan Armstrong*, Joan Ivanov* and Gary Webb*, Toronto, Ontario, Canada

*Discussant: Nicholas T. Kouchoukos, M.D., St Louis, MO*

**OBJECTIVE:** Dilation of the pulmonary autograft (PA) following the Ross procedure is being recognized with increasing frequency. This study was undertaken to determine the magnitude of this problem and its predictive factors.

**METHODS:** From 1990 to 1997, 113 patients underwent the Ross procedure. The diameters of the aortic annulus and sinotubular junction were reduced to those of the PA whenever there was discrepancy in size. There was one operative death, one early failure due to acute dilation of the PA, and one late non-cardiac death. The remaining 110 patients have had annual Doppler echocardiography to assess valve function and to measure the internal diameter of the PA sinuses. The mean follow-up was 44 ± 11 months. A stepwise logistic regression analysis was used to identify predictors of dilation of the PA.

**RESULTS:** No patient had more than mild aortic insufficiency (AI) early post-operatively. At the latest echocardiographic study, 62% had none or trivial AI, 33% had mild AI, and 5% had moderate AI. The diameter of the PA sinuses increased from 29.7 ± 3.7 mm (21 to 38 mm) to 33.7 ± 4.3 mm (range 26 to 46 mm), p=0.001. The PA diameter became abnormal (>39 mm) in 17 patients. PA dilation was an independent predictor of postoperative AI. Bicuspid aortic valve with large aortic annulus (>27 mm) was an independent predictor of PA dilation (odds ratio 12.5). All cases of dilation occurred in patients who had aortic root replacement with a free standing PA (p=0.01).

**CONCLUSIONS:** Patients with bicuspid aortic valve and dilated aortic annulus are at a high risk of PA dilation after the Ross procedure. Aortic root inclusion and subcoronary implantation of the PA may prevent dilation. Late AI is caused by dilation of the PA.

**1:15 a.m. PRESIDENTIAL ADDRESS**

**What the Cardiothoracic Surgeon of the 21st Century Ought To Be**

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

**ADJOURN FOR LUNCH - VISIT EXHIBITS**

**By Invitation*

# MONDAY AFTERNOON, APRIL 19, 1999

## 1:30 p.m. SIMULTANEOUS SCIENTIFIC SESSION A -ADULT CARDIAC SURGERY

Ballroom, Ernest N. Morial Convention Center

*Moderators: Floyd D. Loop, M.D.*

*Eric A. Rose, M.D.*

### 7. Aortic Rupture after Aortic Valve Surgery in Women

Monica L. McDonald*, Nicholas G. Smedira*, Bruce W. Lytle and Delos M. Cosgrove, Cleveland, Ohio
*Discussant: Robert J. Rizzo, M.D., Boston, MA*

**OBJECTIVE:** To examine mortality in women after surgery for aortic regurgitarion (AR) and the contribution of aortic rupture to that mortality.

**METHODS:** 109 women underwent aortic valve replacement (AYR, n=92) or repair (n=17) for pure AR, 1985-96. In 59, concomitant aortic surgery (AOS) was performed by root replacement (n=31) or interposition graft (n=28). Of 50 undergoing isolated procedures, 31 had aortas >3.5 cm. The prevalence of larger aortas increased with age (P=.02). Mean follow-up was 5.7±2.6 yrs.

**RESULTS:** At 5 and 10 yrs, survival was 78% and 44%. 13 died of aortic rupture or sudden death and 3 underwent emergent operation for aortic rupture, 16 late aortic events (AE). Freedom from AE was 86% and 75% at 5 and 10 yrs. Risk factors for AE were older age (P=.07) and increasing ascending aorta diameter (P=.03) in women who had not undergone AOS. Rupture location was ascending aorta in 71% without AOS, and descending aorta in 56% with interposition grafts.

**CONCLUSIONS:** Late mortality following surgery for AR in women is due in part to aortic rupture. The figure depicts the risk of rupture according to AA size normalized to BSA and age, offering guidance for management of high-risk patients.

**By Invitation*

### 8. Aortic Valve Replacement: Is Valve Size Important?

Benjamin Medalion*, Eugene H. Blackstone, Bruce W. Lytle, Delos M. Cosgrove, and Jennifer White*, Cleveland, Ohio

*Discussant: Friedrich W Mohr, Leipzig, Germany*

**OBJECTIVE:** To determine if patient-valve size mismatch affects long-term survival.

**METHODS:** We followed 892 patients (pts) up to 20 years (mean 5±3.9 years) after primary, isolated aortic valve replacement who had received either 1) a mechanical prosthesis (St. Jude, SJM, n=346); 2) a bovine pericardial valve (Carpentier-Edwards, CE, n=463); or 3) a cryopreserved allograft (ALLOG, n=83). Mean BSA was 1.86±0.23, range 1.15-2.57$m^2$. 19mm prostheses were used in 19%. Multivariable analysis was focused on the Z-value for valve size, defined as the

patient-specific number of standard deviations the internal prosthesis diameter departs from predicted normal aortic annulus size based on that individual's body surface area.

**RESULTS:** Internal Z-value ranged from -4.4 to +6.8, with 25% <-2. SJM valves had the smallest Z-value (-2.2±1.0), CE valves -0.5+0.99, and ALLOG the largest (0.2±1.4). Survival was 96%, 86%, 69%, and 49% at 1, 5, 10, and 15 years. Risk factors for death included older age, emergency surgery, endocarditis, coronary artery disease, and comorbidities, but no effect of valve size Z-value for any valve type (P>.2), in early, constant, and late hazard phases.

**CONCLUSIONS:** Survival after aortic valve replacement appears not to be adversely affected by mismatch between prosthesis valve size and body size down to at least -4 SDs below normal valve size.

**By Invitation*

### 9. Minimally-Invasive Reoperative Aortic Valve Replacement Reduces Blood Loss and Transfusion Requirements

John G. Byrne*, Sary F. Aranki, Gregory S. Couper* and Lawrence H. Cohn, Boston, Massachusetts

*Discussant: William A. Gay, Jr., M.D., St Louis, MO*

**OBJECTIVE:** We developed techniques for minimally-invasive (min-inv) *reoperative* (reop) aortic valve replacement (AVR), using an upper hemi-*re*-sternotomy, and compared the results to reop AVR using conventional (conv) full re-sternotomy.

**METHODS:** We retrospectively analysed 19 conv & 20 min-inv isolated elective reop AVRs, performed between 11/96-9/98.

**RESULTS:** Age, sex, FC, EF, previous operations, and Aprotinin® use were not different between groups. Multivariate analysis determined that conv approach predicted greater blood loss, transfusion needs and longer operative (op) duration (Table).

| Table (multivariate analysis) | Conv, n=19 | Min-inv, n=20 | |
|---|---|---|---|
| **Deaths/Valve Morbidity** | 0/19 (0%) | 0/20(0%) | |
| **Injury to Patent CABGs** | 1/19 (5%) | 0/20 (0%) | |
| **Bypass Duration (min)** | 145±53 | 146±52 | |
| **Aortic Clamping Duration (min)** | | | |
| **(3 full root replacements in each group)** | 92±32 | 93±48 | |
| **Total Operative Duration (hours)** | 5.8±1.2 | 5.4±1.2* | *p=0.15 |
| **Blood Loss (ml) 1st 24 hours** | 1071±629 | 472±362† | †p=0.01 |
| **Transfusions (units) PRBC** | 5.2±2.4 | 3.013.0‡ | ‡p=0.10 |
| **Hospital Charges ($)** | 63K±15K | 55K±20K | |
| **Length of Stay (days)** | 7.9±4.9 | 6.9±2.6 | |

**CONCLUSIONS:** Min-inv reop AVR avoids unnecessary lower mediastinal dissection, thereby reducing (1) blood loss, (2) transfusion needs and (3) total op duration. Patent CABGs may be less

prone to injury. These beneficial effects, accomplished without compromising the valve procedure, make min-inv upper hemi-re-sternotomy superior to conv full re-sternotomy for reop AVR.

**By Invitation*

### 10. Mitral Valve Repair and Replacement for Rheumatic Disease

Terrence M. Yau*, Susan Armstrong*, Joan Ivanov*, Yasser A. Farag El-ghoneimi* and Tirone E. David, Toronto, Ontario, Canada

*Discussant: Alain F. Carpentier, M.D., Paris, France*

**OBJECTIVE:** Repair of rheumatic mitral valves (MVs) has generally been associated with poor long-term results. We undertook this study to define the early and late results of surgery for rheumatic MVs.

**METHODS:** From 1978 to 1995, 575 patients (pts) underwent surgery for rheumatic MVs. Demographics and operative data were recorded prospectively. Followup was 97.7% complete (mean 67.5±45.7 mos). Survival and morbidity were evaluated univariately by Kaplan-Meier analysis and multivariately by Cox regression.

**RESULTS:** Mean age was 53.7±14.2 years, 80.5% of pts were female, 54.7% had congestive failure, 23.6% were undergoing redo MV surgery, and 9.7% also underwent coronary bypass. Mitral stenosis was present in 52.5%, regurgitation in 15.4%, and both in 32.1%. Valve repair (REP) was performed in 24.4%, 27.5% had replacement with a bioprosthesis (BIO) and 48.0% had a mechanical valve (MECH).

Operative mortality was 4.1%. Ten-year survival was: REP 88.2±0.04%, BIO 70.2±0.04%, MECH 73.4±0.06% (p=0.0002). Mortality was predicted by age, NYHA class, coronary disease, and reoperation.

Ten-year freedom from reoperation was: REP 72.0±0.05%, BIO 69.0±0.05%, MECH 95.3±0.02% (p=0.005). Type of prosthesis predicted reoperation after replacement. 23 pts underwent reoperation after initial repair, with no operative deaths.

Ten-year freedom from thromboembolic complications was: REP 92.5±0.02%, BIO 93.3±0.02%, MECH 71.6±0.07% (p<0.0001).

**CONCLUSIONS:** Mechanical valves minimize reoperation but limit survival and are prone to thromboembolism. Bioprostheses and repaired valves both have good freedom from thromboembolism and poor freedom from reoperation, but patients selected for valve repair had better late survival.

**Patients with rheumatic MVs should have repairs whenever technically feasible, accepting a risk of late reoperation, to maximize survival and reduce morbidity.**

**By Invitation*

**11. Influence of Size 19mm Aortic Valve Replacement on Survival**

[†]David H. Adams*, Raymond H. Chen*, Sary F. Aranki, Elizabeth N. Allied* and [§]Lawrence H. Cohn, Boston, Massachusetts

*Discussant: Donald B. Doty, M.D., Salt Lake City, UT*

**OBJECTIVE:** Does valve size influence early and late clinical outcomes in patients ‰¥ 70 years of age undergoing aortic valve replacement?

**METHODS:** Between 12/91 and 7/98 408 patients ‰¥ 70 years of age (median age 77, range 70-97,50% male) underwent isolated AVR or AVR combined with CABG, utilizing either Carpentier-Edwards bovine pericardium valves (n=300) or St. Jude mechanical valves (n=108). Univariate methods were used to examine potential confounders (age ‰¥ 75, sex distribution, body mass index, CABG, re-operation, hypertension, myocardial infarction, renal failure, COPD, stroke, diabetes mellitus, congestive heart failure, aortic stenosis, prolonged ischemia/ bypass times) among patients receiving a small (19mm) or a large (‰¥ 21mm) aortic valve. Multivariate logistic regression methods were used to predict operative mortality and multivariate proportional hazards regression methods were used to predict late death (median follow-up 22 months) related to 19mm valve replacement after controlling for potential confounders.

**RESULTS:** Operative mortality was 16% (18/111) for size 19mm valve replacement, 5.8% (8/138) for 21mm valve replacement, and 2.5% (4/159) for ‰¥ 23mm valve replacement (p ‰¤.0005). The unadjusted odds ratio for operative death for 19mm vs. ‰¥ 21mm valves was 4.6 (95% CI:2.1,9.9; p ‰¤.0005). In the final multivariate model adjusted for potential confounders, small valve size remained a significant predictor of operative death (OR 4.2, 95% CI:1.8,9.8; p=.001). 19mm valve usage, however, did not predict late death in either the univariate analysis (HR 0.96,95% CI:0.5,1.9;p=.92) or the multivariate analysis (HR 0.6, 95% CI:0.3,1.4; p=.25).

**CONCLUSIONS:** Implantation of a 19mm aortic valve in a patient ‰¥ 70 years of age appears to be an independejnt risk factor for operative mortality, but does not impact on late death.

**3:15 p.m. INTERMISSION - VISIT EXHIBITS**

*†1992-94 AATS Research Scholar*

*[§]Authors have a relationship with St. Jude Medical & Medtronic*

**By Invitation*

**4:00 p.m. SIMULTANEOUS SCIENTIFIC SESSION A -ADULT CARDIAC SURGERY**

Ballroom, Ernest N. Morial Convention Center

*Moderators: Floyd D. Loop, M.D.*

*Eric A. Rose, M.D.*

**12. Hospital Readmission following Cardiac Surgery: Prevalence, Patterns and Predisposing Factors**

Richard S. D'Agostino*, Jerilynn P. Jacobson*, Lars G. Svensson*, Christina Williamson* and David M. Shahian, Burlington, Massachusetts

*Discussant: Timothy J. Gardner, M.D., Philadelphia, PA*

**OBJECTIVE:** In this study, we examine the incidence of readmission within the 30-day period following hospital discharge after cardiac surgery, the reasons for readmission and pre-discharge factors that predispose to readmission.

**METHODS:** Pre, intra and postoperative clinical data were prosepctively collected for 1027 patients undergoing coronary bypass, valve replacement or combined bypass/valve surgery between 1/1/96 and 7/30/97. Thirty-day data was collected at the time of office visit or by telephone and/or written survey.

**RESULTS:** 163 (15.8%) patients were readmitted to a hospital within the 30-day period following discharge. The clinical problems that prompted read-mission were as follows: dysrhythmia=27 (16.5%), congestive heart failure/ dyspnea/pulmonary edema/pleural effusions=34 (20.8%), chest pain/rule out myocardial infarction=17 (10.4%), wound infection/problem=14 (8.5%), gastrointestinal problem=7 (4.3%), stroke/TIA=2 (1.25%), pneumonia or other non-wound infection=14 (8.5%), deep venous thrombosis or pulmonary embolus=8 (4.9%), peripheral vascular problem=7 (4.2%), anemia/hypotension/transfusion=12 (7.4%) and miscellaneous problems=22 (13.5%). Stepwise logistic regression analysis (Hosemer and Lemeshow Goodness-of-Fit chi square= -5.85; df=8; p=0.6631) showed that advancing age (p=0.04), increasing body mass index (p=0.0032), combined valve/bypass surgery (p=0.0003), longer cardiopulmonary bypass (p=0.0013) and crossclamp time (p=0.000), and longer postoperative length of stay (p=0.005) predispose to readmission.

**CONCLUSIONS:** Readmission within 30 days of discharge after cardiac surgery is a relatively common event. Some patients appear to be at greater risk for readmission and future efforts should focus on risk reduction strategies for these patients. Interestingly, shorter hospital stays do not appear to impact adversely on the risk of readmission.

**By Invitation*

### 13. Mid-term Follow-up of Patients who had LVAD Removal following Cardiac Recovery in End-stage Dilated Cardiomyopathy

Roland Hetzer, Johannes H. Mueller*, Yu-guo Weng*, Thorsten Drews*, Matthias Loebe* and Gert Wallukat*, Berlin, Germany

***Discussant: Eric A. Rose, M.D., New York, NY***

**OBJECTIVE:** Cardiac recovery in end-stage dilated cardiomyopathy (DCM) was demonstrated after temporary LVAD support. However, the durability of such recovery has remained a matter of dispute. We report on our patients who had removal of LVAD up to 3-1/2 years ago.

**METHODS:** Since 3/1995, 21 patients with end-stage DCM who had been supported by LVAD (19 Novacor, 2 TCI) for periods of between 1 and 26 months (mean 6 months)had their LVAD removed following complete or near complete cardiac recovery as documented by echocardiography (LVEF, LVIDd) at time of pump-off trials.

**RESULTS:** Six patients (group A) suffered recurrence of cardiac failure after between 4 and 24 months. Transplantation was successfully performed in five, one died on the waiting list, four patients died from reasons unrelated to cardiac failure after between 3 days and 29 months. Stable

cardiac recovery has been enjoyed by 11 patients (group B) for between 6 and 41 months (mean 20±6). At time of LVAD implant, there were no differences between groups A and B as to age, duration of heart failure, invasive hemodynamic values, LVEF, LVIDd, amount of apoptosis, TNF-u., b-receptor density, and level of autoanti-bodies. However, the degree of cardiac recovery at the time of pump explan-tation was significantly better in group B (A vs. B; LVEF 40±5 % vs. 48±4 % $p < 0.01$; mean LVIDd 62±6 mm vs. 53±5 mm, $p<0.01$).

**CONCLUSIONS:** In a selective group of patients with end-stage DCM complete and lasting cardiac recovery may be achieved after ventricular unloading with LVAD. At present, no reliable pre-LVAD-implant indices have been recognized that would allow prediction of the degree of recovery after implant. However, lasting cardiac recovery seems to be related to complete normalization of cardiac function during the unloading period.

**By Invitation*

## 14. Choice of Non-transplant Cardiac Surgical Procedures for End-stage Cardiomyopathy

Hisayoshi Suma, Tadashi Isomura*, Taiko Horii*, Toru Sato* and Norio Kikuchi*, Kamakura, Kanagawa, Japan

*Discussant: Steven F. Bolling, M.D., Ann Arbor, MI*

**OBJECTIVE:** To treat end-stage cardiomyopathy, endoventricular circular patch plasty (EVCPP), partial left ventriculectomy (PLV) and solo-valvular reconstruction (VR) have been proposed. To find a proper choice of those non-transplant cardiac procedures, we evaluated our 2 years results of those procedures and introduce echo-guided volume reduction test.

**METHODS:** From December 1996 to September 1998, 68 patients with end-stage cardiomyopathy (27 ischemic and 41 non-ischemic) underwent EVCPP±CABG/VR, PLV1VR or Solo±VR. Preoperative characteristics is shown in Table. For ischemic cardiomyopathy, EVCPP(Dor procedure) was used for all patients. In non-ischemic group, Group I indicates early 24 patients treated with PLV±VR (mostly replacement) and Group II includes recent 17 patients treated with different types of procedures depends on intraoperative volume reduction test which observes changes of LV size, wall motion and thickness before and after CPB by echography. When the LV became smaller and the wall increased thickness entirely with LV decompression, solo-VR (mostly annuloplasty) was chosen and if akinetic thin wall remained, PLV or EVCPP was added according to the site of akinesis.

**RESULTS:** Hospital mortality was acceptably low in elective operations in each group whereas the risk was high inongoing shock patients needed emergency operation (Table). With colume reduction test, mortaily reduced from 12% to 0% in non-ischemic group.

| **Etiology** | **Ischemic** | **Non-ischemic Group I** | **Non-ischemic Group II** |
|---|---|---|---|
| **No. of Patients** | 27 | 24 | 17 |
| **Male/Female (Age)** | 21/6(63±7) | 19/5(48±15) | 15/2(51±13) |
| **NYHA class (Pre-»Post)** | 3.3†'1.3 | 3.6†'1.7 | 3.3†'1.2 |
| **EF (%) (Pre-»Post)** | 23†'38 | 18†'31 | 23†'38 |
| **EDVI (mi/m$^2$) (Pre->Post** | 160†'103 | 203†'99 | 162†'119 |

| | | | |
|---|---|---|---|
| **ESVI (mi/m$^2$) (Pre-»Post)** | 118†'72 | 164†'70 | 121†'82 |
| **Hospital Death** | | | |
| **elective op.** | 1/22(5%) | 2/17(12%) | 0/15(0%) |
| **emergency op.** | 3/5(60%) | 6/7(86%) | 1/2(50%) |
| **Late Death** | 3/27(11%) | 4/24(17%) | 1/17(6%) |
| **IABP/LVAD** | 0/0 | 6/0 | 0/1 |

**CONCLUSIONS:** EVCPP is favorable for ischemic cardiomyopathy because septal exclusion is effective. Volume redution test is useful to choose an optimal procedure for non-ischemic cardiomyopathy to avoid excessive LV excision.

**By Invitation*

### 15. Passive Ventricular Constraint With the Acorn Prosthetic Jacket Prevents Progressive Left Ventricular Remodeling and Functional Mitral Regurgitation in Dogs With Moderate Heart Failure

Pervaiz A. Chaudhry*, Gaetano Paone*, Victor G. Sharov*, Takayuki Mishima*, James Hawkins*, §Clif Alferness* and §Hani N. Sabbah*, Detroit, Michigan

*Sponsored By: Norman A. Sttverman, Detroit, Michigan*

*Discussant: Gerald D. Buckberg, M.D., Los Angeles, CA*

**OBJECTIVE:** The purpose of this study was to determine if passive mechanical constraint of the cardiac ventricles with a surgically placed prosthetic jacket prevents progressive left ventricular (LV) remodeling and attenuates functional mitral regurgitation (MR) in dogs with moderate heart failure (HP).

**METHODS:** HF (LV ejection fraction 30-40%) was produced in 12 dogs by multiple sequential intracoronary microembolizations. Six dogs underwent mid-sternotomy and pericardiotomy followed by placement of the prosthetic jacket (a preformed-knitted polyester mesh, Acorn Cardiovascular, Inc.) snugly around the ventricles and anchored at the AV groove. Six untreated HF dogs served as controls (CON). LV end-diastolic (EDV) and end-systolic (ESV) volumes and the presence and severity of functional MR were determined angiographically before (PRE) and 3 months after (POST) treatment. Cardiomyocyte cross-sectional area (CCSA), a measure of myocyte hypertrophy, was assessed histomorphometrically.

**RESULTS:** In CON dogs, EDV increased 15±5 ml between PRE and POST but decreased 7±1 ml in dogs treated with the Acorn prosthetic jacket (P=0.002). Similarly, ESV increased 17±5 ml in CON dogs but decreased 9±1 ml in dogs treated with the jacket (P=0.001). In CON dogs, 4 of 6 had 1+ to 2+ MR that persisted and/or increased after 3 months of follow-up. In contrast, 4 of 6 surgically treated dogs had 1+ to 2+ MR that was completely abolished after 3 months. In dogs treated with the prosthetic jacket, the average CCSA area was smaller than in CON (791+51 vs. 987±37 mm2, p=0.011).

**CONCLUSIONS:** Passive ventricular constraint with the Acorn prosthetic jacket prevents progressive LV remodeling and abolishes functional MR in dogs with moderate HF.

*§Authors have a relationship with Acor Cardiovascular, Inc.*

**By Invitation*

**1:45 p.m. SIMULTANEOUS SCIENTIFIC SESSION B -**

**GENERAL THORACIC SURGERY**

Room 211-213, Ernest N. Morial Convention Center

*Moderators: Thomas R. J. Todd, M.D.*

*Steven J. Mentzer, M.D.*

**16. Secondary Pulmonary Hypertension Does Not Adversely Affect Outcome After Single Lung Transplant.**

Scott Huerd*, Frederick L. Grover, James R. Mault*, Max B. Mitchell*, David N. Campbell, Fernando Torres*, Paul Chetham*, Tony Hodges* and Martin Zamora*, Denver, Colorado

*Discussant: Vaughn A. Starnes, M.D., Los Angeles, CA*

**OBJECTIVE(s):** Elevated pulmonary artery pressures (PAP) have been associated with poor outcomes in patients undergoing SLT. Some groups advocate double lung transplant or the routine use of cardiopulmonary bypass (CBP) for SLT in this population. These recommendations remain controversial. The goal of our study was to determine whether secondary PHTN requires CPB and adversely affects outcomes following SLT.

**METHODS:** We retrospectively reviewed 76 consecutive SLT performed from 1992-98. Patients with primary PHTN and Eisenmenger's syndrome were excluded. Recipients were stratified according to preoperative PAP (PA systolic pressure > 40mm Hg); 58 had low PAP and 18 high PAP (13 with COPD or A1AT deficiency, 5 with idiopathic pulmonary fibrosis).

**RESULTS:** No patient in the high PAP group required CPB or inhaled nitric oxide (iNO) intraoperatively. Postoperatively, no significant differences were seen in length of stay (LOS, 14.6 ±.9 vs.ll.9±1.5 days), 30 day survival (95 vs 89%) or lung injury as measured by CXR score or PaO2/FiO2 or the need for iNO (5(8.6%) in the low and 2(11.1%) in the high group) in the low versus high groups respectively (p=NS). There were also no differences in FEV1 or six minute walk at 1 and 2 yrs or in the incidence of acute rejection, infection or BOS>grade 2. Survival at 1, 2, and 4 yrs posttransplant was 85,79, and 71% in the low group and 78, 78, and 67% in the high group (p=NS).

**CONCLUSIONS:** Our data demonstrate that secondary PHTN is not associatied with significantly increased morbidity, mortality, or LOS after SLT and that double lung transplant or the routine use of CPB are not necessary for patients with pulmonary parenchymal disease and secondary PHTN.

**By Invitation*

### 17. Hyperthermic Pleural Perfusion with Cisplatinum -Preliminary and Midterm Results

Yael Refaely*, David A. Simansky*, Michael Paley* and Alon Yellin*, Tel Hashomer, Israel

*Sponsored By: John R. Benfield, Los Angeles, California*

*Discussant: TBA*

**OBJECTIVE:** Intrapleural chemotherapy and hyperthermia have been investigated separately for the treatment of pleural tumors or rumors with pleural extension . The aim of this study is to conduct a phase I and n study of operation, chemotherapy and hyperthermia in one session for the treatment of pleural cancers.

**METHODS:** Since 1994, 26 pts with the following underlying conditions had intraoperative HPF:Thymoma-11, Mesothelioma-8, others-6. Technique of per-fusion was: Use extracorporal circuit and heat exchanger, circulating the pleural space with 1000-2500 ml/min of Ringer lactate. Cisplatinum (60mg-2 pts, 100mg-2,120mg-1,150mg-18,200mg-3 pts) was added when the inrrapleural temperature stabilized (mean- 40.8ºc). The associated operations were: extended extrapleural pneumonectomy-8, resection of rumor with pleurectomy -10, resection of rumor without pleurectomy -4, exploration and HPF only -4( Thora-cotomy-2, VATS-2).

**RESULTS:** There were no technical problems during the perfusion period. The maximal systemic temperature reached 38ºc. There were no renal or hematological toxicity except one case of thrombocytopenia. One pt died from complications developing after herniation of the stomach through the sutured diaphragm. Additional complications were: bleeding which required re-thoracotomy-1 pt;empyema-3(early-2, late-1); prolonged air leak -2; atrial fibrillation -1. Median postoperative stay was 7 days (range 2-50). four pts died 6-15 months following HPF from systemic disease progression. Two of them had contralateral or peritoneal spread without local reccurence. 20 are alive 1-47 months after surgery. 1-year survival is 81%. 2 and 3-years survival is 70%.

**CONCLUSIONS:** Intraoperative HPF including cisplatinum was adequately safe. Finding to date show that this method offers excellent local control for pleural cancers.

**By Invitation*

### 18. Surgical Treatment of Lung Cancer with Synchronous Brain Metastasis

Peter S. Billing*, Daniel L. Miller*, Mark S. Allen, Claude Deschamps, Victor F. Trastek, and Peter C. Pairolero, Rochester, Minnesota

***Discussant: Joseph I. Miller, Jr., M.D., Atlanta, GA***

**OBJECTIVE:** Although resection of primary lung cancer and metachronous brain metastasis is superior to other treatment modalities in prolonging survival, resection of primary lung cancer and synchronous brain metastasis is controversial.

**METHODS:** From January 1975 to December 1997, 220 patients received treatment at our institution for synchronous brain metastasis and primary non-small cell lung cancer. Twenty-seven of these patients (12.3%) underwent staged surgical resection of solitary synchronous brain metastasis and primary non-small cell lung cancer.

**RESULTS:** There were 18 men and 9 women. Median age was 56 years (range, 35-71). Twenty-four patients (88.9%) presented with neurological symptoms. Craniotomy was performed first in

all 27 patients. Median time between craniotomy and thoracotomy was 13 days (range, 4-67 days). Lobectomy was performed in 21 patients, bilobectomy in 3, and pneumonectomy in 3. There were no operative deaths. Cell type was adenocarcinoma in 11 patients, squamous cell in 8, and large cell in 8. At the time of the pulmonary resection, 14 patients had no evidence of lymph node metastases, 6 had hilar metastases and 7 had mediastinal metastases. Twenty-two patients (81.5%) received adjuvant therapy: whole brain radiation (WBR) alone in 15; WBR/systemic chemotherapy in 4; and WBR, chest radiation and systemic chemotherapy in 3. Follow-up was complete in all patients for a median of 22 months (range, 2-104 months). Median survival was 18.5 months (range, 2-104). Actuarial survival at 1, 2 and 5 years was 60%, 36% and 20%, respectively.

**CONCLUSIONS:** The survival for patients who present with brain metastasis from lung cancer is poor. However, a therapeutic plan including staged resection of synchronous solitary brain metastasis followed by pulmonary resection can be beneficial in a select group of patients.

**By Invitation*

### 19. Chest Wall Resection for Recurrent Breast Carcinoma

Robert J. Downey*, Valerie Rusch, F. Ida Hsu*, David Linehan*, Manjit S. Bains, and Robert J. Ginsberg, New York, New York

*Discussant: Mark S. Allen, M.D., Rochester, MN*

**OBJECTIVE:** To define safety and efficacy of resection of bony thorax for recurrent breast carcinoma

**METHODS:** Retrospective chart review. Survival by Kaplan-Meier, prognostic factors by log rank/Cox regression, *p* significant if ‰¤0.05

**RESULTS:** During 10/87-4/97, 41 women (med age 56yrs) underwent resection of sternum (2), ribs (17), or sternum/ribs (22). Operative mortality 0%, resection rate 80%. Skeletal reconstruction: MMMM(35), MM(2), Gore-tex (1), other/no material(3). Median skin defect 110 $cm^2$ (range 0-448). Soft tissue closure: primary(10), pedicled flap(26), microvascular flap(4), combination(l). At last FU, 24 patients were without local recurrence, 14 with local recurrence, and 3 status unknown. Synch mets, + margins, + lymph nodes, size largest nodule, # nodules, or bony invasion did not correlate with local recurrence. Synch mets, + margins, + lymph nodes, (but not size largest nodule, no. nodules, or bony invasion) correlated with survival.

**Survival after chest wall resection for recurrent breast cancer**

| | 1 YEAR | 3 YEAR | 5 YEAR |
|---|---|---|---|
| *OVERALL* | 97% | 97% | 67% |
| **SYNCHRONOUS METASTASES** | 94% | 94% | 50% |
| **NO SYNCHRONOUS METASTASES** | 100% | 100% | 77% |

**CONCLUSIONS:**

1. Resection/reconstruction of the bony thorax for recurrent breast carcinoma is safe

2. Survival and local control following resection are favorable, even if distant metastases present

**3:05 p.m. INTERMISSION - VISIT EXHIBITS**

*By Invitation

**3:50 p.m. SIMULTANEOUS SCIENTIFIC SESSION B -**

**GENERAL THORACIC SURGERY**

Room 211-213, Ernest N. Morial Convention Center

*Moderators: Thomas R. J. Todd, M.D.*

*Steven J. Mentzer, M.D.*

**20. The Current Role of Mediastinoscopy in the Evaluation of Thoracic Diseases**

Zane T. Hammoud*, Bryan F. Meyers*, Tracey J. Guthrie*, Joel D. Cooper, Charles L. Roper and G. Alexander Patterson, St. Louis, Missouri

*Discussant: Douglas E. Wood, M.D., Seattle, WA*

**OBJECTIVE:** Mediastinoscopy(med) is a commonly employed procedure utilized for the diagnosis of thoracic disease and the staging of lung cancer. We sought to re-evaluate the safety and efficacy of med in an academic thoracic surgical program.

**METHODS:** We conducted a retrospective review of all patients undergoing med on our service between 1/88 and 8/98.

**RESULTS:** We performed med on 2069 patients during the study period, including 1909 cervical meds, 62 anterior mediastinotomies, and 98 combined procedures. A total of 1668 patients underwent med for known or suspected lung cancer. In 427(26%) of these, N2 or N3 disease was identified; only 29(7%) of these patients underwent resection, with a 5 yr. survival of 25%. In 1241(74%) of patients with suspected lung cancer, med was negative; all of these patients underwent exploration. In these patients, 954(77%) had lung cancer. Only 74(8%) of the lung cancer patients were found to have N2 disease at exploration; the 5 yr. survival in this group was 24%. Among the 1241 patients with a negative med, 71(6%) had a non-bronchogenic malignancy and 216(17%) had resection of what proved to be a benign lesion. In 156 patients, the med proved positive for a non-bronchogenic malignancy. Evaluation of mediastinal aden-opathy in the absence of any identifiable intrathoracic tumor in 245 patients led to a definitive diagnosis in 218(89%). In the entire group of 2069 patients, we observed 4(0.2%) perioperative deaths and 10(0.5%) complications. Only one death was attributed to med(tumor invading the aorta). No deaths or complications occurred in patients undergoing med for benign disease.

**CONCLUSIONS:** Mediastinoscopy is a highly effective and safe procedure. We believe that mediastinoscopy should currently be used routinely in the diagnosis and staging of thoracic diseases.

*By Invitation

**21. Bronchoplastic Procedures in Malignant and Non-Malignant Disease - Results of 144 Cases**

Adelheid End*, Peter Hollaus*, Andreas Pentsch*, Michael Mueller*, Ernst Wolner and Franz Eckersberger*, Vienna, Austria

*Discussant: Thomas R. J. Todd, MD., Toronto, Ontario, Canada*

**OBJECTIVE(s):** We present our experience with bronchoplastic procedures in a seven year period.

**METHODS:** Data from 144 patients who underwent bronchoplastic surgery between 1991 and 1997 were collected retrospectively. There were 110 men and 34 women, mean age was 59±9 years (range, 26 - 79 years). Indication for surgery was primary lung cancer (n=130), metastases (n=4) and carcinoid tumors (n=10). Full bronchial sleeve resections were performed in 108 cases, bronchial wedge resections in 28 and sleeve pneumonectomies in 8 cases; 103 procedures were done on the right (72 %) and 41 on the left side (28%). In 15% (n=21) an angioplasty and in 3, 5% (n=5) resection of the thoracic wall was performed. In 3 extended resections cardiopulmonary bypass was used. 61% of patients presented with cardiovascular risk factors, 62% were smokers and 64% had chronic pulmonary obstruction.

**RESULTS:** 30-day mortality was 8 %. Causes of death were cardiac failure and myocardial infarction (n=3), pulmonary embolism (n=2), hepatorenal failure (n=1), stroke (n=1), pneumonia (n=3), gastrointestinal bleeding (n=1) and mesenteric infarction (n=1). Major complications consisted of anastomotic dehiscence in 5 cases beyond the 30th postoperative day, 4 of them died, rethoracotomy for bleeding (n=3) and empyema (n=3). The presence of cardiac risk factors, chronic obstructive disease, N2-disease, R1-resection and performance of sleeve pneumonectomy had an adverse influence on survival ($p<0.05$).

**CONCLUSIONS:** Bronchoplastic procedures have evolved as an alternative to pneumonectomy in high-risk patients with centrally located tumors or in non-malignant disease. However, our mortality rate of 8 % demonstrates that patients should be selected carefully assessing their preoperative risk profile.

**By Invitation*

### 22. M1a/M1b Esophageal Carcinoma: Surgical Relevance

Neil A. Christie*, Thomas W. Rice, Malcolm M. DeCamp *, John R. Goldblum*, David J. Adelstein*, Lisa A. Rybicki* and Eugene H. Blackstone, Cleveland, Ohio

*Discussant: Mark J. Krasna, M.D., Baltimore, MD*

**OBJECTIVE:** The 1997 staging system for esophageal carcinoma subdivides distant metastatic disease (M1) into M1a (non-regional lymph node metastases) and M1b (visceral metastases). This study evaluates the clinical relevance of this classification.

**METHODS:** From our prospective esophageal database, 141 patients (pts) were identified with M1 disease, 37 (26%) with M1a and 104 (74%) with M1b. Histology was adenocarcinoma in 119 (84%) pts, squamous cell in 18 (13%) and adenosquamous in 4 (3%), with a similar distribution for Mia and M1b (P=.3). Forty-seven pts underwent surgery. Of these, 29 (78%) were M1a and 18 (17%) M1b (P<.001). Medical therapy (chemotherapy and/or radiation therapy) was given to 99 (70%) pts; 31 (84%) Mia and 68 (65%) M1b (P=.04).

**RESULTS:** Median and 5-year survival were 11 months and 6% for Mia pts, and 5 months and 2% for M1b pts (P=.002). Surgery provided no evident advantage in M1b and a small benefit after

12 months in Mia. Multivariable analysis demonstrated that M1b pts had 1.7 times the risk of death of M1a pts (CI 1.1-2.5, P=.009), and pts without medical therapy had 2.2 times the risk of those with medical therapy (CI 1.5-3.2, P<.001). Despite the high prevalence of surgery for M1a disease, the analysis suggests that M1a and use of medical therapy, rather than surgery *per se,* account for the small and clinically unimportant differences in survival.

**CONCLUSIONS:** We conclude that: 1) although there are statistically significant survival differences between M1a and M1b pts, these differences are not clinically important; 2) medical therapy is associated with a modest survival benefit; and 3) surgery offers no advantage in the treatment of these pts.

**By Invitation*

### 23. The Role of Positron Emission Tomography in Evaluating the Mediastinum in Patients with Non-Small Cell Lung Cancer

Tracey L. Weigel*, Carolyn C. Meltzer*, David Friedman*, Robert J. Keenan, Peter F. Person* and James D. Luketich*, Pittsburgh, Pennsylvania

*Discussant: Kemp H. Kernstine, M.D., Iowa City, IA*

**OBJECTIVE:** Currently, mediastinoscopy with histologic nodal examination is the gold standard for preoperative assessment of the mediastinum. Positron Emission Tomography (PET) is a new, noninvasive tool that differentiates malignant from benign processes based upon metabolic status rather than anatomic structure. This study compares the accuracy of PET and CT scanning, as compared to histology, in staging the mediastinum in patients with NSCLC.

**METHODS:** Histologic examination of mediastinal lymph nodes (MLNs) was performed on 34 patients with NSCLC by multi-station lymph node sampling at mediastinoscopy and/or mediastinal lymph node dissection at surgical resection. PET scans were read by a single radiologist, blinded to histologic assessment, using CT scans for anatomic localization. CT scans were independently assessed for mediastinal lymphadenopathy.

**RESULTS:** The overall accuracy, sensitivity, and specificity for PET in the determination of metastases to the mediastinum was better than CT when compared to histologic findings (see table). PET scan staging, however, correlated poorly with histologic MLN staging; R=0.553, (p=0.001).

| Scan | Accuracy | Sensitivity | Specificity |
|---|---|---|---|
| PET | 85.3% (29/34) | 80% (4/5) | 86.2% (25/29) |
| CT | 58.8% (20/34) | 60% (3/5) | 58.6% (17/29) |

**CONCLUSIONS:** PET scanning appears to be more accurate than CT for staging the mediastinum in patients with NSCLC. However, PET imaging still fails to identify 20% of histologically-proven MLN metastases. At present, mediastinoscopy should remain the standard of care for pre-operative staging of the mediastinum in patients with NSCLC.

**By Invitation*

**24. Primary Mediastinal Nonseminomatous Germ Cell Tumors: The Influence of Postchemotherapy Pathology On Long-Term Survival After Surgery**

Kenneth A. Kesler*, Karen M. Rieger*, Kristen N. Ganjoo*, Matt Sharma*, Naomi S. Fineberg*, Lawrence H. Einhorn* and John W. Brown, Indianapolis, Indiana

*Discussant: Scott J. Swanson, M.D., Boston, MA*

**OBJECTIVE(s):** Treatment of nonseminomatous germ cell tumors (NSGCT) with cisplatin based chemotherapy (CT) followed by surgical resection of residual disease, represents a successful model for multimodaliry cancer therapy. We reviewed 104 Pts from 1981 to 1997 with primary mediastinal NSGCT to evaluate variables which may influence survival following surgery.

**METHODS:** 27 (26%) Pts did not undergo post-CT resection due to progression during CT (n=25) or had a complete response after CT (n=2). 77 Pts (74%) underwent 80 resections and were further analyzed with respect to multiple variables including pre and post CT pathology and serum tumor markers (STM). All were male between 12 and 49 yrs (mean=28). The majority (95%, 69/73) had elevated STM, 44 of which (64%) demonstrated normalization after primary or secondary CT. 21 Pts (27%) had extra-mediastinal disease at presentation.

**RESULTS:** The finding of tumor necrosis in the resected specimen (n=16) predicted excellent survival which was significant compared to teratoma (n=26), sarcomatous degeneration (n=6) or persistant NSGCT pathologies (n=44). The finding of teratoma predicted significantly better survival than persistent NSGCT. No other variable was significantly predictive.

**CONCLUSIONS:** Primary NSGCT of the mediastinum can be cured with a multimodality approach. Post-CT pathologic findings of tumor necrosis and teratoma predict excellent and intermediate long-term survival respectively. Survival is poor but possible in patients suspected of unfavorable pathology following CT, justifying an aggressive surgical approach.

**ADJOURN**

**By Invitation*

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

Room 208-210, Ernest N. Mortal Convention Center

*Moderators: John J. Lamberti, M.D.*

*John A. Waldhausen, M.D.*

**25. Revision of Previous Fontan Connections to Total Extracardiac Cavopulmonary Anastomosis: a Multicenter Study**

Carlo F. Marcelletti, Frank L. Hanley, Constantine Mavroudis, Mohan V. Reddy*, Raul F. Abella*, Stefano M. Marianeschi*, Francesco Seddio*, Marco Meli*, Fiore S. lorio* and Francis Fontan, Bordeaux, France, Chicago, Illinois, Modena, Italy and San Francisco, California

*Discussant: John E. Mayer, M.D., Boston, MA*

**OBJECTIVE:** Conversion of modified Fontan procedure to a total extracardiaccavopulmonary connection (TECPC)has been proposed as an alternative surgical treatment of pts who showed manifestation of surgical failure. The aim of the present multicenter study is to evaluate the midterm outcome after conversion.

**METHODS:** Between Oct'92 and Sept'98, 24 pts (mean age 21, 1 yrs; range 10-39) underwent revision of the atriopulmonary connection to TECPC in 4 different Institutions. In these pts who have received previously an atriopulmonary connection, complications due to right atrial enlargement were: Fontan pathway obstruction(10), progressive CHF(16), atrial dysrhythmias(19), RA thrombus(2),obstruction of right pulmonary veins(6), subAo stenosis(1), protein losing enteropathy(PLE)(2)and effusions(6). Conversion to a TECPC was carried out with a non-valved conduit from the IVC to RPA.PTFE tube graft was used in 18 pts, homograft conduit in 3 pts and Dacron tube in 3 pts.

**RESULTS:** Three deaths occurred: 1 pt with severe cachexia, in whom transplantation was controindicated for social reasons, died in the early postoperative period of massive effusions. The second patient died for irreversible heart failure during the operation and the third pt died at home 8 months postop for arrhythmia. All surviving pts have improved to NYHA class I (16 pts)or II (4 pts) except 1 pt in NYHA class IV who underwent OHTX. Postop arrhythmias occurred in 10 pts:4 pts underwent PMK implantation and in 6 pts medical therapy was sufficient to manage the symptoms. Two pts with PLE improved within 30 days.

**CONCLUSIONS:** Conversion of failing previous Fontan procedure to TECPC can be accomplished with a low mortality and it appears to be a viable option. However the revision should be undertaken early in symptomatic pts before irreversible ventricular failure ensues.

**By Invitation*

### 26. Repair of the Truncal Valve and Associated Interrupted Arch in Neonates with Truncus Arteriosus

Marjan Jahangiri*, David Zurakowski*, Pedro J. Del Nido, John E. Mayer and Richard A. Jonas, Boston, Massachusetts

*Discussant: Edward L. Bove,* M.D., *Ann Arbor, MI*

**OBJECTIVE:** Truncal valve regurgitation and interrupted aortic arch have frequently been identified as risk factors in the repair of truncus asteriosus. We wished to examine these factors in the current era including the impact of truncal valve repair.

**METHODS:** Between 1992 and August 1998, 50 patients underwent surgical repair of truncus asteriosus. Their ages ranged from 2 days to 6 months (median, 2 weeks). Nine patients had associated interrupted aortic arch. Of the 14 patients (28%) who were diagnosed to have truncal valve regurgitation pre-operatively, five underwent truncal valve repaid and one underwent homograft replacement of the truncal valve with coronary reimplantation.

**RESULTS:** The acturial survival was 96% at 30 days, 1 year and 3 years. There were no deaths in patients with associated interrupted aortic arch. The two deaths in the series occurred in patients with truncal valve regurgitation, neither of whom underwent repair. Postoperative transthoracic echocardiography in patients who underwent valve repair showed minimal residual valvar regurgitation. None of the patients have required reoperation for truncal valve problems or aortic arch stenosis at a mean follow-up of 22 months. Conduit replacement has been performed in 17

patients (34%) after a mean duration of 2 years. The freedom from reoperation for those who had an aortic homograft was 4 years and for those who had pulmonary homograft was 3 years.

**CONCLUSIONS:** Despite the magnitude of surgery, excellent results can be achieved in complex forms of truncus arteriosus. In the current era interrupted aortic arch is no longer a risk factor for repair of truncus. Aggressive application of truncal valvuloplasty methods neutralize the traditional risk factors of truncal valve regurgitation.

**By Invitation*

### 27. Five To Fifteen Year Follow-Up of Fresh Autologous Pericardial Valved Conduits

Andres J. Schlichter*, ††Christian Kreutzer, Rita C. Mayorquim*, Jorge L. Simon*, Maria I. Roman*, Haydee Vazquez*, Eduardo A. Kreutzer* and Guillermo O. Kreutzer*, Buenos Aires, Argentina
*Sponsored by: Richard A Jonas, Boston, Massachustts*

*Discussant: William G. Williams, M.D., Toronto, Ontario, Canada*

**OBJECTIVE:** To evaluate the long term results of an autologous pericardial valved conduit (APVC) in the venous ventricle outflow tract.

**METHODS:** Between 1983 and 1993, 82 conduits were placed in the subpulmonary position. Patients receiving homografts(n=2), heterografts(n=3) or valveless conduits(n=19) and dying within 90 days were excluded. Thus, 54 late survivors of venous ventricle outflow reconstruction with a fresh bicuspid APVC were followed from 5 to 15 years with a median of 7 years. Yearly evaluations by two dimensional echo Doppler were made and 9 patients had cardiac catheterization. Diagnosis include D-transposition of the great arteries (n=16), L-transposition of the great arteries(n=14), Tetralogy of Fallot(n=11), Truncus arteriousus(n=10) and double outlet right ventricle(n=3). Mean age at implantation was 2.9 ± 6.3 years(2 months to 24 years). Cath or surgical intervention was indicated when the gradient exceed 50 mm Hg.

**RESULTS:** In 27 patients the APVC increased its diameter (1 to 7 mm), in 20 it remained unchanged and a reduction of 1 mm was noted in 4. Median conduit diameter at implantation was 16 mm and was 17.5 mm at last evaluation.(p=0.0001) Freedom from reintervention at 5, and 10, years was 89% and 80%. Freedom from reintervention was 100% at 10 years for conduits larger than 16 mm at the time of implantation and it was 85% at 5 years and 72% at 10 years for those 16 mm or less. The valve had good function in the first six months but late failure without obstruction. Conduit related reoperation was required only in 6 cases between 4.5 and 10 years after implantation and 2 patients underwent balloon dilation of APVC. There were 3 late deaths caused by arrhythmia, sepsis and brain tumor.

**CONCLUSIONS:** Pericardial valved conduits show excellent results and compare favorably to those of other conduits.

**2:45 p.m. INTERMISSION - VISIT EXHIBITS**

*††1998-99 AATS Graham Fellow*

**By Invitation*

**3:30 p.m. SIMULTANEOUS SCIENTIFIC SESSION C -**

**CONGENITAL HEART DISEASE**

Room 208-210, Ernest N. Morial Convention Center

*Moderators: John J. Lamberti, M.D.*

*John A. Waldhausen, M.D.*

**28. Pre-Surgical Risk-of-Death Prediction Model in Neonatal Repair of Congenital Heart Disease**

Robert R. Clancy*, Susan A. McGaurn*, James E. Coin*, Gil Wernovsky*, Thomas L. Spray, William I. Norwood, Marshall L. Jacobs*, John D. Murphy*, Deboral G. Hirtz* and J. William Gaynor*, Bethesda, Maryland, Browns Mills, New Jersey, Media, Pennsylvania, Philadelphia, Pennsylvania and Wilmington, Delaware

*Discussant: Marc R. de Leval, M.D., London, U.K.*

**OBJECTIVE:** To generate a pre-surgical risk-of-death prediction model in a population of neonates with congenital heart disease (CHD) undergoing surgery with deep hypothermic circulatory arrest (DHCA).

**METHODS:** We completed a single-center, prospective, randomized, double-blind, placebo-controlled neuroprotection trial in a population of neonates with CHD requiring surgical repair or palliation utilizing DHCA. An extensive 5,500 item database was generated and included pre-surgical, intra- and post-operative variables. Stepwise logistic regression evaluated 49 presurgical variables (delivery, maternal & infant related factors) producing a risk prediction model.

**RESULTS:** Between 7/92 and 9/97, 350 of 481 eligible infants were enrolled of whom 317 were deemed evaluable cases. The overall mortality was 52 of 317 (16.4%), unaffected by the investigational drug. The resulting model contained information on 4 presurgical categories: (i) cardiac anatomical classification (two vs single ventricle, with/without arch obstruction), (ii) 1 min. Apgar score (‰¤5 vs >5), (iii) presence of named genetic syndrome or chromosomal abnormality and (iv) age at hospital admission for surgery (‰¤5 or >5 days). Mortality for two ventricle repair was 4 of 129 (3.1%). Mortality for single ventricle palliation was 48 of 188 (25.5%) but this rate was significantly increased by the presence of low Apgar score, genetic diagnosis and older age at admission. The logistic regression model based on the 4 categories resulted in a prediction accuracy of 81%.

**CONCLUSIONS:** In this population, much of the force of mortality is determined by conditions that exist pre-operatively. The identification of high risk subgroups may impact family counseling, therapeutic intervention and risk stratification for future study designs, (funded by NIH contract NS-NO1-2315)

**By Invitation*

**29. Simplified Single Patch Technique for the Repair of Atrioventricular Septal Defect**

Ian A. Nicholson*, Graham R. Nunn*, Gary F. Sholler*, Richard E. Hawker*, Stephen G. Cooper* and Kai C. Lau*, Westmead, Australia

*Sponsored By: Lawrence H. Cohn, M.D., Boston, Massachusetts*

*Discussant: John* W. *Brown, M.D., Indianapolis, IN*

**OBJECTIVE:** Due to the complexity of traditional one and two patch techniques for the repair of complete atrioventricular septal defect we modified our repair technique to avoid the use of any ventricular septal patch material. We report our prospective experience with this simplified one patch technique.

**METHODS:** Forty seven consecutive patients between September 1995 and August 1998 underwent repair using this technique without modification. All patients were repaired by direct suturing of the common atrioventricular valve leaflets to the crest of the ventricular septum . No division of valve leaflets was necessary. A single pericardial patch was used to close the defect in the atrial septal component. Follow-up included electrocardiography and echocardiographic assessment of ventricular function, AV valve function and the adequacy the left ventricular outflow tract.

**RESULTS:** There were two deaths (4%), only one cardiac related, in the series. There were 17 males and 30 females. Mean age at repair was 5.6 months (median 3.4 months). Associated lesions were repaired in 19 patients (40%). Mean follow-up was 1.85 years ( median 1.9 years). There was no heart block. There were no significant residual ventricular septal defects detected and no left ventricular outflow tract obstruction seen on echocardiography in any patient to date. Mitral valve status post-operatively was assessed as no incompetence in 13 (28%) patients , minimal in 19 (40%), mild in 12 (26%) and moderate in 3 (6%).

**CONCLUSIONS:** Therepair of complete atrioventricular septal defect by direct suturing of the atrioventricular valve leaflets to the crest of the ventricular septum using a single patch technique greatly simplifies the repair and does not lead to left ventricular outflow tract obstruction nor interfere with valve function.

**By Invitation*

### 30. Lung Transplantation in Very Young Infants

Charles Burford Huddleston, George B. Mallory*, Stuart C. Sweet*, Aaron Hamvas* and Eric N. Mendeloff*, St. Louis, Missouri

*Discussant: Thomas L, Spray, M.D., Philadelphia, PA*

**OBJECTIVE(s):** There are rare congenital pulmonary parenchymal and pulmonary vascular diseases that occur in infants resulting in the death of these otherwise normal infants even with aggressive medical therapy. In these circumstances, lung transplantation (LTX) offers the only effective treatment.

**METHODS:** We reviewed our experience with LTX in infants presenting at less than 6 months of age with either end-stage pulmonary parenchymal or pulmonary vascular disease. 25 infants were listed for LTX at an average age of 67 ± 52 days (range = 8-169 days). All were mechanically ventilated and 18 had been so since birth; 12 infants required ECMO and 5 others required high frequency oscillating ventilator prior to transplantation. 7 patients died while awaiting donor organs and thus 18 were transplanted, forming the basis for this review.

**RESULTS:** The average age at transplant was 104 ± 44 days; the average weight was 4.9 ± 1.6 kg. There were 6 early deaths. The 12 surviving hospitalization were ventilated 24 ± 21 days and had

an average post-LTX hospital stay of 58 ± 33 days. The followup for these survivors is 2.6 ±1.8 years (range = 0.5-5 years). Transbronchial biopsies as part of a surveillance protocol or due to clinical indications revealed only 2 episodes of acute rejection (A2 or greater) and only one patient out of the 12 early survivors (8%) has developed bronchiolitis obliterans over an average followup of 2.6 ± 1.8 years. One required re-transplantation for severe respiratory failure following sepsis. Somatic growth has been at eh 20th percentile for length and 25th percentile for weight. There have been 2 late deaths for an overall survival of 56%.

**CONCLUSIONS:** Although high risk, LTX for infants with end-stage pulmonary parenchymal and pulmonary vascular disease is feasible. Acute rejection and bronchiolitis obliterans have not posed significant problems in early followup.

**By Invitation*

### 31. Improved Results with Selective Management in Pulmonary Atresia with Intact Ventricular Septum

Marjan Jahangiri*, David Zurakowski*, David P. Bichell*, John E. Mayer, Pedro J. Del Nido and Richard A. Jonas, Boston, Massachusetts

*Discussant: Frank L. Hartley,* M.D., *San Francisco, CA*

**OBJECTIVE:** Late outcome of neonatal pulmonary atresia with intact ventricular septum remains poor in most reported series. We have followed a selective approach towards either single ventricle repair versus complete or partial biventricular repair based on the presence of right ventricular dependent coronary circulation (RVDCC) and growth of the RV.

**METHODS:** Forty seven patients underwent surgery between January 1991 and September 1998.

**RESULTS:** Sixteen (34%) patients had RVDCC with a tricuspid valve Z score of -3.0+.66 versus -2.0+.95 (p=0.002) for those without RVDCC. A systemic to pulmonary artery shunt only was performed in all patients with RVDCC with one death. Thirteen of the sixteen patients with RVDCC underwent a bidirectional Glenn at a median time of 9 months after their first operation, nine of whom have had a Fontan procedure (no death). One patient has had a 1.5-ventricle repair. In the 31 (66%) patients without RVDCC, 6 patients underwent a systemic to pulmonary artery shunt only, 23 had a shunt and right ventricular decompression and 2 had only a transannular patch. In this group, 10 patients received a 2-ventricle repair, 6 a 1.5-ventricle repair and 8 patients had a Fontan procedure. The overall actuarial survival was 97.7% at 1 month, 1 year, 5 years and 7 years.

**CONCLUSIONS:** If stratified well, excellent survival can be achieved in the treatment of pulmonary atresia with intact ventricular septum. This may be at the price of achieving a 1-ventricle as opposed to a 2-ventricle repair.

**By Invitation*

### 32. Repair of Ebstein's Anomaly Associated with Bidirectional Cavopulmonary Shunt in High Risk Patients

Sylvain Chauvaud*, Jean Francois Fuzellier*, Alain Berrebi* and Alain Carpentier, Paris, France

*Discussant: Jan M. Quaegebeur,* M.D., *New York, NY*

**OBJECTIVE:** Patients suffering from Ebstein's anomaly and severely impaired right ventricular (RV) function often present with difficult postoperative course. The aim of this study is to report the use of bidirectional Cavopulmonary shunt (BCPS) associated with intracardiac repair in this high risk group.

**METHODS:** Since 1980, 125 patients (pts) have been operated on for Ebstein's anomaly. Only 3 pts had a tricuspid valve (TV) replacement and 122 had an intracardiac repair (ICR) using Carpenrier's technique. Among the later group, 67 pts were classified as high risk because of severe RV dysfunction and/or severe arrhythmia. This cohort was divided into 2 groups: GI (45 pts) had isolated ICR and Gil (22 pts) had ICR associated with BCPS. All pts had the same preoperative clinical pattern and age (mean 22y). The ICR was identical in all patients (TV repair and RV plication). The a trial septal defect when present was closed.

**RESULTS:** Operative mortality was 9.8% in the whole group, 24.4% in GI (CL 95%: 13-43) and 0% in Gil (CL 95%: 0-18) $p<0.05$. The main cause of mortality in GI was RV failure (5/11 deaths). Significant residual or recurrent TV insufficiency was present in 12% of both groups. However, the need for a reoperation was more frequent in GI: 11% (5/45) than in Gil: 0%, due to RV preload reduction. No deleterious effect of the BCPS was observed.

**CONCLUSIONS:** Bidirectional Cavopulmonary shunt associated with intracardiac repair decreased the operative mortality in high risk patients suffering from Ebstein's anomaly. It improved clinical tolerance of occasional residual TV insufficiency.

**By Invitation*

## TUESDAY MORNING, APRIL 20, 1999

**7:00 a.m. C. WALTON LILLEHEI RESIDENT FORUM SESSION**

*Supported by an unrestricted educational grant from St. Jude Medical, Inc.*

Ballroom, Ernest N. Morial Convention Center

*Moderators: Fred A. Crawford, Jr., M.D.*

*Timothy J. Gardner, M.D.*

### L1. Angiogenic Therapy with Vascular Endothelial Growth Factor Reverses Pulmonary Hypertension

Andrew I. Campbell*, David A. Latter* and Duncan J. Stewart*, Toronto, Ontario, Canada

*Sponsored By: Richard D. Weisel, Toronto, Ontario, Canada*

**OBJECTIVE:** The role of angiogenic factors in pulmonary hypertension (PH) is controversial. Increased VEGF expression associated with plexiform lesions may contribute to the vasculopathy of this disease, or may represent an incomplete adaptive response to the loss of vascular channels. We hypothesized that VEGF gene transfer could reverse PH in the rat monocrotaline model, possibly by inducing the development of new pulmonary microvessels.

**METHODS:** 18 animals were injected with 80 mg/kg of monocrotaline (MCT) and 14 days later were randomized to receive either $5x10^5$ syngeneic smooth muscle cells (SMC) transfected with

$VEGF_{165}$ (n=10) or with the control vector pcDNAS.l (n=8) via the internal jugular vein. At the time of gene transfer, central venous (CVP) and right ventricular (RV) pressures were measured to confirm the development of pulmonary hypertension. 28 days following MCT injection, CVP, RV, and aortic pressures were measured, and right ventricular/left ventricular weight (RV/LV) ratios obtained.

**RESULTS:** 14 days following MCT delivery, RV pressure was increased to 28mm Hg in both groups. 28 days following MCT injection, RV pressures in control animals were further elevated to 55±5mm Hg, while in the VEGF transfected animals the rise in RV pressure was reduced to 37±3mm Hg (**p<0.01**). CVP was also significantly reduced from 3±0.5 to 1.5±0.5mm Hg (**p=0.05**). Systemic arterial pressure was unaffected by gene transfer in either group. The degree of RV hypertrophy was also significantly attenuated following VEGF transfer (0.4±0.02 in control vs. 0.28±0.01 in VEGF treated animals, **p<0.001**).

**CONCLUSIONS:** VEGF gene transfer is able to significantly reduce the progression of established pulmonary hypertension in the monocrotaline model of PH, and supports a significant therapeutic role for this form of angiogenic gene therapy.

**By Invitation*

### L2. Transforming Growth Factor Beta-1 Gene Transfer Ameliorates Acute Lung Allograft Rejection

Bassem N. Mora*, Carlos H. R. Boasquevisque*, Mariano Boglione*, Jon M. Ritter*, Ronald K. Scheule*, Nelson S. Yew*, Lisa Debruyne*, Lihui Qin*, Jonathan S. Bromberg* and G. Alexander Patterson, Ann Arbor, Michigan, Framingham, Massachusetts and St. Louis, Missouri

**OBJECTIVE:** We have previously demonstrated successful ex vivo transfec-tion of rat lung isografts using reporter genes complexed to liposomes. The aim of the current work was to study the feasibility of functional gene transfer using the gene encoding for Transforming Growth Factor b-1 (TGFb-1), a known immunosuppressive cytokine, on rat lung allograft function in the setting of acute rejection.

**METHODS:** The rat left lung transplant technique was used in all experiments, with Brown Norway donor rats and Fischer recipient rats. Following harvest, left lungs were transfected *ex vivo* with either sense or antisense TGFb-1 constructs complexed to cationic lipids, then implanted into recipients. On postoperative days (POD) 2,5, and 7, animals were sacrificed, arterial oxygenation measured, and acute rejection graded.

**RESULTS:** On POD 2, there were no differences in acute rejection or lung function between TGFb-1 treated animals and controls. On POD 5, oxygenation was significantly improved in grafts transfected with the TGFb-1 sense construct compared to antisense controls: PaO2 = 434.2 ± 198.1 vs. 102.8 ± 85.4 mm Hg, respectively, $p<0.05$. Lung acute rejection scores were also significantly improved, corresponding to decreases in both vascular and airway rejection scores (vascular rejection score: 2.00 ± 0.50 vs. 2.79 ± 0.64; airway rejection score: 1.28 ± 0.67 vs. 2.29 ± 0.76, respectively, $p<0.05$). The amelioration of acute rejection was temporary and decreased by POD 7.

**CONCLUSIONS:** The feasibility of using gene transfer techniques in order to introduce novel functional genes in the setting of lung transplantation is demonstrated. In this model of rat lung allograft rejection, TGFb1 gene transfer resulted in temporary but significant improvements in lung allograft function and acute rejection scores.

*By Invitation

### L3. Mechanical Transmyocardial Revascularization Induced Angiogenic Response

Victor F. Chu*, Jinqiang Kuang*, Amy N. Mcginn*, Adel Giaid*, Stephen Korkola* and Ray Cj-Chiu, Montreal, Quebec, Vancouver, BC, Canada

**OBJECTIVE:** Although it remains controversial, increased expression of an-giogenic growth factors has been proposed as a mechanism of transmyocardial laser revascularization (TMLR). We assessed the effectiveness of less expensive *mechanical* transmyocardial revascularization (TMMR) in a chronically ischemic porcine model by measuring myocardial angiogenic response.

**METHODS:** Ameroid constrictors were implanted around porcine circumflex arteries 6 weeks before transmyocardial revascularization. Group 1 (n=5) recieved TMMR in the ischemic zone with an 18 gauge needle and were harvested at 1 week post-TMMR. Group 2 (n=3) is simliar to Group 1 but were harvested at 4 weeks post-TMMR. Group 3 received sternotomy only with no myocardial puncture and serves as control. Myocardial samples were immunohistochemically stained for VEGF, bFGF, and TGF-b using specific antibodies. Growth factor expression was quantified using computer assisted morphometry. Vascular density was assessed by staining for factor VIII.

**RESULTS:** Significantly increased expression of all three factors in TMMR group were found at 1 week post treatment (VEGF $p<0.001$, bFGF $p<0.001$, TGF-b $p<0.001$). Angiogenic factor levels were lower at 4 weeks but still significantly higher than the baseline (VEGF $p<0.001$, bFGF $p=0.156$, TGF-b $p=0.008$)(Fig. l). Vascular density in TMMR group was significantly higher than control group at 1 week ($p<0.001$). This difference persisted at 4 weeks ($p<0.001$)(Fig. 2).

**CONCLUSIONS:** Mechanical transmyocardial revascularization is associated with significantly increased angiogenic growth factor expression and concomitant neovascularization at up to 4 weeks post treatment. These changes are similar to those observed in TMLR. Myocardial perfusion and functional studies are needed to establish potential clinical significance of these findings on angiogenic responses.

*By Invitation

### L4. Histologic Abnormalities of the Ascending Aorta and Pulmonary Artery in Aortic Valve Disease

Mauro P.L. De Sa*, Jagdish Butany*and Tirone E. David, Toronto, Ontario, Canada

**OBJECTIVE:**

This study was undertaken to examine the histology of the ascending aorta (AA) and pulmonary artery (PA) in patients with aortic valve disease (AVD) in an attempt to clarify dilation of aortic root after the Ross procedure and dilation of the ascending aorta in patients with bicuspid aortic valve (BAV).

**METHODS:**

Histologic examination of the AA and pulmonary artery PA was performed in 15 patients with BAV, 8 patients with tricuspid AVD, and in 6 normal controls. The specimens were divided into 3 groups according to the patients' ages: 21 to 40 years, 41 to 60 years and more than 60 years. The arteries were examined for degenerative changes (cystic medial necrosis, fibrosis, smooth muscle cell orientation, and elastic fragmentation). Each change was quantitated by two independent observers and graded from 0 (none) to 3 (severe).

**RESULTS:**

All 15 pts with BAV displayed various degrees of degenerative changes in the media of the aorta and 7 pts in all age groups had severe changes (cystic medial necrosis and elastic fragmentation). Of the 8 patients with tricuspid AVD, only 2 had severe degenerative changes. None of the controls had more than mild changes in the media of the AA. Severe degenerative change sin the PA were present in all pts with BAV, and in older patients with and without AVD.

**CONCLUSIONS:**

Degenerative changes in the media of the AA and PA are part of the aging process but patients with BAV have premature degeneration. The PA of young patients with BAV have severe degenerative changes similar to those of older patients with or without AVD. This finding explains the late dilation of the aortic root that may occur after the Ross procedure.

**By Invitation*

### L5. A Comparison of Three Fetal Cell Types for Transplantation into a Myocardial Scar To Improve Heart Function.

Tetsuro Sakai*, Ren-ke Li*, Richard D. Weisel, Donald A. G. Mickle*, Zhi-qiang Jia*, Shinji Tomita* and Eung-joong Kim*, Toronto, Ontario, Canada

**OBJECTIVE:** We previously demonstrated that fetal cardiomyocyte transplantation into the myocardial scar improved heart function. The mechanism, however, has not been ellucidated. This study compares cardiac function after cell transplantation with three different fetal cell types: cardiomyocytes (with contractile proteins and gap junction), smooth muscle cells (with contractile proteins, but without gap junction), and fibroblasts (no contractile proteins).

**METHODS:** A left ventricular scar was created by cryoinjury in adult rats. Four weeks after the injury, the rats received an injection into the cardiac scar of cultured fetal cardiomyocytes (CM, n=13), smooth muscle cells (SM, n=10), fibroblasts (FB, n=10), or culture medium (Control). All rats received cyclosporin. Four weeks after injection, cardiac function was examined with an intraventricular balloon, during Langendorff perfusion.

**RESULTS:** All transplanted cells formed a tissue in the cardiac scar. Developed pressure was significantly greater with CM (at a diastolic volume of 0.2 mL, improvement over control was 134±20% for CM; 108±14% for SM; and 106±17% for FB, $p<0.01$). The maximum rate of pressure rise was significantly higher with CM (at 0.2 mL, the improvement was 119±37% for CM; 98±18% for SM; and 92±11% for FB, $p<0.01$). The maximum rate of pressure fall was significantly greater with CM (at 0.2 mL, the improvement was 126±29% for CM; 108±19% for SM; and 99±16% for FB, $p<0.01$).

**CONCLUSIONS:** Fetal cardiomyocytes transplanted into cardiac scar provided better systolic function, contractility and relaxation than smooth muscle cells or fibroblasts. The contractile and elastic properties of cardiomyocytes contributed to the functional improvement.

**By Invitation*

### L6. Delayed Myocardial Preconditioning by Alphal-Adrenoceptors Involves Inhibition of Apoptosis

Kourosh Baghelai*, Laura J. Graham*, Andrew S. Wechsler and Emma R. Jakoi*, Philadelphia, Pennsylvania and Richmond, Virginia

**OBJECTIVE:** Previous studies have demonstrated that Î±l-adrenoceptor activation increases myocardial resistance to ischemic injury 24 hours later. Here we test the hypothesis that phenylephrine induced delayed cardioprotection is associated with limited infarction, decreased apoptosis, and involves altered expression of the anti-apoptotic BCLx and pro-apoptotic BAX proteins.

**METHODS:** Rabbits were treated with phenylephrine (50 mg/kg, I.V., n=6) or an equivalent volume of saline (vehicle, n=6). Twenty four hours after pre-treatment, isolated hearts were perfused with a bovine erythrocyte suspension in modified Krebs solution, subjected to 45 minutes of normothermic global ischemia and reperfused for 120 minutes. Infarct size was determined by triphenyl tetrazolium chloride staining. Apoptosis was quantified by the deoxynucleotidyl transferase mediated dUTP nick end labeling and confocal microscopy. Left ventricular tissue from separate groups of animals (n=5 per group), 24 hours after pretreatment with phenylephrine or vehicle but without ischemia and reperfusion, was analyzed by Western blotting for the content of BCLx and BAX proteins.

**RESULTS:** Hearts pretreated with phenylephrine 24 hours prior showed improved end-reperfusion diastolic recovery (26.7 ± 4.5 vs. 42.3 ± 5.0 mmHg, p=0.04) and developed pressures (56.8 ± 4.9 vs. 36.2 ± 3.9 mmHg, p=0.008), reduced infarct size (9.9 ± 2.4 vs. 42.6 ± 6.3 %, p=0.002) and decreased number of apoptotic nuclei (24.0 ± 4.8 vs. 51.0 ± 4.6 per HPF, p= 0.038). Analysis by Western blotting showed the ratio of BCLx to BAX protein significantly increased in phenylephrine pretreated hearts (2.65 ± 0.5 vs. 1.00 ± 0.1, p=0.008).

**CONCLUSIONS:** Delayed myocardial protection induced by al-adrenoceptor activation involves an increased BCLx to BAX ratio, thereby limiting apoptotic cell death.

**By Invitation*

### L7. Collagen Impregnated Dacron Grafts Resist Infection as Well as Cryopreserved Allografts in the Thoracic Aortic Position

Norman M. Rowe*, Joshua H. Burack*, Nuria M. Lawson*, Paul Impellizzeri*, Yoon D. Kim*, Marcel Sierra*, Peter Homel*, Anthony J. Acinapura and Joseph N. Cunningham, Brooklyn, New York

**OBJECTIVE:** The purpose of this study was to study various graft materials response to placement in the proximal thoracic aorta in the presence of severe intraoperative or postoperative bacteremia.

**METHODS:** Thirteen immature Yorkshire pigs underwent a transverse thoracotomy and were randomized to receive either placement of collagen impregnated dacron grafts (CIDG; n=6) or cryopreserved (CPA; n=7) patch grafts in the ascending aorta. All animals were infused with a control strain of *Staphylococcus aureus* 18-24 hours after surgery. Animals were sacrificed 8 weeks later, the grafts were explanted and analyzed via a combination of microbio-logic culture and a

histologic grading scale for evidence of infection using the Binomial test with significance set at $p<0.05$.

**RESULTS:** The overall mortality rate was 7.7% (1/13) with one animal dying of overwhelming sepsis in the CPA group. The overall infection rate was 38.5% (5/13) with 80% (4/5) of these occurring in the CPA group. The infection rate in the CPA group was 57.2% (4/7) and only 16.7% (1/6) in the CIDG group (see table).

**CONCLUSIONS:** This study demonstrates for the first time that a prosthetic graft in the proximal thoracic aortic position is as effective (*$p<0.0079$) and approaching statistical significance for being superior to allograft in resisting bacterial inoculation. This newly developed model is currently being utilized for controlled investigation of cytokine markers of graft infection.

**Results of Graft Infections**

| | CIDG | CPA | TOTAL |
|---|---|---|---|
| **Infected Grafts** | 1 | 4 | 5 |
| **Non-Infected Grafts** | 5 | 3 | 8 |

**By Invitation*

## L8. Impact of Body Mass Index and Albumin on Cardiac Surgery Morbidity and Mortality

Daniel T. Engelman*, Robert J. Rizzo*, John G. Byrne*, Gregory S. Couper*,Sari F. Aranki*, John J. Collins and *David H. Adams*, Boston, Massachusetts

**OBJECTIVE:** Extremely thin and overly obese patients may not tolerate cardiac surgery as well as other patients. A retrospective study was conducted to determine whether the extremes of body mass index (BMI) [weight/height$^2$ (kg/m$^2$)] and/or cachexia increased the morbidity and mortality associated with cardiac surgery.

**METHODS:** BMI was used to objectively measure thinness (BMI <20)and heaviness (BMI >30); preoperative serum albumin was used to quantify nutritional status and underlying disease. Data were gathered between 1993-1997 from 5168 consecutive patients undergoing coronary artery bypass and/or valve surgery.

**RESULTS:** There was no correlation between BMI and albumin. Low BMI (<20) and low albumin (<2.5) were each associated with increased mortality. Patients with high BMI's (>30) had significantly increased mortality only when associated with low albumin (<2.5). Multivariate analysis demonstrated that a low albumin or low BMI was independently associated with increased reoperation for bleeding, postoperative renal failure, and prolonged ventilation, ICU stay, and total length of stay. High BMI was associated with increased sternal wound infection and saphenous vein harvest site infection.

**Percentage Mortality by BMI and Preoperative Serum Albumin**

| | BMI <20 | BMI 20-30 | BMI >30 | all patients |
|---|---|---|---|---|
| **Albumin <2.5** | 16 (n=49) | 7 (n=589) | 8 (n=130) | 7 (n=768) |
| **Albumin 2.5-3.5** | 9 (n=81) | 3 (n=1365) | 5 (n=464) | 4 (n=1910) |
| **Albumin >3.5** | 7 (n=107) | 3 (n=1777) | 3 (n=606) | 3 (n=2490) |

| | | | | | |
|---|---|---|---|---|---|
| **all patients** | 10 (n=237) | 3 (n=3731) | 4 (n=1200) | 4 (n=5168) | **CONCLUSIONS:** Hypoalbuminemia and low BMI each independently increase morbidity and mortality following cardiac surgery. |

†1992-94 *AATS Research Scholar*

**By Invitation*

**9:00 a.m. SCIENTIFIC SESSION**

Ballroom, Ernest N. Morial Convention Center

*Moderators: Lawrence H. Cohn, M.D.*

*Tirone E. David, M.D.*

*BASIC SCIENCE LECTURE*
Ballroom, Ernest N. Morial Convention Center

**Gene Therapy Strategies for Research Revascularization**

***Victor Dzau,M.D***.

*Hersey Professor of the Theory and Practice of Physic Medicine Harvard Medical School, Chairman, Department of Medicine, Brigham and Women's Hospital, Boston, Massachusetts*

**33. A Clinical Trial Combining Donor Bone Marrow Infusion and Heart Transplantation: Intermediate-Term Results**

Si M. Pham*, Abdul S. Rao*, Adriana Zeevi*, Kenneth R. McCurry*, Robert L. Kormos, Paulo Fontes*, Brack G. Hattler*, John J. Fung*, Thomas E .Starzl* and Bartley P. Griffith, Miami, Florida and Pittsburgh, Pennsylvania

*Discussant: Verdi J. DiSesa, M.D., Chicago, IL*

**OBJECTIVE:** We have demonstrated that donor cell microchimerism is augmented by the infusion of donor bone marrow at the time of solid organ transplantation. We report herein the intermediate-term results of a trial combining donor bone marrow (BM) insusion and heart transplantation.

**METHODS:** From 9/93 to 9/97, 29 patients receiving concurrent heart transplant and infusion of unmodified donor donor bone marrow ($3.6 \times 10^8$ cells/ kg). During that period, 24 heart recipients who did not received bone marrow (because a lack of consent for bone marrow donation) served as comptemporaneous controls. Initial immunosuppression regimen consisted of two drugs: tacrolimus and steroids. A third drug (azathioprine or mycophenolic mofatil) was added as needed (for rejection or sparing renal toxicity). The mean follow-up were 844 ± 506 and 824 ± 327 days for the BM, and control groups, respectively.

**RESULTS:** The overall survival rates for the BM and control groups were 90% (26/29) and 88% (21/24), respectively. No complication or death was associated with bone marrow infusion. Of those patients who had been followed for ‰¥ 1 years, 72% (18/25) of the BM group never experienced an episode of moderate to severe acute rejection (grade ‰¥3A) during the 1st year as compared with 32% (6/19) in the control group ($p < 0.05$). There was no difference in the rate of donor-specific immunomodulation between two groups by mixed lymphocyte reaction assay (18%

in BM group vs 15% in controls). However there is a trend toward less lymphocyte growth from heart biopsies in the BM group (28/150 biopsies {18%} in BM group versus 27/102 {26%}in controls)

**CONCLUSIONS:** Infusion of donor bone marrow at the time of heart transplantation appears to reduce the acute rejection rate. Its impact on coronary allograft vasculopathy is yet to be determined.

**By Invitation*

**34. Autologous Cardiomyocyte Transplantation Improved Porcine Heart Function after a Myocardial Infarction**.

[§]Ren-ke Li*, [§]Richard D. Weisel, [§]Donald A.G. Mickle*, Terrence M. Yau*, Robert Burns*, Robert J. Cusimano*, Leonard Schwartz*, Eung-ioong Kim*, Tetsuro Sakai*, Shinji Tomita*, Patty Boylen* and Zhi-qiang Jia*, Toronto, Ontario, Canada

*Discussant: Chu-Jeng (Ray) Chiu, M.D., Montreal, Quebec, Canada*

**OBJECTIVE:** We previously demonstrated that fetal cardiomyocyte transplantation into a cardiac scar improved heart function. However, the allograft was rejected despite cyclosporin therapy. Therefore, we evaluated autologous cardiomyocyte transplantation in adult swine.

**METHODS:** In 19 adult pigs a myocardial infarction was created by occlusion of the distal LAD with an inrraluminal coil. Four weeks after infarction, ECHO showed a dyskinetic anterorapical region and a SPECT MIDI scan showed minimal perfusion and very few viable myocytes in the infarcted region. The ejection fraction was 26±4%. Cardiomyocytes were isolated and cultured from the right ventricular septum at the time of infarction and the number of cells was expanded *in vitro* for 4 weeks. Through a left thoracotomy either cells (N=12) or culture medium (N=7) were injected into the infarcted region.

**RESULTS:** Eight weeks after transplantation, SPECT MIDI scans demonstrated improved perfusion, motion and thickening of the infarcted region in the transplanted group and no improvement in the control group. Ejection fraction increased in the transplant group(38±6%) but not in the control group (28±5%, $p<0.05$). Conductance and intravenrricular pressure catheters revealed better preload recruitable stroke work and end-systolic elastance in the transplanted hearts ($p<0.05$). Histological studies revealed transplanted cardiomyocytes within the infarcted region in the transplanted group but not the control group. The transplanted pigs were more active and gained more weight ($p<0.01$) than control pigs. The clinical benefit of autologous cell transplantation was dramatic.

**CONCLUSIONS:** Autologous cardiomyocyte transplantation may improve perfusion and restore heart function after an extensive myocardial infarction.

**10:10 a.m. INTERMISSION - VISIT EXHIBITS**

*[§]Authors have a relationship with Genzyme, Corp.*

**By Invitation*

**10:55 a.m. SCIENTIFIC SESSION**

Ballroom, Ernest N. Morial Convention Center

*Moderators: Lawrence H. Cohn, M.D.*

*Tirone E. David, M.D.*

**C. WALTON LILLEHEI RESIDENT FORUM**

**AWARD PRESENTATION**

### 35. Human Factors and Surgical Outcomes. A Multicentre Study

†Marc R. De Leval, Jane Carthey*, David J. Wright*, Vernon T. Farewell* and James Reason*, London, England

*Discussant: John J. Lamberti, M.D., San Diego, CA*

**OBJECTIVE:**
Surgical failures could be analysed as accidents in complex socio-technical systems (eg aviation) in which human factors (HF) play a determinant role. This was tested on the arterial switch operation.
**METHODS:**
All 230 arterial switch operations performed in the UK over 18 months were studied. As well as clinical data, individual, team, situational and organisation HF data were collected: these included questionnaires and lists of negative events (NE) with lists of human compensations (HC). Outcomes (OC) were divided into: OC1 extubated within 72 hrs; OC2 delayed extubation; OC3 serious complications; OC4 death. HF data were added to baseline logistic regression analyses of clinical data.
**RESULTS:**
Mortality was 6.5%. A subset of coronary patterns (CP) was the most determinant risk factor of death ($p<0.001$). Here, surgeon awareness of CP carried a risk of 8.3% against 30.4% in misdiagnosed cases. There were 155 NE; but no deaths occurred with HC against 39.4% mortality without HC. Without HC the estimated odds of death increased by a factor of 5 per NE (95% CL 2-12). OC4 and OC3 combined occurred in 25.2% of cases. They include 87.9% of NE without HC. Without HC the estimated odds of OC4 and OC3 per NE increased by a factor of 37 (95% CL 9-44).
**CONCLUSIONS:**
HF influence OC, particularly in high-risk cases. HC can prevent NE becoming serious failures. Applying accident theories to surgery, a novel form of audit, could improve safety and results, and help understand individual and institutional differences.
*†1973-74 AATS Graham Fellow*

**By Invitation*

### 36. Lung Transplantation for Pulmonary Fibrosis: A Ten Year Institutional Experience

Bryan F. Meyers*, John P. Lynch*, Elbert P. Trulock*, Joel D. Cooper, and G. Alexander Patterson, St. Louis, Missouri

*Discussant: R. Morton Bolman, III, M.D., Minneapolis, MN*

**OBJECTIVE(s):** Between 7/88 and 7/98 we performed 433 lung transplants, including 48 transplants in 47 patients for pulmonary fibrosis (PF). The operations for PF include 15 bilateral

sequential lung transplants (BLT) and 33 single lung transplants (SLT). Our objective of this study is to retrospectively review this experience and compare the suitability of SLT versus BLT for pulmonary fibrosis.

**METHODS:** A retrospective review including inpatient hospital charts, outpatient clinic records and telephone contact to patients to verify current health status.

**RESULTS:** All recipients had severe restrictive lung disease and required continuous supplemental oxygen pre-transplant. Peri-operative mortality was 4 patients (8.5 percent). One patient underwent unsuccessful redo BLT for irreversible reperfusion injury and graft failure after an initial SLT. The median hospitalization, intensive care unit stay, and time on mechanical ventilation were 22 days, 5 days, and 4 days.

Actuarial survival for these 47 patients at 1, 2, and 5 years was 75.4 percent, 72.7 percent, and 49.9 percent, slightly poorer than our institutional survival for recipients of all diagnoses: 82 percent, 76 percent, and 53 percent. Eighteen of 43 operative survivors have died. Late causes of death include obliterative bronchiolitis with progressive respiratory failure (9), malignancy (3), and CMV pneumonitis (2).

Hospital mortality was 3/33 (9.1 percent) after SLT and 1/14 (7.1 percent) after BLT. There have been 12/30 (40 percent) late deaths after SLT with a mean time to death of 1115 days. After BLT, there have been 6/13 (46 percent) late deaths with a mean time to death of 592 days. There was no difference between SLT and BLT with regard to median hospital stay, ICU stay, or intubation. Four of the 33 (12 percent) SLT patients required tracheostomy while 3 of 14 (21 percent) BLT recipients required tracheostomy.

**CONCLUSIONS:** We conclude that either SLT or BLT offer viable surgical therapy for patients with end-stage pulmonary fibrosis. We can demonstrate no benefit conferred by BLT over SLT for patients with this diagnosis.

**PRESENTATION OF SCIENTIFIC ACHIEVEMENT AWARD**

Michael E. DeBakey, M.D., Houston, TX

**11:45 a.m. ADDRESS BY HONORED SPEAKER**

Ballroom, Ernest N. Morial Convention Center

**Experimental and Clinical Application of Angiogenesis Research**

***Judah Folkman, M.D.***

*Andrus Professor of Pediatric Surgery, Professor of Cell Biology Harvard Medical School, Senior Associate in Surgery, Children's Hospital, Boston, Massachusetts*

**ADJOURN FOR LUNCH - VISIT EXHIBITS**

*By Invitation

# TUESDAY AFTERNOON, APRIL 20, 1999

**1:45 p.m. SIMULTANEOUS SCIENTIFIC SESSION A-2 -**

**ADULT CARDIAC SURGERY**

Ballroom, Ernest N. Morial Convention Center

*Moderators: Bruce W. Lytle, M.D.*

*D. Glenn Pennington, M.D.*

**37. The Impact of the Maze Procedure on the Incidence of Stroke Due to Atrial Fibrillation**

James L. Cox, Niv Ad*, Washington, DC

*Discussant: Hartzell V. Schaff, M.D., Rochester, MN*

**OBJECTIVE:** The incidence of stroke in non-anticoagulated patients with atrial fibrillation is 1-12 % per year, depending on associated risk factors. Warfarin therapy dramatically decreases the incidence of stroke but despite its effectiveness, the number of strokes due to atrial fibrillation in the U.S.A. remains at approximately 75,000 per year. This study evaluates the effect of abolishing atrial fibrillation with the Maze procedure on the subsequent incidence of stroke in both low-risk and high-risk patients.

**METHODS:** Between September, 1987 and September, 1998, we performed the Maze procedure on 275 patients with intermittent or chronic atrial fibrillation. Fifty-two (19 %) of those patients (Group I) had suffered at least one previous stroke, TLA, or systemic embolic episode directly attributable to atrial fibrillation prior to surgery. Two hundred twenty-three patients (Group II) (81%) had not experienced any evidence of thromboembolism preoperatively. These groups were compared to the expected rate of stroke in anticoagulated patients with atrial fibrillation.

**RESULTS:** During the follow-up period of up to 11 years (mean: 50.1+34.1 months), statistics would predict that even with appropriate warfarin therapy in all patients, 24 patients would have had a thromboembolic stroke. However, there were no thromboembolic strokes in either Group I or Group II patients undergoing the Maze procedure during the follow-up period despite the lack of anticoagulation postoperatively.

**CONCLUSIONS:** The Maze procedure abolishes the risk of stroke associated with atrial fibrillation.

*By Invitation

### 38. T-Grafts With Bilateral ITA Versus Left ITA And Radial Artery: Flow Dynamics In The ITA Mainstem

Olaf Wendler*, Benno Hennen*, Torsten Markwirth*, Dietmar Tscholl*, Qi Huang*, Erfane Shahangi* and Hans-Joachim Schsfers*, Homburg Saar, Germany

*Sponsored by: Hans G. Borst, M.D., Munich, Germany*

*Discussant: Alfred J. Tector, M.D., Milwaukee, WI*

**OBJECTIVE:** Complete arterial coronary artery bypass grafting (CABG) with two grafts may be attained in triple vessel disease when a T-configuration is employed. There is still scepticism wether the coronary flow reserve (CFR) in the ITA-mainstem is sufficient to supply more than one anastomosed coronary vessel.

**METHODS:** Between 3/96 and 9/98 118 patients (pts) (102 male; mean age 59 years) with triple vessel disease received complete arterial CABG with T-grafts. In 57 bilateral skeletonized ITAs (group I) and in 61 pts left skeletonized ITA and RA (group II) were used as conduits. A mean of 4.04 (3-6) (I) versus 4.33 (3-6) (II) coronary vessels were anastomosed per patient. One week postop-eratively resting flow in 32 (I) and 30 pts (II) was measured in the proximal ITA using a doppler guide wire (Cardiometrics FloWire; 0.014 inches). Maximum flow was determined after injection of adenosin (30 mg/kg). Six months later the investigation was repeated in 12 (I) and 9 pts (II).

**RESULTS:** The in-hospital mortalities were 3.5%(I) versus 0% (II). No case of bleeding, sternal wound infection or dehiscence occurred. On angiography 94.6% (I) versus 96.4% (II) of vessels were patent. ITA-mainstem flow after stimulation with adenosin increased significantly in all pts (p‰¤0,0001). There was no significant difference between **base-line flow, maximum flow and CFR** in the ITA-mainstem between the two groups. **1 week: (I)**78.3±33.8 / 136.0+54.8 / 1.81±0.30 **(II)** 64.0±30.3 / 115.1±49.3 / 1.87±0.34 **6 months: (I)**42.9±15.2 / 118.9±37.3 / 2.89±0.82 **(II)** 67 .6±40.5 / 142.9±80.3 / 2.49±0.51 (flow data in ml/min±sd) CFR, however, increased significantly in both groups after the first six postoperative months (p‰¤0,0001).

**CONCLUSIONS:** Bilateral ITA or left ITA and RA as T-grafts produce complete arterial revascularization with good perioperative results. Using the T-graft configuration the CFR of ITA-mainstem is identical with puplished results after single CABG with ITA. Therefore revascularization with T-grafts using two arteries (ITA or RA) results in adequate coronary perfusion and portends good long-time prognosis of arterial revascularization.

**By Invitation*

### 39. Isolated LITA to LAD: Late Consequences of Incomplete Revascularization

Roslyn Scott*, Eugene H. Blackstone, Patrick M. McCarthy, Bruce W. Lytle, Floyd D. Loop, Jennifer White* and Delos M. Cosgrove, Cleveland, Ohio

*Discussant: Antonio Maria Calafiore, M.D., Cheiti, Italy*

**OBJECTIVE:** Early and long-term outcome of isolated left internal thoracic artery (LITA) to the left anterior descending coronary artery (LAD) is the benchmark for minimally invasive and percutaneous intervention. However, changing indications for revascularization according to extent

of disease over the years presents the opportunity to assess the impact of existing residual non-LAD system stenoses at the time of LITA-LAD.

**METHODS:** 2072 pts underwent primary isolated LITA-LAD 1971-1997. Using a 50% stenosis criterion, 26% and 13% would nowadays be considered 2-or 3-system disease, respectively. Incomplete revascularization fell from 60% in the early 1970s to 20-25% from the 1980s onward. Multivariable analysis of detailed coronary stenosis variables was conducted in the hazard-function domain for the 24,300 patient-years of follow-up (mean 12±6.0).

**RESULTS:** Survival was 99%, 86%, and 57% at 1,10, and 20 yrs. In addition to expected risk factors, presence of a 70% or greater lesion in the circumflex (LCx), but not in the right coronary (RCA) systems, was a late-phase risk factor (70% and 38% survival at 10 and 20 yrs with LCx disease vs. 89% and 62% without, P<.0001). Any left main disease unfavorably affected survival (P=.003). In contrast, residual stenoses, particularly in the RCA (P=.001), were unrelated to survival and only weakly predictive of later reintervention (60% free at 20 yrs).

**CONCLUSIONS:** Residual stenoses in non-LAD systems after LITA-LAD are poorly predictive of late reintervention, but residual LCx or left main disease importantly reduces late survival.

**By Invitation*

### 40. Prospective Randomized Comparative Study of Brain Protection in Total Aortic Arch Replacement: Deep Hypothermic Circulatory Arrest with Retrograde Cerebral Perfusion or Selective Antegrade Cerebral Perfusion

Yutaka Okita*, Kenji Minatoya*, Osamu Tagusari*, Motomi Ando* Kazuyuki Nagatsuka* and Soichiro Kitamura, Osaka, Japan

*Discussant: Randall B. Griepp, M.D., New York, NY*

**OBJECTIVE:** Comparing results of total aortic arch replacement with two different methods of brain protection, particularly in view of neurological outcomes.

**METHODS:** From June 1997 to September 1998, 47 consecutive patients who underwent total arch replacement through a midsternotomy were randomly allocated based upon the two methods of brain protection: deep hypothermic circulatory arrest with retrograde cerebral perfusion (RCP: 22 patients) and selective antegrade cerebral perfusion (SCP: 25 patients). Patients who had acute aortic dissection or ruptured aneurysm were excluded. Mean ages were 69±8 years. Pre- and postoperative (3 weeks) brain CT scan, neurological examination, and cognitive function tests were performed. Serum 100b protein was assayed before and after cardiopulmonary bypass, 24 hours, and 48 hours after the operation. The whole bypass time was shorter in the RCP (176±53 vs 218±89 min. p=0.05). Duration of the RCP was 46±15 min. and SCP was 116+42 min. No differences existed in patients' demography and other details of surgery.

**RESULTS:** Hospital mortality was noted in 2 patients only in the SCP (8.0%, p=0.18). New strokes occurred in 1(4.5%) in the RCP and in 1 (4.0%) in the SCP (p=0.93). The incidence of transient brain dysfunction was significantly higher in the RCP (8,36.3% vs 2,8.0%, p=0.02). Excluding the patients with strokes, S-100b values were identical in two groups, (RCP:SCP, pre-bypass 0.01±0.03: 0.002±0.01, post-bypass 1.90±0.97:1.96±1.00, 24 hours 0.67±0.58: 0.59+0.26, 48 hours 0.39±0.30: 0.31±0.13 mg/L, respectively. p=0.69). Postoperative wake-up time (RCP 6.1±3.3; SCP 5.2±2.4 hours, p=0.32) and extubation time (RCP 13.4±7.5; SCP 12.3±5.7 hours, p=0.60) were equal. Declined scores of the memory (RCP 0.74±0.99; SCP 0.55±1.19, p=0.59), orientation (RCP 1.11±1.29; SCP 0.50+.0.76, p=0.08), and intellectual function (RCP 1.21±1.27;

SCP 1.05±1.15, p=0.68) showed no difference. Postoperative CT showed no abnormalities in either group. Stay in the ICU (RCP 3±1; SCP 4±3 days, p=0.15) and in the hospital (RCP 34±21; SCP 29±10 days, p=0.36) was equivalent.

**CONCLUSIONS:** Both methods of brain protection for patients undergoing total arch replacement provided acceptable mortality and morbidity. However, the incidence of transient brain dysfunction was significantly higher in patients with the RCP.

**3:05 p.m. INTERMISSION**

**By Invitation*

**3:50 p.m. SIMULTANEOUS SCIENTIFIC SESSION A-2 - ADULT CARDIAC SURGERY**

Ballroom, Ernest N. Morial Convention Center

*Moderators: Bruce W. Lytle, M.D.*

*D. Glenn Pennington, M.D.*

### 41. Competing Risks after Bypass Surgery: The Influence of Death on Reintervention

Eugene H. Blackstone and Bruce W. Lytle, Cleveland, Ohio

*Discussant: Robert H. Jones, M.D., Durham, NC*

**OBJECTIVE:** Extensive arterial grafting lowers the incidence of reintervention (REINT), but is being performed in older, higher risk patients. Is reduced REINT real or simply the passive result of dying before needing REINT?

**METHODS:** Multivariable competing risks analysis was performed of 2001 patients undergoing CABG using bilateral internal thoracic artery (ITA) conduits (BITA) and 8123 receiving single ITA conduits (SITA) for the events death before REINT, REINT by angioplasty (PTCA), and redo CABG. Mean follow-up was 9.7±3.0 yrs and 10.8±5.2 yrs for the BITA and SITA groups, respectively.

**RESULTS:** BITA provided better survival (difference of 5% at 10 yrs, P<.0001) and fewer REINT (difference of 5% in redo CABG, P<.0001, but no difference in PTCA), while older age was associated with poorer survival (P<.0001) and fewer REINT (P<.0001). Death impacted estimates of REINT prevalence more often in SITA than BITA because of the simultaneous effects of decreased mortality and REINT.

**% in Categories at 12 Years**

| | Age 35 | | Age 70 | |
|---|---|---|---|---|
| **Category** | **SITA** | **BITA** | **SITA** | **BITA** |
| **Alive, no REINT** | 23 | 65 | 37 | 56 |
| **Dead before REINT** | 8 | 8 | 35 | 28 |
| **PTCA** | 43 | 24 | 20 | 12 |
| **Redo CABG** | 26 | 3 | 8 | 3 |

**CONCLUSIONS:** Across all ages, after accounting for death's confounding influence, more extensive arterial grafting was associated with fewer REINTs. However, at older ages, its influence on redo CABG narrows considerably.

**By Invitation*

### 42. Clinical Benefits of Endoscopic Vein Harvesting in Coronary Artery Bypass Patients With Risk Factors for Saphenectomy Wound Infections.

Phillip A. Carpino*, Kamal R. Khabbaz*, Robert M. Bojar*, Hassan Rastegar*, Kenneth G. Warner*, Richard E. Murphy* and Douglas D. Payne*, Boston, Massachusetts

Sponsored By: Benedict D. T. Daly, Boston, Massachusetts

*Discussant: Robert J. March, M.D., Chicago, IL*

**OBJECTIVE:** The impact of the use of endoscopic techniques on the incidence of complications in the saphenectomy incision after coronary artey bypass surgery (CABG) is not defined for patients with higher risks for developing wound infections (WI).

**METHODS:** In 1473 CABG patients who had the saphenous vein harvested by either a continuous incision or skip incisions leaving intact skin bridges, the incidence of WI was 9.6%. The following variables were entered into a logistical regression analysis to identify the significant risk factors that are predictors of WI: Diabetes (DM), peripheral vascular disease, obesity, renal failure, steroid use, age, gender, and type of closure. We then randomized prospectively 132 patients found to be at high risk of WI to either endoscopic vein harvesting approach (ENDO) or continuous open incision (OPEN).

**RESULTS:** A univariate analysis showed female gender (p=0.0314), DM (p=0.002), and obesity (p=0.002) to be predictors of WI. In a mulrivariate model, only DM (p=0.022) and obesity 9p=0.025) were independent predictors. The incidence of wound infection in the high-risk group was 4.5% for the ENDO group vs. 20% for the OPEN group (p=0.002). However, the vein procurment time was longer in the ENDO group (65 min vs. 32 min.) and so was the number of vein repairs required (2.5 vs. 0.8).

**CONCLUSIONS:** The use of endoscopic vein harvesting decreases the incidence of post operative saphenectomy infections in patients with DM and or Obesity, independent predictors of that problem. Wether this translates into an economic benifit that justifies the additional cost of theat technology requires further complex analysis.

**By Invitation*

### 43. Systematic Off-Pump Coronary Artery Revascularization in Multi-Vessel Disease: Experience of 230 Cases

Raymond Carrier*, Stacey Brann*, Francois Dagenais*, Raymond Martineau* and Yves Leclerc*, Montreal, Quebec, Canada

*Sponsored By: Michel Carrier, Montreal, Quebec, Canada*

*Discussant: Stephen E. Colvin, M.D., New York,* NY

**OBJECTIVE:** We report our recent experience with systematic off-pump coronary artery revascularization for multi-vessel disease.

**METHODS:** Between September 1996 and June 1998, 230 off-pump revascularization representing 80% of all revascularizations done during this time frame and 95% since January 1998 were performed by a single surgeon (RC) at the Montreal Heart Institute. There were 184 men and 46 women averaging 63.2±10.9 years old. Sixteen (7%) procedures were reoperative surgery. Main indication was unstable angina (61%). An average of 2.88±0.6 (1-5) grafts/ patient was performed, majority (70%) being triple and quadruple bypasses.

**RESULTS:** Complete revascularization was achieved in 91 % of the patient and single or double internal thoracic artery, saphenous vein, radial, and gastro-epiploic arteries were employed in respectively 95, 84, 10 and 1% of the patients. Coronary artery mechanical stabilization (Coroneo Corvasc. patent pending) and heart ventricalizing technique were used to reach circumflex area. Average total ischemic time was 29.8±0.9 (8-65) min. 68% of the patient did not require transfusion. Four percent of the patients were reexplored for bleeding, 2.5% experienced transmural myocardial infarction and only one required postoperative aortic counterpulsation assistance. There were two operative death, one due to multi-organ failure non-cardiac related and one from malignant arrhythmia already present prior to the surgery. Three patients experimented early recurrent angina and two among them had negative investigation. Early angiograms performed on the first 12 patients confirmed 100% patency with excellent run-off (95%).

**CONCLUSIONS:** With adequate surgical technique, complete coronary revascularization can be achieved without extracorporeal circulation in a majority of patients with excellent result and low morbidity and mortality.

**4:50 p.m. EXECUTIVE SESSION (MEMBERS ONLY) - Ballroom**

**6:30 p.m. MAGIC OF MARDI GRAS RECEPTION**

**MARDI GRAS WORLD**

**By Invitation*

**1:45 p.m. SIMULTANEOUS SCIENTIFIC SESSION B-2 - GENERAL THORACIC SURGERY**

Room 211-213, Ernest N. Morial Convention Center

*Moderators: David J. Sugarbaker, M.D.*

*Carolyn E. Reed, M.D.*

**44. Induction Chemotherapy Prior to Surgery for Early Stage Lung Cancer - A Novel Approach**

Katherine M. W. Pisters*, Robert J. Ginsberg and Paul A. Bunn*, Houston, Texas, New York, New York and Denver, Colorado

*Discussant: Mark K. Ferguson, M.D., Chicago, IL*

**OBJECTIVE:** Patients (Pts) with clinical stage Ib, II and T3 N1 non-small cell lung cancer(NSCLC) have a poor survival following with surgery-less than 50% are cured. Adjuvant treatment has had little impact. Induction chemotherapy(CT) for N2 disease improves long-term

survival. Newer CT has proven tolerable, user-friendly, and effective in advanced NSCLC. A phase II trial assessed the feasibility-measured by response rate, toxicity, resectabil-ity rate, and surgical morbidity and mortality-of perioperative paclitaxel and carboplatin in pts with early stage NSCLC-proven by mediastinoscopy and imaging studies.

**METHODS:** Pts with T1 N0 or superior sulcus tumors were excluded. All pts required adequate medical parameters to undergo induction CT and surgery. CT consisted of paclitaxel:225mgm/$M^2$3hr infusion, and carboplatin:AUC=6 every 21 days for 2 cycles prior to Surgery. Three further cycles of CT were given to RO pts. Rl,2 pts were deemed off-study.

**RESULTS:** Between 06/97 and 07/98, 94 pts were entered, M/F = 65/29, median age 64yrs. All pts have completed therapy. Major (CR&PR) responses occurred in 50 of 92 pts (54%) eligible for surgery. 9 pts were deemed off-study prior to surgery because of : progression(3), CT reaction(2), death (1), MI(1), lost to follow-up(1), unresectable(1). Of 83pts (90%) explored, 75(82%) were completely resected. Two postop deaths have occurred. Four(4%)pathologic CRs have been observed. There was no increased or unexpected toxicity or surgical morbidity.

**CONCLUSIONS:** Induction CT is feasible and paclitaxel/carboplatin has a high response rate without morbidity in early stage NSCLC. These results have stimulated development of a randomized intergroup trial comparing induction CT and surgery to surgery alone in early stage lung cancer.

*** For the Bimodality Lung Oncology Group (BLOT)**
**By Invitation*

### 45. Pulmonary Hemodynamics Contribute to Indicate Priority for Lung Transplantation in Patients with Cystic Fibrosis

Federico Venuta*, Erino A Rendina*, Giorgio Delia Rocca*, Tiziano De Giacomo*, Francesco Pugliese*, Anna Maria Ciccone* and Giorgio F. Coloni*, Rome, Italy

*Sponsored by: G. Alexander Patterson, M.D., St. Louis, Missouri*

*Discussant: Robert Duane Davis, M.D., Durham, NC*

**OBJECTIVE:** Lung transplantation is a viable therapeutic option for patients with cystic fibrosis (CF). Timing of referral and priority for transplantation are crucial to improve results and minimize mortality on the waiting list. The current strategy, based on pulmonary function tests and deterioration of quality of life, results in a high waiting list mortality. We reviewed the CF population accepted for lung transplantation in our program to ascertain if pulmonary hemo-dynamics could contribute to enhance referral and priority in the waiting list.

**METHODS:** Forty - two CF patients were accepted: 22 were transplanted, 10 died in the waiting list and 10 are still waiting. At the time of evaluation we recorded $FEV_1$, FVC, $FEF_{25-75}$, supplemental $O_2$, $PaO_2/FiO_2$, $PaCO_2$, 6-minute walking test, Right Ventricular Ejection Fraction (RVEF) and pulmonary hemodynamics with and without inhaled nitric oxide. We also recoreded the age at the time of diagnosis, gender, body weight and Schachman score. We compared the data from patients dying on the waiting list (Group I) with patients undergoing lung transplantation (Group II). A comparison was also made within Group II between the data collected at the time of evaluation and at the time of transplantation to quanitfy the functional deterioration during the waiting time.

**RESULTS:** Mean waiting time for Groups I and II was respectively 121 (1-281) and 112 (28-238) days. Age at time of diagnosis, gender, weight, Schachman score, spirometry, 6-minute walking test, RVEF and response to inhaled nitric oxide did not differ between Group I and II. A statistically significant was found for $PaO_2/FiO_2$ (191±54 mmHg in Group I vs 274±63 mmHg in Group II), $PaCO_2$ (64±23 vs 45±5, mmHg), mean pulmonary artery pressure (35±12 vs 23±6, mmHg) cardiac index (4.6±1 vs 3.5±0.6, LÎ£minÎ£$m^2$), pulmonary wedge pressure (6.6±2.4 vs 3±2, mmHg) and intrapulmonary shunt (31±7 vs 23±3m %). The comparison within Group II showed a significative deterioration of pulmonary hemodynamics during the waiting time.

**CONCLUSIONS:** We conclude that pulmonary hemodynamics should contribute to indicate priority for lung transplantation in patients with cystic fibrosis.

**By Invitation*

### 46. A Prospective Randomized Trial Comparing Suction to Water Seal for Air Leaks

Robert J. Cerfolio*, Ramu P. Tummala*, William L. Holman, George L. Zorn*, Charles R. Katholi* and Albert D. Pacifico, Birmingham, Alabama

*Discussant: Claude Deschamps, M.D., Rochester, MN*

**OBJECTIVE:** To compare whether suction or water seal for chest tubes is better at stopping air leaks

**METHODS:** One hundred forty consecutive pt who underwent elective pulmonary resection, were randomized to receive suction or water seal to their chest tubes after postoperative day (POD) #2. On the morning of POD #3, they were randomized to suction or seal. Chest tubes were checked daily for air leaks and were scored from 1 (least) to 7 (greatest) by a leak meter. Air leaks were also classified as forced expiratory, expiratory, inspiratory or continuous. Pt with air leaks that continued after POD #4 who had been randomized to suction were then placed on seal. Pt who had been randomized to seal who developed a pneumothorax were placed to -10 cm of suction.

**RESULTS:** There were 140 pt (96 men). On POD #1, 35 pt had an air leak. It was a forced expiratory leak in 21 pt (60%) and expiratory in 14 (40%). On POD #2, 33 pt had an air leak. It was a forced expiratory leak in 19 pt and expiratory in 14. On POD #3, 33 pt had air leaks, 18 pt were randomized to seal and 15 to suction. Of the 18 pt randomized to seal, air leaks resolved in 12 (66%) by the next morning. Four of the 6 other pt had air leaks greater than 3/7. In the suction group, only 1 pt's air leak resolved. The remaining 14 pt were placed to seal on POD #4 and 13 pt's leaks resolved after 24 hours. Eight pt who were placed on seal had a pneumothorax and 6 had leaks of 3/7 or greater.

**CONCLUSIONS:** Placing chest tubes on water seal is superior to suction for sealing air leaks after pulmonary resection (p=0.001). Water seal does not stop expiratory leaks that are greater than 4/7. Pneumothorax, although rare, can occur after placing chest tubes to water seal, especially with leaks greater than 4/7.

**2:45 p.m. INTERMISSION - Visit Exhibits**

**By Invitation*

**3:30 p.m. SIMULTANEOUS SCIENTIFIC SESSION B-2 - GENERAL THORACIC SURGERY**

Room 211-213, Ernest N. Morial Convention Center

*Moderators: David J. Sugarbaker,* AID.

*Carolyn E. Reed, M.D.*

### 47. Surveillance Transbronchial Lung Biopsies: Implication for Survival after Transplantation

Scott J. Swanson*, John R. Reilly*, Steven J. Mentzer, Malcolm M. Decamp*, Edward P. Ingenito*, Raphael Bueno*, Lester Kobzik*, Jeanne M. Lukanich*, Michael T. Jaklitsch* and David J. Sugarbaker, Boston, Massachusetts

*Discussant: Thomas M. Egan, M.D., Chapel Hill, NC*

**OBJECTIVE:** Does early rejection(rej) after LTX by TBBX predict survival.

**METHODS:** 113 pts had LTX from 1990-1998. We have minimum 1-yr follow-up and results of first 3 TBBX on 89 consecutive pts. Survival was tabulated using Kaplan-Meier lifetable and statistical analysis done by Log-Rank Test. Surveillance TBBX was done in 1st mo then at 3mo and 6mo. Standard immunosuppression was induction therapy with either Minnesota Antilymphocyte Globulin or Antithymocyte Gammaglobulin and methylprednisone and triple drug maintenance: prednisone,CyA,azathioprine. Acute rej was treated with methylprednisolone Igm/dx 3d, persistent acute rej (>2 consecutive) with total lymphoid irradiation and maintenance change to tacrolimus and mycophenolate in 5/9 pts. Blinded grading was done retrospectively using ISHLT classification.

**RESULTS:** l-yr survival for 89 is 79%,51% at 3yr. Survival was not significantly different in subset with rej the 1st (n=36), 1st and 2nd (n=16), or lst, 2nd, 3rd (n=9) or no rej on lst, 2nd, 3rd(n=20) post-LTX TBBX. 61 positive biopsies were graded, 11/36 pts showing > 1 moderate/severe episodes. Survival for this group was not statistically different(p=0.10).

| Rejection # | 1yr% | 2yr% | 3yr% | 5yr% | *p* |
|---|---|---|---|---|---|
| 1 n=36 | 78 n=29 | 62 n=19 | 49 n=10 | 49 n=2 | 0.89 |
| 1,2 n=16 | 93 n=15 | 76 n=9 | 61 n=5 | 61 n=3 | 0.30 |
| 1,2,3 n=9 | 100 n=9 | 76 n=7 | 57 n=4 | 57 n=2 | 0.87 |
| No 1,2,3 n=20 | 85 n=18 | 69 n=14 | 58 n=10 | 45 n=6 | 0.84 |

**CONCLUSIONS:** Surveillance and aggressive treatment of persistent early acute rej leads to survival comparable to pts who do not exhibit early acute rej.

**By Invitation*

### 48. Does Pneumonectomy for Lung Cancer Adversely Influence Long-Term Survival?

Mark K. Ferguson and Theodore Karrison*, Chicago, Illinois

*Discussant: Leslie J. Kohman, M.D., Syracuse, NY (not confirmed)*

**OBJECTIVE:** The increased operative mortality associated with pneumonectomy has stimulated the use of lung sparing operations such as sleeve lobectomy. Whether pneumonectomy adversely affects long-term outcome after resection for lung cancer is unknown.

**METHODS:** We reviewed lobectomy/bilobectomy and pneumonectomy performed for stages I-III non-small cell lung cancer from 1980-97. Kaplan-Meier survival curves were compared using the log-rank test. Covariates were determined for operative mortality and survival using logistic regression analysis and Cox proportional hazards estimation.

**RESULTS:** 258 men and 179 women with a mean age of 62 yrs underwent lobectomy/bilobectomy (334) or pneumonectomy (103). 209 were stage I, 99 were stage II, and 129 were stage III. Operative mortality was 36 (8.2%) overall, 22 (6.6%) for lobectomy/bilobectomy and 14 (13.6%) for pneumonectomy. Mean follow-up was 41 mos (range 0 - 222). Median survival was worse for pneumonectomy (stage II: 17.9 vs 36.3 mos, p=0.05; stage III 11.4 vs 21.1 mos, p=0.07), an effect that was not significant excluding operative deaths (stage II: 21.7 vs 37.8 mos, p=0.14; stage III 14.4 vs 22.0 mos, p=0.17). Covariates for operative mortality were pneumonectomy (relative risk 2.7; 95% C.I. 1.3-5.6) and performance status (2.6; 1.5-4.7). Covariates for survival (operative deaths included, stratified by stage) were age (1.3; 1.1-1.4), performance status (1.4; 1.1-1.8), and postoperative predicted $FEV_1$% (1.2; 1.1-1.3). Pneumonectomy did not achieve statistical significance as a covariate for survival whether operative mortality was included (1.2; 0.8-1.8) or excluded (1.4; 0.9-2.1).

**CONCLUSIONS:** The adverse effect of pneumonectomy on survival relates primarily to its immediate operative risk. We demonstrated no significant long-term adverse influence of pneumonectomy on survival.

*†1998-99 AATS Graham Fellow*

**By Invitation*

### 49. Surgical Resection of Unilateral Lung Metastases: Unilateral or Bilateral Thoracotomy?

Riad N. Younes*, Jefferson L. Gross* and Daniel Deheinzelin*, Sao Paulo, Brazil

*Sponsored by: Adib D. Jatene, M.D., Sao Paulo, Brazil*

*Discussant: Joseph S. Friedberg, M.D., Philadelphia, PA*

**OBJECTIVE:** To evaluate the need for bilateral thoracotomy in patients diagnosed with unilateral lung metastases.

**METHODS:** A retrospective evaluation of a prospective data base from a single institution(1990-1997) of all consecutive patients (n=267) diagnosed on admission with unilateral (n=179) or bilateral(n=88) lung nodules. Ipsilateral thoracotomy was performed on all patients with unilateral disease; contralateral lung was only explored if *de novo* nodules were detected. Bilateral thoracotomy was performed on all patients with bilateral lung metastases. Histology: adenocarcinoma(25%), osteosarcoma(23%), squamous cell carcinoma(18%), soft tissue sarcoma(18%). Median follow-up was 17 months. Contralateral-disease free survival and overall survival were determined. Univariate and multivariate analyses were performed to determine

prognostic factors for overall and contralateral-disease free survival. The 2 groups of patients with confirmed bilateral metastases (synchronous or metachronous) were compared.

**RESULTS:** Acruarial overall 5-year survival was 34.9%. Contralateral-recurrence free 6 months, 12 months, and 5 year survival were 95%, 89%, and 78%, respectively. Patients who recurred in contralateral lung within 3, 6 and 12 months had an overall 5-year survival of 24%, 30%, and 37%, respectively. When patients who recurred in contralateral lung were compared to patients with bilateral metastases on admission, there was no significant difference in overall survival. Only histology and the number of pathologically-proven metastases significantly ($p<0.05$) predicted recurrence in contralateral lung.

**CONCLUSIONS:** Bilateral exploration for unilateral lung metastases is not warranted. Most patients will only have unilateral disease, and delaying contralateral thoracotomy until radiologically detected disease does not affect outcome.

**By Invitation*

### 50. Long-term Results of Cricopharyngeal Myotomy for Muscular Disease

Talat S. Chughtai*, Long-qi Chen*, Dimitrios Nastos*, Raymond Taillefer*, Pasquale Ferraro*, and Andre C. Duranceau, Montreal, Quebec, Canada

*Discussant: Antoon E. M. R. Lerut, M.D., Leuven, Belgium*

**OBJECTIVE:** Muscular disease may cause progressive oropharyngeal dysphagia and tracheobronchial aspiration. When these symptoms are present, short-term improvement has been consistently documented following Cricopharyngeal myotomy. Our aim is to analyze the long-term effects of this operation in patients where muscular dystrophy is responsible for the dysphagia.

**METHODS:** 13 dystrophic patients having undergone Cricopharyngeal myotomy for more than 10 years were retrospectively assessed. The effects of myotomy were measured clinically, using a symptom score (0: no symptom to 3: severe or frequent). Radiologic, manometric and radionuclide pharyngeal emptying studies were obtained. All parameters were measured for both short-term (<6 years) and long-term (>6 years) results.

**RESULTS:**

| | Pre-op | p | Short-term | p | Long-term |
|---|---|---|---|---|---|
| **Symptom (0 to 3)** | | | | | |
| **Dysphagia to solids** | 2.92 | *0.0001* | 0.46 | *0.003* | 1.77 |
| **Regurgitation** | 1.46 | *0.0005* | 0 | *0.002* | 1.23 |
| **Aspiration with meals** | 1.15 | *0.03* | 0.15 | *0.007* | 1.31 |
| **Voice change** | 0.08 | *NS* | 0.15 | *0.006* | 0.85 |
| **Limb weakness** | 0.08 | *NS* | 0.15 | *0.02* | 0.85 |
| **Esophago-gram (%)** | | | | | |
| **Abnormal c-p relaxation** | 69.2 | *0.0003* | 0 | *NS* | 0 |
| **Stasis** | 30.8 | *NS* | 23.1 | *0.02* | 85.7 |
| **Aspiration** | 30.8 | *NS* | 15.4 | *NS* | 57.1 |

| | | | | | |
|---|---|---|---|---|---|
| **Manometry (mm Hg)** | | | | | |
| **UES Resting Pressure** | 35 | *0.01* | 20.89 | *NS* | 25 |
| **UES Closing Pressure** | 51.33 | *0.03* | 31.67 | *NS* | 45.2 |
| **Emptying Scintiscan** | | | | | |
| **% Stasis at 2 min.** | 10 | *NS* | 14.38 | *NS* | 18.57 |

**CONCLUSIONS:** When dystrophy causes debilitating dysphagia, Cricopharyngeal myotomy results in significant and consistent early improvement. Physiologic alterations include a decrease in resting and closing pressures at the pharyngoesophageal junction. Late assessment reveals: 1) the unchanged physiologic effects of the operation 2) reappearance of oropharyngeal symptoms 3) manifestation of dystrophy in previously intact peripheral muscle groups 4) increasing hypopharyngeal stasis with time. Cricopharyngeal myotomy is a palliation in the natural evolution of the dystrophic process.

**4:50 p.m. EXECUTIVE SESSION (MEMBERS ONLY) - Ballroom**

**6:30 p.m. MAGIC OF MARDIGRAS RECEPTION**
**MARDI GRAS WORLD**

**By Invitation*

**1:45 p.m. SIMULTANEOUS SCIENTIFIC SESSION C-2 - CONGENITAL HEART DISEASE**

Room 208-210, Ernest N. Morial Convention Center

*Moderators: Frank L. Hartley, M.D. John E. Mayer, M.D.*

**51. Surgical Reintervention of Neopulmonary Arteries after Complete Unifocalization in Patients with Ventricular Septal Defect, Pulmonary Atresia, and Major Aorto Pulmonary Collaterals**

Vadiyala Mohan Reddy*, Zahid Amin*, Phillip More*, David F. Teitel* and Frank L. Hanley, San Francisco, California

*Discussant: Roger B. B. Mee, M.D., Cleveland, OH*

**OBJECTIVE:** With early and complete one stage unifocalization of major aortopulmonary collaterals and the use of native collaterals, there is concern about the growth of the native collateral tissue and the need for subsequent reintervention. The purpose of this report is to examine the patterns of surgical reintervention after unifocalization and the outcomes.

**METHODS:** Between July 1992 and September 1998,81 patients (median age 7 months, range 10d to 37 yrs)have undergone complete one stage unifocalization with (groupI n=54) or without (groupII n=27) ventricular septal defect closure. All group I patients were evaluated by lung perfusion scans, echocardiography and when indicatedcardiac catheterization. All group II patients underwent elective cardiac catheterization 3 to 6 months after complete unifocalization.

**RESULTS:** Seventy early survivors were followed. There were 4 nonsurgical late deaths. Among the 66 survivors (group I n=46; group II n= 20). In group 1,6 patients (6/46; 13%)required balloon angioplasty and five of these patients also required surgical neopulmonary augmentation. In group II, 15 patients have undergone balloon angioplasty and 12 patients have undergone surgical neopulmonary artery augmentation with successful closure of the VSD 13 patients. The stenosis

were primarily at anastamotic sites,in the central neopulmonary arteries or the native distal stenotic segments of the collateral vessels which could not be surgically addresses (in group II) patients.

**CONCLUSIONS:** Up to 6 year followup shows that incidence of neopulmonary artery reintervention in completely repaired patients is very low. In patients with complicated anatomy neopulmonary rehabilitation is successful in the majority with good hemodynamic outcome

**By Invitation*

### 52. Twenty-five Year Experience with Rastelli Repair for Transposition of the Great Arteries.

†Christian Kreutzer*, Julia De Vivie*, Kimberley Gauvreau*, Guido Oppido*, Jaqueline Kreutzer*, Michael Freed*, John E. Mayer, Richard A. Jonas and Pedro J. Del Nido, Boston, Massachusetts

*Discussant: Gordon K. Danielson, M.D., Rochester, MN*

**OBJECTIVE:** To evaluate the outcome of the Rastelli repair for transposition of the great arteries (TGA).

**METHODS:** From 3/73 to 4/98, 101 pts with d-TGA and ventricular septal defect (VSD) underwent a Rastelli repair. Pts with double outlet right ventricle and bilateral conus were excluded. The mean age at operation was 4.9 ± 5.7 yrs (ld-27 yrs) and the mean weight 16.8 ± 13.3 kg (3.3-71 kg). Pulmonary stenosis was present in 73 and pulmonary atresia in 18; 4 pts had multiple VSD's.

**RESULTS:** Right ventricle to pulmonary artery continuity was achieved by the use of 39 aortic homografts, 18 pulmonary homografts, 18 Hancock conduits, 11 Carpentier Edwards, 6 Dacron, 4 Tascon and 5 right ventricle-pulmonary artery direct anastomosis. The VSD was enlarged in 48 pts. There were 7 early deaths(6.9%) with none in the last 7 yrs. Risk factors for early death by univariate analysis included complete heart block (p=0.02), straddling tricuspid valve (p=0.04), longer cardiopulmonary bypass (p=0.02)and cross clamp times (p=0.04). At a mean follow-up of 6.6 ± 5.7 yrs (1 m to 22 yrs), there were 14 late deaths (5 sudden deaths), and 1 heart transplant. Late arrhythmias developed in 9 pts. Reoperations for conduit stenosis were performed in 45, for left ventricular outflow tract obstruction (LVOTO) in 10, and 31 had catheter interventions for conduit obstruction. Overall freedom from death or transplant (Kaplan-Meier) was 82.5%, 79.1% and 62.7 % at 5, 10 and 15 yrs respectively. Freedom from death or reintervention (transcatheter or surgical) was 43.6%, 16.0% and 8.0% at 5,10 and 15 years of follow up.

**CONCLUSIONS:** Rastelli repair for TGA and LVOTO can be performed with low early mortality. However, there is significant late morbidity and mortality associated with conduit stenosis, LVOTO and arrhythmias.

*†1998-99 AATS Graham Fellow*

**By Invitation*

### 53. Results of Norwood Stage-one Operation: Comparison of Hypoplastic Left Heart Syndrome with Other Malformations

Sabine H. Daebritz*, Georg D. A. Nollert*, Philipe N. Khalil*, John E. Mayer, Pedro J. Del Nido and Richard A. Jonas, Boston, Massachusetts

*Discussant: William I. Norwood, M.D., Wilmington, DE*

**OBJECTIVE:** Norwood stage-one operation is performed in hypoplastic left heart syndrome (HLHS) and other complex malformations with ductus dependent systemic circulation. We investigated the outcome in these two groups of patients.

**METHODS:** Between 1990 and 1998, eight surgeons performed the Norwood stage-one procedure in 194 patients at a median age of 5 days (weight 3.5+/-2.5kg; 32.3% female). Malformations in 131 patients were classified as HLHS in the presence of aortic and mitral atresia or severe stenosis with normal seg-mental anatomy (SDS), intact ventricular septum and hypoplasia of the left ventricle; 63 had other lesions with aortic outflow obstruction: hypoplastic left ventricle and VSD (n=18), unbalanced complete AV-canal (n=9), complex double outlet right ventricle (n=14), single LV or double inlet LV (n=11), tri-cuspid atresia with transposition of the great arteries (n=6), and others (n=5) including heterotaxia.

**RESULTS:** Operative and one year survival was significantly lower for patients with HLHS compared to those with other lesions (63.4% versus 81%, p=0.013 and 55.7% versus 73%, p=0.027, respectively). The presence of a non-hypoplastic left ventricle (n=27) was associated with significantly higher operative survival in uni- and multivariate analysis (96.3% versus 64.7%, p=0.001). Other echocardiographic measurements of anatomical structures such as size of the ascending aorta were not found to have an impact on operative survival. Prematurity was the only additional patient related risk factor (p=0.022).

**CONCLUSIONS:** The outcome of patients with malformations other than HLHS after Norwood stage-one procedure is better than of those with HLHS. The presence of an anatomically left ventricle is the single most important predictor of survival.

**By Invitation*

## 54. Repair of the Hypoplastic Left Heart: Survival, Quality-of-Life, and Cost.

Deborah L. Williams*, Judy H. Ng*, Emily Crawford", Annetine C. Gelijns*, Alan J. Moskowitz*, Constance J. Hayes*, Mark E. Galantowicz* and Jan M. Quaegebeur, New York, New York

*Discussant: John L. Myers, M.D., Hershey, PA*

**OBJECTIVE:** The debate about the hypoplastic left heart syndrome (HLHS) is moving from *whether* to *how* to treat patients with this defect. Beyond survival, little is known about the QoL and costs of alternative treatment approaches. This paper analyzes these endpoints for the Norwood staged repair.

**METHODS:** Between 1993-98, 62 patients underwent staged repair for HLHS (Stage 1:62; Stage 2:25; Stage 3:7; 2 patients required conversion to cardiac transplantation). Survival was analyzed by the Kaplan Meier method, QoL was measured by the Infant/Toddler Child Health Questionnaire (I/T CHQ), developmental status measured by the Ages and Stages Questionnaire (ASQ). Inpatient costs were calculated with the ratio-of-cost-to-charges approach, outpatient costs were calculated using payments.

**RESULTS:** Overall survival at 4.7 years was 56%; survival beyond stage 2 and 3 was 96% and 100%, respectively. QoL ratings (mean 1.9 years, 0-100 scale) were as follows: global health

(89.1±18.7); physical abilities (82.9±21.1); soc. interactions (68.6±9.9);and health percept.(53.9±21.5). However, 47% scored below established norms on the overall ASQ. The mean inpatient cost for stage 1, 2, and 3 repairs was $59,280 (±114,605), $26,700 (±13,215), $38,925 (±26,013), respectively. Total outpatient costs were less than 2% of total costs.

**CONCLUSIONS:** Despite progress, survival after stage 1 remains uncertain and needs improvement. QoL is surprisingly high by parents' standards but developmental status lags behind peers at this early stage. The majority of costs are inpatient costs (which are comparable to cardiac transplantation), while outpatient costs, by contrast, are low.

**2:45 p.m. INTERMISSION - VISIT EXHIBITS**

**By Invitation*

**3:25 p.m. SIMULTANEOUS SCIENTIFIC SESSION C-2 - CONGENITAL HEART DISEASE**

Room 208-210, Ernest N. Morial Convention Center

*Moderators: Frank L. Hartley, M.D.*

*John E. Mayer,* M.D.

**55. Biventricular Repair for Aortic Atresia or Hypoplasia and Ventricular Septal Defect**

Richard G. Ohye*, Koji Kagisaki*, Lisa Lee*, Ralph S. Mosca*, Caren Goldberg* and Edward L. Bove, Ann Arbor, Michigan, Osaka, Japan

*Discussant: Richard A. Jonas, M.D., Boston, MA*

**OBJECTIVE:** Aortic valve atresia or hypoplasia can present with a VSD and a normal mitral valve and left ventricle. These patients may be suitable for a biventricular repair (BVR). The optimal management of aortic atresia/hypoplasia with VSD remains uncertain.

**METHODS:** From 1991-1998, 17 patients with aortic atresia/hypoplasia and VSD underwent BVR. Aortic atresia was present in 5 patients and 12 had aortic valve hypoplasia. Among the group with aortic hypoplasia, Z scores for the diameter of the aortic annulus ranged from -8.8 to -2.7. Associated anomalies were common and included interrupted aortic arch (10), coarctation (5), AP window (1), and heterotaxy (1). Eight patients were staged with an initial Norwood procedure followed by BVR, while 9 were corrected with a single procedure.

**RESULTS:** Among the 8 patients undergoing staged repair, there were no deaths after the Norwood procedure and one death after BVR due to low cardiac output and sepsis. For the 9 patients having a primary BVR, there was one early death due to low cardiac output, and two late deaths from non-cardiac causes. Follow-up ranged from 1 to 85 months (mean, 28 months). Actuarial survival for the entire group was 76 ± 12% at 5 years and was not significantly different for the staged repair group (88%) when compared to the patients undergoing primary BVR (67%). There was no significant morbidity among late survivors.

**CONCLUSIONS:** Both primary and staged BVR for patients with aortic atresia or hypoplasia and VSD may be performed with good late survival. Although the superiority of either approach was not clearly established in this series, patients with diminished pulmonary function, who would

tolerate shunt dependent pulmonary blood flow poorly, should be considered for primary repair. Morbidity and mortality is largely related to associated anomalies.

**By Invitation*

### 56. Does the Degree of Cyanosis Affect Myocardial Bioenergetics and Function?

Hani K. Najm*, Jack Wallen*, Michael P. Belanger*, John G. Coles, Glen S. Van Arsdell*, Michael D. Black*, William G. Williams, and Carin Wittnich*, Toronto, Ontario, Canada
*Discussant: Bradley S. Allen, M.D., Oak Lawn, IL*

**OBJECTIVE:** Animal studies indicated detrimental effects of exposure to chronic hypoxia on myocardial metabolism and function. Whether the presence or the degree of cyanosis adversely affects myocardial bioenergetics, ventricular function and clinical outcome in children.

**METHODS:** 48 children undergoing repair of tetralogy of Fallot were divided according to their preoperative saturation: group I; ‰¥ 90% (n=14), group II; ‰¥80 - 89% (n=16) and group III; 65-79% (n=18). ATP was measured from RV biopsies taken (a) before ischemia, (b) 15 minute of ischemia, (c) end ischemia and (d) 15 minute reperfusion. Ventricular function was assessed by echocardiography in the pre, intra and early postoperative period.

**RESULTS:** Group III had lower ATP levels at baseline (15.1 vs 19.1 vs 21.4 µmol/g/dry wt, group III, II, I respectively, P <0.01 ) and at 15 minutes of ischemia (11.2 vs 14.77 vs 17.6 Î¼mol/g/dry wt, group III, II, I respectively, P <0.01). With reperfusion both cyanotic groups lost further ATP from end is-chemic level compared to an actual recovery in the acaynotic group (-22% vs 20% vs 18%, group III, II, I respectively, P <0.01). Cyanotic children also had lower preoperative ejection fraction (59 vs 66 vs 65%, group III, II, I respectively, P <0.01). Clinical outcome of children in group III was complicated as evidenced by longer ventilatory support (85 vs 31 vs 40 hours, group III, II, I respectively, P 0.07), inotropic support ( 86 vs 38 vs 36 hours, group III, II, I respectively, P <0.01) and intensive care unit stay (160 vs 60 vs 82 hours, group III, II, I respectively, P 0.02).

**CONCLUSIONS:** Cyanotic children undergoing cardiac surgery are at a precarious metabolic and functional status, and these children should be identified to be at a potentially higher risk of complications.

**By Invitation*

### 57. Selective Cerebral Perfusion in Infants/Neonates Undergoing Complex Aortic Arch Reconstruction

Michael D. Black*, Bruno Bissonette* and Vivek Rao*, Toronto, Ontario, Canada

*Sponsored By: Bruce A. Reitz, Stanford, California*
*Discussant: Ross M. Ungerleider, M.D., Durham, NC*

**OBJECTIVE:** Repair of complex congenital heart defects (CHD)involving the aortic arch usually requires deep hypothermic circulatory arrest (DHCA). Unfortunately, DHCA has been associated

with significant postoperative neurologic abnormalities. To avoid DHCA, a novel cardiopulmonary bypass (CPB) technique using selective antegrade cerebral perfusion has been employed.

**METHODS:** We reviewed the clinical records of 17 children who underwent univentricular (n=3) or biventricular(n=14) repair of complex CHD requiring surgery on the aortic arch. In addition to clinical outcomes, we reviewed the postoperative requirement for inotropic support and the adequacy of systemic perfusion as assessed by serial measurements of arterial lactate concentrations.

**RESULTS:** DHCA was completely avoided in 15 children while 2 children (1 Norwood and 1 interrupted aortic arch) required a limited interruption of cerebral blood flow. Aortic x-clamp was avoided in all children without concomitant intra-cardiac anomalies (n=7). The type of aortic repair included patch aortoplasty (n=6), extended end-end anastamosis (n=7), Norwood procedure (n=2) and repair of interrupted aortic arch (n=2). There was one death in a child with univentricular physiology who succumbed to abdominal sepsis (NEC). There were no postoperative neurologic events. Postoperative inotropic support was limited to dopamine and nitroprusside in all patients. The mean postoperative lactate was 3±4 mmol/L(range 1-15).

**CONCLUSIONS:** Repair of complex CHD involving the aortic arch is possible without the use of DHCA. Avoiding DHCA should lower the incidence of postoperative neurologic complications. In addition, the use of selective antegrade perfusion avoids myocardial injury secondary to DHCA and is associated with lower inotropic requirements and improved systemic perfusion.

**4:35 p.m. EXECUTIVE SESSION (Members Only)**
Ballroom, Ernest N. Morial Convention Center

**6:30 p.m. MAGIC OF MARDI GRAS ATTENDEE RECEPTION**

**Mardi Gras World**

**By Invitation*

## WEDNESDAY MORNING, APRIL 21, 1999

**7:00 a.m. GENERAL THORACIC SURGERY FORUM SESSION**

Room 211-213, Ernest N. Morial Convention Center

*Moderators: Mark K. Ferguson, M.D.*

*Valerie W. Rusch, M.D.*

**F1. A Rational Approach to Substitution of The NOcGMP Pathway in Lung Transplantation - Overview of a Series of Experiments**
Ralph Alexander Schmid*, Sven Hillinger*, Gabriele Schoedon* and Walter Weder*, Zurich, Switzerland

*Sponsored By: G. Alexander Patterson, St. Louis, Missouri*

**OBJECTIVE(s):** Impairment of the NO pathway accelerates ischemia/reperfusion injury following lung transplantation (LTPL). Direct application of NO is not ideal because of its short half-life and toxic side effects. Tetrahydrobiopterin (BH4) is the essential coenzyme of the NO synthase (NOS). 8-Br-cGMP is a membrane permeable analogue of cGMP, second messenger of

the NO pathway. We evaluated the effect of several treatment modalities with these compounds on posttransplant lung edema in a large animal model of LTPL.

**METHODS:** Unilateral left LTPL was performed in 25 weight matched out-bred pigs (all groups n=5). Donor lungs were flushed with 1.51 cold LPD solution and preserved for 20h at 1°C. All donors (except group III) received 250mg PGE1 into the pulmonary artery prior to flush. Treatment in experimental groups was as follows: I: BH4 (20mg/kg) to recipient over 30 min, starting before reperfusion; II: as in group I plus continuous dose BH4 (10mg/kg/h); III: 8-Br-cGMP (1mg/kg) to flush solution; IV: 8-Br-cGMP (0.2mg/kg/h) continuously to recipient; V: control. Extravascular lung water index (EVLWI) and hemodynamic parameters (PAP, PVR, CO) were assessed during a five hour observation period. Lipid peroxidation (TEARS) and neutrophil migration (MPO) in the allograft were measured at the end of the assessment.

**RESULTS:** EVLWI (ml/kg) in group I: 8.4±0.9; II: 7.0±.0.5; HI: 6.7±1.0; IV: 8.2±0.3; V: 10.1±0.6, MPO (DOD/mg/min) in group I: 1.1±0.2; II: 1.0±0.1; III: 0.9±0.1; IV: 1.0±0.2; V: 1.7±0.3, TEARS (pmol/g) in group I: 68.2±11.3; II: 65.7±7.9; III: 65.6±7.9; IV: 61.8±12.3; V: 120.8±7.2. No effect on pulmonary and systemic hemodynamic parameters could be detected with either treatment.

**CONCLUSIONS:** Our results show that substitution of the NO pathway by BH4 or 8-Br-cGMP reduces posttransplant pulmonary edema, neutrophil migration, and lipid peroxidation in the allograft. Addition of 8-Br-cGMP to the flush solution is superior to PGE1. Based on pharmacologic considerations of both substances and the presented results, administration of cGMP in the flush solution and BH4 during reperfusion seems to be very promising.

**By Invitation*

### F2. Cyclooxygenase-2 is Up-regulated in Lung Cancer

Fan Zhang*, Amit Kumar*, Yu-chang Wu*, Eun K. Yang*, Robert A. Soslow*, Jamie L. Masferrer*, Alane Koki-Goodson*, Andrew J. Dannenberg* and Nasser K. Altorki, New York, New York, St. Louis, Missouri

**OBJECTIVE:** Prostaglandin (PGs) are important in the pathogenesis of cancer because they affect mitogenesis, cellular adhesion, immune surveillance and apoptosis. Epidemiological studies have suggested that nonsteroidal anti-inflammatory drugs (NSAIDs), known inhibitors of PG synthesis, protect against lung cancer. Two cyclooxygenase (COX) isoenzymes catalyze the conversion of arachidonic acid to PGs: constitutive COX-1 and inducible COX-2. The recent finding that a COX-2 null mutation protected against the formation of intestinal and skin tumors suggests that COX-2 represents an important target for anti-cancer therapy. The goal of this study was to determine whether COX-2 is a potential target for preventing or treating lung cancer.

**METHODS:** The expression and activity of COX-2 were compared in human lung cancer vs. adjacent nontumorous tissue. Levels of $PGE_2$ were determined by enzyme immunoassay. Amounts of COX-2 protein (immunoblotting, im-munohistochemistry) and mRNA (quantitative RT-PCR) were measured in surgical specimens.

**RESULTS:** Levels of $PGE_2$ were increased by about 4-fold in cancerous vs. adjacent non-rumorous tissue (n=19, $p<0.001$). A corresponding increase in amounts of COX-2 protein was detected in both adenocarcinoma and SCC of the lung. Amounts of COX-2 mRNA were also increased in the majority of cases of lung cancer. Immunohistocehmical evaluation indicated that COX-2 protein was expressed in 18/20 lung cancers and localized to malignant epithelial cells.

**CONCLUSIONS:** Levels of COX-2 and PGs are increased in adenocarcinoma and SCC of the lung. These results may explain the prior epidemiological findings that NSAIDs protect against lung cancer. Newly developed selective inhibitors of COX-2 may prove useful in preventing or treating lung cancer.

**By Invitation*

### F3. Proliferation of Airway Epithelial Cells Secondary to Anti-HLA Antibodies: A Potential Mechanism for Bronchiolitis Obliterans Syndrome.

Scott I. Reznik*, Andres Jaramillo*, Wan J. Zhang*, G. A. Patterson, Joel D. Cooper and T. Mohanakumar*, St. Louis, Missouri

**OBJECTIVE:** Development of antibodies to donor HLA after lung transplantation is associated with earlier onset of bronchiolitis obliterans syndrome (BOS) and decreased graft survival. We sought to determine the mechanism by which anti-HLA antibodies effect chronic chronic lung allograft rejection. We postulate that anti-HLA antibodies bind HLA class I molecules on the surface of donor lung epithelium and stimulate phosphorylation and proliferation.

**METHODS:** A549, a lung epithelial carcinoma cell line, was cultured in serum deficient media for 48 hours to produce static growth. Cells were then treated with complete media (15% FBS), media containing HLA-sensitized serum from lung transplant recipients, pooled HLA-sensitized sera, non-sensitized sera or deficient media containing either W6/32 anti-HLA monoclonal antibody or mouse IgG. [$^{3}$H-]-thymidine was determined at 24,48 and 72 hours. In phosphorylation studies, cells treated as above were assayed for tyrosine phosphorylation at one minute by western blot analysis.

**RESULTS:** Cells treated with HLA-sensitized sera exhibited proliferation at 24,48 and 72 hours equivalent or greater than cells treated with complete media. Cells treated with non-sensitized serum showed significantly less proliferation. Treatment with W6/32 induced proliferation at levels equal to or greater than cells treated with complete media. Increased phosphorylation of cellular proteins was observed in cells treated with HLA-sensitized sera and W6/32.

**CONCLUSIONS:** These data indicate that anti-HLA antibodies stimulate lung epithelial cells and may play important role in the development of BOS. Immunosupression of the humoral immune response may be pivotal in the delaying the onset of BOS in patients with HLA reactive antibodies.

**By Invitation*

### F4. Transgenic Swine Lungs Expressing hCD59 in a Pig-to-Human Model of Xenotransplantation

David M. Kulick*, Christopher T. Salerno*, Agustin P. Dalmasso*, Soon J. Park*, Manuel Guzman-Paz*, [§]William Fodor*, [§]Steven Squinto* and R. M. Bolman, III, Minneapolis, Minnesota, New Haven, Connecticut

**OBJECTIVE:** Pulmonary Xenotransplantation is currently limited by hyper-acute rejection mediated in part by xenoreactive natural antibody and complement. Transgenic swine organs, expressing the human complement regulatory protein CD59 (hCD59), have demonstrated improved survival in models of pig-to-primate Xenotransplantation. Our objective was to evaluate

transgenic swine lungs expressing hCD59 in an *ex-vivo* model of pig-to-human Xenotransplantation.

**METHODS:** Transgenic swine lungs (n=4, experimental group) and outbred swine lungs (n=6, control group) were perfused with fresh, whole, human blood via a centrifugal pump on an *ex-vivo* circuit. Functional data were collected throughout each perfusion. Immunoglobulin and comlpement studies were performed on perfusate samples, and both histologic and immunofluorescent analyses were performed on tissue sections.

**RESULTS:** Mean lung survival for the experimental group was significantly increased when compared to controls, 240±0 min vs. 35.3±5.9 min respectively, $p<0.01$. A decreased rise in pulmonary vascular resistance was observed in the experimental group 380±75 dynes-sec-cm$^{-5}$ in contrast to the control group 985±338 dynes-sec-cm$^{-5}$, $p<0.01$. Lung compliance was improved for the experimental group vs. the control group, 8.7±.97 ml-cm$^{-2}$ $H_2O$ and 4.8±1.2 mi-cm$^2$ $H_2O$, respectively, $p<0.05$. SC5b9 levels measured by ELISA were significantly lower for the experimental group vs. controls 901±224 units and 4069±828 units repectively, $p<0.01$. Immunofluorescent examination of tissue sections demonstrated equivalent deposition of IgG, IgM, C1q, and C3 in both groups, with reduced deposition of C9 in the experimetnal group.

**CONCLUSIONS:** Transgenic swine pulmonary xenografts expressing hCD59, demonstrate improved function and survival in an *ex-vivo* model of pig-to-human Xenotransplantation.

*§Authors have a relationship with Alexion Pharmaceuticals*

**By Invitation*

### F5. Prolonged Discordant Lung Orthotopic Xenograft Survival

Paolo Macchiarini*, Rafael Oriol* and Philippe Dartevelle, Robinson Le Plessis, France, Villejuif, France

**OBJECTIVE(s):** We used a pig-to-goat orthotopic lung xenograft model to test whether depletion of goat xenoreactive antibodies (XNA) against pig red blood cells would prolong pig lung xenograft survival and to study the late phase of discordant lung xenograft rejection.

**METHODS:** Adult goats with anti-pig XNA underwent left pneumonectomy followed by orthotopic transplantation of pig left lung (group 1) or immunodepletion of XNA by extra-corporeal right pig lung perfusion before transplantation without (group 2) or with (group 3) complete occlusion of the right pulmonary artery (RPA). In group 4, goat left lungs were orthotopically transplanted into pigs and served as negative controls (pig serum does not have anti-goat XNA). Each study group included 5 animals. Immunosuppression in surviving recipients included cyclosporin and azathioprine. Open chest xenograft biopsies were made on the 2nd postoperative day and at sacrifice.

**RESULTS:** Group 1 recipients died 7±3 hours following xenograft reimplantation due to severe pulmonary hypertension and right heart failure, with little evidence of histological xenograft injury. Group 2 xenografts had significantly ($p<0.001$) lower pulmonary vascular resistances and higher blood flow compared to group 1 animals, and recipients survived for 9±4 days. Group 3 animals tolerated also complete occlusion of the RPA, and the xenografts assured the total respiratory support for 4±1 days. After immunodepletion, goat serum showed no detectable titers of XNA which reappeared on the 2nd postoperative day. On the 2nd postoperative day, all immunodepleted xenografts showed features of delayed humoral (perivas-cular stromal cells, microvasculature thrombosis, interstitial hemmorrhages) and cellular (perivascular and interstitial lymphocyte

infiltration) vascular rejection (DVR). At sacrifice, all xenografts were completely necrosed being the contralateral lungs intact. Group 4 xenografts showed scattered features of acute rejection.

**CONCLUSIONS:** Immunodepletion prolongs left lung xenograft survival, even after occlusion of the RPA. Despite immunosuppression, DVR and xenograft necrosis occurred and corresponded to the return of XNA titres.

**By Invitation*

### F6. Functional Interleukin-4 Receptor and Interleukin-2 Receptor Common Gamma Chain on Human Non-Small Cell Lung Cancers: Novel Targets for Directing Immune Therapy

Richard Essner*, Young Huynh*, Tung Nguyen*, Donald L. Morton and Dave S. B. Hoon*, Santa Monica, California

**OBJECTIVE:** IL-4R has been demonstrated on human non-small cell lung carcinoma cell lines (NSCLC) and rumor specimens. IL-4 causes $G_1$ phase cell-cycle arrest of lung cancer cells expressing IL-4R; the effect directly correlates with expression of IL-4R and is seen within 48 hr after treatment. We examined signal transduction pathways employed by IL-4R, which may account for growth arrest of established line LUst and but had no effect on a second cell line SK-MES-1.

**METHODS:** Western blot analysis was performed on both cell lines cultured up to 48 hr in the presence of IL-4 (500 U/ml). Cells were lysed, protein-extracted, and electroblotted; blots were then probed with murine monoclonal antibodies to specific intracellular proteins.

**RESULTS:** Western blotting of cell lines with anti-phosphotyrosine antibody (4G10) demonstrated multiple (140, 100-130, and 65 kD) phosphoproteins in IL-4 treated LUst but not in IL-4 treated SK-MES-1. Immunoprecipitation and blotting of LUst with specific secondary antibodies showed that the 140 kD phosphoprotein was IL-4R, the 100 kD phosphoprotein was nuclear transcription factor STAT 6, the 130 kD phosphoprotein was janus kinase (JAK-1), the 120 kD phosphoprotein was JAK-3, and the 65 kD phosphoprotein was IL-2R common gamma chain (IL-2Rgc). Specific binding was not observed in SK-MES-1, suggesting the absence of a functional IL-2Rgc. Southern blotting with cDNA probes to IL-2Rgc confirmed absence of this cytokine receptor on SK-MES-1.

**CONCLUSIONS:** These results suggest that human NSCLC may express functional cytokine receptors, including the IL-2Rgc commonly associated with lymphocyte IL-2R. These receptors may be novel targets for directing cytokine-based immune therapy for NSCLC.

**By Invitation*

### F7. Paclitaxel Induces Apoptosis in Lung Cancer Cells by Increasing Caspase-3 Activity But May Not Require Fas and Fas Ligand

Christine Odoux*, Michael Lotze*, Peter K. Kim*, Andy Amoscato*, James D. Luketich*, Robert J. Keenan and Tracey L Weigel*, Pittsburgh, Pennsylvania

**OBJECTIVE:** The Fas receptor when bound by its ligand (FasL) induces programmed cell death (apoptosis). Both Fas and FasL have been documented on the cell surface of lung cancer cell lines

(LCCLs). Paclitaxel (PA), one of the most active chemotherapeautic agents for NSCLC, has been shown to induce apoptotic cell death.

**METHODS:** To elucidate the role of Fas/FasL in PA induced apoptosis of LCCLs, we cultured 2 squamous (A549, H226) and 1 bronchoalveolar (H358) patient-derived, NSCLC cell lines in 10 uM paclitaxel. Cell morphology was assessed @ 24 hrs for nuclear and cytoplasmic condensation by methylene blue/Azure A/eosin staining. Apoptosis was confirmed by DNA laddering. Caspase-3 (cytoplasmic protease in the apoptotic cascade) activity was measured by a Z-DVED cleavage assay. Expression of Fas and FasL was assessed immunohistochemically with human anti-Fas and anti-FasL antibodies.

RESULTS:Paclitaxel consistently induced apoptosis in LCCLs as determined by morphological analyses, DNA laddering and increased Caspase-3 activity. The effect of PA on Fas/FasL expression was variable (see table).

| Cell Line | Caspase-3 act. (%)* | Fas (%) | Fas (%) |
|---|---|---|---|
| **A549** | 138.0 ± 3.7 (p=0.03)* | 85.7 ± 9.9 (NS) | 110.9 ± 9.9 (NS) |
| **H358** | 240.7 ± 77.6 (p=0.05) | 111.6 ± 5.8(NS) | 82.7 ± 10.4 (p=0.03) |
| **H226** | not determined | 57.4 ± 6.6 (p=0.05) | 108.4 ± 0.6 (p=0.05) |

* *Values are expressed as percentages of controls from untreated cells.*

** *Mann-Whitney U test, n = 4 experiments, NS = not significant*

**CONCLUSIONS:** Caspase-3 activity was significantly increased in all LCCLs after culture in PA. Upregulation of Fas and FasL did not appear requisite for PA induced apoptotis of LCCLs.

**By Invitation*

### F8. Respiratory Failure after Thoracic Surgery: The Incidence of Aspiration can be Limited by GI Tract Management

John R. Roberts*, Karla R. Christian*, Richard R. Pierson*, Davis C. Drinkwater and Walter H. Merrill, Nashville, Tennessee

**OBJECTIVE:** Respiratory failure is the major cause of mortality after general thoracic surgery. However, respiratory failure may result from two very different causes: aspiration, which results from ileus and reflux, and pneumonia, which results from poor pain control and weak cough. Epidural catheters help control pain and prevent pneumonia, but contribute to ileus and may increase aspiration. We compared our results after thoracotomy before and after a change in management designed to decrease the risk of aspiration.

**METHODS:** All patients undergoing thoracotomy by a single surgeon over three years were evaluated for aspiration, pneumonia, morbidity, and mortality. For the first eighteen months, patients (N=129) did not receive an intraoperative NG tube and were started on "advance as tolerated" diet postoperatively. For the second 18 months (N=141) NG tubes were placed intraoperatively and diets advanced slowly. Patients were considered to have pneumonia if they developed infiltrates in a single or adjoining lobes and grew a dominant organism from their sputa. Patients were considered to have aspirated if they developed diffuse infiltrates and multiple organisms. Chi-square testing was used to test significance.

**RESULTS:** Two hundred seventy patients underwent thoracotomy at five different hospitals over a three-year period. Seven patients (5.43%) developed respiratory failure on ad lib diets in the first eighteen months, five due to aspiration and two due to pneumonia. Three (2.11%) developed respiratory failure in the second eighteen months, all due to pneumonia. The difference was highly significant (p=0.0183).

**CONCLUSIONS:** Careful GI tract management decreased the incidence of aspiration in this cohort of patients undergoing thoracotomy who received epidurals for pain control.

**By Invitation*

### F9. Sequence-dependent Enhancement of Taxol Sensitivity in Non-Small Cell Lung Cancer by the erbB-2 Tyrosine Kinase Inhibitor NSC 330507.

Dao Nguyen*, Aaron Chen*, Arnold Mixon* and David S Schrump*,
Bethesda, Maryland

*Sponsored By: Jack A. Roth, M.D., Houston, Texas*

**OBJECTIVE:** Overexpression of the erbB-2 oncogene may contribute to chemoresistance in NSCLCs. The aim of this study was to determine if down regulation of this oncogene by NSC 330507 (NSC) could enhance taxol sensitivity in NSCLCs.

**METHODS:** Four NSCLC lines (H460, H1299: low erbB-2; H322, H358: high erbB-2) were treated with taxol(90 minutes exposure, 0.016 to 16 mM) and NSC (96 hrs continuous exposure, 20 to 80 nM) as follow: taxol + NSC (schedule 1) or 24 hrs NSC treatment prior to NSC (schedule 2). Taxol IC50 values of each groups were calculated. Apoptosis was evaluated by ApoBrdU technique.

**RESULTS:** Sensitivity to taxol was significantly enhanced (Ã˜IC50) only in high erbB-2 cells treated with the schedule 1 combination compared to taxol alone controls: 0.12±0.08mM vs 0.45±0.13mM and 0.75±0.12mM vs 1.9±0.4mM in H322 and H358 cells respectively (n=4, $p<0.01$). Significant apoptosis was noted in H322 cells treated with the schedule 1 combination of taxol (0.5mM) and NSC (40nM): control:<1%, NSC:<2%, taxol:16.0±1.4%, taxol+NSC:48.5±8.0% ($p<0.01$, n=3). Moreover, all cells treated with the schedule 2 became less sensitive to cytotoxic effects of taxol, possibly related to G1 cell cycle arrest induced by NSC (as previously observed) prior to taxol treatment.

**CONCLUSIONS:** Downregulation of erbB-2 gene function by NSC results in sensitization of high erbB-2 NSCLC cells to taxol. This synergistic effect is sequence-dependent which has important implication in development of combination chemotherapy regimens for lung cancer. Mechanism(s) of this salutary effect of NSC 330507 is currently under investigation.

**By Invitation*

**F10. Decitabine-mediated Induction of NY-ESO-1 and MAGE-3 Cancer-Testis Antigen Expression in Lung Cancer Cells and EBV-transformed Lymphocytes**

David S. Schrump*, Li Lee*, Dao Nguyen*, Rong F. Wang*, Xiang Wang* and Steven A. Rosenberg*, Bethesda, Maryland

*Sponsored by: Jack A. Roth, M.D., Houston, Texas*

**OBJECTIVE:** Previously we observed expression of NY-ESO-1 in 40% of NSCLC lines, and demonstrated that expression of this tumor antigen can be induced by the demethylating agent, decitabine (DAC). Recently we further defined the kinetics of NY-ESO-1 (and MAGE-3) induction in lung cancer cells.

**METHODS:** CALU-6 NSCLC cells, normal human bronchial epithelial (NHBE) cells, and EBV-transformed lymphocytes (EBV-L) were exposed to DAC at 4 mM x 8 h, 2 mM x 24h, 1 mM x 24h, and 1 mM x 96h. RT-PCR techniques were utilized to evaluate NY-ESO-1 and MAGE-3 expression 96h following initiation of DAC treatment. Cytokine release assays were utilized to evaluate recognition of EBV-L by an HLA restricted TIL clone specific for NY-ESO-1.

**RESULTS:** NY-ESO-1 expression was detected in CALU-6 cells following 8h DAC exposure, and induction following 24h treatment was comparable to that observed after continuous 96h exposure. MAGE-3 expression was similarly augmented under these conditions. No induction of NY-ESO-1 or MAGE-3 was observed in near confluent NHBE cells or EBV-L following similar treatment; however, expression of NY-ESO-1 could be detected in log phase NHBE cells and EBV-L following DAC exposure at 1 mM x 96h and 2 mM x 8d, respectively. EBV-L expressing NY-ESO-1 were recognized by an autologous melanoma TIL clone specific for this antigen.

**CONCLUSIONS:** NY-ESO-1 and MAGE-3 cancer-testais antigen expression can be induced under conditions which are known to facilitate p16 expression and have been achieved in leukemia patients. Demethylating agents may be useful for simultaneously enhancing immunogenicity and cell cycle arrest in lung cancer cells. Furthermore, decitabine treated EBV-L may prove efficacious for the immunotherapy of lung cancer patients whose tumors express NY-ESO-1.

*By *Invitation*

## WEDNESDAY MORNING, APRIL 21, 1999

**7:00 a.m. ADULT CARDIAC SURGERY FORUM SESSION**

Ballroom, Ernest N. Morial Convention Center

*Moderators: Verdi J. DiSesa, M.D.*

*Edward D. Verrier, M.D.*

*F11. Cardiac Allograft Long-Term Functional Tolerance*
**Without Immunosuppressive Drugs in Mice Transgenic For a Suicide Gene**

Eric Bratmberger*, Nathalie Raynal-raschilas*, David Klatzmann*, Olivier Boyer*, Patrick Bruneval*, Jean-Noel Fabiani*, Denis Glotz* and Alain Carpentier, Paris, France

**OBJECTIVE:** Life-long immunosuppression is a major cause of mortality and morbidity in transplant recipients. Gene therapy could provide new ways to obtain tolerance and avoid indefinite immunosuppression.EpTK mice are derived from the FVB/N strain (H2q) and express the Thymidine Kinase gene of the Herpes virus in all mature T cells. Thus, any mature dividing T cell can be killed in the presence of Ganciclovir (GCV). We investigated the survival of allo-incompatible C57B1 /6 (H2b) hearts heterotopically transplanted into EpTK mice given only GCV from day 0 to day 7 or 14.

**METHODS:** Abdominal cardiac transplantation was performed in 22 controls (untreated FVB n=15, GCV treated FVB n=5, untreated EpTK mice n=2), and in 28 EpTK mice given GCV from day 0 to day 7 (n=15) or 14 (n=13). Rejection was defined as complete cessation of cardiac beats. Histological examination of the grafts was performed at rejection or at day 100. Lymphocyte proliferation assays (Con A stimulation or Mixed Lymphocyte Reaction) were performed at day 100.

**RESULTS:** All control animals rejected in 7 days (5-9), whereas survival >100 days was observed in 89% of the GCV treated EpTK group, irrespective of the duration of GCV treatment. Graft histology showed extensive cellular infiltrates with myocyte necrosis and arteritis in the controls, but only a mild infiltrate without necrosis or arteritis in the GVC treated EpTK group. The proliferative responses of the tolerant mice at day 100 were identical to naive mice, including a preserved proliferation against the donor's lymphocytes in MLR.

**CONCLUSIONS:** Functional transplantation tolerance of a fully incompatible heart could be achieved without immunosuppressive drugs in this model of suicide gene therapy.

**By Invitation*

### F12. Development of an Autologous Bioengineered Cardiac Graft.

SRen-ke Li*, Zhi-qiang Jia*, §Richard D. Weisel, §Donald A. G. Mickle*, Eung-Joong Kim*, Shinji Tomita* and Tetsuro Sakai*, Toronto, Ontario, Canada

**OBJECTIVE:** Patients with congenital heart defect frequently require graft material for cardiac reconstruction. Currently available grafts lack growth potential, are not contractile and are thrombogenic. We developed a beating three dimensional mesh from fetal cardiomyocytes, which successfully repaired a cardiac defect but the cells were rejected. Then we developed a graft derived from autologous cardiomyocytes which could be employed to replace a cardiac defect.

**METHODS:** BEATING MESH: Fetal rat cardiomyocytes were seeded into a gelatin mesh and cultured. The cells formed a tissue in the mesh and after 7 days the mesh was beating regularly and spontaneously in culture. We transplanted the beating mesh into the subcutaneous tissue of an adult rat leg (N=14). After 21 days, the graft continued to beat with a fractional shortening of 35±9%. We attached the beating mesh to a cryoinjured rat heart scar (N=10). After 5 weeks, the mesh contracted synchronously with the heart and the gelatin matrix dissolved. Unfortunately, the fetal cardiomyocytes were rejected 24 weeks later.

**RESULTS:** AUTOLOGOUS GRAFT: We isolated and cultured juvenile rat cardiomyocytes which were seeded into a gelatin mesh and formed a tissue after 7 days. The tissue engineered mesh was then exposed to mechanical stretch and relaxation. After 7 days of contraction, the cardiomyocytes in the mesh joined together and oriented in the direction of the stress.

**CONCLUSIONS:** Fetal cardiomyocytes were grown into a three dimensional graft which contracted *in vitro* and *in vivo.* Juvenile cardiomyocytes were grown in a mesh which oriented to stretch. These studies suggest that an autologous cardiac graft could be employed to replace congenital cardiac defects.

*[§]Authors have a relationship with Genzyme, Corp.*

**By Invitation*

### F13. Novacor LVAS versus HeartMate as a Long-term Mechanical Circulatory Support Device in Bridging Patients: a Prospective Randomized Study

Aly El-banayosy*, Latif Arusoglu*, Lukas Kizner*, Heinrich Koertke*, Michael Koerner*, Michel Morshuis*, Ullrich Schuett*, Peter Sarnowski*, Oliver Fey*, Kazutomo Minami* and Reiner Koerfer, Bad Oeynhausen, Germany

**OBJECTIVE:** The aim of our study was to investigate the reliability of both devices and to compare them with regard to morbidity following implantation.

**METHODS:** From October 1996 to March 1998, we randomized patients (pts) needing mechanical left ventricular support as a bridge-to-transplant procedure alternatingly between Novacor and HeartMate. Altogether, 40 pts were included in the study, 20 of whom were supported with Novacor and 20 with the VE HeartMate. Both groups were comparable as to age, preoperative laboratory parameters, hemodynamics and risk factors.

**RESULTS:** Mean duration of support in Novacor pts was 137.7 days, in HeartMate pts 134.6 days. 15 Novacor pts and 13 HeartMate pts could be discharged home while under support. A bleeding complication occurred in 15 pts (8 Novacor, 7 HeartMate), minor and major neurological disorders in 9 pts (7 Novacor, 2 HeartMate), device related infection complications (driveline infections, pocket infections, conduit endocarditis) in 16 pts (6 Novacor, 10 HeartMate), sepsis in 6 pts (2 Novacor, 4 HeartMate). 2 pts (1 Novacor, 1 HeartMate) suffered severe right heart failure requiring the implantaion of an additional right VAD (Novacor + Thoratec VAD, HeartMate + Medos HIA-VAD). Pump failure occurred in 1 HeartMate pt, a controller change was necessary 14 times in the HeartMate group and twice in the Novacor group. Driveline rupture was found in 2 HeartMate pts and in 1 Novacor pt.

**CONCLUSIONS:** Both systems have proved to be reliable devices for long-term support with slight advantage of the Novacor LVAS, which is also superior to the HeartMate device in terms of infection complications, whereas HeartMate patients suffer less neurological disorders.

**By Invitation*

### F14. Tissue Engineered Three Leaflet Heart Valves

Ulrich A. Stock*, Mitsugi Nagashima*, Philipe N. Khalil*, Georg D. Nollert*, Adrian M. Moran*, Tanja Herden*, Jason S. Sperling*, [§]David P Martin*, Jamie G. Lien*, Frederick J. Schoen*, Joseph P. Vacanti*,and John E. Mayer, M.D., Boston, Massachusetts and Cambridge, Massachusetts

**OBJECTIVE:** Bioprosthetic, mechanical valves or valved conduits have the disadvantage that they are not able to grow, repair or remodel. In an attempt to overcome these shortcomings, we have evaluated the feasibility of creating 3 leaflet pulmonary conduits from autologous ovine cells and biodegradable polymers.

**METHODS:** Endothelial cells(EC)were harvested from carotid artery segments using collagenase instillation and myofibroblasts(MF)by mincing of the remaining artery wall. Scaffolds of polyglycolic acid and polyhydroxyalkanoates (Metabolix Inc) were formed into a 3 leaflet valved conduit (18mm ˆ†,2cm length) and were seeded on 4 consecutive days with MF and finally once with EC. With the heart beating, 6 constructs were implanted 31 days(±3) after the vessel harvest using cadiopulmonary bypass. A segment of the native pulmonary artery and all 3 leaflets were removed and 5 seeded and 1 unseeded construct implanted. The animals received no postoperative anticoagulation. Valve function was evaluated by echocardiography before chest closure and before explantation after 1, 2, 4, 6, and 8 weeks. The conduits were evaluated histologically and biochemically for DNA, collagen(4-OHP) and elastin.

**RESULTS:** ALL animals survived the procedure. Postoperative echocardiography of the seeded constructs demonstrated no thrombus formation with mild non progressive valvular regurgitation up to 8 weeks after implantation. Histology showed progressive tissue formation. Biochemical assays revealed increasing DNA, 4-OHP and elastin content.The unseeded construct showed severe thrombus formation on all 3 leaflets after 1 week.

**CONCLUSIONS:** This preliminary experiment showed that 3 leaflet heart valves constructed from autologous cells and a biodegradble matrix can function in the pulmonary circulation. Tissue consisting of cellular and extracellular matrix increased over the observed time period.

*[§]Author has a relationship with Metabolix, Inc.*

**By Invitation*

### F15. Transplantation of Fetal Cells Limits Postinfarct Ventricular Expansion: An Echocardiographic Study

Marcio Scorsin*, Alain A. Hagege*, Jean-Thomas Vilquin*, Francoise Marotte*, Marc Fiszman*, Jane-Lise Samuel*, Ketty Schwartz*, Lydie Rappaport* and Philippe Menasche, Paris, France

**OBJECTIVE:** Transplantation of fetal cardiomyocytes has been shown to improve function of the infarcted myocardium but the patterns of this improvement have not yet been documented by longitudinal studies.

**METHODS:** Myocardial infarction was created in 18 rats by ligation of the left coronary artery. One week later, the animals were reoperated on and randomly assigned to receive fetal cardiomyocytes (5 x $10^6$ cells harvested from 19-day fetuses and suspended in 250 mL) injected intramyocardially in the core and at the borders of the infarct or an equivalent volume of culture medium. Left ventricular function was assessed by echocardiography immediately before transplantation and 1 month later using a custom-made 13 MHz 2D transducer allowing to display 180 frames/sec. All rats were immunosuppressed by cyclosporin. Data were compared by Student's *t* tests and are reported as mean ± SD.

**RESULTS:** Left ventricular enddiastolic volume (LVEDV) significantly increased in controls (from 0.47±0.12 mL to 0.68±0.17 mL, p<0.003) while cell-treated hearts dilated to a lesser extent with LVEDV being 0.45±0.16 mL at baseline and 0.62±0.25 mL 1 month later, p=NS. Likewise, control hearts increased their endsystolic volumes to a much greater extent than cell-treated hearts (at 1 month: 0.44+0.17 mL and 0.36±0.19 mL, respectively). As a result, ejection fraction did not change in controls (from 37.7±14.4% to 34.2±9.4%) whereas it significantly increased after fetal cell grafting (from 33.9±12.7% to 44.9±13.7%, p<0.04 vs baseline).

**CONCLUSIONS:** Transplantation of fetal cardiomyocytes significantly improves function of the infarcted myocardium, primarily through a limitation of ventricular remodeling, and might represent a potential rescue therapy for the ischemically-damaged heart.

### F16. Emboli Management with a Novel Aortic Filtration System: Histopathological Confirmation of Atheromatous Plaque Capture in Cardiac Surgery

Hermann H. Reichenspurner*, Jose Navia*, Gerald J. Berry*, Robert C. Robbins*, §Jefferey P. Gold and Bruno Reichart*, Munich, Germany, Buenos Aires, Argentina, New York, New York and Stanford, California

**OBJECTIVE:** Significant neurological morbidity following cardiac surgery is associated with aortic emboli released upon removal of the cross clamp. Initial clinical experience with an aortic filtration system was previously evaluated and shown to be safe and feasible for emboli capture in 58 patients. Further study was performed on 20 patients to evaluate the presence of atheromatous plaque.

**METHODS:** Elective cardiac surgery patients underwent cardiopulmonary bypass (CPB) with an aortic filtration system (average age; 63, female; 26%). Intra-aortic filters were inserted via a modified 24F arterial cannula (EMBOLÎ£X, Mt.View, CA) immediately prior to releasing the cross clamp and were deployed for an average of 23 minutes (range 5-30). Filters were subsequently examined by scanning electron microscopy (SEM) (11) and histology (20).

**RESULTS:** The insertion and removal of the filtration system was safe and uneventful in all patients. No morbidity due to stroke or neurological events was noted. Upon removal, visual inspection of the filters revealed particulate debris. SEM was performed and no thrombus formation was evident (few activated platelets 4/11 filters; and few fibrin strands 4/11 filters ). Histologic examination of 7/20 samples showed evidence of atheromatous material: mature collagen (4/20 filters); myofibroblasts compatible with fibroatheromatous caps (2/20 filters); aggregates of cholesterol grumous material and choleterol fragments indicative of atheromatous plaque (1/20 filters).

**CONCLUSIONS:** The intra-aortic filtration system can be safely deployed during CPB and captures atheromatous parriuclate material without generating thrombus. The impact of aortic fitlration on neurological outcomes is to be determined in prospective randomized studies.

*§Author has a relationship with Embolx*

**By Invitation*

## F17. Sutureless Coronary Artery Bypass with Biologic Glued Anastomoses: Preliminary In Vivo and In Vitro Results

[§]Steven R. Gundry, [§]Kirby S. Black* and [§]Hironori Izutani*, Atlanta, Georgia and Loma Linda, California

**OBJECTIVE:** As heart surgery becomes increasingly focused upon minimally invasive techniques, conventional anastomosis techniques will need to be severely altered. We developed and tested coronary artery bypass graft anastomoses using a biologic glue formulated from bovine albumin and glutaralde-hyde and using a double-balloon catheter temporary internal stent to create and seal the anastomosis during gluing.

**METHODS:** Anastomoses were made between cryopreserved human saphenous vein segments and coronary arteries in vitro on 12 intact bovine hearts. A total of 42 anastomoses were created using the catheter system introduced into the distal end of the graft exiting the back wall and entering the anterior wall of the coronary artery. Two balloons (one in the graft and one in the coronary) held the anastomosis stable while the biologic glue was applied externally and allowed to set for 2 min. The balloon catheter was then removed from the end of the graft. The open end of the graft was clipped, turning the anastomosis into an end to side graft.

**RESULTS:** A pressure transducer was then attached to the graft and saline forcefully infused. All grafts easily held 300 mmHg pressure. Distal and proximal coronary artery patency was checked by examining flow out of the coronary ostia and by cutting arteries distal to the grafts. All anastomoses were patent upon opening. Subsequently, three LIMA to LAD anastomoses have been constructed in goats, with ligation of the proximal LAD. Survival now exceeds 7 months, with normal echo function.

**CONCLUSIONS:** A biologic glue and catheter system has been developed which allows for a high bursting strength coronary anastomosis to be performed. When further developed and tested, truly minimally invasive heart surgery may be possible.

*[§]Authors have a relationship with Cryolife, Inc.*

**By Invitation*

## F18. Hypothermic Retrograde Venous Perfusion Cools the Spinal Cord and Reduces Paraplegia During Thoracic Aortic Surgery

Scott D. Ross*, John A. Kern*, James J. Gangemi*, Char R. St. Laurent*, Kimberly S. Shockey*, Irving L. Kron and Curtis G. Tribble, Charlottesville, Virginia

**OBJECTIVE:** We evaluated the utility of retrograde venous perfusion to cool the spinal cord and protect neurological function during aortic clamping. We hypothesized that hypothermia-induced electrical silence would preserve the spinal cord during ischemia.

**METHODS:** Six swine(GroupI) underwent thoracic aortic occlusion for 30 minutes at normothermia. A second group(GroupII) underwent spinal cord cooling by retrograde perfusion of the paravertebral veins with a hypothermic(4°C) saline solution during 30 minutes of aortic occlusion. The spinal cords of a third group(GroupIII) were cooled with a hypothermic adenosine solution in a similar fashion. Intrathecal temperature was monitored and measurements of

somatosensory evoked potentials(SSEP) assessed the functional status of the spinal somatosensory pathways during clamping, cooling, and recovery.

**RESULTS:** Spinal cooling without systemic hypothermia significantly improved neurological Tarlov scores in groupIII(4.7±0.2) and groupII(3.8±0.4) when compared to groupl(1.3±0.6)($p<0.001$). Furthermore, 5 of the 6 animals in groupIII displayed normal neurological function, whereas only 1 animal in groupII and no animals in groupI did($p=0.005$). SSEP were lost 10.6±1.4 minutes after ischemia in groupI. In contrast, spinal cooling resulted in rapid cessation of neural transmission with loss of SSEP at 6.9±1.2 minutes in groupll and 7.0±0.8 minutes in groupIII($p=0.06$). SSEP amplitudes returned to 85% of baseline in groupIII and 90% of baseline in groupII compared to only 10% of baseline in groupI($p=0.01$).

**CONCLUSIONS:** We conclude that retrograde cooling of the spinal cord is possible and protects against ischemic injury. Its efficacy may be attributed to induced electrical silence of the cord during ischemia, similar to cardiac electrical silence during cold cardioplegia.

**By Invitation*

### F19. Experimental Aortomyoplasty Improves Systolic and Diastolic Performance in Chronic Ischemic Cardiomyopathy

Thierry G. Mesana*, Olivier Y. Ghez*, Jeffrey S Martin*, Mark H. Danton*,

Sergey D. Grachev*, Kathryn Q. Flores*, Rita G.. Laurence*, Lawrence H.

Cohn and John G. Byrne*, Boston, Massachusetts

**OBJECTIVE:** Aortomyoplasty (AMP) may have a role in the treatment of chronic ischemic cardiomyopathy (CIC), but has not yet been evaluated for this purpose. Therefore we tested whether AMP improves global and regional myocardial performance and regional myocardial blood flow (RMBF), in an experimental model of CIC.

**METHODS:** Ameroid constrictors were placed on the proximal circumflex artery in goats. After six weeks, AMP (n=6) was performed by wrapping untrained latissimus dorsi muscle around the descending thoracic aorta and coun-terpulsation established at 1:2. Measurements were made with and without AMP-powered counterpulsation.

**RESULTS:**

| | **AMP Off** | **AMP On** |
|---|---|---|
| **Subendocardial Viability Ratio** | 65.9 ± 6.9 | 83.1 ± 18.1* |
| **Diastolic Aortic Pressure (mmHg)** | 37.0 ± 9.1 | 44.0 ± 10* |
| **t (Diastolic Relaxation Time Constant, ms)** | 20.9 ± 7.7 | 21.2 ± 8.5 |
| **Diastolic dP/dt (mmHg/sec)** | -543 ± 217 | -618 ± 213* |
| **Minute Stroke Work Index (J/min/m$^2$)** | 12.23 ± 2.73 | 13.5 ± 3.16t |
| **Power Max (mmHgÎ£cc/min)** | 7868 ± 1241 | 8445 ± 1339* |
| **Cardiac Index (L/min/m$^2$)** | 1.74 ± 0.27 | 1.81 ± 0.31* |
| **RMBF non-ischemic area (ml/min/gm) (n=3)** | 0.97 ± 0.33 | 1.13 ± 0.49* |

| | | |
|---|---|---|
| **RMBF ischemic area (ml/min/gm) (n=3)** | 0.68 ± 0.32 | 0.79 ± 0.43* |
| **Regional Shortening Ischemic Area** | 8.8 ± 10 | 9.4 ± 10 |

**p<0.05 †p<0.01*

**CONCLUSIONS:** Aortomyoplasty improves global systolic and diastolic performance and RMBF in this model of chronic ischemic cardiomyopathy.

**By Invitation*

## F20. Cardiac Performance After Deep Hypothermic Circulatory Arrest In Cyanotic Neonatal Lambs

Mitsugi Nagashima*, Georg Nollert*, Ulrich Stock*, Jason Sperling*, Dominique Shum-tim*, Koh Takeuchi*, Arther Nedder* and John E Mayer, Boston, Massachusetts

**OBJECTIVE(s):** Acute hypoxic stress before ischemia results in increased free radical production and depressed recovery of cardiac function. The relevance of the acute hypoxic stress model to chronic cyanosis in patients remains unclear. This neonatal lamb study assessed the effect of 1 week of cyanosis on recovery of cardiac function and free radical production after deep hypothermic circulatory arrest (DHCA).

**METHODS:** Cyanosis was created in 8 lambs (age 4.8 days) by an anastomosis between the pulmonary artery and the left atrium. Seven controls underwent thoracotomy and pericardiotomy. One week later, all animals underwent CPB with 90 min of DHCA at 18°C. Preload recruitable stroke work (Mw) was measured as LV contractility before CPB ($FiO_2$=1.0 and $FiO_2$=0.21) and post CPB at 2 hr of reperfusion ($FiO_2$ =1.0). Malondialdehyde (MDA) levels in coronary sinus were measured. LV antioxidant reserve capacity (ARC) was assessed at 2 hr of reperfusion by measuring thiobarbituric acid reactive substance (TEARS) in LV biopsies incubated with t-butylhydroperoxide (t-BHP).

**RESULTS:** The cyanosis model produced a significantly ($p < .01$) lower arterial oxygen tension (35 ± 3 mmHg vs. 93 ± 9 in control, FiO2 = 0.21). Mw and MDA are shown in the Table (Data are mean ± SE. * = $p < .05$ vs. control, † = $p < .05$ vs FiO2 = 0.21 baseline). LV ARC was not significantly different between groups (TEARS, 1.5 ± 0.2 in cyanosis vs. 1.4 ± 0.1 mmol/g protein in control at 4mM t-BHP).

| | **Cyanosis-Base (21% O2)** | **Base (100% O2)** | **Rep2h** | **Control-Base (21% O2)** | **Base (100%02)** | **Rep 2h** |
|---|---|---|---|---|---|---|
| **Mw (mmHg)** | 36.9 ± 3.8* | 56.1 ± 3.3† | 36.3 ± 5.7* | 59.0 ± 5.2 | 62.3 ± 5.1 | 55.3 ± 6.6 |
| **MDA in CS(mM)** | | 3.4 ± 0.6 | 2.5 ± 0.2 | | 2.5 ± 0.1 | 27 ± 0.3 |

**CONCLUSIONS:** After one week of chronic cyanosis, acute increases in arterial oxygen before CPB result in improved LV function. Recovery of LV function after DHCA was worse in cyanotic animals than controls. The absence of a significant difference in coronary sinus MDA or LV antioxidant reserve capacity suggests that free radical injury may not responsible for worse LV functional recovery in chronically cyanotic hearts.

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Baldwin, John C

**Jaffrey**

Woods, Francis M

**Lebanon**

Nugent, William C

Sanders, John H, Jr

**Windham**

Burbank, Benjamin

**Santa Teresa**

Glass, Bertram A

**Silver City**

Waddell, William R

Gerard, Franklyn P

**Browns Mills**

Fernandez, Javier

McGrath, Lynn B

**Camden**

Camishion, Rudolph C

DelRossi, Anthony J

**East Orange**

Auerbach, Oscar

**Hackensack**

Hutchinson, John E, III

**Jersey City**

Demos, Nicholas J

**Millburn**

Parsonnet, Victor

**Moorestown**

Morse, Dryden P

**Morristown**

Parr, Grant V S

**Neptune**

Roberts, Arthur J

**New Brunswick**

Lewis, Ralph J

MacKenzie, James W

Scholz, Peter M

**Newark**

Donahoo, James

Gielchinsky, Isaac

Swan, Kenneth G

**Pittstown**

Garzon, Antonio A

**Short Hills**

Hochberg, Mark S

**Tenafly**

Gerst, Paul H

**Wyckoff**

## NEW YORK

**Albany**

Foster, Eric D

Moores, Darroch W. O

**Bay Shore**

Ryan, Bernard J

**Bellport**

Finnerty, James

**Bronx**

Attai, Lari A

Brodman, Richard F

Fell, Stanley C

Ford, Joseph M

Prater, Robert W M

Gold, Jeffrey? Hirose, Teruo

Veith, Frank J

**Brooklyn**

Acinapura, Anthony J

Cunningham, Joseph N, Jr

Levowitz, Bernard S

Sawyer, Philip N

**Buffalo**

Bhayana, Joginder N

Dietl, Charles A

Guiraudon, Gerard M

Hoover, Eddie L

Lajos, Thomas Z

Salerno, Tomas A

**East Amherst**

Andersen, Murray N

**East Quogue**

McCormack, Patricia M

**Fayetteville**

Bugden, Walter F

Effler, Donald B

**Floral Park**

Adler, Richard H

**NEW MEXICO**

**Albuquerque**

Edwards, W. Sterling

Wernly, Jorge A

**Buena Vista**

Thai, Alan P

**Santa Fe**

Davila, Julio C

Crastnopol, Philip

**Honeoye Falls**

Graver, William L

**Larchmont**

Steichen, Felicien M

**Lido Beach**

Hines, George L

**Millerton**

Green, George E

**New Rochelle**

Rubin, Morris

**New York**

Adams, Peter X

Altorki, Nasser K

Anagnostopoulos, C E

Bains, Manjit S

Bloomberg, Allan E

Boyd, Arthur D

Cahan, William G

Clauss, Roy H

Colvin, Stephen B

Conklin, Edward F

Cuffiford, Alfred T

Ergin, M Arisan

Friedlander, Ralph

Galloway, Aubrey C, Jr

Ginsberg, Robert J

Griepp, Randall B

Isom, O. Wayne

King, Thomas C

Kirschner, Paul A

Krieger, Karl H

**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

Bryant, Lester R

Lansman, Steven L

Litwak, Robert S

Martini, Nael

McCord, Colin W

Michler, Robert E

Oz, Mehmet C

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

**Plattsburgh**

Potter, Robert T

**Rochester**

DeWeese, James A

Hicks, George L

Schwartz, Seymour I

Stewart, Scott

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**

Robicsek, Francis

Selle, Jay G

**Durham**

Anderson, Robert W

Glower, Donald D

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

**Morehead City**

**Columbus**

Davis, J Terrance

Kakos, Gerard S

Meckstroth, Charles

Williams, Thomas E, Jr

**Dayton**

DeWall, Richard A

Kerth, William J

**Southern Pines**

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

Permington, D. Glenn

**OHIO**

**Blacklick**

Myerowitz, P. David

**Chagrin Falls**

Ankeney, Jay L

Cross, Frederick S

**Cincinnati**

Albers, John E

Callard, George M

Flege, John B, Jr

Gonzalez, Luis L

Helmsworth, James A

Hiratzka, Loren F

Ivey, Tom D

Wilson, James M

Wright, Creighton B

Yee, Edward S

**Cleveland**

Blackstone, Eugene H

Cosgrove, Delos M

Groves, Laurence K

Kay, Earle B

Kirby, Thomas J

**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

Zuhdi, M Nazih

**OREGON**

**Ashland**

Campbell, Daniel C, Jr

**Days Creek**

Miller, Arthur C

**Portland**

Cobanoglu, Adnan

Krause, Albert H

Lemmer, John H, Jr

Okies, J. Edward

Poppe, J Karl

Starr, Albert

**PENNSYLVANIA**

**Bryn Mawr**

Haupt, George J

Templeton, John Y, III

**Camp Hill**

Pennock, John L

**Carlisle**

DeMuth, William, Jr

Loop, Floyd D

Lytle, Bruce W

McCarthy, Patrick M

Rice, Thomas W

Snow, Norman J

Van Heeckeren, Daniel W

**Hershey**

Campbell, David B

Damiano, Ralph J, Jr

Myers, John L

Pae, Walter E, Jr

Pierce, William S

Waldhausen, John A

**Johnstown**

Kolff, Jacob

**Lancaster**

Bonchek, Lawrence I

Rosemond, George P

Witmer, Robert H

**Norristown**

Dunn, Jeffrey M

**Philadelphia**

Addonizio, V. Paul

Bowles, L Thompson

Brockman, Stanley K

Diehl, James T

Edie, Richard N

Edmunds, L. Henry, Jr

Fineberg, Charles

Gardner, Timothy J

Goldberg, Melvyn

Guerraty, Albert J

Hargrove, W Clark, III

Kaiser, Larry R

**Darby**

McKeown, John J, Jr

**Wayne**

Lemmon, William M

**Wilkes-Barre**

Cimochowski, George E

**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

Kratz, John M

Parker, Edward F

Reed, Carolyn E

Sade, Robert M

**Columbia**

Almond, Carl H

**Hilton Head Island**

Humphrey, Edward W

**Isle of Palms**

Mullen, Donald C

**Landrum**

MacVaugh, Horace

Mannion, John D

Nemir, Paul, Jr

Sink, James D

Spray, Thomas L

Wechsler, Andrew S

Whitman, Glenn J R

**Pittsburgh**

Bahnson, Henry T

Clark, Richard E

Griffith, Bartley P

Hardesty, Robert L

Keenan, Robert J

Kormos, Robert L

Landreneau, Rodney J

Magovern, George J

Magovern, George J, Jr

Magovern, James A

Pontius, Robert G

Rams, James J

Siewers, Ralph D

**Rydel**

Frobese, Alfreds

Stayman, Joseph W

**Spartanburg**

Utley, Joe R

**TENNESSEE**

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**Knoxville**

Blake, Hu Al

Brott, Walter H

Domm, Sheldon E

**Memphis**

Cole, Francis H

Hughes, Felix A, Jr

McBurney, Robert P

Pate, James W

Robbins, S Gwin, Sr

Rosensweig, Jacob

Shochat, Stephen J

Watson, Donald C

**Nashville**

Alford, William, Jr

Bender, Harvey W, Jr

Drinkwater, Davis C

Gobbel, Walter G, Jr

Merrill, Walter H

Randolph, Judson G

Rankin, J. Scott

Sawyers, John L

Stoney, William S

Thomas, Clarence, Jr

Safi, Hazim J

Walker, William E

Wukasch, Don C

**Kemp**

Davis, Milton V

**Lubbock**

Bricker, Donald L

Feola, Mario

Wallsh, Eugene

Marble Falls

Hood, R Maurice

**TEXAS**

**Amarillo**

Sutherland, R Duncan

**Austin**

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 C, Jr

**Dilley**

Hood, Richard H, Jr

**Galveston**

Conti, Vincent R

Derrick, John R

Zwischenberger, Joseph B

**Houston**

Beall, Arthur C, Jr

Burdette, Walter J, PhD

Cooley, Denton A

Coselli, Joseph S

DeBakey, Michael E

Espada, J. Rafael

Frazier, O. Howard

Hallman, Grady L

Henly, Walter S

Lawrie, Gerald M

Mattox, Kenneth L

**San Antonio**

Calhoon, John H

Cohen, David J

Dooley, Byron N

Heaney, John P

Treasure, Robert L

Trinkle, J. Kent

**Temple**

Brindley, G. Valter, Jr

**Woodville**

Harrison, Albert W

**UTAH**

**Salt Lake City**

Doty, Donald B

Karwande, Shreekanth V

Liddle, Harold V

McGough, Edwin C

Mortensen, J D

Nelson, Russell M

**VERMONT**

**Richford**

Grondin, Claude M

**West Dover**

Humphreys, George H, II

**VIRGINIA**

**Altavista**

Pierucci, Louis, Jr

**Annandale**

Akl, Bechara F

Burton, Nelson A

Lefrak, Edward A

**Arlington**

Klepser, Roy G

**Aylett**

Gwathmey, Owen

Ott, David A

Overstreet, John W

Putnam, Joe B, Jr

Reardon, Michael J

Reul, George J, Jr

Roth, Jack A

**Charlottesville**

Dammann, John F

Daniel, Thomas M

Kron, Irving L

Minor, George R

Muller, William H, Jr

Nolan, Stanton P

Spotnitz, William D

Tribble, Curtis G

**Fredericksburg**

Armitage, John M

**McLean**

Conrad, Peter W

Gomes, Mario N

Mills, Mitchell

Wallace, Robert B

**Norfolk**

Baker, Lenox D

**Reston**

Boyd, Thomas F

**Richmond**

Bosher, Lewis H, Jr

Brooks, James W

Cole, Dean B

Lower, Richard R

## WASHINGTON

**Belfair**

Jones, Thomas W

Merendino, K. Alvin

Miller, Donald W, Jr

Rittenhouse, Edward

Sauvage, Lester R

Thomas, George I

Verrier, Edward D

Wood, Douglas E

**Spokane**

Berg, Ralph, Jr

## WEST VIRGINIA

**Charleston**

Walker, James H

**Huntington**

Ferraris, Victor A

**Morgantown**

Graeber, Geoffrey M

Gustafson, Robert A

Hill, Ronald C

Murray, Gordon F

Warden, Herbert E

**Parkersburg**

Tarnay, Thomas J

## WISCONSIN

**Altoona**

McEnany, M Terry

**Madison**

Chopra, Paramjeet S

Cochran, Richard P

**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**

Aldea, Gabriel S

Allen, Margaret D

Anderson, Richard P

Hill, Lucius D

Lupinetti, F. Mark

Manhas, Dev R

Mansfield, Peter B

Young, William P

**Marshfield**

Myers, William O

Ray, Jefferson F, III

Sautter, Richard D

**Mequon**

Narodick, Benjamin

**Milwaukee**

Johnson, W. Dudley

Litwin, S Bert

Olinger, Gordon N

Tector, Alfred J

**West Bend**

Gardner, Robert J

**WYOMING**

**Teton Village**

Kaunitz, Victor H

## OTHER COUNTRIES

**ARGENTINA**

**Buenos Aires**

Favaloro, Rene G

**AUSTRALIA**

**QUEENSLAND**

**Brisbane**

O'Brien, Mark F

**SOUTH AUSTRALIA**

**Beaumont**

Sutherland, H D'Arcy

**VICTORIA**

**Melbourne**

Karl, Tom R

**AUSTRIA**

**CANADA**

**ALBERTA**

**Calgary**

Bharadwaj, Baikunth

Miller, George E

**Edmonton**

Gelfand, Elliot T

Koshal, Arvind

Penkoske, Patricia A

Rebeyka, Ivan M

Sterns, Laurence P

**BRITISH COLUMBIA**

**Vancouver**

Ashmore, Phillip G

**Leonding**

Bruecke, Peter E

**Salzburg**

Unger, Felix H

**Vienna**

Wolner, Ernst

**BELGIUM**

**Bertem**

Sergeant, Paul T

**Leuven**

Lerut, Antoon E. M. R

**BRAZIL**

**Rio de Janeiro**

Meier, Milton A

**Sao Paulo**

Jatene, Adib D

Jamieson, W. R. Eric

Tyers, G. Frank O

**Victoria**

Field, Paul

Stenstrom, John D

**MANITOBA**

**Winnipeg**

Barwinsky, Jaroslaw

Cohen, Morley

**NOVA SCOTIA**

**Halifax**

Murphy, David A

**ONTARIO**

**Collingwood**

Heimbecker, Raymond

**London**

McKenzie, F Neil

Menkis, Alan H

Novick, Richard J

**North York**

Goldman, Bernard S

**Nottawa**

Key, James A

**Oakville**

Allen, Peter

**Ottawa**

Hendry, Paul J

Keon, Wilbert J

**Toronto**

Baird, Ronald J

Bigelow, Wilfred G

Christakis, George T

Coles, John G

David, Tirone E

**Herts**

Lennox, Stuart C

**Liverpool**

Donnelly, Raymond J,.

**London**

Braimbridge, Mark V

Feindel, Christopher M

Frernes, Stephen E

McKneally, Martin F

Mickleborough, Lynda L

Pearson, F. Griffith

Scully, Hugh E

Todd, Thomas R J

Trimble, Alan S

Trusler, George A

Weisel, Richard D

Williams, William G

**Westbrook**

Lynn, R Beverley

**QUEBEC**

**Montreal**

Blundell, Peter E

Carrier, Michel

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

Tchervenkov, Christo I

**Sainte-Foy**

DesLauriers, Jean

**CENTRAL AMERICA**

**Guatemala**

Castaneda, Aldo R

**DENMARK**

**Copenhagen**

de Leval, Marc R

Goldstraw, Peter

Lincoln, Christopher R

Ross, Donald N

Stark, Jaroslav F

Taylor, Kenneth M

Yacoub, Magdi

**Oxford**

Westaby, Stephen

**Somerset**

Abbey-Smith, R

**FINLAND**

**Kauniainen**

Mattila, Severi P

**FRANCE**

**Bordeaux**

Fontan, Francis M

**Bordeaux-Pessac**

Baudet, Eugene M

**Creteil**

Loisance, Daniel

Le Plessis Robinson

Binet, Jean-Paul

Dartevelle, Philippe G

Lacour-Gayet, Francois

**Lyon**

Champsaur, Gerard L

**Marseille**

Metras, Dominique R

**Montpellier**

Thevenet, Andre A

**Paris**

Blondeau, Philip

Cabrol, Christian E A

Carpentier, Alain F

Menasche, Philippe

Pettersson, Gosta B

**ENGLAND**

**Bath, Avon**

Belsey, Ronald

**Cambridge**

Kennedy, John H

Wallwork, John

Piwnica, Armand H

Planche, Claude

Weldon, Clarence S

**Pessac**

Couraud, Louis

**Suresnes**

Bachet, Jean E

**GERMANY**

**Aachen**

Messmer, Bruno J

**Bad Oeynhausen**

Korfer, Reiner

**Berlin**

Alexi-Meskishvili, Vladimir

Hetzer, Roland

**Freiburg**

Beyersdorf, Friedhelm

**Hannover**

Haverich, Axel

**Loiching**

Sebening, Fritz

**Munich**

Borst, Hans G

**Neuss**

Bircks, Wolfgang H

**GREECE**

**Kallithea, Athens**

Palatianos, George M

**GUATEMALA**

**Guatemala City**

Herrera-Llerandi, Rodolfo

**IRELAND**

**Dublin**

O'Malley, Eoin

**JAPAN**

**Kamakura**

Suma, Hisayoshi

**Kanazawa**

Watanabe, Yoh

**Kanazawa-city**

Iwa, Takashi

**Kitakyushushi**

Miyamoto, Alfonso T

Minoo, Osaka

Kawashima, Yasunaru

**Osaka**

Kitamura, Soichiro

Matsuda, Hikaru

**Sendai**

Mohri, Hitoshi

**Shinjuku-ku**

Imai, Yasuharu

**Tokyo**

Koyanagi, Hitoshi

Wada, Juro J

**KOREA**

**Seoul**

Cho, Bum-Koo

**MONACO**

**Monaco Cedex**

Dor, Vincent

**ITALY**

**Bergamo**

Parenzan, Lucio

**Milan**

Peracchia, Alberto

**Naples**

Cotrufo, Maurizio

**Pisa**

Bortolotti, Uberto

**Rome**

Marcelletti, Carlo

**NETHERLANDS**

**Wassenaar**

Brom, A Gerard

**NEW ZEALAND**

**Waiwera Auckland**

Barratt-Boyes, Brian G

**P. R. OF CHINA**

**Beijing**

Ying-Kai, Wu

**PORTUGAL**

**Coimbra**

Antunes, Manuel J

**Lisbon**

Machado 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, Rarruro

**Santander**

Revuelta, Jose Manuel

**SWEDEN**

**Sollentuna**

Bjork, Viking

**Umea**

Aberg, Torkel H

**SWITZERLAND**

**Arzier**

Hahn, Charles J

**Lausanne**

von Segesser, Ludwig K

**Pully**

Naef, Andreas P

**Zurich**

Senning, Ake

Turina, Marko I

**VENEZUELA**

**Caracas**

Tricern, Fernando E

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Edward R. Baldwin
Walter M. Boothby
William Branower
Harlow Brooks
Lawrason Brown
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J. Frank Corbett
Armistead C. Crump
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Edmond Melchior Eberts
Max Einhorn
Herman Fischer
Albert H. Carvin
Nathan W. Green
John R. Hartwell
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Chevalier Jackson
H.H. Janeway
James H. Kenyon
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Howard Lilienthal
William H. Luckett
Morris Manges
Walton Martin
Rudolph Matas
E.S. McSweeney
Samuel J. Metzler
Willy Meyer (Founder)
James Alexander Millei
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Leo S. Peterson
Eugene H. Pool
Walter 1. Rathbun
Martin Rehling
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Charles 1. Scudder
William H. Stewart
Franz Torek
Martin W. Ware
Abraham O. Wilensky
Sidney Yankauer

Back to Annual Meeting Program

# AMERICAN ASSOCIATION
*FOR THORACIC SURGERY*
# THE BY-LAWS

## 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 111, Section 8.

Section 3. At the conclusion of the annual meeting, the retiring President shall automatically become a Councilor for a one-year term office. One of the other four Councilors shall be elected at each annual meeting of the Association to serve for a four-year term of office in the place of the elected Councilor whose term expires at such meeting, but no Councilor

may be reelected to succeed himself. Any Councilor so elected shall take office upon the conclusion of the annual meeting at which he is elected.

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

**ARTICLE V. Officers**

Section 1. The officers of the Association shall be 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 offcio 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 reselected to succeed himself in the same office, unless such officer is filling the unexpired term of an officer previously elected to such office. The Secretary and the Treasurer shall be elected for a one-year term of office and may be reelected for not more than four additional terms.

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

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

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

Section 7. The Treasurer of the Association shall perform all duties customarily pertaining to the office of Treasurer. He shall serve as Treasurer of the Association.

Section 8. The Editor of the Association is not an officer of the Association. He shall be appointed by the Council at its annual meeting; provided, however, that such appointment shall not become effective until approved by the Association at the annual meeting of the Association. The Editor shall be appointed for a five-year term and may not be appointed to more than two successive terms; provided, however, that an Editor completing two years or less of the unexpired term of a previous Editor may be appointed for two successive five-year terms. The Editor shall serve as the Editor of the Official Journal and shall be ex officio the Chairman of the Editorial Board and a member of the Council of the Association without vote.

Section 9. Vacancies occurring among the officers named in Section I 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 VIE, Section 4, of these By-Laws.

Section 3. The Program Committee shall consist of at least 13 members: the President, the Vice President, the Secretary and the Editor and at least 9 members-at-large, three each representing 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 or two-year terms. 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. The Chairman shall be the member-at-large serving his fourth year. The duties of the Committee shall be to recommend Fellowship candidates to the Graham Education and Research Foundation and to carry out other business pertaining to the Fellowship and Fellows, past, present, and future.

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

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

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

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

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

## ARTICLE VII. Finances

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

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

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

Section 4. Funds derived from the payment of initiation fees shall not be available to current expenses and shall be placed in a special fund, to be invested and reinvested in legal securities, to be held intact.

## ARTICLE VIII. Meetings

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

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

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

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

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

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

1. Appointment of necessary committees.

2. Miscellaneous business of an urgent nature.

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

1. Reading or waiver of reading of the minutes of the preceding 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, May, 1998

# Meetings of the American Association for Thoracic Surgery

1918-Chicago, IL..... President, Samuel J. Meltzer

1919-Atlantic City, NJ..... President, Willy Meyer

1920-New Orleans, LA..... President, Willy Meyer

1921-Boston, MA..... President, Rudolph Matas

1922-Washington, DC..... President, Samuel Robinson

1923-Chicago, IL..... President, Howard Lilienthal

1924-Rochester, MN..... President, Carl A. Hedblom

1925-Washington, DC..... President, Nathan W. Green

1926-Montreal, QUE..... President, Edward W. Archibald

1927-New York, NY..... President, Franz Torek

1928-Washington, DC..... President, Evarts A. Graham

1929-St. Louis, MO..... President, John L. Yates

1930-Philadelphia, PA..... President, Wyman Whittemore

1931-San Francisco, CA..... President, Ethan Flagg Butler

1932-Ann Arbor, MI..... President, Frederick T. Lord

1933-Washington, DC..... President, George P. Muller

1934-Boston, MA..... President, George J. Heuer

1935-New York, NY..... President, John Alexander

1936-Rochester, MN..... President, Carl Eggers

1937-Saranac Lake..... President, Leo Eloesser

1938-Atlanta, GA..... President, Stuart W. Harrington

1939-Los Angeles, CA..... President, Harold Brunn

1940-Cleveland, OH..... President, Adrian V. S. Lambert

1941-Toronto, ONT..... President, Fraser B. Gurd

1944-Chicago, IL..... President, Frank S. Dolley

1946-Detroit, MI..... President, Claude S. Beck

1947-St. Louis, MO..... President, I. A. Bigger

1948-Quebec, QUE..... President, Alton Ochsner

1949-New Orleans, LA..... President, Edward D. Churchill

1950-Denver, CO..... President, Edward J. O'Brien

1951-Atlantic City, NJ..... President, Alfred Blalock

1952-Dallas, TX..... President, Frank B. Berry

1953-San Francisco, CA..... President, Robert M. Janes

1954-Montreal, QUE..... President, Emile Holman

1955-Atlantic City, NJ..... President, Edward S. Welles

1956-Miami Beach, FL..... President, Richard H. Meade

1957-Chicago, IL..... President, Cameron Haight

1958-Boston, MA..... President, Brian Blades

1959-Los Angeles, CA..... President, Michael E. De Bakey

1960-Miami Beach, FL..... President, William E. Adams

1961-Philadelphia, PA..... President, John H. Gibbon, Jr.

1962-St. Louis, MO..... President, Richard H. Sweet (Deceased 1-11-62)

..... President, O. Theron Clagett

1963-Houston, TX..... President, Julian Johnson

1964-Montreal, QUE..... President, Robert E. Gross

1965-New Orleans, LA..... President, John C. Jones

1966-Vancouver, BC..... President, Herbert C. Maier

1967-New York, NY..... President, Frederick G. Kergin

1968-Pittsburgh, PA..... President, Paul C. Samson

1969-San Francisco, CA..... President, Edward M. Kent

1970-Washington, DC..... President, Hiram T. Langston

1971-Atlanta, GA..... President, Thomas H. Burford

1974-Las Vegas, NV..... President, Lyman A. Brewer, III

1975-New York, NY..... President, Wilfred G. Bigelow

1976-Los Angeles, CA..... President, David J. Dugan

1977-Toronto, ONT..... President, Henry T. Bahnson

1978-New Orleans, LA..... President, J. Gordon Scannell

1979-Boston, MA..... President, John W. Kirklin

1980-San Francisco, CA..... President, Herbert Sloan

1981-Washington, DC..... President, Donald L. Paulson

1982-Phoenix, AZ..... President, Thomas B. Ferguson

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

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

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

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

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

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

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

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

1991-Washington, DC..... President, Keith Reemtsma

1992-Los Angeles, CA..... President, John A. Waldhausen

1993-Chicago, IL..... President, John L. Ochsner

1994-New York, NY..... President, Aldo R. Castaneda

1995-Boston, MA..... President, Robert B. Wallace

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

1997-Washington, DC..... President, David B. Skinner

1998-Boston, MA..... President, Floyd D. Loop

## GRAHAM EDUCATION AND RESEARCH FOUNDATION

13 Elm Street, Manchester, Massachusetts 01944, (978) 526-8330

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### EVARTS A. GRAHAM MEMORIAL 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, 46 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 | Adarl. Hallen Uppsala, SWEDEN |
| 13th | 1964-65 | Stuart C. Lennox London, ENGLAND |
| 14th | 1964-65 | Elias Carapistolis Thessaloniki, GREECE |
| 15th | 1965-66 | Gerhard Frichs Graz, AUSTRIA |

| | | |
|---|---|---|
| 16th | 1965-66 | Ary Blesovsky |
| | | London, ENGLAND |
| 17th | 1966-67 | C. PeterClarke |
| | | Fitzroy, AUSTRALIA |
| 18th | 1966-67 | G.B. Parulkar |
| | | Bombay, INDIA |
| 19th | 1967-68 | Claus Jessen |
| | | Copenhagen, DENMARK |
| 20th | 1969-70 | Peter Brucke |
| | | Linz-Puchenau, AUSTRIA |
| 21st | 1970-71 | Michel S . Slim |
| | | New York, NY, 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.DeWetLubbe |
| | | Cape Town, 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. Vargas |
| | | 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, 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, CHINA |
| 46th | 1998-99 | Christian Kreutzer |
| | | Buenos Aires, ARGENTINA |

| | | |
|---|---|---|
| 47th | 1999-00 | Andes Franco-Cereceda<br>Stockholm, SWEDEN |

## THE THORACIC SURGERY FOUNDATION FOR RESEARCH AND EDUCATION

## ORGANIZATION

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

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

The mission of The Foundation is to: support research and education initiatives to increase knowledge and enhance treatment of patients with cardiothoracic diseases; develop the skills of cardiothoracic surgeons as surgeon-scientists and health policy leaders; and, strengthen society's understanding and trust in the profession.

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*Executive Director Development Associate*

Mari Glass

*Administrative Assistant*

# 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 cardiothoratic surgery. Administered by the Graham Education and Research Foundation, the program is undertaken within the first three years after completion of an approved cardiothoraric 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 Mehmet 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

***DWIGHT HARKEN RESEARCH SCHOLARSHIP***

**"Chimeric Hearts Test the Role of Antigen Presenting Cells in Rejection and Tolerance"**
1998-2000 Bruce Rosengard

The University of Pennsylvania

**SECOND EDWARD D. CHURCHILL RESEARCH SCHOLAR**

**"The Role of respiratory Muscle Adaptation in Lung Volume Reduction Surgery"**
1999-2001 Joseph B. Shrager, M.D.

Philadelphia, Pennsylvania

# INTERNATIONAL TRAVELING FELLOWSHIP

The AATS Traveling Fellowship was established in 1997 by the American Association for Thoracic Surgery. Administered through the Graham Education and Research Foundation, it provides grants to young North American Cardiothoratic Surgeons who are within two years of the completion of their formal cardiothoracic surgery training. The award allows the recipient to study abroad for one year to intensify training in different disciplines and to travel to several sites to broaden the overall training and increase 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.

1st 1998-99 Lishan Aklog

West Roxbury, MA

## SCIENTIFIC ACHIEVEMENT AWARD

The American Association for Thoracic Surgery Scientific Achievement Award was established by the Association in 1994. The award serves to honor individuals who have achieved scientific contributions in the field of thoracic surgery worthy of the highest recognition the Association can bestow. Honorees receive a Medallion for Scientific Achievement from the Association presented by the president at the Annual Meeting and the honoree's name and biography is printed in the Journal of Thoracic and Cardiovascular Surgery.

### SCIENTIFIC ACHIVEMENT AWARD RECIPIENTS

1995 John W. Kirklin, Birmingham, Alabama

1998 Norman E. Shumway, Stanford, California

1999 Michael E. DeBakey, Houston, TX